Inpatient Geriatric Psychiatry

Optimum Care, Emerging Limitations, and Realistic Goals

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To our aging veterans

Foreword 1

Most people are aware of the rapidly changing demographics. In the USA, 10,000 Baby Boomers are turning 65 every single day, and this will continue until 2030 [1]. What is not well appreciated is that the numbers of older people with mental illnesses are rising even more rapidly [2, 3]. The number of Americans with Alzheimer's disease and other dementias is expected to increase from 4 million today to 15 million by 2050 [4]. A large majority of the persons with dementia have behavioral problems including psychosis, depression, and severe agitation that are often multi-determined and difficult to manage at home, especially in acute states. There are also problems in diagnosing illnesses in older neuropsychiatric patients because of nearly universal comorbidities. These patients need multidisciplinary evaluation including psychiatric, neurological, general medical, neuropsychological, nursing, and social assessments. A number of the patients also require continuous observation for several days. A common source of acute syndromes such as delirium and dehydration is polypharmacy, which may warrant discontinuation of some medications, which often cannot be done safely in an outpatient setting.

For all these reasons, geriatric inpatient units are a critical component of geriatric psychiatry, and their value will increase even further in the near future. A book on *Inpatient Geriatric Psychiatry* is, therefore, a very timely addition to the literature. We are delighted and honored to write the Foreword to this outstanding volume, edited with remarkable innovation and efficiency by Howard H. Fenn, with Ana Hategan and James A. Bourgeois as coeditors, and a superb cast of authors in various relevant areas. The comprehensive approach to inpatient geriatric psychiatry makes this the first textbook specific to geriatric inpatient clinical operations. It would serve as a guide to the best clinical practices in the evaluation and the treatment of psychiatric symptoms in older inpatients. The focus of the chapters is on inpatient settings including inpatient psychiatry units, medical units, and postoperative units. In clinical practice, it is often challenging to balance efficiency and thoroughness of evaluation with the constraints of time, length of stay, resources, and cost, imposed by the healthcare system. Numerous medical and psychosocial factors can interfere with the quality care that is needed for older adults with neuropsychiatric disorders. We applaud the concept of "emerging limitations, realistic goals" that is at the center of the approach presented in this book.

We are also pleased that, instead of having separate chapters on treatment of psychosis, depression, and anxiety, which are well covered in many other volumes, the editors have covered topics that are highly relevant to inpatient settings but are typically not addressed together in one book, such as communication barriers, sleep disorders, pain management, and legal issues. The structure of the chapters is well designed, and a unique creative feature is a summary algorithm/flowchart for each chapter, along with clinical vignettes and take-away messages in the form of bullet points.

At the University of California San Diego, we have had a remarkably patient- and family-friendly geriatric psychiatry inpatient unit for two decades, developed and operationalized by our colleague Daniel Sewell, MD. In addition to excellent multidisciplinary healthcare, the unit also offers training in several relevant fields including geriatric psychiatry and medicine, neuropsychology, social work, and family therapy, to name a few. We have also published papers on topics such as unrecognized medical disorders (e.g., a new inpatient diagnosis of pancreatic cancer that had previously been misdiagnosed as major depression in outpatient setting) [5] and the use of iPads to manage severe agitation among inpatients with advanced dementia as a non-pharmacologic technology-based approach [6].

Other investigators too have used data from geriatric psychiatry inpatient units for research publications in peer-reviewed journals. For example, Weintraub and Mazour [7] compared demographic and clinical characteristics of 198 admissions to a psychogeriatric inpatient unit in 1988 and 1998. The recent cohort was more likely to have dementia with agitation or psychosis, be older and taking more psychiatric medication on discharge, having a shorter length of stay, and less likely to be discharged home. These changes have implications for the delivery of treatment on inpatient units.

In another study of 424 consecutive admissions to a university-based geriatric psychiatry inpatient unit over a 20-month period, Woo et al. [8] examined factors associated with repeat hospitalizations, using multivariate logistic regression analysis. Significant predictors of rehospitalization were single relationship status, male gender, and a diagnosis of bipolar disorder. Such information, which is readily obtainable on admission to an inpatient unit, may provide a useful indication of the risk for frequent psychiatric hospitalizations leading to use of readmission prevention strategies at the time of discharge.

An important function of this book will be to document evidence for the best practices in inpatient geriatric psychiatry, in order to work with regulators of the hospitals such as The Joint Commission (TJC) to ensure that the regulations are reasonable and pragmatic. An illustration of its value is the recent announcement by TJC in 2017 that it is increasing scrutiny during surveys on ligature, suicide, and self-harm risks in psychiatric hospitals and inpatient psychiatric units of medical/surgical hospitals. Specifically, TJC surveyors will determine if the hospital has already identified these risks and, if so, what action plan has been developed and is being practiced to remove or mitigate those risks [9, 10]. While the goal of this initiative is laudable, there are several important practical issues in implementing it that need to be

considered with the input of key stakeholders. Also, it is necessary to realize that the risk of suicide cannot be totally eliminated [11].

To conclude, we want to compliment the editors and authors of *Inpatient Geriatric Psychiatry* for making an invaluable contribution to the literature on a vital component of the practice of geriatric psychiatry. We believe that the readers of the book will benefit from clinical, training, research, and policy perspectives, resulting in improved care and well-being of geriatric psychiatry patients.

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Foreword 2

If you're a geriatric psychiatrist, this text will answer all your questions, and you should keep it on your desk. It will be of interest not only to geriatric psychiatrists and other specialists accustomed to providing care to the elderly but also to internists, hospitalists, family medicine specialists, general psychiatrists, neurologists, and trainees of all specialties who consult on an inpatient unit. Due to the growing proportion of the aged among our inpatient population, healthcare providers will be increasingly confronted with the complex psychiatric and medical symptoms prevalent among geriatric inpatients.

The editors have wisely focused on a range of topics which are often not addressed in one volume yet are commonly seen in clinical care of aging patients. Such issues include, for example, surreptitious or involuntary medication interventions, psychotherapies within the inpatient setting, medication strategies such as tapering and withdrawal, identification of discontinuation syndromes, and delivery of medical nursing care. The editors have chosen to include essential topics like pharmacological overview, delirium, the medical workup, adverse medical events, and the management of major neurocognitive disorder (MNCD) with behavioral disturbance. They have not included, as separate topics, issues that are already well covered in many other fine texts, such as treatment of psychosis, anxiety, and depression. Instead, they have woven these widely discussed subjects within other chapters, such as when, for example, to use ECT and neuromodulation for major depressive disorder or MNCD. Each chapter refers to other chapters which may expand on a related topic, so that the book is as cohesive and integrated as possible.

The text is supplemented by graphics which summarize the key points simply and attractively. In addition, flowcharts at the beginning of each chapter guide the reader through the topics which follow. These flowcharts offer suggestions for clinical decision-making, but without the "and/or" format of a decision tree, which often does not include the many nuanced choices available in actual clinical care. This is especially true in the geriatric patient, wherein the research evidence may not be available to provide definitive direction in each individual case.

I believe that this book could also be titled, “The Geriatric Psychiatry Inpatient: An Anatomy of Care,” and I commend it to anyone who provides treatment or consultation to the elderly in the psychiatric inpatient setting.

Stanford, CA, USA Ira D. Glick, MD

Preface

Inpatient Geriatric Psychiatry: Optimum Care, Emerging Limitations, and Realistic Goals

Treatment of the geriatric patient on an inpatient psychiatric unit is among the most complex challenges in clinical psychiatry. The patient is typically at the nexus of a number of systemic medical, psychiatric, psychosocial, and legal problems, all interacting in ways that are often impossible to parse and to address. Physiological factors of aging, acute and chronic medical conditions, acute onset of psychiatric illness and/or exacerbation of chronic psychiatric conditions (including neurocognitive disorders), medication interactions, family dynamics, personal loss, and legal matters may all contribute to the hospitalization. In addition, many conditions have no certainty of definitive cure or even amelioration. As a result, expectations from patients, family members, and others for clarification, sustained improvement, and/or resolution of problems may be unrealistic. Finally, adequate time for evaluation and treatment may be constrained by the current fiscal and health care delivery environment, which makes hospitalization days increasingly precious and limited by utilization review/insurance coverage in the USA.

Our review of the geriatric psychiatry literature and our combined clinical experience has convinced us that to search only for discrete, treatable diagnoses, though necessary, often does not encourage a more thorough exploration of the overlapping and interwoven nature of these problems nor the development of creative and balanced interventions. Acknowledging the practical limitations of inpatient geriatric care can help direct clinical attention to find interventions which can make small but meaningful improvements in quality of life. In light of this understanding, we have chosen to subtitle the book *Optimum Care, Emerging Limitations, and Realistic Goals*.

The challenge of unraveling complex symptoms and signs is placed before the inpatient team, with the attending physician taking the ultimate responsibility. And the evidence base for guidance in the assessment and management of these issues in the geriatric population is not robust. The psychiatrist is often left feeling as if only she/he were more competent, experienced, skilled, or knowledgeable, this could be accomplished within the limited time available.

The goal of this book, therefore, is to offer an accessible and realistic overview of common clinical problems which arise among geriatric psychiatry inpatients, along with current practices and novel approaches.

Content

With an eye to the practicing clinician, chapters in this book address common problems that arise in the inpatient setting, rather than address only specific diagnoses and their treatments. Therefore, we have chosen:

- *Not* to include the very important disease categories (e.g., major depressive disorder, schizophrenia, generalized anxiety disorder, bipolar I and II disorders) as distinct chapters. We refer readers to other sources, such as many fine texts and guidelines for illness-specific information (e.g., Hategan A, Bourgeois JA, Hirsch CH, Giroux C. (Eds.) Geriatric Psychiatry: A Case-Based Textbook. Springer; 2018).
- To discuss other non-illness-specific issues which often arise as problems in the inpatient psychiatric setting, e.g., suicide, legal aspects, involuntary interventions, and pain.
- To include chapters on topics that are covered less often in psychiatry textbooks, such as delivery of medical nursing care, communication barriers, telepsychiatry, and placement.
- To integrate the topics by cross- referencing their discussion in other chapters.
- To include colorful graphics which clearly illustrate concepts and show where topics overlap or are addressed elsewhere.

Flow Charts

At the beginning of each chapter we include a chart which provides an overview of the topics covered. These flow-charts are not intended as strict algorithms or prescriptive decision-trees of the only way to proceed. Rather, they illustrate one way an experienced clinician might approach a particular issue. The reader is encouraged to attend to the ‘fine print’ in each chart, which offers details to consider at each step. This process of rational decision-making can strengthen clinical judgement, especially in the complex context of the geriatric inpatient.

Terminology

- We adhere to standard DSM-5 diagnostic classifications for all psychiatric illnesses.
- We use the terms first-generation antipsychotic (FGA) or second-generation antipsychotic (SGA) rather than "typical" or "atypical."
- We do not use the term "neuroleptic" except in neuroleptic malignant syndrome, consistent with DSM-5.
- We use the DSM-5 term "major neurocognitive disorder," but occasionally add "dementia" in parentheses to help readers who are not familiar with DSM-5 usage.
- We have attempted to use consistent terminology across the chapters but have also respected the preferences of the chapter authors to use a term that they find meaningful.

Palo Alto, CA, USA — Howard H. Fenn
Hamilton, ON, Canada — Ana Hategan
Temple, TX, USA — James A. Bourgeois

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Part I

Foundations of Inpatient Geriatric Psychiatry

Essential Medical Work-Up and Rule Outs

1

Mirela S. Bucur, Heather Sylvester, and Ana Hategan

1.1 Introduction

Geriatric patients with psychiatric illness are at higher risk of developing medical disorders over the course of their lifetime, and the burden of medical disorders increases with age [1]. The Centers for Disease Control and Prevention (CDC) in the United States has found that nearly 80% of adults 65 years or older have at least one chronic systemic medical illness and 50% of them have at least two conditions; screening geriatric patients admitted to a psychiatric inpatient unit for underlying systemic medical illnesses is imperative [2].

For a number of reasons, it is easy to misdiagnose a symptomatic geriatric patient as having only an exacerbation of a psychiatric condition, to the exclusion of any contribution from other medical conditions. The possibility of cognitive decline, limited ability to articulate history, and accumulation of comorbid medical illnesses and medications make the process of evaluation more complex. Yet, many cognitive, emotional, or behavioral aspects of chronic medical conditions can be identified by thorough medical evaluations and review of medication regimens (Chap. 17: Medication Strategies). At the time of admission to an inpatient psychiatric unit, a standard and thorough diagnostic work-up for geriatric patients is essential. As the vignettes demonstrate, vigilance to the possibility of a medical, treatable condition applies also to patients transferred from medical settings to inpatient psychiatry or recently discharged from general medical settings. Figure 1.1 illustrates factors to consider in the essential medical work-up.

1.2 Clinical Vignettes

1.2.1 Clinical Vignette 1

A 73-year-old woman without any previous psychiatric history had a lower extremity cellulitis which was treated with an antibiotic and corticosteroid medication. She developed an infection with *Clostridium difficile* and respiratory distress, which resulted in a 5-day admission to internal medicine unit. Her discharge problem list included "lethargy, anxiety, and headaches." Medical history included type 2 diabetes mellitus, ischemic heart disease with dilated cardiomyopa-

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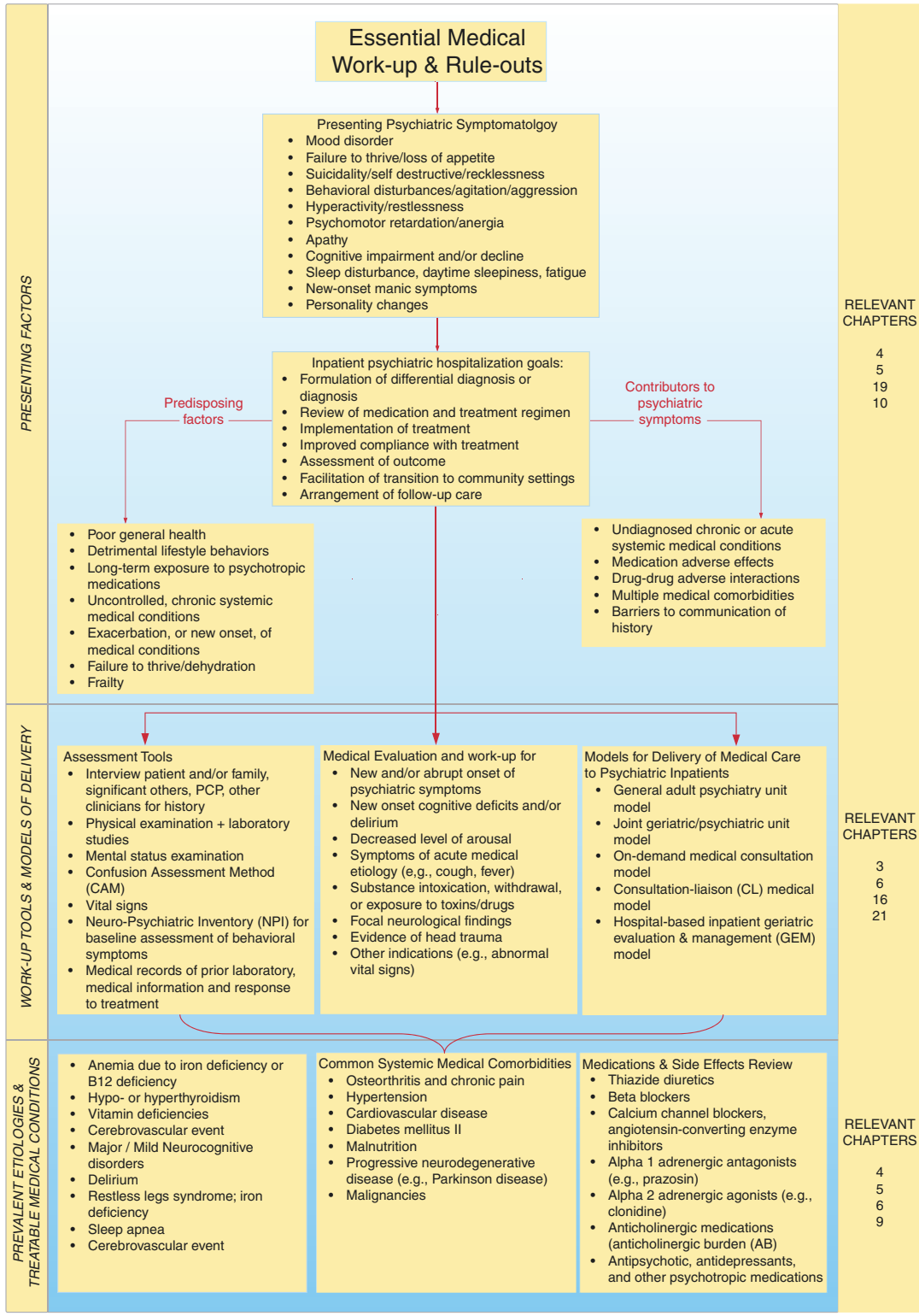

Fig. 1.1 Flowchart

thy and atrial fibrillation, asthma, sleep apnea, and anemia.

Five days after discharge from internal medicine, her anxiety symptoms worsened, her sleep-wake cycle reversed, and she developed confusion. She was admitted to the psychiatric unit in the same hospital. A complete medical work-up showed unremarkable laboratory investigations except for a poor glycemic control with a glycosylated hemoglobin (HbA1C) of 8.1%. The physical examination revealed residual skin rash on her right shoulder. Family members now reported that she had had varicella in childhood and, most recently, had shingles (infection with *varicella zoster* virus), 3 weeks prior to the current admission. This information had not been reported during her previous medical hospitalization.

The Montreal Cognitive Assessment (MoCA) scores fluctuated between 5/30 and 22/30 during a 24-hour period, with variable level of arousal and deficits in calculation, repetition, verbal fluency, attention, concentration, and delayed recall. Magnetic resonance imaging (MRI) coronal T2-weighted image showed a high-intensity signal in the temporal lobes including hippocampal formations and para-hippocampal gyri, insulae, and right inferior frontal gyrus. An electroencephalogram (EEG) showed a diffuse sharp-and-slow-wave complex. A lumbar puncture confirmed a diagnosis of herpes zoster encephalitis. The patient was transferred to internal medicine for further treatment; the consultation-liaison psychiatry team remained involved for management of neuropsychiatric symptoms caused by viral encephalitis.

Discussion The medical work-up of headaches, anxiety, fluctuating cognitive changes, and altered levels of consciousness diagnosed delirium due to encephalitis. Several viruses can cause encephalitis, including *varicella zoster* (varicella virus can reappear decades later as shingles). The reactivation of a latent *varicella zoster* virus manifesting as shingles and subsequently as encephalitis was triggered by the immunosuppressive effect of the corticosteroid. Neuroimaging showed characteristic changes in the temporal lobes. Electroencephalograms characteristic of herpes viral encephalitis usually show focal abnormalities, with periodic lateralized epileptic discharges. A definitive diagnosis came with testing of the cerebrospinal fluid by a lumbar puncture for presence of the virus.

This case illustrates the importance of in-depth medical evaluation of any geriatric patient, especially one without any psychiatric history, who presents with abrupt onset of new, disabling, yet unexplained, psychiatric symptomatology. The psychiatric symptoms in this case were not explained as part of aging nor as manifestations of a primary psychiatric etiology.

1.2.2 Clinical Vignette 2

A 69-year-old man with a long-standing history of bipolar I disorder was stable on lithium carbonate 900 mg/day (serum lithium level 0.8 mEq/L, or 0.8 mmol/L). His primary care physician started a nonsteroidal anti-inflammatory drug (NSAID) for unexplained peripheral neuropathy. Within 2 weeks he developed an acute fluctuating confusional state, which precipitated admission to internal medicine for evaluation of delirium. His medical history also included hypothyroidism, controlled with levothyroxine 125 mcg/day, and chronic kidney disease, with tubulointerstitial nephritis, both believed to be secondary to lithium therapy.

The physical examination upon admission showed altered mental status, fluctuating level of arousal, attention deficits, temporal spatial disorientation, tremor, dysarthria, and an ataxic broad-based gait. There was no muscular rigidity, and the serum creatine phosphokinase (CPK) was normal. His MoCA scored between 0/30 and 10/30. Laboratory results showed an elevated serum lithium level of 2.2 mEq/L (2.2 mmol/L), elevated serum sodium of 165 mmol/L and creatinine of 2.3 mg/dL, decreased urine osmolality of 180 mOsm/kg, and normal urine sodium of 42 mmol/day. The new hypernatremia was attributed to nephrogenic diabetes insipidus secondary to lithium toxicity. A computed tomography (CT) scan was noncontributory, with mild cortical atrophy, nonspecific white matter changes, but no

acute bleed or mass lesion. The electrocardiogram (ECG) had T wave inversion, and EEG showed a diffuse slowing of the background rhythm consistent with delirium.

Lithium was discontinued, intravenous hydration provided, with normalization of sodium, and he was transferred to inpatient psychiatry service for further review of psychotropic medication. After 5 days on the psychiatry service, his serum lithium was undetectable, after decreasing steadily; his sensorium was clear with full level of consciousness (LOC) and MoCA of 15/30.

Valproic acid (750 mg/day) was started to replace lithium for mood stabilization. Complete blood count (CBC), liver-associated enzymes, ammonia, and thyroid-stimulating hormone (TSH) were unremarkable, whereas serum valproate level was 50 μg/mL, a therapeutic level. Serum creatinine level remained unchanged in keeping with his chronic kidney failure. He was discharged home in stable condition.

At 2-month follow-up, his peripheral neuropathy, cerebellar impairment, and mild cognitive impairment (MoCA of 19/30) were still present. He now had new parkinsonian symptoms. MRI of the brain showed small areas of flair hyperintensities consistent with demyelination. A neurological consultation led to the diagnosis of Syndrome of Irreversible Lithium-Effectuated Neurotoxicity (SILENT).

Discussion The common side effects of lithium span a continuum from early reversible lithium neurotoxicity, to irreversible lithium neurotoxicity. *Reversible* lithium neurotoxicity resolves without any permanent neurologic sequelae. However, some patients develop SILENT syndrome, with cognitive impairment, sensorimotor peripheral neuropathy, cerebellar dysfunction, and extrapyramidal symptoms, all of which tend to persist longer than 2 months following an episode of lithium toxicity [3].

In published case reports of lithium toxicity in older adults (mean age, 71.4 years), manifestations of lithium toxicity varied, with neurotoxicity being the most common, followed by renal and cardiovascular toxicity [4]. Neurotoxicity has been reported to occur any time during the duration of lithium therapy. Early signs of lithium neurotoxicity (e.g., peripheral neuropathy in this patient) can be missed, and patients can progress to severe neurotoxicity, especially when other precipitating factors are present (e.g., drug-drug interactions which increase the lithium level, as in this case with a newly added NSAID). Although inconsistently reported, advanced age has been one of the precipitating factors for lithium neurotoxicity [3]. Medical comorbidity (76.3%) and polypharmacy (63.2%) have been reported as factors which precipitate lithium toxicity [4].

Most reported cases of lithium neurotoxicity showed lithium levels *within* the therapeutic range, and the condition was reversible, suggesting that serum levels may not exhibit a linear relationship with the intracellular lithium level. Serum levels *above* the therapeutic range may be present in the irreversible cases [3].

Patients with chronic lithium treatment are more susceptible to neurological adverse effects after an acute increase in lithium level. Neurotoxicity can persist despite falling serum levels of lithium, perhaps because of lithium's propensity to accumulate in the brain. As in this case, neurological sequelae can become more apparent after the improvement of LOC or remission of delirium. Some authors recommend red blood cell lithium level monitoring in addition to serum level, a more sensitive index of brain lithium levels [3]. Regardless of lithium level value, other laboratory investigations may show a low anion gap or a low urine-specific gravity suggesting lithium toxicity due to sodium loss.

Sodium levels should be monitored closely, as hypernatremia, other than due to lithium-associated nephrogenic diabetes insipidus, may also occur when large amounts of normal saline are administered. A complete thyroid function panel and renal function panel should be obtained in patients presenting with symptoms suggestive of hypothyroidism and in any case of suspected lithium toxicity.

Lumbar puncture is needed in patients with delirium and high suspicion of central nervous system infection. Neuroimaging studies are necessary to rule out structural etiologies. The vignette patient's MRI findings of demyelination at multiple sites reflect the proposed mechanism of SILENT [5]. The most common abnormal investigation associated with lithium neurotoxicity reported in the literature has been an abnormal EEG, consistent with delirium [3]. Although there are no standardized ECG guidelines for lithium therapy, cardiac side effects can result in both benign ECG changes and near-fatal arrhythmias [6]. As in this case, a systematic review has shown that T wave inversion is the most frequently reported lithium-induced ECG finding [6].

Indications for medical hospitalization may include signs of severe neurotoxicity requiring hemodialysis, a history of chronic lithium therapy with serum lithium levels > 2 mEq/L, and significant signs or symptoms of neurotoxicity, including any evidence of delirium, *regardless* of serum lithium levels. Intravenous hydration and hemodialysis provide good results. *Even when serum lithium concentrations are within reported therapeutic levels*, *regular clinical monitoring of lithium levels and renal function of patients on lithium therapy and appropriate caution should be accomplished*, *especially in older adults.* Figure 1.2 summarizes the EXTRIP (extracorporeal treatment for poisoning) workgroup recommendations for extracorporeal treatment of patients with lithium toxicity [7].

1.3 Models of Medical Care Delivery in the Geriatric Psychiatry Inpatient Unit

Geriatric patients who cannot be safely managed in community settings require complex and comprehensive care in inpatient psychiatric units or similar units. One important goal of inpatient treatment is to ensure safe care during treatment for complex psychiatric disorders and multiple comorbid systemic medical illnesses. Another goal is to determine a patient's ability to function safely and independently in a less restrictive setting after discharge. These goals are crucial for frail patients with neuropsychiatric symptoms [8].

Aging adults with psychiatric illnesses tend to be hospitalized for longer duration compared with younger adults, have increased hospital readmission rates, and tend to be admitted on an urgent basis, with increasing resource utilization and cost [8, 9]. Comorbid systemic medical conditions are more common among *readmitted* psychiatric patients than single admission patients, and their association with *readmission* can vary according to the nature of psychiatric disorders and characteristics of study population [9].

The need to increase effectiveness of hospitalization and prevent readmission focuses on clinician knowledge, increased clinical skills, and the integration of psychiatric and internal medicine inpatient services. The fragmentation of healthcare systems, with problematic separation of "physical" from "psychiatric" care, is

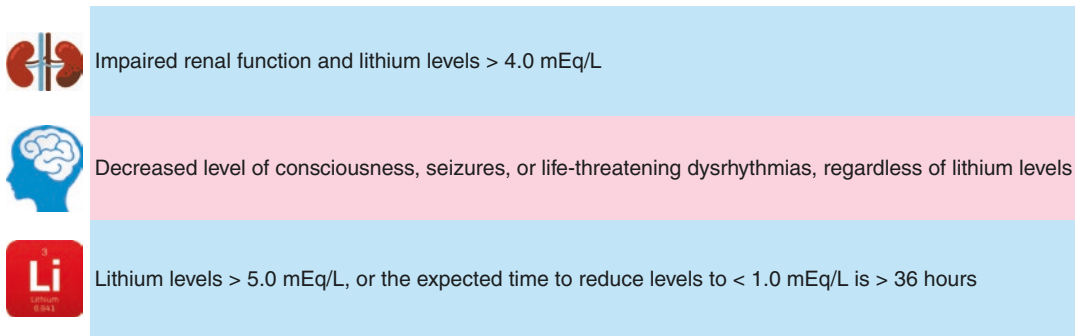

Fig. 1.2 Clinical recommendations for EXTRIP (extracorporeal treatment for poisoning) in lithium toxicity [7]

Table 1.1 Common models of psychiatric care in the general hospital setting [8, 10, 11]

Model of care	Description
General adult psychiatric unit model	Treatment of older adults on general psychiatry units. Studies that compare treatment in general psychiatry and psychogeriatric units are limited, but the paucity of outcome studies suggests that it is unknown whether psychogeriatric care provides more effective care than general psychiatry ward model
Joint geriatric/psychiatric unit model	Patients are treated in a secure environment which facilitates rehabilitation. The patient remains on the same unit. This model avoids missing systemic medical and psychiatric illnesses, care is person-centered, and family and caregivers are involved in care planning. Patients have access to acute investigations and hospital-based treatment. It ensures continuity of care with good community links and psychiatric and other medical staff to facilitate safe discharge. Specialized psychogeriatric units have been mostly affiliated with university hospitals. It may provide more thorough assessments than general psychiatry units, but it is not known whether it provides more effective care than general psychiatry units
On-demand psychiatric consultation model[a]	Usual care delivery of psychiatric service in general hospitals without specific dedicated consultation-liaison services and provides assessments and gives advice to clinicians. The limitations of this model: it is reactive and has a slow response to referrals, and general hospital referrals have a low priority
Consultation-liaison (CL) psychiatry model[a]	Consultation-liaison subspecialists have dedicated time for general hospital work, have a rapid response to referrals, and have an emphasis on teaching and training. The CL psychiatrist and the liaison nurse operate in a collaborative way. Studies tend to suggest that CL geriatric psychiatry services are more likely to consider post-discharge follow-up and community services as treatment options than pure CL services
Hospital-based inpatient geriatric evaluation and management (GEM) model[a]	An interdisciplinary diagnostic process and assessment of medical, psychosocial, and functional problems; it develops an overall plan for treatment and long-term follow-up. This model has proven to be successful in functional recovery particularly for patients admitted to medical units, with successful discharge outcomes and cost-effectiveness

[a]It describes a psychiatric consultation model to medical/surgical units, which does not operate on inpatient geriatric psychiatry units; however, it can be helpful in admitting patients and following after discharge

often detrimental. Multiple studies identified the insufficiency of financial and human resources to meeting the needs of hospitalized frail older patients [10, 11]. Table 1.1 summarizes the common models of psychiatric care in the general hospital setting [8, 10, 11].

It should be noted that models of care are being developed to replace full-service hospitalization. These include free-standing surgery centers and similar units that specialize in specific procedures, for which full hospitalization is not necessary. It is not clear yet whether these models can provide adequate services for the geriatric patient in need of close monitoring, round-the-clock care, and providers with expertise in geriatrics. The chapter on Information Technology (see Chap. 20) addresses the advantages of emerging technologies for care to outpatient and inpatient geriatric care.

1.4 Domains of Clinical Evaluation

Four sources of information comprise a full geriatric psychiatry evaluation: (a) interview and observation of the patient; (b) medical evaluation; (c) interviews of those with knowledge of the patient (e.g., family, significant others, primary care physician, other clinicians); and (d) medical records.

Laboratory studies should be considered individually, with a clear rationale: to rule in or rule out a diagnosis, to aid in the choice of treatment, and to monitor treatment effects and assess for adverse effects. Tests of hematological, thyroid, renal, liver, and cardiac function in a patient with bipolar disorder help the clinician choose among mood-stabilizing medications. Baseline and follow-up glucose levels and lipid panels may iden-

tify adverse effects of some antipsychotics, particularly second-generation antipsychotics. Blood levels of psychotropic medications are monitored for effectiveness, toxicity, and/or adherence. A cardiac evaluation may be important in the pre-work-up evaluation for electroconvulsive therapy (Chap. 16 Neuromodulation Interventions). Baseline and follow-up electrocardiograms may be required to identify adverse effects of lithium and certain antipsychotic or antidepressant medications on cardiac conduction.

Neuropsychological testing can ascertain the presence and rate of cognitive decline, and neuroimaging can determine the presence of a structural brain abnormality (Chap. 2: Neuropsychological Assessment).

Factors such as level of arousal, language, culture, and education level can influence the choice of diagnostic approaches. In the frail, aging patient, potential benefits of identifying and treating a condition need to be weighed against costs and physical and emotional stress of testing.

1.5 General Medical Examination and Common Medical Conditions/ Symptoms

Geriatric patients with abnormal vital signs, disturbance of arousal, agitation, acute or subacute disorientation, or other symptoms consistent with delirium are also likely to have a systemic medical etiology. The outcome can be fatal if untreated [12, 13] (Chap. 12: Delirium).

1.5.1 Arthritis and Chronic Pain

Osteoarthritis or degenerative arthritis is the most common form of arthritis, and its impact suggests a need for integrated management strategies based on the biopsychosocial model [14]. If not adequately addressed, chronic pain can contribute to disruptive behavior, depressive disorders, sleep disruption, and demoralization [14] (Chap. 14: Pain and Chap. 6: Major Neurocognitive Disorder with Behavioral Disturbance). Treatment for pain is symptomatic, using scheduled doses of acetaminophen, with nonsteroidal anti-inflammatory drugs (NSAIDs) as second line of treatment. NSAIDs are associated with gastrointestinal complications, particularly in aging adults. Patients should be encouraged to avoid smoking, alcohol, and use of laxatives to further reduce the gastrointestinal side effects of NSAIDs. Misoprostol, a prostaglandin analog, can help prevent gastrointestinal effects of NSAIDs. Cyclooxygenase-2 (COX2) inhibitors (e.g., celecoxib) cause less gastric mucosal irritation but can cause renal dysfunction and should be avoided in patients with liver disease. Codeine should be avoided in the geriatric patient due to the side effects of sedation, cognitive impairment, delirium, and constipation. Long-term codeine use has been associated with depressive symptoms [15]. Conservative measures such as physiotherapy and joint injection with chondroitin and glucosamine may help.

1.5.2 Hypertension

Hypertension is the second most common chronic systemic medical condition in the geriatric population. According to the 2017 guidelines, hypertension stage 1 is defined as systolic blood pressure between 130 and 139 mm Hg *or* diastolic pressure between 80 and 89 mm Hg, whereas stage 2 is defined as systolic pressure at least 140 mm Hg *or* diastolic pressure at least 90 mm Hg [16, 17].

Although the previous 2014 hypertension guidelines classified 140/90 mm Hg as stage 1 hypertension, this level is *now* classified as stage 2 hypertension under the 2017 guidelines. Under the 2017 guidelines, medication is recommended for stage 1 hypertension only if a patient has already had a cardiovascular event (e.g., heart attack, stroke) or is at high risk of heart attack or stroke based on risk factors (e.g., age, comorbid diabetes mellitus, comorbid chronic kidney disease, calculation of atherosclerotic risk) [16]. Pseudo-hypertension, which can be seen in older patients, is defined as falsely elevated cuff pressure due to calcification of the brachial artery.

Secondary causes should be considered if there is new onset of hypertension after age 50. These include renal artery stenosis and aldosterone-producing adenoma which presents with hypokalemia. Treatment of hypertension in the geriatric population is particularly cost-effective [18]. Studies using the 2014 hypertension guidelines conducted in older adults with hypertension have confirmed that antihypertensive therapy prevents fatal and nonfatal myocardial infarction and improves overall cardiovascular survival [18].

Education about non-pharmacological therapy and risk factors for hypertension can begin during an inpatient hospitalization. Patients can be trained to use proper technique to measure their own vital signs. Regular, daily use of calibrated devices on an inpatient unit can establish blood pressure baselines and thereafter accurately assess response to medication treatment or lack thereof [19, 20]. A study conducted in hospitalized and ambulatory patients has shown that a high prevalence of older-old patients regarded as normotensive are true hypertensive (17.1% of hospitalized patients and 50% of ambulatory patients), or masked hypertensive (28.6% of hospitalized patients and 10.3% of ambulatory patients), with a higher risk of cardiovascular morbidity and mortality [20].

Choosing the antidepressant category in the geriatric hypertensive patient often rests upon the adverse effect profile of the medication; e.g., venlafaxine can precipitate hypertension [21]. The rise of blood pressure is dose dependent, more often at higher doses of venlafaxine, although the effect on blood pressure is weak [21]. The half-life of venlafaxine is relatively short, which explains the normalization of blood pressure shortly after discontinuation of venlafaxine. Although patients with cardiovascular disease are also at increased risk for developing depressive disorder, treatment with beta-blockers did not appear to further increase their risk [22]. Table 1.2 lists some potential side effects of antihypertensives in geriatric patients.

Table 1.2 Potential adverse effects of common antihypertensive agents

Medication	Adverse effects
Thiazide diuretics	Frequent urination, orthostatic hypotension, dehydration, hyponatremia, hypokalemia, hyperglycemia, hyperuricemia, hyperlipidemia
Beta blockers	Drowsiness, disturbed sleep, bradycardia, glucose intolerance, heart block, bronchospasm
Calcium channel blockers, angiotensin-converting enzyme inhibitors	Fatigue, dizziness, headache, hypotension, hyperkalemia, cough, nausea, renal impairment, constipation
Alpha 1 adrenergic antagonists (e.g., prazosin)	Depression, restlessness, sedation, dizziness, headache, fatigue, nausea, heart palpitations, edema, orthostatic hypotension, syncope, vertigo, rash, priapism
Alpha 2 adrenergic agonists (e.g., clonidine)	Anxiety, sedation, fatigue, dizziness, headache, orthostatic hypotension, xerostomia, nausea, constipation, atrioventricular block, hyperglycemia

1.5.3 Cardiovascular Disease and Common Circulating Biomarkers

Lipid abnormalities these have shown to have predictive potential as markers for primary cardiovascular events [23]. Markers for secondary cardiovascular events are more associated with ischemia. Fibrinogen has been a strong predictor for primary stroke [23].

Gamma-glutamyl transferase (GGT) a well-established serum marker for alcohol-related liver disease. Circulating serum GGT has also been linked to an array of chronic conditions and all-cause mortality [24]. GGT has been involved in the pathogenesis of cardiovascular diseases, especially coronary artery disease, and the prognosis of cardiovascular disease may be predicted by increasing GGT levels. Studies show that

GGT levels are related to cardiovascular emergencies in congestive heart failure, in which an elevated GGT level is an independent predictive maker for cardiac death [25]. The predictive value of serum GGT applies beyond current liver disease; it is also a superior marker for future disease risk.

1.5.4 Diabetes Mellitus

Between 10% and 20% of geriatric patients have diabetes mellitus. Most have type 2 diabetes mellitus with onset after age 40. Common symptoms of type 2 diabetes mellitus include polydipsia and polyuria. Women may experience recurrent vaginal candidiasis. Comorbidities of hypertension and hyperlipidemia are often present in type 2 diabetes mellitus.

Recurrent hypoglycemia is common in older patients with diabetes mellitus and is likely to be less recognized and under-reported [26]. In an inpatient psychiatric setting, worsening psychiatric symptomatology may be due to hypoglycemia, including nocturnal hypoglycemia causing insomnia. Recurrent hypoglycemia is likely to lead to frailty, and this relationship between hypoglycemia and frailty is likely bidirectional and mediated through several factors, including under-nutrition [26]. Because the brain is highly dependent on glucose for its metabolism, each hypoglycemic episode can cause major cognitive changes, including post-hypoglycemic delirium [26]. Therefore, recurrent hypoglycemia may be associated with subsequent development of major neurocognitive disorder.

The pathophysiological mechanisms of hypoglycemia include post-hypoglycemic neuronal damage, inflammation, coagulation defects, endothelial abnormalities, and synaptic dysfunction of hippocampal neurons [27]. A retrospective study of 16,667 patients (mean age, 65 years) with type 2 diabetes mellitus and episodes of severe hypoglycemia (defined as hypoglycemia needing a hospital admission or emergency department visit) found a graded increase in risk for major neurocognitive disorder (dementia) by 26% for 1 episode (hazard ratio (HR) 1.26, 95% CI 1.10–1.49), 80% for 2 episodes (HR 1.80, 95% CI 1.37–2.36), and 94% for 3 or more episodes (HR 1.94, 95% CI 1.42–2.64). This finding was *independent* of glycemic control, medications, and comorbidities [28]. The *excess* attributable risk of major neurocognitive disorder was 2.39% per year (95% CI 1.72%–3.01%) in patients with a history of hypoglycemia vs. those without a history of hypoglycemia [28]. Whether recurrent moderate hypoglycemia episodes increase risk of major neurocognitive disorder remains unknown.

Nearly 65% of deaths in older diabetic patients are caused by ischemic heart disease and cerebrovascular accidents [29]. Vigilant blood glucose control and control of hypertension, hyperlipidemia, and renal function can decrease these risks in diabetic patients. It has been found that improving energy intake, maintaining muscle mass, increasing physical activity, and a more conservative approach to glycemic targets in frail older patients with diabetes mellitus may be worthwhile [29].

Although some second-generation antipsychotics are associated with weight gain and diabetes mellitus in younger patients, it is unknown whether these medications have a similar effect on weight gain or glucose metabolism in the geriatric population. In a retrospective chart review of 1678 older adults in long-term care facilities, there was no evidence that short-term use (median 13.1 weeks) of second-generation antipsychotics was associated with the onset or worsening of diabetes mellitus [30].

1.5.5 Constipation

Because constipation is a subjective symptom-based complaint, what constitutes "normal" bowel function varies among individuals. The prevalence of constipation increases with age and differs across clinical settings. In institutional settings including long-term care facilities, the

prevalence is as high as 80% [31]. The comorbidity of constipation in patients with depressive and anxiety disorders is higher than in the general population. Healthy older adults may have a bowel frequency from three times a day to three times a week, similar to younger adults. Although several definitions developed by expert consensus are available for diagnosing constipation, constipation is defined as unsatisfactory defection due to infrequent stools or difficult or incomplete evacuation [31]. Clinicians often rely on the frequency or consistency of bowel movements to identify constipation, although patients tend to use terms such as straining, hard stools, and bloating [31].

Factors which aggravate constipation in the geriatric population include decreased fluid and fiber intake, inactivity, bowel lesions (including hemorrhoids), endocrine and metabolic disorders, and medication side effects (e.g., peripheral anticholinergic side effects of antipsychotics, opioids). Medical work-up may include a digital rectal exam looking for perianal disease such as fissures or hemorrhoids. A family history of colon cancer, hemoccult-positive stools, or weight loss should prompt a colonoscopy.

In aging adults, there is usually more than one mechanism for constipation, requiring an individualized but multifactorial treatment approach. Treatment includes cessation/dose reduction of contributing medications, when feasible, and addressing underlying causes for constipation. Dose reduction of antipsychotic medication may ameliorate anticholinergic effects, including constipation. Changing to an antipsychotic agent with a lesser anticholinergic profile can also improve symptoms (Chap. 17: Medication Strategies).

Although osmotic laxatives such as polyethylene glycol remain the mainstay of management, several new agents that target different mechanisms appear promising such as lubiprostone (a chloride-channel activator), linaclotide (a guanylate cyclase-C agonist), prucalopride (a 5HT4 receptor agonist), and alvimopan and methylnaltrexone (peripherally acting μ-opioid receptor antagonists) for opioid-induced constipation [32]. In inpatient settings, efforts to increase appropriate physical activity should be encouraged at least for general health outcomes and may improve symptoms of constipation, but definitive evidence for improvement in constipation is lacking [33]. Promoting fluid intake to improve symptoms of constipation has not been supported by the literature [33].

1.5.6 Weight Loss and Malnutrition

Major depressive disorder can result in significant weight loss due to anorexia. Other causes of weight loss must be ruled out, including medications that decrease appetite or affect the taste and smell of food, poor oral health, ill-fitting dentures, low income, smoking, alcoholism, disability, and neglect of older adults who have neurocognitive disorders or physical disabilities and who cannot function independently.

A loss of greater than 5% of body mass in 3 months or 10% in 6 months is consistent with increased risk of malnutrition [34]. However, malnutrition can easily be missed. Aside from unintentional weight loss, malnourished patients can present with fatigue, muscle weakness or loss of strength, bone fractures, anemia, and delirium. Malnutrition is most common in the older age group. The estimated prevalence rate for the general hospital population is between 29% and 61% of older adults [34].

Although body composition changes with aging, it also changes during malnutrition, with the loss of both fat and muscle tissue [34]. The changes which occur because of aging can be distinguished from malnutrition-related alterations. Sarcopenia is a loss of muscle mass, which may be an intrinsic part of the aging process rather than the effect of age-associated disease. Although physical inactivity is not the sole cause in the development of sarcopenia, increased activity reduces the muscle loss [34]. A direct relationship between the degree of malnutrition and increased hospital length of stay, treatment costs, and hospital readmission rates has been found [34].

Treatment of malnutrition in hospitalized geriatric patients remains an important factor influencing the outcome of clinical diseases; it

is vital for interspecialty and multidisciplinary approaches to help develop effective treatments.

1.6 Medical Evaluation Prior to Psychiatric Admission

1.6.1 Medical Evaluation in the Emergency Department (ED)

The term "medical clearance" is no longer in line with current terminology in the emergency departments. According to the 2017 American Association for Emergency Psychiatry Task Force on medical evaluation of adult psychiatric patients, a thorough history and physical examination, including vital signs and mental status examination, are the minimum necessary elements in the evaluation of psychiatric patients in emergency department [35].

Prior to transfer to psychiatric emergency services, psychiatric inpatient units, or other psychiatric settings, a transfer note should indicate that the patient is "medically stable," appropriate for treatment in a psychiatric setting, along with details of the assessment, results, the medical decision-making process, and recommendations for further assessment and treatment of any active systemic medical problems.

The ED assessment should include the conclusion that the patient's behavioral disturbance is unlikely to be due to a systemic medical condition or physical trauma and that medical treatment for any concurrent conditions is within the capabilities of the psychiatric receiving facility [35]. The facility which provides medical evaluation should define the continuing care required for both systemic medical and psychiatric disorders. This implies that necessary care will be available within a reasonable time frame at the receiving facility.

In the United States, it may be necessary to document that the patient is medically stable for transfer per the Emergency Medical Treatment and Labor Act (EMTALA) guidelines, although these guidelines should be considered the minimum rather than the standard level of care [35]. Medical capability varies widely among psychiatric facilities. Many psychiatric settings have limited medical capability, and, therefore, receiving physicians may still ask for "routine laboratory testing" for those patients otherwise deemed medically appropriate for transfer. These requests should be honored without further delaying the transfer. It may be reasonable to provide results of routine laboratory testing after the patient is transferred to the receiving facility through a proper communication process.

Table 1.3 Clinical situations for further medical evaluation [35]

Advanced age (65 years and older)
New onset psychiatric/behavioral symptoms
Cognitive deficits or delirium
Altered level of arousal
Positive review of systems indicative of a physical etiology (e.g., cough, fever)
Substance intoxication, withdrawal, or exposure to toxins/drugs
Focal neurological findings
Evidence or history of recent head trauma

The American College of Emergency Physicians Clinical Policies Subcommittee has developed a clinical guideline on evaluation of adult psychiatric patients and proposed that routine laboratory testing is unnecessary for asymptomatic, alert, and cooperative patients [35]. Many medical screening algorithms have been proposed; however, a simple algorithm with broad applicability to psychiatric patients in EDs has yet to be validated and widely adopted [36]. Table 1.3 lists clinical situations that may require further medical evaluation (e.g., laboratory evaluation, neuroimaging) [35].

1.7 Medical Evaluation in Inpatient Psychiatry Unit

Studies have also found that for the geriatric population, a comprehensive geriatric assessment (CGA), comprised of a multidimensional, interdisciplinary diagnostic inpatient process, including laboratory, physical function, and medical/physical assessment, can improve outcome at 1-year follow-up. A meta-analysis of 22 relevant randomized controlled trials (RCT) across six

countries, covering 10,315 frail geriatric patients, found that patients who underwent CGA were more likely to be alive and in their own homes at the end of scheduled follow-up, odds ratio 1.16 (95% CI 1.05–1.28; $P = 0.003$; number needed to treat 33) at a median follow-up of 12 months versus odds ratio 1.25 (1.11–1.42; $P < 0.001$; number needed to treat 17) at a median follow-up of 6 months, compared with patients who received general medical care. Patients who had a CGA were also less likely to die or experience deterioration (0.76, 0.64–0.90; $P = 0.001$) and were more likely to experience improved cognition (standardized mean difference 0.08, 0.01–0.15; $P = 0.02$) [37].

Patients aged 65 or older, those with neurocognitive disorders, low socioeconomic status, and history of poor outpatient follow-up often have laboratory abnormalities upon admission [38]. In asymptomatic patients admitted to psychiatric unit, some studies have concluded that the most warranted tests for limited screening were serum glucose, electrolytes, blood urea nitrogen (BUN), creatinine, and urinalysis [37, 38]. Patients on psychotropic medications should also be monitored for side effects of that specific medication.

The inpatient clinician must consider clinical guidelines and the rationale for any blood tests ordered, as well as laboratory tests that are *not* ordered but should have been (Chap. 5: Legal Aspects). Complaints or legal action can result if there is no appropriate follow-up for abnormal values, especially if adverse effects occur, and treatable conditions could have been addressed with the appropriate laboratory information.

In the inpatient setting, it is also important to discover if the geriatric patient has received regular screening tests such as breast exams, prostate exam, and/or colonoscopy.

When starting a patient on a new psychotropic medication, laboratory tests and other studies, including drug blood levels, may also be indicated. Common psychotropic medications can cause abnormalities in CBC (e.g., some antipsychotics, antiepileptics), electrolytes (e.g., selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, antiepileptics), renal function tests (e.g., lithium), liver function tests (e.g., antipsychotics, antiepileptics, antidepressants), thyroid function tests (e.g., lithium), and lipids (e.g., antipsychotics). One can argue that this entire battery of tests can be ordered just in case the patient needs one of these psychotropics.

A rationale for baseline laboratory tests is to screen for potentially treatable systemic medical disorders that may contribute to a psychiatric presentation. Laboratory evaluation for inpatients may be reserved for those with high pretest probabilities of having a systemic medical illness, particularly aging adults who present with systemic medical comorbidity, those with substance user disorders, patients with no prior psychiatric history, and patients who present with clear histories of recent systemic medical problems.

For example, thyroid dysfunction associated with alterations in mood and cognition has long been recognized. The yield of active case finding of clinical hypothyroidism in psychiatric patients is low. In a study of 200 patients with major depressive disorder, 2.6% of cases had subclinical hypothyroidism, and no overt cases of hypothyroidism were identified [39]. Interestingly, these patients were treated openly with fluoxetine for 12 weeks, and there was no association between response rate and thyroid status [39]. More explicitly, the presence of subtle thyroid function abnormalities did not have an impact on treatment outcome of depressed patients [39].

Subtle age-related changes in thyroid-stimulating hormone (TSH), free triiodothyronine, and free thyroxine occur in individuals who remain euthyroid [40]. Subclinical hypothyroidism remains the most prevalent thyroid dysfunction in older adults and is associated with depressive symptoms [40, 41]. In a study of 725 geriatric patients with major depressive disorder or dysthymic disorder and available TSH results, only 5 patients (0.7%) had high TSH (> 10 mIU/L) levels, but patients with elevated TSH did not differ from patients with normal TSH (≤5 mIU/L) results in the severity or symptom pattern of depressive disorder [42]. Borderline TSH results may not be the primary cause of a patient's depressive disorder [42].

However, others have demonstrated that subclinical hypothyroidism increases the risk for

depression. In a study of 323 individuals aged 60 years or older, subclinical hypothyroidism increases the risk for depressive disorder more than four times (odds ratio 4.8, 95% CI 2.7–8.6), which emphasized the importance of thyroid screening tests in older adults [41]. In a cross-sectional study of 1171 individuals aged 23 years–102 years, subclinical hypothyroidism and subclinical hyperthyroidism were more prevalent in older than in younger adults (subclinical hypothyroidism, 3.5% vs. 0.4%, $P < 0.03$; subclinical hyperthyroidism, 7.8% vs. 1.9%, $P < 0.002$) [40]. Older adults with subclinical hyperthyroidism had lower scores on Mini-Mental State Examination than euthyroid subjects (22.61 vs 24.72, $P < 0.03$) [40]. In adjusted analyses, those with subclinical hyperthyroidism were significantly more likely to have cognitive dysfunction (HR 2.26, $P = 0.003$) [40].

Treatment of overt thyroid dysfunction has been shown to largely resolve associated disturbances in mood and cognition [43]. However, the subtle detrimental effects on cognition in the context of overt hypothyroidism may not fully resolve [43]. Until future randomized controlled trials are conducted to confirm causality and guide the assessment of benefits vs. risks of intervention in those with subclinical thyroid disease, evaluating the thyroid function in older hospitalized psychiatric patients remains relevant to management.

With regard to neuropsychological assessment, these studies can help direct the appropriate care in the presence of altered sensorium or other cognitive impairments (Chap. 2: Neuropsychological Assessment). But collateral information from a reliable informant is necessary to determine whether these symptoms were present before hospitalization or have developed since inpatient admission.

1.8 Rationale for Laboratory Studies

Medical consultation is indicated for any aging inpatient, especially in those with systemic medical comorbidity. If this is not readily available, the psychiatrist should perform the physical exam, conduct a thorough medical review of systems, and order a battery of relevant screening evaluation tests.

Consider an electrocardiogram for surveillance of long QTc if initiating medications that may cause specific cardiac abnormalities (e.g., citalopram, ziprasidone). For those at higher risk for sexual transmitted infections, venereal disease research laboratory test (VDRL) or human immunodeficiency virus (HIV) testing may be needed.

Whether the cost of a test is acceptable depends on whether a patient is likely to actually benefit. For example, ordering electrolytes for a patient starting a selective serotonin reuptake inhibitor (SSRI) is reasonable for monitoring of blood sodium level (due to risk of SIADH), whereas ordering a CBC in every patient starting mirtazapine is probably not cost-effective, because agranulocytosis is an extremely rare side effect of that antidepressant, and it does not routinely require monitoring of white blood cell counts [44]. But if a patient on treatment with mirtazapine presents with symptoms and signs of infection, a white blood cell count may be obtained.

Checking blood ammonia level before starting valproic acid is not recommended because the baseline value is extremely unlikely to be abnormal. However, valproate-induced hyperammonemic delirium may occur in patients with normal liver function, despite normal doses and serum levels of valproic acid [45]. Therefore, ammonia level should be checked promptly in patients taking valproic acid who present with delirium. Note: laboratory testing recommendations are a framework of suggestions and not strict guidelines.

The most common reasons psychiatrists may order laboratory tests on their geriatric inpatients are presented in Table 1.4. Common routine tests to consider in ruling out systemic medical conditions in patients presenting with specific psychiatric symptoms are listed in Table 1.5. When starting a patient on a psychotropic medication, what common tests should be ordered at baseline and what should be ordered over time are presented briefly in Table 1.6.

Table 1.4 Rationale for laboratory tests in geriatric psychiatric inpatients

Reasons	Comments
Assessment of general health	Look for metabolic syndrome; think of preventive healthcare and refer the patient to a primary care physician post-hospitalization; and ensure bilateral sharing of laboratory results between attending and primary care physician
Differential with systemic medical conditions that may contribute to patient's presentation	Consider tests in psychiatric patients who have not responded to multiple medication trials or have pronounced somatic features
Prevention/management of drug-drug interactions	Assess whether the patient has liver or renal disease which may affect the pharmacokinetics of most prescribed medications
Obtain baseline values before starting specific medications	It depends on what adverse effects a medication can cause; hepatic function tests and serum creatinine are commonly recommended beyond general medical evaluation for medications known to cause liver and renal impairment

Table 1.5 Common rule-outs and laboratory investigations in geriatric psychiatric inpatients

Clinical presentation	Condition to rule out	Test
Depression, anergia	Anemia of any cause	CBC
Depression, anergia, weight gain	Hypothyroidism	TSH
Depression while on lithium prophylaxis	Hypothyroidism	TSH
Cognitive impairment	Hypo- or hyperthyroidism	TSH
	Hypo- or hyperparathyroidism	PTH, calcium
	Vitamin deficiencies	B_{12}, folate, 25-hydroxy vitamin D
	Cerebrovascular event	Neuroimaging
	Neurocognitive disorder	Neuroimaging, LP for CSF biomarkers (amyloid-beta and tau)
New-onset psychosis	Hypo- or hyperthyroidism	TSH
	Neurocognitive disorder	Neuroimaging
	Delirium	*Routinely*: CBC, electrolytes, glucose, renal and liver function tests, TSH, B_{12}, urinalysis *Not routinely*: urine drug screen, HIV and other infectious causes, calcium-binding protein S-100 B *When indicated*: neuroimaging, EEG, chest radiograph, LP, oximetry, ECG
	Anemia due to B_{12} deficiency	B_{12}
Rapid-cycling bipolar disorder	Hypo- or hyperthyroidism	TSH
Akathisia	Restless legs syndrome; iron deficiency	Ferritin
Sleep disturbance, daytime sleepiness, fatigue, or depression	Sleep apnea	Polysomnography
New-onset manic symptoms or personality change	Hyperthyroidism	TSH
	Behavioral-variant frontotemporal neurocognitive disorder	Neuropsychological testing, neuroimaging
	Cerebrovascular event	Neuroimaging
	Anemia due to B_{12} deficiency	B_{12}
Suspected alcohol abuse	Occult history of alcohol abuse	GGT, other LAEs

Note: *CBC* complete blood count, *CSF* cerebrospinal fluid, *ECG* electrocardiogram, *EEG* electroencephalogram, *GGT* gamma-glutamyl transferase, *LAEs* liver-associated enzymes, *LP* lumbar puncture, *TSH* thyroid-stimulating hormone, *PTH* parathyroid hormone

Table 1.6 Monitoring of selective psychotropic medications in geriatric patients

Medication	Tests before prescribing	Tests after prescribing
Citalopram/ escitalopram	ECG for those with cardiac disease, dose >20 mg/d of citalopram or 10 mg/d of escitalopram, concurrent use of QT-interval prolonging drugs (e.g., quetiapine, ziprasidone, levofloxacin, quinidine)	ECG if cardiovascular symptoms appear
All SSRIs	Electrolytes for hyponatremia (esp. if on diuretic) Routine bone density screening (suggested)	Electrolytes for hyponatremia if unexplained fatigue, dizziness, cramping, confusion; test within 30 days of starting the SSRI CBC if bleeding risk (not through platelet dysfunction)
Venlafaxine	BP	Regular BP; dose-dependent; close monitoring at dose ≥225 mg/d
Duloxetine	BP LAEs if suspect liver disease	Regular BP LAEs if suspect liver disease (avoid use in any liver disease)
Mirtazapine	BMI, fasting lipids	BMI periodically, fasting lipids after 1 year
Tricyclic antidepressants (secondary amine)	ECG	ECG after dose change, then q yearly Nortriptyline level (therapeutic window of 50–150 ng/mL)
Lithium	CBC, TSH, BUN, eGFR/creatinine[a], calcium, ECG	Li level q 1 week after dose change, then 6–12 months CBC, BUN, eGFR/creatinine, calcium q 6–12 months TSH q 2 weeks, 6 months, then yearly
Valproic acid	CBC, LAEs	VA level q 1 week after dose change, then 6–12 months CBC yearly LAEs q 2 weeks, 6 months, then yearly NH3 if present with delirium
Topiramate	Bicarbonate	Bicarbonate q yearly
Antipsychotics (second generation)[b] (see below for specific drugs)	Metabolic syndrome[c]: waist circumference, BP, glucose, lipids	Waist circumference[d] q monthly × 3, then yearly BP q 3 months, then yearly Glucose q 3 months, then yearly Lipids q 3 months, then yearly, then q 5 years if normal
Risperidone	Glucose, lipids	Waist circumference, glucose, lipids Prolactin if indicated
Olanzapine	Glucose, lipids	Waist circumference, glucose, lipids
Ziprasidone	ECG if cardiac disease	ECG after full dose in high-risk patients
Clozapine	WBC and absolute neutrophil count Weight/height Vitals signs ECG	WBC and absolute neutrophil count q week × 6 months, then q 2 weeks for up to 1 year, then q monthly Weight/height q monthly, then q yearly Cardiovascular, metabolic, and gastrointestinal risk assessment

(continued)

Table 1.6 (continued)

Cholinesterase inhibitors (donepezil, rivastigmine, galantamine)	HR	HR q 1 month after each dose increase If HR > 60 bpm and asymptomatic: repeat HR q 6 months; if satisfactory HR: repeat HR q yearly If syncope or seizure occur, stop the medication and refer to specialist If no causal relationship is found/if pacemaker is fitted, medication may be resumed
Memantine	Creatinine	Creatinine; if creatinine clearance <30 mL/min, reduce dose

Note: *BMI* body mass index, *BP* blood pressure, *BUN* blood urea nitrogen, *CBC* complete blood count, *ECG* electrocardiogram, *eGFR* estimated glomerular filtration rate, *HR* heart rate, *LAEs* liver-associated enzymes, *Li* lithium, *NH3* ammonia, *SSRIs* selective serotonin reuptake inhibitors, *TSH* thyroid-stimulating hormone, *VA* valproate

[a]Serum creatinine is used to calculate the estimated glomerular filtration rate (eGFR), which is a more precise measure of renal function

[b]Some guidelines recommend glucose and lipid monitoring with all second-generation antipsychotics

[c]Three or more criteria must be met for metabolic syndrome

[d]Women: > 35 inches; Men: > 40 inches

1.9 Specific Laboratory Investigations

1.9.1 Hematocrit

Anemia can cause anergia, which can be misdiagnosed as a symptom of major depressive episode. If symptoms of fatigue in a depressed patient are not improving with treatment, it is advisable to check a complete blood count.

1.9.2 White Blood Cell Count

White blood cells serve as the primary defense against infection. Neutrophils make up the majority of circulating white blood cells. Neutropenia is defined as an absolute neutrophil count of $< 1.5 \times 10^9$/L. Agranulocytosis refers to severe neutropenia (i.e., absolute neutrophil count $< 0.5 \times 10^9$/L) and is a potentially fatal side effect of several psychotropic medications, most notably clozapine and carbamazepine. Conversely, lithium can cause a benign elevated white blood cell count (i.e., leukocytosis). This effect appears to be common and is unrelated to plasma lithium levels [46]. CBC check along with a lithium level, renal panel, calcium, and TSH should be performed for patients on lithium (see Table 1.6).

1.9.3 Platelets

Many psychotropic medications can cause platelet count alterations. Valproate and carbamazepine can cause thrombocytopenia, defined as a reduction in platelet count to less than 100×10^9/L [47]. Women are significantly more likely to develop thrombocytopenia [47]. Additional risk factor is a lower-baseline platelet count. Research has shown that the probability of developing thrombocytopenia also increases at trough valproate levels above 100 μg/mL in women and above 130 μg/mL in men [47]. In order to exert an antimanic effect, the plasma therapeutic level of valproate should be in the range of 45–150 μg/mL [47]. Gastrointestinal intolerance to valproate may occur in some patients, especially in the acutely ill, which may preclude oral administration of the drug.

Although not studied in the geriatric population, IV injectable valproate has been shown to be an effective mood stabilizer for mania [48]. It is recommended to obtain a baseline platelet count, then 1 week after starting the medication, followed by monitoring the platelets every 6 months or annually, with an increased caution exerted for patients with bleeding disorders.

Selective serotonin reuptake inhibitors (SSRIs) can affect platelet aggregation possibly by decreasing the amount of serotonin reuptake in platelets, and this can lead to an increased risk of bleeding. Because of this effect, it is unclear whether SSRIs reduce the risk of primary and secondary ischemic disorders, which are common in geriatric age [49]. However, depressive disorder itself is a cardiovascular risk factor that may have been a major confounding factor in studies on SSRIs and thrombotic risk. Older adults appear to be the group in which both the risks and the benefits of SSRIs can be the greatest [49].

Unfortunately, there is no established laboratory value to monitor the bleeding risk of SSRIs. Clinicians must consider this adverse effect in patients on therapy with SSRIs, especially when there is concomitant use of oral anticoagulants and NSAIDs. One study on various antidepressant classes demonstrated that an increased risk of bleeding was strongly associated with the degree of serotonin reuptake inhibition [50]. In this study, low-risk medications for bleeding included mirtazapine, trazodone, nortriptyline, and bupropion; among intermediate-risk medications were venlafaxine, fluvoxamine, and citalopram, whereas the highest-risk medications included fluoxetine, sertraline, clomipramine, and paroxetine [50]. In a large population-based cohort of 1,363,990 incident users of antidepressants, SSRIs and more generally antidepressants with strong inhibition of serotonin reuptake were associated with an increased risk for spontaneous intracranial hemorrhage, particularly during the first 30 days of use (incidence rate ratio 1.68, 95% CI 0.90–3.12) and when used concomitantly with oral anticoagulants (incidence rate ratio 1.73, 95% CI 0.89–3.39) [51].

1.9.4 Sodium Levels for Hyponatremia

Most antidepressants are associated with hyponatremia due to syndrome of inappropriate antidiuretic hormone secretion (SIADH). The association is strongest with citalopram and lowest with duloxetine, venlafaxine, and mirtazapine [52]. Even mild hyponatremia is associated with cognitive impairment, gait instability, falls, osteoporosis, and increased morbidity and mortality [52].

Risk factors for the development of hyponatremia with SSRIs include older age, female sex, concomitant use of diuretics, low body weight, and lower-baseline serum sodium concentration [53]. In a cross-sectional study of older adult users of antidepressants, 23 of 30 patients with hyponatremia and available data on serum and urinary sodium were classified as having either SIADH or probable SIADH [54]. The authors of this study concluded that the mechanism of hyponatremia produced by antidepressants might be either an elevated antidiuretic hormone or an increased renal response to antidiuretic hormone. The incidence of hyponatremia in older adults due to use of SSRIs varies between 0.5% and 32% [53]. Hyponatremia generally develops within the first few weeks of treatment initiation (3–120 days) and is reversible between 2 and 28 days after treatment discontinuation [55]. Criteria for a SIADH diagnosis include serum sodium below 135 mEq/L (135 mmol/L), serum osmolality below 280 mOsm/L, volume expansion (without clinical hypervolemia or edema), hypertonicity of urine as compared to blood, and absence of dehydration. It is worth noticing that a correct diagnosis requires thyroid, adrenal, and renal functions to be normal [56].

Clinical guidelines have not been developed to direct screening for antidepressant-associated hyponatremia and whether a finding of

hyponatremia requires a medication change or other specific treatment [57]. Some have reported that treatment of euvolemia hypotonic hyponatremia associated with SSRI use should include fluid restriction to about 1 or 1 1/2 liters per day and mild diuresis with a loop diuretic [53]. Higher doses of loop diuretics and hypertonic saline are required in more severe cases [53]. The definitive treatment is to stop the medication, and most cases resolve within 2 weeks. Some reports have noted that re-challenging with the same or another SSRI or substitution of another agent from a different therapeutic class produced recurrent hyponatremia in some, but not in all cases [53]. Vigilance is needed to avoid the emergence of hyponatremia, a potentially life-threatening adverse event in older adults especially if other risk factors for developing hyponatremia are present.

1.9.5 Calcium and Vitamin D Levels

Both are necessary for bone mineral density and prevention of osteoporosis. Studies suggest that antidepressants, particularly SSRIs, may be associated with low bone density in both men and women, which can potentially lead to osteoporosis and stress fractures [58]. Older patients who take SSRIs and who have other risk factors for osteoporosis should receive screening, which includes bone mineral density testing and calcium and 25-hydroxy vitamin D levels by their primary care physician once discharged from hospital occurs.

1.9.6 Thyroid Panel

In psychiatry, thyroid levels are ordered to rule out hypothyroidism or hyperthyroidism as a cause of psychiatric symptoms or when thyroid hormone is used as an augmentation strategy to antidepressants for major depressive disorder. Furthermore, symptoms of underlying thyroid disease can present similarly to symptoms of psychiatric illness in this patient population. Therefore, screening geriatric patients is cost-effective.

The thyroid gland produces T3 (triiodothyronine) and T4 (thyroxine). T4 is considered a prohormone that is transformed into T3, the active form of thyroid hormone, in the peripheral tissue [59]. Both T3 and T4 are regulated by the release of TSH from the pituitary gland. Even subtle decreases in T4 levels often lead to substantial increases in TSH, making TSH an ideal screening test for thyroid function.

Typical symptoms of hypothyroidism include cognitive impairment, depressive disorder, psychosis, neuromuscular dysfunction, hair loss, high cholesterol levels, myocardial dysfunction, decreased exercise tolerance, and cold intolerance. In older adults, low thyroid function can mimic normal aging and other conditions as previously noted. Symptoms of hyperthyroidism include anxiety, mania, psychosis, tremor, sweating, diarrhea, rapid or irregular heartbeat, and heat intolerance.

Only a quarter of older hyperthyroid patients have typical symptoms, and this incidence decreases with increasing age [60]. In some, the clinical presentation of hyperthyroidism is similar to that seen in hypothyroidism and can be masked by other existing diseases such as diabetes mellitus. Not uncommonly, older patients with hyperthyroidism can present with "apathetic hyperthyroidism," with characteristic symptoms of apathy, depression, and weakness [60].

Serum total T3 and T4 levels include both "free" and "bound" forms, but the free form levels give a better estimate of the amount of thyroid that can be used by the tissues. T3 is used in psychiatry as augmentation strategy to antidepressants, while T4 is used in endocrinology to treat hypothyroidism. If T3 is used as augmentation to an antidepressant, start with a dose of 25 mcg daily with an average dose range of 25–50 mcg daily. Before starting T3, check a baseline thyroid panel with a TSH and a free T4 and free T3. If the values are normal, consider starting T3, and once or twice a year thereafter, check a thyroid panel. There are some potentially concerning side effects of T3. Older adults, especially those with pre-existing cardiac disease, can develop a T3-associated arrhythmia. In older patients, before starting the hormone, confirm that an ECG

has been done within the past 12 months and is normal. Chronic use of thyroid hormones can lead to bone loss, osteoporosis, and fractures. If T3 is started in inpatient setting, older adults should have their primary care physician monitor their bone density and consider calcium and vitamin D supplementation.

1.9.7 Investigations for Metabolic Syndrome

Metabolic syndrome is prevalent in older adults and increases the risk of cardiovascular disease. Metabolic syndrome requires not only monitoring for glucose and lipids but also for waist circumference and blood pressure (see Table 1.6). Second- and third-generation antipsychotics (e.g., clozapine, olanzapine, quetiapine, risperidone, ziprasidone, aripiprazole) variably increase the risk of metabolic syndrome and present many challenges for psychiatrists. Olanzapine and clozapine are more strongly associated with metabolic risks, whereas aripiprazole and ziprasidone are less associated [61]. Screening for metabolic syndrome is an important element of the psychiatric evaluation of patients who are prescribed antipsychotic treatment. (See Table 1.6 for the screening and monitoring of antipsychotic medication use.)

Elevated blood glucose and/or abdominal obesity have been present in all psychiatric patients with metabolic syndrome [61]. Aside medication, other secondary causes of metabolic abnormalities should be ruled out; e.g., elevated low-density lipoprotein (LDL) cholesterol can be caused by diabetes mellitus, hypothyroidism, liver disease, or chronic renal failure [61]. Some research has shown that the prevalence of metabolic syndrome in adult patients (such as high prevalence of obesity and triglyceride levels and low high-density lipoprotein (HDL) cholesterol levels) was significantly higher in patients with bipolar disorder compared with the general population [62]. Therefore, identification of these clinical parameters at treatment initiation and continuation of an antipsychotic medication is necessary.

The prevalence of metabolic syndrome in patients with schizophrenia in the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) studies was high and represented a significant source of cardiovascular risk, especially for women [63, 64]. Note: antipsychotic-associated metabolic complications have been produced across age groups, but research on the metabolic effects of antipsychotics specifically in older adults is still needed [65]. Nonetheless, it is presumed that metabolic side effects would be at least as likely to occur in older adults. Notably, the antipsychotics are not the only cause of increased metabolic risk, but they can lead to increased risk in combination with hereditary factors and other medical conditions (Chap. 5: Legal Aspects).

1.9.8 Prolactin

Research has shown that in older patients with schizophrenia who take antipsychotics, measuring serum prolactin levels may be a good way to monitor for toxicity [66]. Older patients have a greater likelihood of developing hyperprolactinemia on exposure to antipsychotic medications. Clinicians need to order prolactin levels if screening questions indicate possible hyperprolactinemia. Signs and symptoms of elevated prolactin levels in antipsychotic-treated older patients include decreased libido and decreased bone mass, whereas galactorrhea is very uncommon in postmenopausal women [67].

Among antipsychotics, olanzapine appears to cause only a modest prolactin increase when compared with first-generation antipsychotics in general or the second-generation antipsychotic risperidone [68]. Aripiprazole (a partial dopamine D_2 receptor agonist) was associated with a decrease in serum prolactin level [69].

1.9.9 Cardiac Investigations

A baseline and follow-up ECG for geriatric patients are required when using antipsychotics and antidepressants known to potentially affect cardiac conduction. Studies have shown that

there are no dose-response changes in the QTc interval in patients on aripiprazole, which makes this antipsychotic a safer medication for older adults with psychotic syndromes [69]. ECG monitoring is also important in lithium therapy. Although T wave inversion is the most commonly reported lithium-induced ECG finding, some cases have shown more serious cardiac adverse events such as ST elevation myocardial infarction, heart blocks, and the Brugada pattern [6]. Lithium-induced cardiac electrical changes have been dependent on both duration of treatment and the serum lithium level [6].

Baseline vital signs are necessary for treatment initiation with a cholinesterase inhibitor (donepezil, rivastigmine, or galantamine). This class of medications is contraindicated in second- or third-degree heart block in the un-paced patient, in those with QTc prolongation (men > 450 ms, women > 470 ms), and bradycardia of < 50 beats/minute. Clinicians should prescribe these medications cautiously if pulse is 50–60 beats/minute and the patient is asymptomatic [70] (see Table 1.6). In patients with left bundle branch block, unexplained syncope, and those on concomitant use of rate-limiting drugs (e.g., calcium channel blockers, beta-blockers), these medications should only be used with careful monitoring. If calcium channel blockers or beta-blockers are used to treat hypertension, alternative antihypertensive agents may be considered to allow the initiation of cholinesterase inhibitors.

1.9.10 Investigation of Motor Side Effects

The development of parkinsonism in aging adults treated with antipsychotic medications may represent an "unmasking" of underlying Parkinson disease (Chap. 11: Psychiatric Symptoms Comorbid with Neurological Syndromes). It remains unclear if exposure to the antipsychotic medication has any impact on the underlying neurodegenerative disease progression. However, in animal models, Parkinson knockout mice (considered a susceptible model for Parkinson disease rather than a disease model) are more likely to manifest the neurotoxic effects of dopamine antagonists, suggesting that there is a risk to potentiate human parkinsonism or unmask symptoms with exposure to such medications [71].

The concept that antipsychotics may unmask Parkinson disease is supported by observations that parkinsonian symptoms may worsen or progress in patients who have been exposed to antipsychotics despite cessation of the agent [72, 73]. A 15-year prospective study involving older adults found a 3.2-fold higher risk of Parkinson disease among those previously treated with antipsychotics [72]. A functional neuroimaging study of 50 patients suggested that persistent drug-induced parkinsonism in those with visually normal dopamine transporter imaging within 12 months may be associated with subtle decrement of dopamine transporter activity [73].

1.10 Summary

An essential medical work-up for geriatric psychiatry inpatients includes medical consultation, physical examination, laboratory, and other studies to identify and rule out medical conditions which may contribute to the presenting symptoms. Treatable medical conditions can be missed in the context of factors which often accompany the geriatric patient, including overlapping behavioral, psychiatric, and physical symptoms. An extensive meta-analysis of 22 studies, comprising a total of 10,315 frail geriatric patients, has shown that a comprehensive geriatric assessment (CGA) can have a positive impact on outcome at 1-year follow-up, in terms of return to home and possible improved cognition [37]. As the vignettes exemplify, even patients admitted from medical settings, or with recent hospitalizations and full medical work-ups, may still have medical conditions which require further medical and laboratory investigation in order to institute appropriate treatment.

Take-Away

- Geriatric patients with psychiatric illness are at higher risk of developing comorbid systemic medical disorders. In the United States, nearly 80% of adults aged 65 years or older have at least one chronic systemic medical disorder, and 50% of them have at least two chronic systemic medical disorders.
- The burden of systemic medical illnesses in psychiatric patients increases with age.
- Identification and management of systemic medical conditions can delineate, stabilize, and improve a geriatric patient's behavioral, psychiatric, and neurocognitive symptoms.
- Geriatric patients admitted to a psychiatric inpatient unit benefit from comprehensive laboratory and medical screening for conditions which precipitate, exacerbate, or mimic psychiatric disorders.
- Geriatric patients with psychiatric illness tend to be hospitalized for longer periods of time, have increased readmission rates, and tend to be admitted on an urgent basis, increasing resource utilization and cost.
- The frail geriatric patient is at high risk for medical complications, and hospitalization itself is a risk factor for decline.
- Patients with neurocognitive disorders, low socioeconomic status, and poor outpatient follow-up often reveal laboratory abnormalities during hospitalization.
- Patients on psychotropic medications should be monitored for adverse effects of those medications.
- A collaborative approach among hospital-based psychiatrists, other hospital physicians, and primary care physicians in the community can provide comprehensive, timely work-up, and optimal post-discharge follow-up.

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Neuropsychological Assessment

2

Heather E. McNeely and Jelena P. King

2.1 Introduction

Characterizing the quality and severity of cognitive impairment among geriatric inpatients in psychiatric and medical units can assist in diagnosis, treatment planning, and placement. Cognitive difficulties may reflect normative age-related decline, and/or episodic symptoms of pre-existing neuropsychiatric disorders: psychotic, depressive, or anxiety symptoms, transient variations related to situational stressors, changes secondary to an acute medical event, or decline due to mild or major neurocognitive disorders. Thorough neuropsychological assessment can help evaluate and establish:

- A baseline of cognitive function, by which to assess the progression of neurocognitive decline and the outcome of interventions.
- Disruptive behavior and its association with cognitive deficits.
- Specific cognitive and language deficits, such as aphasia, in order to fashion individualized interventions and improve communication for the delivery of medical care.
- Diagnoses of etiological subtypes of mild and major neurocognitive disorder to help in treatment planning, placement, interpersonal interventions, psychoeducation, and psychotherapy.
- Evidence relevant for legal capacity assessments, to be completed by professionals not involved in treatment (Chap. 5: Legal Aspects).
- Normative cognitive changes, differentiated from any cognitive deficits associated with preexisting neuropsychiatric disorders, transient changes due to medical conditions, or progression of mild or major neurocognitive disorder.

The vignette below provides an example of the contribution of neuropsychological testing to diagnosis and treatment planning.

2.2 Vignette

A 65-year-old right-hand dominant married female with a high school education sustained an ischemic stroke 6 months prior to her inpatient admission. Diffusion-weighted brain MRI showed an infarct in the right middle cerebral artery (MCA) territory in the posterior region, with some anterior cerebral artery (ACA) distribution, and evidence of a mass effect with a midline shift to the left. The patient demonstrated left-sided hemiparesis of her

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arm initially, but she made modest gains in physiotherapy. There was no pre-morbid psychiatric history. Medical history included hypothyroidism, low vitamin B12, and iron deficiency. There was no evidence of delirium.

Following the stroke, the patient was admitted to a Skilled Nursing Facility (SNF) where she stabilized medically but had difficulty coping, possibly due to a urinary tract infection (UTI). She did not participate in rehabilitation, she ruminated about appointments, and she repeatedly asked staff what time family would visit. She had difficulty doing exercises with the physiotherapist and struggled to use the white board and clock. Since the stroke she had lost interest in hobbies and needed reassurance.

The patient was admitted to acute inpatient psychiatry for evaluation and treatment of depressive and anxiety symptoms.

The patient's estimated pre-morbid general intellectual functioning, based on a psychometric measure, registered in the average range compared to appropriate normative data (i.e., women her age with 12 years of education). The husband described his wife at baseline as very organized and independent; she handled everything apart from finances (which he had always managed). He rated her comprehension of spoken material as intact, unless there were unusual technical or medical terms, or if a large volume of information was presented.

Once admitted to inpatient psychiatry, she was deemed able to participate in neuropsychological assessment. But during testing, she was distracted by environmental stimuli and required cuing. She was unable to tolerate more than 45 minutes in a single session; testing was divided into four shorter sessions over 3 days to accommodate fatigue and maximize her performance. Tasks requiring bi-manual motor involvement could not be administered due to her left-sided hemiparesis. Screening of visual perceptual acuity was intact, and despite her left hemiparesis, there was no left-side neglect. Her thought processes were concrete, requiring simplification of instruction. The patient answered direct questions about educational and occupational history. She struggled to compose answers to open-ended questions. She denied word finding problems, speech, or manual motor apraxia with her dominant hand. She was tearful and turned frequently to her husband for support. When the husband would leave, she perseverated about his return.

2.2.1 Neuropsychological Assessment Results for Vignette

(Please see full results in Appendix) Relatively intact performance: Simple auditory attention span, language including expressive vocabulary, and confrontation naming, as well as praxis with her dominant hand.

- Significant impairments across all measures: Visual spatial and constructional skill, including drawing a clock and a complex figure. When presented with a clock face without numbers with hands set to 10 past 11, ability to tell the time fell below the 2nd percentile; with numbers on the clock her performance improved slightly to the mildly impaired range.
- Mild to moderately impaired: Verbal and visual measures of complex attention, memory, and executive functions: deficits on both.
- Mildly impaired: Verbal fluency; practical reasoning-judgment.
- Overwhelmed: Memory poor when presented with objective memory tasks, with both verbal and visual stimuli; some measures were discontinued.
- Perfect score: When presented with forced choice recognition of the pictures used in the confrontation naming task.

2.2.2 Interpretation of Neuropsychological Test Results

- Evidence of a significant globalized decline in general intellect over her estimated pre-morbid baseline, with some spared but predominantly impaired cognition.
- Spared abilities were those cognitive functions sub-served by the relatively intact left

hemisphere, including language, dominant manual motor skills, and praxis.
- Verbal memory and executive skills were impaired though to a less severe degree compared to visual memory and visuospatial/perceptual mediated executive functions.
- Basic visual perceptual ability was intact; no evidence of left neglect of visual environment based on both qualitative observation and intact performance on a line cancellation task.
- Impairment on more complex visual tasks, including perception of angles, construction, visual attention, memory for visual stimuli, and reasoning/problem solving with visual stimuli.
- Performance on visual tasks was hampered by executive deficits, for example, tended to perseverate on unsuccessful strategies and lost mental set on numerous occasions, in keeping with staff observations during daily functioning and physiotherapy.
- Additional measures of frontal lobe functioning: Evidence of impairment in complex alternating attention and working memory, with both verbal and non-verbal stimuli.
- Learning and memory profile: Significant for impairment on tasks that required explicit learning, such as a list of words or geometric design.
- Memory benefitted from increased meaningfulness of stimuli (e.g., better able to remember faces vs. geometric shapes) and from the provision of cues. This pattern suggested that executive difficulties were hampering her memory performance.
- Anxiety impaired memory performance; when told she would need to learn and remember information she became anxious; the story memory task was then discontinued. When memory was tested in a less overt manner, for example, when asked if she recognized the pictures shown earlier during confrontation naming, recognition memory was errorless. Techniques were developed to minimize anxiety and facilitate new information acquisition when presented with new tasks in physiotherapy.
- Pattern of spared and impaired cognition indicated both right hemisphere and frontal lobe impairment consistent with what would be expected following a right MCA stroke with ACA involvement and watershed regions in right dorsolateral pre-frontal cortex.
- Medical, collateral history, and test findings were consistent with DSM-5 diagnosis of major neurocognitive disorder due to vascular disease.
- While anxiety contributed to cognitive difficulties on testing, particularly objective memory testing, significant visuospatial and executive deficits were prominent also in the absence of anxiety.
- Deficits consistent with the functional difficulties: Although staff had provided a white board and clock to keep track of appointments and visits, testing revealed that her ability to organize visual space was poor. The patient was no longer able to read an analogue clock accurately.
- Unless provided clear and concise instructions and continuous cuing to remain on task, she tended to perseverate and lose mental set. These frontal deficits would lead to difficulties in a wide variety of daily tasks, for example, problem solving and new learning.

2.2.3 Specific Recommendations for Inpatient Care and Discharge Planning

- Digital clock for prominent visual and executive deficits, to decrease visual distractions/clutter.
- Simple, concise verbal terms during physiotherapy exercises; new learning in very small steps, and frequent verbal cues and feedback to maintain engagement.
- Cognitive rehabilitation to reinforce relatively intact verbal skills, with word search and cross-word puzzles.
- Visual spatial skills improved with very simple visual puzzles.
- Outpatient medical care to treat cardiovascular risk factors that might contribute to further cognitive decline.

2.2.4 Neuropsychological Testing of the Vignette Patient Provided

- Specific recommendations for accommodation to the inpatient setting, and help with discharge planning.
- An understanding of the cognitive limitations in order to avoid less productive attempts to reverse semi-permanent cognitive deficits, in favor of strengthening domains of relatively intact cognitive functioning.
- Prompts for further medical assessment/interventions and functional supports.

2.3 Goals for Neuropsychological Assessment

Cognitive impairment can interfere with inpatient treatment engagement and clinical outcome, but it is often not specific and can reflect both *static* and *dynamic* factors. Normative aging, mental health conditions, and general medical conditions can negatively affect cognition, but whether these factors are interactive or additive is often difficult to determine. For an aging adult with a pre-existing mental health disorder, an acute or chronic medical event can add a layer of complexity to the cognitive profile. This can be challenging to disentangle, yet essential for treatment optimization and discharge planning. Neuropsychological testing can also help parse out the aforementioned factors and determine a baseline by which to assess treatment interventions.

Neuropsychological assessment may help determine:

1. Evidence for the diagnosis of mild or major neurocognitive disorder
2. Domains of cognitive strengths and weaknesses, to assist treatment planning or modify approach to communication
3. Identification of potential etiological contributors that may warrant further specialized assessment
4. Recommendations for discharge planning, safety, and legal-capacity issues

Figure 2.1: The process of the neuropsychological evaluation of the geriatric inpatient.

When mild neurocognitive disorder (NCD) or major neurocognitive disorder (MNCD) is identified or suspected, neuropsychological assessment provides data to help determine a specific etiology. Such information can inform the design of treatment plans, communication interventions, psychoeducation, and psychotherapeutic/non-pharmacological approaches (Chap. 18: Psychotherapies and Non-pharmacological Interventions). Standardized neuropsychological assessment offers relatively objective information to parse out the impact of normative age-related changes, level of education, and other factors that impact test performance. Figure 2.2 lists some goals of neuropsychological assessment.

Any change from the patient's own cognitive baseline is especially important for patients who pre-morbidly fall in the upper or lower ends of the normal distribution. Cognitive screening may be sufficient to determine if there has been a likely change from an "average" patient's normative baseline. An in-depth neuropsychological assessment is especially useful when the patient is likely to have been "non-normative" pre-morbidly, in order to accurately inform medical, psychiatric, or neurodegenerative etiological considerations [1]. Figure 2.3 lists some causes of cognitive change.

2.4 Evaluation Prior to Neuropsychological Assessment

Medical factors that impact cognition should be investigated *prior to* formal neuropsychological testing. Documentation of such factors may be available in previous medical workups.

- Relevant studies of neurobiological changes include a magnetic resonance imaging (MRI) study, which has advantages over a computed tomography (CT) scan. Both MRI and CT provide a gross estimate of cerebral volume loss. Advantages to an MRI scan include nuanced detail of neurodegenerative changes in the brain and localization of neurodegeneration, important for differential diagnosis

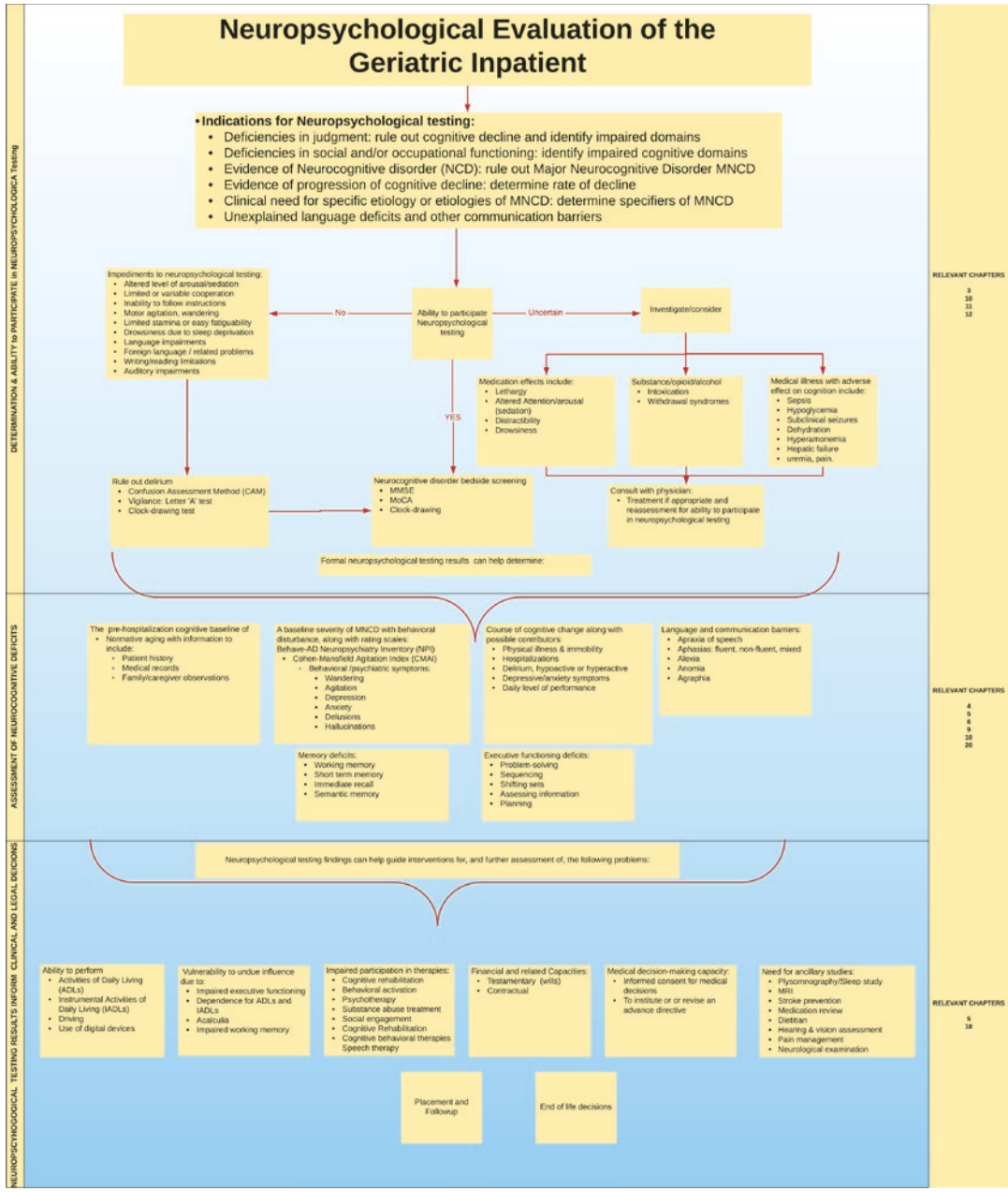


Fig. 2.1 Flowchart of neuropsychological evaluation of geriatric inpatient

(e.g., frontotemporal degeneration vs. global cell loss, space occupying lesions, or acute intracranial hemorrhage). MRI is more sensitive than CT in characterizing microvascular changes, including their severity and localization. Sub-cortical microvascular damage, in excess of that expected for normative aging, associated with executive deficits found with neuropsychological testing, would be an indicator of NCD due to vascular disease.

- Low vitamin B12 levels negatively impact memory ability, or can be an indicator of medication toxicity.

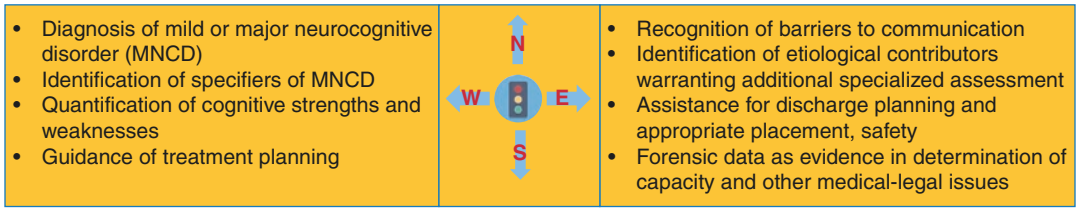
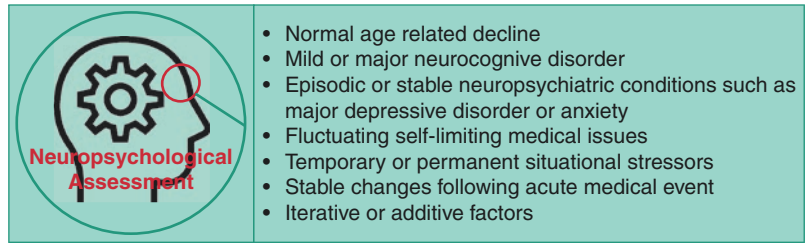


Fig. 2.2 Goals for neuropsychological assessment

Fig. 2.3 Causes of cognitive change



- History of, and prior laboratory evidence of, alcohol or substance use, and behavioral assessments are used to rule out withdrawal symptoms.
- Abrupt onset of confusion may indicate delirium due to reversible medical factors.

To ensure valid results, formal neuropsychological assessment should be completed *after* medical and psychiatric functioning has been stabilized. Unlike cognitive screening instruments like the Montreal Cognitive Assessment (MoCA) [2] or Mini Mental Status Examination (MMSE) [3], a comprehensive neuropsychological evaluation cannot be repeated after short intervals due to time and resource constraints; there is also a need to avoid the confounding effects of repeated practice (Chap. 6: Major Neurocognitive Disorder (MNCD) with Behavioral Disturbance).

It is also important to consider factors that may limit participation of the geriatric inpatient in neuropsychological testing: anxiety, fatigue, low motivation, motor deficits, reduced stamina, medical illnesses, and anxiety about the implications of test results. Systemic limitations within an inpatient unit may include time constraints, competition for hospital resources, and access to reliable collateral information.

2.5 Indications for Neuropsychological Testing

2.5.1 Distinction between Normative Aging vs. Neurocognitive Disorder

Brain volumes decrease in normative aging beginning with pruning in late adolescence that progresses across the lifespan, and accelerates after the fifth decade of life. While there is some degree of global atrophy, frontal and superior parietal regions are most impacted in healthy older adults [4]. Volume loss is due to a lesser extent from brain cell or gray matter loss, but is more strongly related to loss of white matter integrity. As white matter integrity is essential for neural signaling between and within brain regions, information processing efficiency declines as a result of these changes. Changes in intercellular connectivity and neuronal integrity translate into declines in processing speed,

immediate and delayed memory, and executive functions in aging individuals [5].

2.5.2 Attention and Executive Cognitive Changes

While normative aging does not impact focused attention or attention span (e.g., as measured by the forward digit span test), other aspects of attention are impacted. The research evidence regarding sustained attention in geriatric patients, however, is mixed.

Healthy older adults are impaired on measures of cognitive inhibition/inhibitory control; for this group, more effort in mental control is required to suppress attention to task-irrelevant material. Working memory, the ability to hold information in mind over a relatively brief period of time (up to about 3–5 minutes), while actively using that information to perform a mental operation (e.g., mental arithmetic), is also an aspect of executive attention that is impacted in normative aging. Aging adults may require longer to process information, may experience a reduction in sustained attention, and have slips in working memory.

Some results show no difference between aging adults and younger adults on specific sustained attention tasks in the laboratory. However, there are also well-established findings that show decrements in the inhibitory control aspects of attention, which could have down-stream negative effects—with age—on sustained attention during working memory tasks.

2.5.3 Memory-Related Changes

While forgetfulness is often considered part of normative aging, "memory loss" is not. Rapid forgetting is a pathognomonic indication of the most common presentation of mild NCD or MNCD due to Alzheimer disease (AD). Standardized neuropsychological measures can help distinguish normative aging from memory impairment secondary to most psychiatric disorders. Memory is not a unitary construct; normative aging impacts some aspects of memory but not others [6].

Memory is comprised of three primary processes:

(i) Encoding (learning: getting information in)
(ii) Consolidation (storage: keeping information in)
(iii) Retrieval (recall: getting information out).

Normative aging effects are most notable at encoding and retrieval. Retrieval cues can assist healthy older adults and those with major depressive disorder to access information in memory storage, such as a word list heard earlier, whereas the aging adult with mild NCD or MNCD due to AD will *not benefit* and may not recall having ever been read a list; this is a classic indication of the "rapid forgetting" characteristic of the amnestic presentation of mild NCD or MNCD due to AD (Chap. 6: MNCD with Behavioral Disturbance).

2.5.4 Mild and Major Neurocognitive Disorders

According to the *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition (DSM-5) [7], MNCD is evidenced by cognitive decline from a previous level of performance in one or more cognitive domains. Normative age-related decline versus pathological decline can be distinguished using objective neuropsychological measures, as the normative data is stratified based on age. A typically aging adult may be slower than they were during their middle age, but this will be reflected in the normative data from their age-matched peers. In contrast, an older adult with MNCD will evidence declines relative to their own baseline *and* the normative data for their age group. Mild NCD is characterized by mild and often isolated cognitive changes in the absence of significant functional declines. In contrast, MNCD is characterized by more severe and often multi-domain cognitive changes, accompanied by a global functional decline in activities of daily living compared to pre-morbid levels.

The aforementioned cognitive difficulties of mild NCD and MNCD are not better accounted for by delirium or another neuropsychiatric disorder (e.g., depressive disorder, anxiety disorder,

schizophrenia), thus the pre-assessment medical screening is crucial, as is psychiatric stabilization at the time of neuropsychological testing (Chap. 12: Delirium). Although changes in cognition may be based upon concerns of the patient, a family member, or a clinician, according to the DSM-5 "substantial impairment in cognitive performance [is] preferably documented by standardized neuropsychological testing" [7]. Numerous etiological subtypes of mild NCD or MNCD exist with variations in the patterns of cognitive impairments, nature of onset, and course of decline.

Formal neuropsychological assessment is often more relevant in the context of a suspected *mild* NCD, because impairment may be subtler and it is challenging to discriminate mild NCD from normative aging, or from the effects of common neuropsychiatric disorders.

2.6 Information and Techniques to Facilitate Valid Assessment

2.6.1 Information Supportive of Neuropsychological Test Interpretation

A medical chart review, as well as direct contact with the patient, caregivers, and family members, can provide a context within which to understand neuropsychological findings. Data includes: prior diagnoses; treatment responses; demographics; developmental, educational, and psychosocial history; and information pertaining to current concerns or complaints [8]. The patient may be unaware of her/his cognitive limitations or decline; and information should be sought from other reliable sources. Establishing rapport and engaging the patient/family in the assessment process is essential to ensure valid information.

2.6.2 Anxiety, Rapport, and Ethics

The neuropsychological evaluator may help establish a rapport with the patient by acknowledging that the hospitalization may have resulted from stressful events, an episode of confusion, delirium, fall, or other accidents. And hospitalization itself is a stressor. Aging adults within the inpatient context may be particularly worried about the implications of testing as it relates to potential loss of independence, for example, ability to return to their previous living environment or maintain a driver's license. Geriatric inpatients are also more likely than their younger counterparts to experience anxiety related to unfamiliarity with standardized testing. Orienting the patient to the assessment process, use of technology (if any), the purpose of the assessment, and allowing sufficient time for questions can all help minimize uncertainty and anxiety, and ensure more valid test results.

A point on ethics: the authors believe that it is ethical practice, even if not legally mandated, to inform the patient and family as to how the results of neuropsychological testing may impact legal/capacity determinations regarding independence and quality of life. Informing the patient and family of these potential consequences of neuropsychological testing, which may have significant impact on the patient, may be overlooked, because there is no physical risk involved in neuropsychological evaluation. If fully informed, some patients and family may choose to decline the testing, due to its impact on these factors. The dilemma is that patients *with most apparent and intact capacity to accept or refuse neuropsychological testing* may possess subtle cognitive deficits in greatest need of neuropsychological assessment (Chap. 5: Legal Aspects).

Figure 2.4 provides a summary of the factors that interfere with the validity of neuropsychological testing.

2.6.3 Collateral Information

Collateral information is most valuable if derived from those *most* familiar with, and able to observe the patient *prior to* the admission and *prior to* the onset of suspected cognitive changes. This may be particularly important if the patient lacks insight into her/his cognitive changes (common in some forms of NCD) or after an acquired

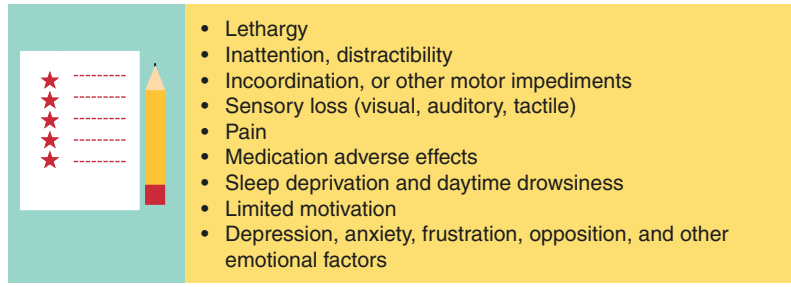


Fig. 2.4 Factors that can interfere with the validity of testing results

brain injury (such as frontal ischemic stroke). The helpfulness of collateral information is also enhanced if the patient deliberately minimizes or attempts to conceal difficulties because of her/his concerns of how this information might negatively impact their independence.

Aging adults with a history of neuropsychiatric illness that impacts cognition may have difficulty recalling specific details regarding their cognitive status. Here the collateral informant can indicate whether the current presentation is *consistent with* previous psychiatric episodes or *represents a change*. In situations where patient stamina is limited, with the patient's consent, the collateral informant may provide background information, and thereby free up the patient's time and stamina to be allocated to cognitive testing. Also, consultation with inpatient staff, and detailed review of medical record documentation can provide valuable information without taxing patient stamina.

2.6.4 Information on Pre-morbid Level of Functioning

The patient's pre-morbid general intellectual ability is a baseline against which cognitive decline is judged. The assessment of this baseline is complicated by the wide variation in individual differences in the general population. For a patient who was pre-morbidly high functioning, scores on standardized testing falling in the *average* range may in fact reflect a decline. Conversely, for patients with pre-morbidly *lower* functioning, impaired performance on standardized testing may be consistent with prior ability and not reflective of a decline [1].

The recommended method for establishing pre-morbid general intellectual ability is through a combination of standardized psychometric testing and demographic information, such as educational and occupational attainment [9]. The most commonly used psychometric estimate of pre-morbid ability is the single word reading test such as the National Adult Reading Test (NART) [10] and the North American adaptations of the NART, the American Version of the National Adult Reading Test (AMNART) [11] and the North American Adult Reading Test (NAART) [12], or the Advanced Clinical Solutions Test of Pre-morbid Function (TOPF) [13]. Single word reading is generally reliable in estimating pre-morbid baseline, since word reading correlates highly with IQ in healthy adults and is relatively resistant to decline in patients with various brain disorders [14].

However, in situations where the pre-morbid general intellect was low, the patient speaks English as a second language, or there was an undiagnosed verbal learning disability, poor performance on a reading test *may not reflect a decline*. Questions to include when probing educational and occupational history include: acceleration or delayed acquisition of basic academic skills, grades skipped or failures/repetitions, level of formal education completed, advanced degrees completed, and level of occupational attainment.

2.6.5 Cognitive, Emotional, and Physical Complaints

The most common cognitive concern among aging adults is memory [15]. Many patients may not be aware of or able to spontaneously generate a description of changes. Probing questions about

memory and other cognitive changes associated with the main etiological subtypes of NCDs are helpful. Memory complaints may actually be due to other cognitive difficulties, most frequently, poor attention or concentration. Physical problems or changes may also lead to cognitive difficulties. Determine, either through the interview, collateral or medical chart review, the presence of balance or motor problems, sensory loss (visual, auditory, tactile), pain, potential medication side effects, sleep quality, and daytime fatigue.

Emotional functioning, including mood, anxiety, and frustration, should also be assessed: symptoms of depression are often difficult to distinguish from a neurodegenerative process in the absence of formal testing. Anxiety or situational distress can lead to inattention and associated memory difficulties. When patients or collaterals endorse difficulties, it is important to verify whether this is a change from baseline, or a worsening or re-emergence of a longstanding difficulty.

2.7 Pre-morbid Daily Functioning

The diagnosis of *mild* versus *major* NCD is based, in part, on a *modest* (mild NCD), versus a *significant* (MNCD) decline in cognitive performance in one or more cognitive domains, in addition to either the absence (mild NCD) or presence of functional decline (major NCD).

A functional assessment can help determine whether there have been any changes to the patient's ability to independently perform basic activities of daily living (ADLs) and instrumental activities of daily living (IADLs). Basic activities of daily living include self-care tasks, while *instrumental* activities of daily living involve more complex tasks such as managing finances and preparing meals.

In order for a patient with documented cognitive changes to meet criteria for a diagnosis of *major* NCD, there must be *a substantial change or decline* in daily functioning, including ADLs and IADLS, compared to pre-morbid levels [7]. This can be very difficult to assess during an inpatient admission wherein the patient is temporarily removed from a usual environment, especially in the case of minor NCD where basic activities of daily living may remain intact. Again, seeking collateral information about possible changes to activities of daily living prior to the admission can assist in this determination.

2.7.1 The Course of Cognitive Performance Changes

It is not unusual for cognitive difficulties to first emerge or become apparent in an inpatient hospital setting. When cognitive, motor, sensory, or emotional changes or concerns are endorsed, the next step is to determine the onset and progression of these changes over time: sudden, insidious, progressive, step-wise, or static. Collateral information from a reliable observer may be the only way to verify the onset and time-course of cognitive difficulties.

Cognitive changes associated with certain forms of MNCDs such as Alzheimer disease (AD) are more likely to begin insidiously and gradually worsen over years. Patients and their family members may be unobservant of these insidious changes, or fail to appreciate their clinical significance, especially in the context of the patient's routine environment. A medical event or inpatient admission, or the removal of the patient from a routine environment, may "unmask" these longstanding difficulties. In contrast, the onset of cognitive difficulties associated with acute medical conditions such as myocardial infarction or stroke are more abrupt and static in course. A sudden onset and step-wise course of cognitive changes is often the profile associated with MNCD due to vascular disease, secondary to accumulation of cerebral damage caused by numerous small vessel and/or transient ischemic events. Cognitive difficulties associated with depression or anxiety disorders may be more state-dependent, waxing and waning with mood and anxiety fluctuations, but might also persist during periods of euthymia and worsen with age.

2.8 Cognitive Screening

Bedside testing or cognitive screening with tools such as the MoCA [2] or MMSE [3] is the recommended first step in the inpatient setting. These screening tools are brief, can be repeated to monitor progress over the course of admission, and inform the need for lengthier cognitive testing. Procedures to ensure valid and representative results include: (1) reviewing instructions with the patient prior to testing, (2) a quiet, distraction-free setting, (3) ensure the patient is alert, comfortable, and wearing corrective lenses or hearing aids (if required), and (4) use of native language or most commonly used current language.

General assessment of mental status and orientation is necessary and entirely feasible at the inpatient's bedside. Orientation questions include those addressing person, place, and time: full name, date of birth, place of birth, and current residence. Place can be assessed by asking the patient to name the state/province and city they reside in, and asking them to name first the type of building they are in (i.e., hospital) and, more specifically, the name of the hospital. Many patients will be able to surmise they are in a hospital based on their surroundings, but may have no idea which hospital they are in. If the patient is unable to state the type of building, multiple choice options may be offered, for example, "Is it a school, a hospital, or a library?" If the patient cannot spontaneously report they are in a hospital, but recognize the word from multiple choice, this suggests potential retrieval issues.

Orientation to time is assessed by asking the patient to state the day, month, and year, as well as the time of day. Ensure there is no visible calendar, clock, or watch before testing. Examiners should not provide leading questions or offer hints beyond multiple choice options. Bedside screening of memory is also easily conducted. To screen verbal memory, the examiner may state their name, ask the patient to repeat it back and remember it, and ask them again after a few minutes have passed. A short list of three simple words may be given to the patient with the preface, "I am going to tell you three words, I want you to listen carefully and repeat them back to me: Car, Banana, Paper." The patient repeats the words back several times to ensure they have attended to the items and encoded them. Inattention is often mistaken for memory impairment, as such, ensuring patients are attentive during the initial intake phase is important. After a period of 5–10 minutes, the examiner will ask the patient to recall the three words. To test visual memory, the examiner may show the patient three small objects found in the room or on their person, such as a pencil, pen, paperclip, newspaper, etc. The examiner informs the patient, "You see this pencil, I am going to hide it here (inside the tissue box). I want you to remember where I put it." This is repeated with the other items. After 5–10 minutes, the examiner asks the patient to either report or show where the items are hidden. Any gaps in recall would be indicators of memory impairment. Using visual memory items circumvents potential language barriers to memory testing, for example, with a patient who is non-English speaking or who has significant language disturbance. Bedside testing specific to aphasia/language disturbance is described below.

Bedside screening is a sensitive but not specific measure of cognitive impairment. More specialized neuropsychological assessment, as described below, should be reserved for those trained in the administration and interpretation of standardized psychometric measures in relation to appropriate normative data corrected for factors known to impact cognition, including age, gender, and level of formal education. In addition to special knowledge and training related to test selection, scoring, and interpretation, specialized training in neuropsychological assessment also involves appraising clinical status and test taking behaviors.

Qualitative observations of test taking behavior are often as important as the test scores. Moreover, these observations in combination with test scores should be interpreted by a clinical neuropsychologist with licensed competence assessing elderly adults, as there can be multiple reasons why a final total score is below expectation. For example, an aging adult being assessed

to evaluate frontal lobe impairment may perform relatively well on structured testing but between tests is observed to make socially inappropriate comments that are out of character or they may perseverate on previous topics of conversation. Although these latter factors are not objectively scored, they are important behavioral indicators of frontal compromise that might not be captured on structured and examiner-led standardized testing with elderly adults.

2.9 Neuropsychological Assessment Batteries

The clinical interview, collateral informant, referral source, medical history/chart review, in combination with logistical constraints (such as patient stamina, length of admission), and resource allocation can all guide the assessment in inpatient settings. In comparison to the lengthier outpatient assessment that may require an entire day, inpatient assessments will generally be divided into shorter testing sessions to accommodate patient fatigue and limited stamina. This also allows for flexibility and adjustment of the test battery, as needed based on patient performance and logistical constraints.

The assessment initially begins with a determination of the pre-morbid level and current general intellectual ability to identify any significant discrepancy that would raise the likelihood of decline consistent with an NCD. As described above, the pre-morbid function is typically psychometrically determined using an algorithm based on performance on a test of single word reading such as the NAART [12] or TOPF [13]. However, in cases of English as a second language (ESL) or known verbal learning disability, establishing pre-morbid intellectual ability from single word reading performance would not be appropriate, and in these cases, demographic information such as educational or occupational attainment should be used instead. Pre-morbid intellectual ability is compared to current intellectual ability most often obtained from the Wechsler Adult Intelligence Scale, Fourth Edition [16]. An abbreviated version is advised in geriatric inpatient settings when stamina is an issue, such as a two or four subtest Wechsler Abbreviated Scale of Intelligence, Second Edition [17].

Rather than using a number of stand-alone assessment tools, which tend to be more time consuming, neuropsychological assessment in the inpatient setting can be completed more efficiently with brief cognitive test batteries designed for geriatric populations. The results obtained from such batteries can be compared to standardized normative data, and is often sufficient for differential diagnosis when combined with clinical history and qualitative observations.

In certain instances, the results may also be used to direct the need for additional testing. For example, the Kaplan Baycrest Neurocognitive Assessment (KBNA) [18] was developed for a geriatric population and includes normative data for ages up to 89 years. The cognitive domains assessed with the KBNA are sufficient to obtain objective cognitive performance relevant to the diagnosis of the main etiological subtypes of neurocognitive disorder. Domains measured include: Orientation, Praxis, Attention/Concentration, Word List Memory Encoding/Immediate Recall, Delayed Word List Memory Recall, Delayed Word List Memory Recognition, Visual Memory, Delayed Visual Memory Recall and Recognition, Visual Spatial and Construction, Confrontation Naming, Verbal Fluency, Auditory Comprehension, Sentence Repetition, Practical Reasoning, and Conceptual Shifting. Additional tests may be added to a screening battery such as the KBNA to further evaluate particular clinical presentations, such as language disturbance.

2.10 Language Assessments: Aphasia

Indicators of aphasia in patients with a behavioral disturbance may include: communication deficits, confusion, right-sided weakness or sensory loss, impaired, unintelligible, or labored speech. Aphasia can be assessed by observation of the

patient's ability to produce, perceive, comprehend, hold in mind, and respond to spoken and written material, or with an aphasia screening test. Bedside screening of this cognitive domain can be conducted without the use of extensive formal testing [19]. Neurological consultation is crucial in order to make the subtle distinction between *verbal apraxia, non-fluent aphasia, other aphasias, and dysarthria* (Chap. 19: Medical Nursing Care and Communication Barriers).

2.10.1 Auditory Attention

Screening for aphasia should first assess auditory attention. The examiner stands behind the patient and makes a sound by rubbing the fingers together beside the patient's head, first both sides simultaneously and then unilaterally, varying single and simultaneous stimulation randomly. The patient is to indicate where the sounds are occurring: both, right, or left. If auditory *inattention* is detected, this may indicate types of lateralized brain lesions. It may also be caused by hearing loss that warrants further investigation and assistance with hearing aids or other devices. Audiology services may need to be consulted. Make sure hearing aids are turned on and working.

2.10.2 Expressive Speech Production

Evaluation of expressive speech begins qualitatively by evaluating the patient's spontaneous speech and assessment of the extent to which the speech is fluently produced. The presence of articulatory errors or atypical rhythms and intonations (dysprosody), poor grammar, errors of syntax (agrammatic), or misspoken words (paraphasic errors) may indicate *verbal apraxia.* Phonemic paraphasic errors are misspoken words that sound like the desired word (e.g., saying "asparag*rass*" for "asparagus") or semantic (e.g., saying "dominoes" instead of "dice").

Expressive speech is most often formally assessed with naming tasks such as the Boston Naming Test (BNT) currently in its second edition [20]. The BNT contains a short form for screening and a longer form for more detailed assessment. It involves a systematic presentation of a series of line drawings of items ranging from common (e.g., a toothbrush) to less common (e.g., a tripod). Patients are asked to name each item, and they are evaluated based on the amount of time taken to generate a response, the quality of the spoken response (i.e., evidence of phonemic or semantic paraphasias), or if phonemic or semantic cues or multiple choice, recognition cues are required. Normative data should be used for comparison purposes. Given the complexities in administration and evaluation of responses, the BNT should be administered by trained neuropsychologists. At the bedside, the clinician may ask the patient to name various items the examiner points to (e.g., cup, pencil), colors of items, or to name single digit numerals or letters shown on paper. Repeating a phrase or sentence, for example, "no ifs, ands, or buts", elicits disorders of articulation and sound sequencing [19]. If patients demonstrate difficulties with these screening tasks, formal neuropsychological assessment should be completed to obtain severity indices compared to normative data, using standardized aphasia batteries, such as the Boston Diagnostic Aphasia Examination [21].

As mentioned, assessment of the distinction between dysarthric speech, language deficits such as speech apraxia, or non-fluent aphasia should optimally include a thorough neurological examination and an evaluation by a Speech Language Provider (SLP) (Chap. 19: Medical Nursing Care and Communication Barriers).

2.10.3 Receptive Language Comprehension

Comprehension of spoken or written language can be assessed at the bedside using simple one-step auditory commands involving a motor response, for example, "Pick up the pencil," or "Show me your knee." Asking patients closed-ended "yes-no" questions is another way to screen for

comprehension, for example, "Is a ball round?" More extensive testing of language receptivity and comprehension involves presenting sentences or commands of increasing complexity or numerous steps, such as the Token Test originally developed by De Renzi and Vignolo [22]. Several modified, shortened versions of the Token Test have been developed, and a version is included in the Multilingual Aphasia Exam (MAE) [23]. Token Test stimuli include shapes, such as circles and squares, in two sizes and several colors. Commands increase in complexity from very simple, for example, "show me a circle," to more complex, for example, "Put the white circle in front of the blue square." Scores are based on accuracy of the steps and components of steps completed. Normative data for adults and older adults is available. Token test scores are sensitive to even minor impairments in receptive language [24] though globalized brain impairment and attention deficits may also lead to poor performance. Thus, performance on a Token Test should be contextualized within the overall clinical picture.

2.11 Visuospatial Assessments

Visuospatial deficits are often less readily apparent as compared to language deficits, however, these may occur as part of MNCD or mild NCD. For example, NCD due to vascular disease or to alcohol use disorder often presents with visuospatial and visual reasoning deficits prior to other cognitive changes. Visual-perceptual difficulties also typically manifest later in the progression of MNCD due to AD. Deficits in visuospatial and perceptual functioning, especially the ability to *speed up visual attention*, and to quickly scan the environment have important implications for driving. Declines in visual perceptual organization, as measured by drawing tasks such as the cube, interlocking pentagons, clock or complex figure copy raise concerns regarding the potential for visual disorientation during navigation in the environment. Slowed visual scanning/attention in excess of that expected for age, as measured by the Trail Making Test B raises concerns about driving safety, as patients may be unable to safely and efficiently scan the road ahead while alternating their attention to the instrument panel of the vehicle, for example. In general, Trail Making B total time over 3 minutes and/or three or more errors raises significant concerns about driving safety. In the case of clear MNCD, visual perceptual and visual attention impairment warrants removal of driving privileges. In the case of mild NCD with isolated findings on cognitive testing, an on-road test post-discharge can better inform functional driving ability.

2.12 Practical Considerations for Testing in the Inpatient Setting

2.12.1 Stamina and Fatigue Issues

Decreased mental stamina and fatigue, common with aging adults, becomes obvious in inpatient settings. Testing should capture the patient's best ability, thus, managing fatigue during the assessment is critical: allow extra time and break the testing into a number of shorter sessions with sufficient rests. Testing is best when the patient is most alert, usually in the morning, especially for patients with suspected MNCD.

2.12.2 Sensory Motor Deficits

An acutely admitted patient may come to hospital without prescription eye glasses and/or hearing aids, common in patients with mild or MNCD. Poor vision can interfere with performance on many tests; and missing words during a verbal memory test may be due to poor hearing, rather than inattention or impaired working memory (Chap. 19: Medical Nursing Care and Communication Barriers). Aging adults might

not mention the need for sensory aids. Standard visual and hearing aids should be available: inexpensive "reader glasses," test material where visual stimuli are printed in large format, and assistive hearing devices such as a pocket talker. Assessment of manual sensory or gross motor impairment is important for manual motor and praxis testing, and interpreting test results from timed tasks requiring a visual-motor response such as the Trail Making Test. Patients with arthritis, carpel tunnel, or fingertip sensory loss secondary to diabetes mellitus or peripheral neuropathy will often perform poorly on tests requiring manual motor responses. These impairments might reflect peripheral and not central nervous system deficits and could be disentangled with appropriate tests to make this determination.

2.12.3 Anxiety

Anxiety related to testing can increase in unfamiliar surroundings that unmask cognitive impairment, which was latent in a more familiar environment. Anxiety also revolves around the implications of cognitive testing for independence and driving ability. Digital technology and unfamiliar technologies can also exacerbate anxiety (Chap. 20: Information Technology). Attention to these issues can improve engagement and validity of formal neuropsychological assessment.

2.12.4 Time Constraints

Treatment and stabilization of acute psychiatric symptomatology, as well as a thorough medical evaluation, can take time in inpatient settings and delay completion of neuropsychological assessment. There may be pressure to complete neuropsychological testing prior to a planned discharge. Anticipation of the need for neuropsychological testing can avoid a suboptimal process and incomplete testing results. A brief and focused neuropsychological evaluation tailored to a patient's unique circumstances may be appropriate.

2.12.5 Foreign Language and Cultural Barriers

Foreign languages and/or cultural differences can be barriers to neuropsychological testing. Assess the level of formal education attained, nature of the education system, and age at which the patient first learned and spoke English fluently. Aging adults who learned English as a second language later in life may begin to lose their English language skills with the onset of cognitive changes. In contrast, some who have spoken primarily English for most of their adult life may be distressed at having lost fluency in their native language.

Language fluency is important for differential diagnosis of mild NCD and MNCDs, including AD and language variant frontotemporal dementias. English as a Second Language (ESL) individuals may exhibit apparent paraphasic word finding errors that may not be due to a language deficit per se but rather articulation challenges due to lack of fluent English pronunciation, or lack of familiarity with depicted test items common in North American society but *not common* in other cultures (e.g., some items on the BNT [21]). A professional interpreter for non-fluent ESL patients is recommended instead of a family member, who may be tempted to assist with the answers. Even with an interpreter, examiners should take care in selecting tests and interpret results with caution, because cultural influences may negatively impact performance and underestimate true abilities.

2.13 Systems-Related Factors

2.13.1 Resources

A thorough neuropsychological assessment can require as much as six or more hours of professional

time, exclusive of time devoted to interpretation of results and report-writing. In acute or tertiary medical settings, neuropsychology services can be limited due to time and cost. Allocation of neuropsychological assessment resources should be prioritized for geriatric inpatients wherein a neuropsychological assessment can inform diagnosis, identify safety concerns, or assist with treatment planning post discharge. An objective neuropsychological assessment is frequently requested to differentiate normative aging from NCD.

But not every aging inpatient needs formal neuropsychological assessment; often a thorough medical and psychiatric investigation will be sufficient to determine etiological factors, inform treatment, and discharge planning. For example, a geriatric patient with a 2-year course of worsening mood, fatigue, poor memory, and functional decline at home may appear to be an ideal candidate for a neuropsychological assessment. However, screening may reveal non-compliance with CPAP treatment for sleep apnea. Cognitive screening with a MoCA might be appropriate immediately, and comprehensive formal neuropsychological assessment only *after* reinstating sleep disorder treatment to eliminate the contribution of sleep apnea to the neuropsychiatric symptoms.

2.13.2 Access to Collateral Information

Collateral information provided by someone familiar with the patient prior to the onset of their medical or cognitive difficulties is of significant value; it can determine pre-morbid cognitive baseline, clarify suspected personality changes, and verify the extent to which a patient was functionally independent prior to admission. When a reliable collateral source is not available, differential diagnosis is more challenging; if mild or minor cognitive decline is suspected, repeat neuropsychological assessment to monitor for further cognitive decline and progression to major NCD.

2.14 Interpretation of Neuropsychological Findings

2.14.1 Differential Diagnosis and Interventions

Neuropsychological test findings are frequently used to inform diagnostic possibilities, prompt further workup, and develop treatment and environmental/safety recommendations. Treatment recommendations may include: cognitive rehabilitation and emphasizing important health and lifestyle changes to maintain or promote healthy aging. Patients diagnosed with mild NCD may be appropriate for interventions aimed at promoting cognitive resilience, such as Learning the Ropes for Living with MCI (Mild Cognitive Impairment) [25]. Learning the Ropes is a manualized intervention program that involves 6 weekly sessions and a follow-up session at 1 and 3 months. The intervention aims to improve healthy lifestyle behaviors in all participants, functional memory skills in participants with MCI, and adaptive coping skills in close relatives.

Multi-disciplinary healthcare staff familiar with cognitive impairment of mild NCD may be trained to deliver this program through online or in-person workshops (www.baycrest.org). Recommendations are provided for lifestyle changes that promote healthy aging and maintenance of cognitive function. These may include activities to increase behavioral activation, improve sleep hygiene and diet, reduce substance use, and remain cognitively and socially active. For patients with significant mood or anxiety symptoms, referral for psychiatric treatment should be initiated, as untreated psychiatric symptoms can mimic NCD in older adults. For patients without NCD with first onset of psychiatric symptoms in older age, referral to a health provider specialized in monitoring cognition, such as a geriatric psychiatrist or geriatrician, is recommended; onset of psychiatric symptoms in the aging adult is a risk factor for NCD (Chap. 18: Psychotherapies and Non-pharmacological Interventions).

2.15 Further Investigations

Neuropsychological assessment in the geriatric inpatient setting can identify cognitive findings that warrant further medical investigations. If neuroimaging has not been completed, an MRI of the brain may be warranted. This may be particularly relevant in those cases where cognitive deficits are identified, in order to investigate underlying neurological changes that could substantiate a diagnosis of mild or MNCD.

Neuroimaging may also aid in differential diagnosis, for example, by confirming pronounced frontal atrophy in a patient suspected of behavioral variant frontotemporal disorder who displays behavioral disinhibition and personality change, but who may have performed within normal limits on standard cognitive testing. Pre-morbidly high functioning individuals with post-graduate degrees and professional careers are able to sustain cognitive resilience despite advanced neuronal compromise, likely due to greater brain reserve. MRI would also be warranted in patients with numerous cardiovascular risk factors to assess the extent of chronic microangiopathic changes and identify potential remote infarcts that may have been undetected but are now affecting cognition.

Patients with a pattern of vascular cognitive impairment and/or risk factors may benefit from psychoeducation toward reducing vascular risks and/or referral to a stroke prevention program for monitoring. Other medical investigations can include: a sleep study in the case of probable untreated sleep apnea or other sleep disorder; medication review; assessment of vision or hearing; dietary review; smoking, alcohol, or substance use cessation; and treatment of chronic pain. All of these factors can contribute to cognitive deficits in excess of those expected for age. Targeting some of these factors with appropriate treatment could yield cognitive and functional improvement and prevent further decline.

2.16 Medical-Legal Issues

Formal neuropsychological assessment of geriatric inpatients can identify cognitive changes that have medical-legal implications. For example, a diagnosis of MNCD, or mild NCD, may be accompanied by deficits revealed in the cognitive skills required for safe driving, such as visual perceptual organization, the ability to divide and alternate visual attention between stimuli, quickly scan the visual environment, and respond appropriately. Many jurisdictions require health care providers to inform government transportation offices when clinical evaluation shows deficits that may impact driving safety. Depending upon the jurisdiction, an on-road driving safety test may also be recommended.

Neuropsychological testing also provides evidence with which to assess various decision-making capacities. Testing results may encourage adoption of a Power of Attorney, or a judicial appointment of Substitute Decision Maker, a surrogate, especially for health care decisions. Patients with only mild neurocognitive impairment and intact decision-making may be able to assert their decisions, through an advance directive, ahead of any future decline, allowing their wishes to be honored in the future when they no longer have decision-making capacity. Significant memory impairments or major neurocognitive disorder (MNCD) with behavioral disturbances may encourage arrangements for a controlled environment, such as a senior facility with a secure unit to prevent wandering (Chap. 21: Placement). Patients living at home may need a personal identification band with name, address, and phone number. Figure 2.5 provides a summary of some data helpful for incorporating into assessment of neuropsychological functioning.

2.17 Summary

Neuropsychological testing is an important component of a comprehensive inpatient geriatric psychiatry evaluation and can be considered the gold

Useful Information for Interpretation of Neuropsychological Findings

Source of data	Information to consider
Interview of patient	• Demographics • Developmental information • Level of education • Psychosocial history
Collateral Interview	• Current and past patient history from family members and others who know patient well
Premorbid Functioning Level	• Determination of Individualized premorbid baseline of functioning • Accommodate for any history of cognitive deficits
Current Daily Functioning	• Assess ability to manage activities of daily living - self-care, finances and meal preparation
Nature and Course of Cognitive Changes	• Assess if changes are sudden or insidious, progressive or static
Special Considerations	• Patient factors • Stamina and fatigue issues • Sensory motor deficits • Anxiety or adjustment issues • System related factors • Time constraints • Resources access to collateral information

Fig. 2.5 Information for interpretation of neuropsychological findings

standard for documenting subtle and/or significant impairments in cognitive performance. A full battery of neuropsychological instruments, individualized to the specific patient, can help delineate differential diagnoses, track treatment outcome, determine specific neuropsychological functional deficits, and provide treatment recommendations. The effective use of neuropsychological consultation in the acute inpatient setting can also be the first step in the process of monitoring degenerative neurocognitive disease progression, the patient's response to treatment, cognitive decline, and the course of psychiatric/behavioral symptoms.

Take-Away

- A large proportion of geriatric inpatients present with some degree of cognitive impairment.
- Comprehensive neuropsychological assessment in the inpatient setting adds useful clinical data in order to develop:
  - A full differential diagnosis as well as a treatment plan with psychosocial interventions, to enhance safe and effective placement.
  - An opinion as to whether cognitive changes are transient and reversible, or more enduring.
  - A formulation of the nature, onset, and course of cognitive changes, and whether these changes coincide with acute medical or psychosocial stressors.
  - A baseline from which to distinguish normative changes of aging from mild or major neurocognitive disorder.
- Neuropsychological assessments of geriatric patients are supported by reliable collateral information, history of past behavior, and history of past functioning.
- Developmental history, psychosocial history, educational attainment, and occupational attainment, all obtained in the context of neuropsychological testing, can help establish an accurate pre-morbid baseline against which cognitive and functional decline can be measured.
- Bedside testing and cognitive screening can be administered in the inpatient geriatric setting. Bedside testing can be sensitive to cognitive impairment, but not specific.
- The presence of medical and psychiatric symptoms may affect neuropsychological testing results.
- The evaluator's ability to establish a rapport can ease anxiety and enhance the validity of neuropsychological testing.

Appendix: Table of Vignette Patient's Neuropsychological Results

Domain	Functional area	Test	Performance level[1]
Intellectual Functioning	Global Intellectual Functioning	WASI Estimated IQ	*Borderline*[2]
	Estimated Pre-morbid IQ	Test of Pre-morbid Function	*Average*
Attention	Attention	WAIS-III Digit Span	*Low Average*[3]
		KBNA Verbal Sequencing	*Mildly Impaired*
	Visual Attention/Visual Search	Albert's Lines KBNA Symbol Cancellation	*Intact*
	Processing Speed	Stroop Word Reading	*Moderately Impaired*
Executive Functioning	Phonemic Fluency	KBNA Phonemic Fluency	*Mildly Impaired*
	Semantic Fluency	KBNA Semantic Fluency	*Mild – Moderately Impaired*
	Logical Reasoning	KBNA Practical Problem Solving; NAB Judgment	*Impaired*[4]
	Initiation	Go-No-Go Task	*Impaired*[5]
	Cognitive Inhibition/Selective Attention	Stroop Interference	*Mildly Impaired*
Memory	Verbal Acquisition, Verbal Delayed Memory, and Delayed Recognition	HVLT List Learning Total	*Moderately Impaired*
		HVLT List Delayed Recall	*Moderately Impaired*
		List Delayed Recognition	*Mildly Impaired*
		RBANS Story Immediate Recall	*Discontinued*[6]
	Visual Acquisition, Visual Delayed Recall, and Visual Delayed Recognition	RBANS Figure Immediate Recall	*Severely Impaired*
		RBANS Figure Delayed Recall	*Severely Impaired*
		Warrington Facial Recognition	*Mildly Impaired*
		KBNA Picture Naming Delayed Recognition	*Intact*[6]
Language	Expressive Vocabulary	WASI Vocabulary	*Low Average*[7]
	Confrontation Naming	Boston Naming Test	*Intact*
Visuospatial Processing	Visual Perception	VOSP Screening	*Intact*
	Visual Construction	Clock Drawing, DRS Construction	*Moderately Impaired*
	Visual Abstraction	WASI Matrix Reasoning; Clock Reading; Hooper	*Mild to Moderately Impaired*
	Visual Orientation	JLO	*Severely Impaired*
	Right-Left Discrimination	Self	*Intact*
		Confronting Other	*Severely Impaired*
Praxis	Manual and Buccofacial Praxis	KBNA Praxis	*Intact*

[1]The descriptive labels of performance based on comparison to normative data. *Very superior* (≥98th percentile); *superior* (91st to 97th percentile range); *high average* (68th to 90th percentile range); *average* (30th to 67th percentile range); *low or "below" average* (16th to 29th percentile range); *mildly impaired* (6th to 15th percentile range); *mildly-to-moderately impaired* (2nd to 5th percentile range); *moderately impaired* (0.6th to 1.9th percentile range); *moderately-to-severely impaired* (0.1st to 0.5th percentile range); and *severely impaired* (<0.1st percentile)

Qualitative Results:

[2]According to the Wechsler classifications of IQ scores, a Borderline IQ includes scores between the 2nd and 8th percentile (IQ 70 to 79) and falls between the Low Average and Extremely Low classifications. In the vignette patient, the global IQ is less meaningful, as her verbal skills were intact (low average) but she did significantly more poorly on visuospatial tasks secondary to her stroke, and this resulted in a decline in her overall IQ score

[3]The patient performed in the average range on Digit Span Forwards, her performance on Digit Span Backwards and Sequencing (placing random numbers in consecutive sequence in her head) was mildly impaired. This was likely due to problems with working memory, but also a tendency to lose set and perseverate on a previous response mode (i.e., if not reminded each time what she was required to do on the task, she would revert to repeating the digits forwards)

[4]The patient was concrete when answering questions about what she would do in certain common situations (e.g., "what would you do if you smelled smoke in your home"). She repeatedly said that she would rely on her husband to handle the situation and, even when reminded that this was a hypothetical situation, was unable to improve on her answer. When similar questions were presented in the third person (e.g., "what should *one* do …") she no longer gave dependent responses, but her performance was still mildly impaired relative to expectations

[5]While the Go/No Go task does not have norms, qualitatively, the patient failed to respond to the stimuli on four occasions, suggesting a problem with initiation rather than inhibition

[6]When given two trials to learn a short story, the patient became anxious and overwhelmed. She showed better performance and less anxiety, when smaller amounts of information were presented at one time, when she attended well to a task, when she was not aware it was going to be a memory task, and when her memory was tested by recognition methods. For example, she made no errors on a delayed recognition task of 20 pictures that she had been shown during confrontation naming earlier in the session

[7]On an expressive vocabulary task in which she had to define words of increasing difficulty, the patient was noted to be concrete and had difficulty understanding that the task required her to give the *defining* features of the word

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3 Pharmacological Overview in Geriatrics: Pharmacodynamics, Pharmacokinetics, Laboratory Monitoring

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3.1 Introduction

Medical advances and life-prolonging measures have increased life expectancy for individuals in developed countries, including the United States and Canada. The US Centers for Disease Control and Prevention predicts that by 2030, approximately 20%, or 72 million Americans, will be 65 years or older [1]. As the general population ages, one can expect a substantial increase of aging adults with serious mental disorders [2]. Of patients admitted to inpatient psychiatric facilities and geriatric psychiatric units over the last few decades, a greater number have been medically complicated due to co-morbid medical diagnoses [3]. In addition, data suggests that about 25% of seniors take more than four prescribed drugs. This number of prescriptions correlates with a greater incidence of drug-related complications in advanced age [4, 5]. This increase in pharmacodynamic burden impacts a group already vulnerable to adverse drug reactions.

The controlled environment of the inpatient unit permits medication administration based upon reliable drug delivery and direct observations of outcome. Gradual medication adjustment, in concert with monitoring for target symptoms, adverse effects, laboratory findings, and physical examination, ensures the most efficacious results. The authors have found that after a geriatric patient is hospitalized, careful adjustment and discontinuation of medications is as important as adding new medications (Chap. 17: Medication strategies: Switching, Tapering, Cross-Over, Overmedication, Drug-Drug Interactions, Discontinuation Syndromes). Figure 3.1 provides and overview of the pharmacology issues pertaining to the geriatric inpatient.

3.2 Clinical Vignette

A 78-year-old man was admitted for exacerbation of congestive heart failure (CHF), worsening shortness of breath (SOB), loss of energy, weight gain of 15 pounds, anxiety, depressed mood, reduced level of arousal, limited attention, and cognitive decline.

History included: ischemic cardiomyopathy with an ejection fraction of 30%, hypertension, hyperlipidemia, chronic back pain, and coronary artery disease with a myocardial infarction at age 65. The patient stopped his 40-pack years of tobacco use at age 65. Medications on admission: aspirin 325 mg/day, metoprolol 50 mg twice daily, simvastatin 40 mg/day, lisinopril 20 mg/day, furosemide 40 mg/day, tramadol 50 mg three times per day, sertraline 200 mg/day, bupropion XL 300 mg/day, trazodone 100 mg/day, and alprazolam 0.5 mg nightly as needed for insomnia.

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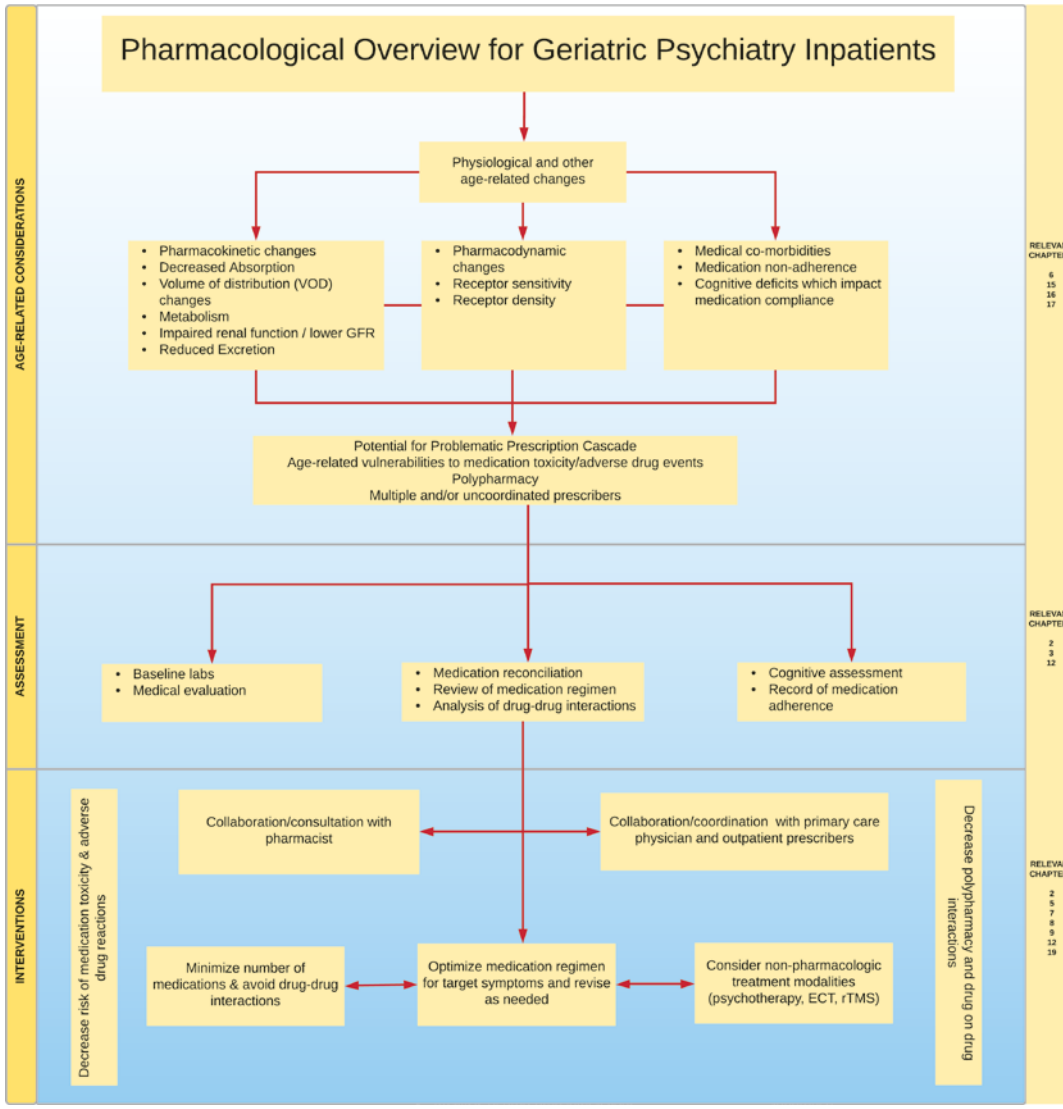

Fig. 3.1 Overview of pharmacology for the geriatric inpatient

The patient was widowed 12 months prior to admission, lived alone, and his children took him to medical appointments. He was behind in paying bills and often did not refill prescriptions. His family believed that he did not understand his medication schedule. Patient was eating adequately, but children had concerns regarding nutrition and follow-up for medical conditions. There was no history of suicidal attempts, but 1 week prior to admission the patient reported that he wished he was dead (Chap. 8: Suicide).

The dose of sertraline at 200 mg/day had been started by the primary care physician 4 months before admission. The depressive symptoms did not improve, and 300 mg/day of bupropion XL was added. Trazodone 50 mg at bedtime for insomnia was started 2 months prior to admission. The patient often awakened with orthopnea and felt anxious, even though his propranolol dose mitigated peripheral symptoms of anxiety. Alprazolam 0.5 mg at bedtime as needed was added 1 month prior to admission for anxiety.

It was not clear how often the patient used the alprazolam.

In the emergency department (ED), a disagreement arose among providers as to whether the patient would be admitted to inpatient psychiatry or internal medicine. The patient's dysphoric mood and suicidal ideation tipped the balance toward admission to the inpatient psychiatric unit with cardiology consultation.

Discussion The effectiveness of the patient's outpatient medication regimen, as well as his compliance, were in doubt. There was no practical way to assess his psychiatric symptoms, medication regimen, or to develop a treatment plan outside of a controlled setting. It was difficult to differentiate depressive symptoms from apathy, lethargy due to heart failure, anxiety, and/or sedation due to medications.

The first focus was a thorough review of the medication regimen and its impact on medical and psychiatric symptoms. A risk of serotonin syndrome, due to serotonin-active medications including sertraline, bupropion, tramadol, and trazodone, was discussed. The inpatient team discontinued bupropion, which had been started for symptoms of apparent intractable depression. The depressive symptoms overlapped with his low energy due to CHF exacerbation. The noradrenergic stimulation of bupropion had worked antagonistically with metoprolol and may have contributed to progression of heart failure. This medication combination and orthopnea contributed to insomnia, which had prompted a prescription for trazodone and alprazolam.

The internal medicine and cardiology teams continued cardiac medications. Intravenous furosemide was started on hospital day 1 to facilitate diuresis since the absorption of PO furosemide was not optimal due to hepatic congestion and gastrointestinal edema. Blood pressure, creatinine, daily weights, and intake/output were monitored. Over the next 5 days of diuresis, the patient lost 5 pounds, and furosemide was converted to PO dosing. Orthopnea became less frequent at night, and anxiety symptoms diminished slightly. Uninterrupted sleep became more frequent.

Concurrent with the aforementioned interventions, trazodone was continued on an as-needed basis for sleep at a dose of 25–50 mg at bedtime. Instead of the maximum dose, sertraline was restarted at 50 mg daily to minimize the risk for serotonin syndrome. The risk of a selective serotonin reuptake inhibitor (SSRI) discontinuation syndrome was considered, but deemed unlikely due to the relatively short-term duration of treatment with an SSRI. Over the next 5 days following admission, tramadol was tapered to a nighttime dose of 50 mg to minimize daytime sedation; the prior daytime dose had interfered with the level of arousal. Acetaminophen was added on an as-needed basis for pain.

Alprazolam was continued for the first 4 days, at a reduced dose of 0.5 mg per night. It was suspected that the patient had been taking alprazolam intermittently, often 3 or 4 times a night, and none on some nights. Over the next 5 days, alprazolam was tapered to 0.25 mg, then discontinued. By hospital day 10, the patient became more anxious at bedtime, and he began to express intense suicidal thoughts. A nighttime sitter was provided.

The inpatient team hypothesized that inconsistent outpatient medication adherence contributed to worsening CHF and depressive symptoms. Increase in congestive heart failure resulted in hepatic congestion, gastrointestinal edema, and acute kidney failure, which affected absorption, metabolism, and renal elimination of various medications. An outpatient prescribing cascade had contributed to worsening psychiatric symptoms after the addition of medications for depressive symptoms, insomnia, and anxiety.

After 10 days in the hospital, patient's level of arousal improved and he was more attentive and able to participate in a full neurocognitive assessment (Chap. 2: Neuropsychological Testing). Deficits in executive functioning were found, but no other evidence supported a major neurocognitive disorder (MNCD). This helped clarify that the contribution of depression to his cognitive decline

was minimal. His cognition had been impacted by medication effect and heart failure.

The patient's anxiety symptoms became less intense along with stabilization of heart failure. The patient no longer alluded to suicidal ideation. He was discharged to a skilled nursing facility which managed his medications. His family felt that he was more accessible and he could participate in group meetings. His heart failure continued to progress, however, and he passed away quietly within 2 years of discharge.

3.3 Pharmacokinetic Changes with Aging

Pharmacokinetics is defined as the processing of a drug or "what the body does to a drug": absorption, metabolism, distribution, and excretion [6]. With age, changes in the physiology of major organ functions affect the body's ability to process drugs. Pharmacokinetic changes are summarized in Table 3.1.

3.3.1 Absorption

Absorption is defined as the movement of a drug from outside the body into the bloodstream and tissues. Since most drugs are prescribed as oral formulations, the rate of drug absorption is influenced by factors that affect gastrointestinal (GI) motility, pH, and various other factors (e.g., expression of epithelial transporters). The rate of absorption of oral drugs is decreased in geriatric adults due to a decreased rate of gastric emptying, decreased intestinal motility, and reduced gastric acidity. In addition, many aging adults are prescribed medications that affect GI motility and pH, such as proton pump inhibitors, opiates, or pro-kinetic agents. Nutritional status may

Table 3.1 Pharmacokinetic changes with aging

Pharmacokinetic process	Changes with aging	Implications	Interventions
Absorption	Delayed gastric emptying Delayed intestinal motility Reduced gastric acidity (especially with proton pump inhibitors)	Reduced medication absorption Longer time to reach steady state or effective serum drug levels	Longer interval before titrating medication—more time between dose changes Different formulations may promote better absorption (e.g., IM or SL)
Distribution	Lipophilic drugs: increased volume of distribution Hydrophilic: decreased volume of distribution	Lipophilic: longer time to reach steady state and slower rate of elimination; longer elimination half-life. Hydrophilic drugs: lower dose required to reach effective therapeutic levels	Lipophilic drugs: longer interval before increasing dose Close monitoring of fluid status in patients sensitive to fluid shifts (e.g., on diuretics, vomiting, diarrhea)
Metabolism	Slower metabolism Decreased cellular metabolic activity	Increased parent drug/metabolite ratio Potential accumulation of unmetabolized drugs	Slower titration of medications to avoid accumulation Consider administration of metabolite Consider administration of drugs that do not require phase I metabolism
Excretion	Decreased GFR with age	Longer elimination half-life of renally excreted drugs	Start medications at lower dose, longer titration period, monitor medication levels closely; monitor serum creatinine

Note: *GFR* glomerular filtration rate, *IM* intramuscular, *SL* sublingual

also affect the rate of absorption of medications (e.g., the rate of absorption may be decreased in malnourished patients with lower expression of intestinal transporters) [7].

Delayed gastric absorption increases the time it takes to achieve serum steady-state levels even though the effective serum levels may not be significantly different [8]. There is a longer lag time to achieve effective serum levels before the need to increase medication doses. Alternate formulations may be considered, if there is poor absorption of oral medications and immediate efficacy is needed (e.g., intramuscular or sublingual formulations) (Chap. 13: Involuntary Interventions). Certain medications need to be administered with food (e.g., ziprasidone, lurasidone) to promote absorption.

3.3.2 Distribution

During aging, the percentage of body water decreases while body fat composition increases. Older adults have an average of 30% more body fat compared to younger adults [9]. As a result, the volume of distribution (VOD) of lipophilic drugs increases with age; while the VOD of hydrophilic drugs decreases. Consequently, the time it takes for lipophilic drugs to reach steady-state levels increases, as does the time it takes to *eliminate* them.

Malnourishment is a factor to consider when estimating distribution rates: the malnourished patient may have reduced fat composition, wherein lipophilic drugs reach steady-state, and are eliminated faster than anticipated. Nutritional status also affects the distribution of protein-bound drugs. Levels of free (active) drugs may be elevated disproportionately to the protein-bound (inactive) drug in patients with protein malnutrition or hypoalbuminemia, affecting its distribution between the aqueous and protein-bound environment [10]. As an example, the effective serum level of valproic acid may appear abnormally low in patients with hypoalbuminemia, as laboratory values represent only the protein-bound fraction of the drug [11]. Providers must specifically request *free* valproic acid levels if albumin levels are low.

Given the decreased VOD for hydrophilic drugs, lower doses are needed to reach therapeutic drug levels in older adults. In geriatrics, a *dose reduction* is usually needed for hydrophilic drugs. The decreased aqueous VOD in seniors makes them more sensitive to dehydration and rapid fluid shifts (e.g., aggressive diuresis, severe diarrhea, vomiting, with drug entry into third spaces such as ascites and pleural effusions). Normal therapeutic levels can quickly reach toxic concentrations in these conditions. Drugs such as lithium need to be used with extreme caution and monitored closely. In the inpatient medical setting, a patient's volume status may change from day to day, especially in those on parenteral fluids, and on medications such as diuretics. Close monitoring is warranted, especially in patients with renal and hepatic disease(s).

3.3.3 Metabolism

Once absorbed into the bloodstream, medications are metabolized by the liver through phase I oxidation via cytochrome P450 enzymes, and phase II glucuronidation to make them more soluble and more amenable to renal excretion (see Fig. 3.2). While most drugs require phase I biotransformation before phase II can occur, some drugs bypass phase I directly, and move to phase II modification. Genetic polymorphisms affect the expression of genes encoding for cytochrome P450 enzymes that may influence the rate of metabolism of different drugs. Genetics may play a role: about 8% of Caucasians are poor metabolizers of CYP2D6 substrates compared with the general population [12]. Cytochrome P450 enzymes can also be induced or inhibited by a variety of drugs. Any drug's half-life and concentration should be considered when co-administered with medications that are potential metabolic inducers or inhibitors. This is a concern in seniors who are prescribed a variety of medications.

Cellular metabolic activity is reduced with aging [13]. Hepatic insufficiency reduces cellular metabolism further, leading to decreased drug

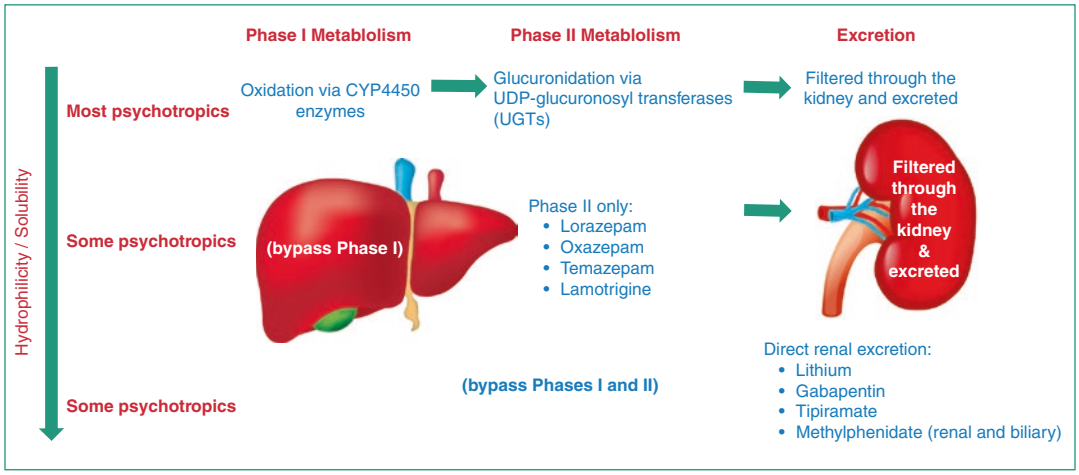

Fig. 3.2 Process of metabolism and excretion of drugs

metabolism, accumulation of unmetabolized drugs, and adverse drug reactions (Chap. 17: Medication Strategies).

In the geriatric patient, administration of a metabolite of the parent drug, if available, or a medication that bypasses phase I metabolism, can be useful. The metabolites of some drugs have bioactive properties and can be equipotent to the parent compound. For example, both the parent compound and metabolites of risperidone (paliperidone), bupropion (hydroxybupropion), and venlafaxine (desvenlafaxine) are highly active at their respective target receptor sites [14, 15] and amenable to renal clearance. Medications such as benzodiazepines, lorazepam, oxazepam, and temazepam, that do not require phase I biotransformation, are recommended for use in patients with liver disease.

3.3.4 Elimination

Drug excretion is most significantly determined by the glomerular filtration rate (GFR), which declines with aging for a variety of reasons. Microscopic, macroscopic, and functional changes in the kidney impair its ability to withstand and recover from injury, increasing the susceptibility of geriatric patients to acute and chronic kidney disease [16]. Even in healthy individuals, GFR can decline by up to 50% between the age of 30 and 80 years [14] and a GFR of 30–60 ml/min, equivalent to stage 3 kidney disease, has been observed in 15–30% of individuals aged 65 and older [17]. Some nephrotoxic drugs further impair renal function (e.g., NSAIDS) and further reduce GFR (e.g., Angiotensin-converting enzyme (ACE) inhibitors). The rate of excretion of hydrophilic drugs and metabolites is significantly decreased with aging, increasing the risk of toxic accumulation of these drugs.

3.4 Pharmacodynamic Changes in Aging

Pharmacodynamics is defined as the effects of a drug on the body or "what the drug does to the body" via targeted action on specific cellular receptors. Table 3.2 summarizes age-related pharmacodynamic changes that lead to increased sensitivity to medication action as well as their side effects [18, 19]. These include decreased receptor density, decreased receptor binding, and increased or decreased receptor sensitivity to the drug. Neurotransmitter receptor density decreases with age, and the central nervous systems of the geriatric patient becomes more vulnerable to agents that affect neurotransmission (most psychotropic drugs). The same medication doses administered to a geriatric patient may lead to greater effects as compared to a younger patient.

Table 3.2 Pharmacodynamic changes and increased sensitivity and side effects to medication

Age-related physiologic changes	Consequences
Decreased baroreceptor responsiveness and decreased sensitivity of the adrenergic system to adrenergic agonists and antagonists	Variable and less predictable responses to adrenergic agonists and antagonists leading to increased risks of both orthostatic hypotension and hypertension
Decreased dopamine-2 receptor density	Increased risk of parkinsonism and extrapyramidal symptoms in response to antipsychotics
Decreased cholinergic activity	Increased sensitivity to anticholinergic side effects of medications
Increased sensitivity of the GABA-ergic system	Increased risk of adverse effects from benzodiazepines (e.g., falls, imbalance, memory loss, sedation)
Decreased serotonin reuptake receptor binding and density of 5-HT1A and 5-HT2A receptors	Increased mood dysregulation and anxiety

Data derived from [14] and [15]

As a result of the pharmacodynamic changes, lower initial starting doses and target doses are often adequate to achieve the same therapeutic effect in older adults. In geriatric patients, increased sensitivity of the GABA-ergic system to benzodiazepines may only need to achieve 30–50% of serum levels to achieve the same therapeutic effect compared to younger patients [20]. Similarly, lower target doses of antipsychotics are recommended: only 50–60% receptor occupancy is required for optimum effects in older patients with schizophrenia, compared with the 65–80% occupancy recommended for younger patients [21]. Lower target doses of antidepressants have been recommended to avoid adverse drug reactions such as falls, bleeding, and cardiovascular events [19] (Chap. 7: Acute Medical Events). Given the overall decreased cholinergic activity with aging, older adults are sensitive to anticholinergic medications [22]. Geriatric patients are vulnerable to delirium from anticholinergic effects, or side effects of many psychotropic drugs (Chap. 12: Delirium). It is best to minimize the use of anticholinergic medications in this population when possible.

3.5 Categories of Psychotropic Medications

Tables 3.3, 3.4, and 3.5 offer prescribing recommendations and dose adjustments to moderate side effects of psychotropic medications in the geriatric patient [6, 23].

3.5.1 Antidepressants

After sedative-hypnotics, antidepressants are the most commonly prescribed class of psychiatric medications. Patients who are admitted to an inpatient geriatric psychiatry unit may already be on a medication regimen that includes an antidepressant for treatment of depressive or anxiety disorders. The outpatient doses need to be scrutinized to make sure they are compatible in the geriatric patient. Often, lower starting doses and a lower target dose are required in the geriatric patient due to pharmacokinetic and pharmacodynamics factors. High or maximum dosing of antidepressants requires attention to potential side effects and drug-drug interactions when evaluating the patient. If antidepressants are not tolerable, non-pharmacological interventions should be considered (Chap. 16: Neuromodulation Interventions: ECT, rTMS, and Novel Treatments; Chap. 18: Psychotherapies and Non-pharmacological Interventions).

Selective-serotonin reuptake inhibitors (SSRIs) are generally well tolerated in the geriatric patient, but certain SSRIs are high inhibitors of cytochrome P450 enzymes and have a higher risk of inducing drug-drug interactions (e.g., fluvoxamine, fluoxetine, paroxetine). Paroxetine is not recommended based on the Beers criteria, due to high anticholinergic burden, sedation, and orthostatic hypotension [24]. Low inhibitors include citalopram, escitalopram, and sertraline. Citalopram carries a risk for prolonging QTc measures and should be used with caution in

Table 3.3 Antidepressants

Medication	Dose adjustments needed		Anti-cholinergic side effects	Risk of drug-drug interaction	Risk of falls	Effects in geriatric patients
	Renal impairment	Hepatic impairment				
Antidepressants[a]						
Sertraline	No	↓ dose and frequency	*Low*	*Low.* Substrate of 2C19, 3A4. Weak inhibitor of 2D6.	Yes	↑ risk hyponatremia, bleeding
Citalopram	No	Yes	*Low*	*Low.* Substrate of 2C19, 3A4. Weak inhibitor of 2C19, 2D6.	Yes	↑ risk hyponatremia, bleeding ↑ risk prolonged QTc
Escitalopram	No	↓ max dose	*Low*	*Low.* Substrate of 2C19, 3A4. Weak inhibitor of 2D6.	Yes	↑ risk hyponatremia, bleeding Lower risk of prolonged QTc compared to citalopram
Fluoxetine	No	↓ dose and frequency	*Low*	*High.* Substrate of 2D6. Strong inhibitor of 2C19, 2D6. Weak inhibitor of 2C9.	Yes	↑ risk hyponatremia, bleeding
Paroxetine	No[b]	No[b]	High	*High.* Substrate of 2D6. Strong inhibitor of 2D6.	Yes	↑ risk hyponatremia, bleeding
Fluvoxamine	No[b]	No[b]	Low	*High.* Substrate of 2D6. Strong inhibitor of 1A2, 2C19. Weak inhibitor of 3A4.	Yes	↑ risk hyponatremia, bleeding
Venlafaxine	Yes	Yes	Low	*Low.* Substrate of 2D6, 3A4.	Yes	Dose-dependent risk of HTN and tachycardia ↑ risk hyponatremia, bleeding
Duloxetine	Avoid	Avoid	Low to Mod	*Mod.* Substrate of 1A2, 2D6. Moderate inhibitor of 2D6.	Yes	↑ risk hyponatremia, bleeding, useful for neuropathic pain and fibromyalgia
Bupropion	↓ dose and frequency	↓ dose and frequency	Low	*High.* Substrate of 2B6. Strong inhibitor of 2D6.	N/A	Lowers seizure threshold (avoid in seizure disorder), potential for precipitating psychosis
Mirtazapine	↓ dose and frequency	↓ dose and frequency	Low to Mod	Low. Substrate of 1A2, 3A4.	Yes	Sedation (at low doses), weight gain, constipation, leukopenia (rare)

[a]Serotonergic side effects: GI upset, decreased libido, erectile dysfunction, increased risk of bleeding, hyponatremia/syndrome of inappropriate anti-diuretic hormone (SIADH)
[b]Decrease dose of immediate release or extended release formulation in renal and hepatic impairment

Table 3.4 Antipsychotics

Medication	Dose adjustment needed		Anti-cholinergic side effects	Risk of drug-drug interaction	Risk of falls	Effects in geriatric patients
Antipsychotics[a, b]						
Haloperidol[c, f]	No	Mild to moderate: No Severe: contraindicated	Low	*Low.* Substrate of 1A2, 2D6, 3A4.	Yes[c]	↑ EPS ↑ QTc (avoid if QTc >500 ms)
Olanzapine[b, c, e, f]	No	No	High	*Low.* Substrate of 1A2.	Yes[c]	↑ metabolic syndrome (avoid in diabetes mellitus)
Risperidone[b, f], Paliperidone[f]	↓dose	↓dose	Low	*Low.* Substrate of 2D6, 3A4.	Yes[c]	↑EPS Hyperprolactinemia
Ziprasidone	No	No	Low	*Low.*	Yes[c]	↑ QTc (avoid if QTc >500 ms) Take with food for optimal absorption
Quetiapine	No	No[d]	High	*Low.* Substrate of 3A4.	Yes[c]	↑ metabolic syndrome, sedation
Clozapine[b, e]	Not defined	Not defined	High	*Low.* Substrate of 1A2, 3A4.	Yes[c]	↑ orthostatic hypotension, metabolic syndrome, sedation, lowers seizure threshold
Aripiprazole[b, c, e, f]	No	No	Low	*Low.* Substrate of 2D6, 3A4.	Yes[c]	↑ akathisia
Lurasidone	Yes	Yes	Low	*Low.* Substrate of 3A4.	Yes[c]	Take with food for optimal absorption
Asenapine[b, e]	No	No, but contraindicated in severe liver failure	Low	*Low.* Substrate of 1A2. Weak inhibitor of 2D6.	Yes[c]	↑ QTc (avoid if QTc >500 ms)

[a]Use lower doses for patients aged > 75 or those > 60 with frailty or multiple medical co-morbidities
[b]Class-wide side effects: extrapyramidal symptoms, akathisia, anticholinergic symptoms, sedation, orthostatic hypotension, metabolic side effects, cognitive decline
[c]Antipsychotics increase the risk of falls as a general medication category; increased parkinsonism and sedation side effects may contribute to fall risk
[d]Decrease dose of immediate release or extended release formulation in renal and hepatic impairment
[e]Available in sublingual formulation
[f]Available in long-acting injectable formulation

those with cardiac disease. Older adults are also more susceptible to hyponatremia, gastrointestinal or other bleeding, and falls with the use of serotonergic antidepressants [25].

Serotonin/norepinephrine reuptake inhibitors (SNRIs) carry an increased risk of hypertension and tachycardia due to noradrenergic stimulation and should be used with caution in older adults.

Table 3.5 Mood stabilizers

Medication	Dose adjustment needed		Anti-cholinergic side effects	Risk of drug-drug interaction	Risk of falls	Effects in geriatric patients
Mood stabilizers						
Lithium	Yes. Relative contraindication with renal impairment.	Not defined	*Low*	*Low.* Not metabolized by CPY450. High risk of toxic levels with NSAIDs, diuretics, ACE inhibitors, and other nephrotoxic drugs	Low	↑ hypothyroidism, diabetes insipidus, tremor, benign leukocytosis. Laboratory: BMP, complete blood count (CBC), GFR, thyroid stimulating hormone (TSH), free T4, lithium level, ECG
Valproic acid	No	Yes[a] ↓dose	*Low*	*Mod.* Substrate of UGT1A4. Weak inhibitor of 2C9 and UGT2B7.	Low	Tremor, ataxia, nausea, vomiting, weight gain, thrombocytopenia, pancreatitis, hyponatremia, hyperammonemia Laboratory: CBC, LAEs, valproic acid level, check ammonia if altered mental status occurs
Lamotrigine[b]	Yes	Yes ↓dose	Low	UGT1A4 substrate (increased half-life) when combined with valproic acid)	Low	↑ risk Stevens-Johnson Syndrome

BMP basic metabolic panel, *GFR* glomerular filtration rate, LAEs liver-associated enzymes
[a]Contraindicated in severe liver disease; avoid in pancreatitis, hyperammonemia, thrombocytopenia
[b]UGT1A4 Glucuronosyltransferase

Venlafaxine is often avoided in older adults as it was found to be less well tolerated and less safe; but not more effective than sertraline in a randomized controlled trial with frail geriatric patients [26]. Duloxetine is a commonly prescribed drug in geriatric patients as it has FDA indications for neuropathic pain and fibromyalgia, in addition to depression and anxiety. It has low inhibitory activity on cytochrome P450 enzymes but its levels may be increased when combined with CYP1A2 and 2D6 inhibitors. A small study showed that at a low dose, duloxetine had comparable safety profiles between older and younger adults despite being eliminated at a slower rate [27].

Even though it is generally well tolerated, bupropion, which is a norepinephrine and dopamine reuptake inhibitor (NDRI), may cause or worsen psychotic symptoms due to its dopaminergic activity. Bupropion carries increased risks of seizures and falls [28, 29] and should be used with caution in older adults, a population with a high prevalence of seizures.

The Beers criteria recommends avoiding tricyclic antidepressants that have a high anticholinergic burden as well as known cardiotoxic properties [24]. Oral formulations of monoamine oxidase inhibitors should be avoided due to the risk of hypertensive crises in combination with tyramine-rich foods. Selegiline transdermal patch in small doses does not require a restricted diet as the drug bypasses the liver, but more research is needed to determine its safety in the geriatric population with major depressive disorder [30].

3.5.2 Antipsychotics

The decreased density of dopamine receptors in older adults may contribute to greater sensitivity to extrapyramidal side effects (EPS) of antipsychotic treatment. This negatively affects patients who have been treated chronically with antipsychotics, and patients who develop a co-morbid movement disorder such as Parkinson disease, or

mild or major neurocognitive disorder (MNCD) with Lewy bodies.

Patients on long-term antipsychotic medications who develop these side effects may need a dose reduction to decrease or eliminate EPS. Dose reduction may often be preferable to treatment of these symptoms with anticholinergic agents such as benztropine, due to risk of delirium. Gradual dose reduction of the antipsychotic should be tried in order to avoid rebound psychosis, maintain the safety and functional stability of the patient, while addressing movement dysfunction.

Atypical antipsychotics (second generation antipsychotics; SGA) have been linked with an increased risk of falls as a result of orthostatic hypotension due to adrenergic receptor antagonism. One study showed a modest increase in the 90-day risk of falls and fractures in geriatric patients with a new prescription of an atypical antipsychotic (specifically quetiapine, olanzapine, and risperidone) [31]. Another recent study re-visited the question but failed to find significant results, likely due to a different approach in statistical analysis [32]. To prevent side-effect-related falls in the geriatric patient, include slow titration of medications, in small increments, and monitor orthostatic vital signs.

A black box warning in the US has been mandated by the FDA for all antipsychotics, highlighting the risk of cerebrovascular events, and mortality for treatment of acute agitation in patients with major neurocognitive disorders (Chap. 6: Major Neurocognitive Disorder with Behavioral Disturbance). It is unclear if this risk exists for geriatric patients who have been taking antipsychotics on a long-term basis (e.g., in those with schizophrenia). Metabolic risks are more relevant in patients taking antipsychotics chronically.

The Beers criteria recommends avoiding the use of typical and atypical antipsychotics in the geriatric population except for the treatment of schizophrenia, bipolar disorder, or short-term use as an antiemetic during chemotherapy [24] (Chap. 12: Delirium; Chap. 5: Legal Aspects for Informed Consent Issues).

Medication absorption is reduced in aging adults, especially in those with poor nutritional status, and alternative formulations of antipsychotic drugs may need to be considered (Table 3.4). Sublingual formulations improve administration. There is evidence of long-term stability of individuals with schizophrenia with long-acting injectable formulations of antipsychotics [33], though specific pharmacokinetic profiles in the geriatric population have not been studied. In emergencies, intramuscular formulations of antipsychotics may need to be administered, though lower doses are recommended in the geriatric patient (Chap. 13: Involuntary Treatment).

3.5.3 Mood Stabilizers

The hepatic and renal functioning of an aging patient on mood stabilizers deserves attention. Given that GFR is decreased in older adults, treatment with lithium should start at a lower dose, titrated at a slower rate, while monitoring lithium levels (e.g., with every dose change, and monthly thereafter) to prevent toxicity. Patients should avoid nephrotoxic drugs, for example, non-steroidal anti-inflammatory drugs, ACE inhibitors, diuretics, and angiotensin-receptor blockers. If these medications are unavoidable, consider stabilizing the patient on valproic acid or an atypical antipsychotic.

Case studies have reported valproic acid-induced extra-pyramidal symptoms and cognitive impairment that is reversible with discontinuation of the drug [34–36], though the mechanism has not been characterized. Valproic acid is also linked to hyperammonemia and hyperkalemia [37–39], potentially causing delirium. Monitoring of serum chemistries, liver function, complete blood counts, ammonia, and valproic acid levels is warranted. As previously discussed in the pharmacodynamics section, levels of *free* valproic acid should be obtained in those with low albumin levels to avoid being misled by abnormally low protein-bound valproic acid measures [11].

Carbamazepine is an autoinducer of CYP3A4 and can increase or decrease the half-lives of many drugs through drug-drug interactions. Carbamazepine should be avoided in the geriatric patient. If a patient with mania has both hepatic

and renal impairment that precludes treatment with lithium or valproic acid, low doses of an atypical (second-generation) antipsychotic should be considered.

3.5.4 Benzodiazepines

Benzodiazepines carry a significant risk of harm for the geriatric patient. Side effects of benzodiazepines include cognitive impairment, falls, and increased mortality. The Beers criteria recommends avoidance of benzodiazepines in the geriatric patient, reserving the use of some long-acting benzodiazepines (e.g., clonazepam, diazepam, chlordiazepoxide), only for brief periods, in the treatment of seizure disorders, alcohol withdrawal, benzodiazepine withdrawal, or pre-procedural anesthesia [24].

In the inpatient geriatric setting, benzodiazepine use should be short-term, at the lowest possible dose, and in acute situations, wherein the patient is not responding to high or maximum doses of antipsychotics or mood stabilizers. In patients with a long-term history of benzodiazepine use or benzodiazepine dependence, taper the medications over a course of time that corresponds with the period of dependence (Chap. 10: Alcohol and Substance Use Disorders in the Geriatric Psychiatry Inpatient).

3.6 Problematic Situations: Polypharmacy, Non-compliance, and the Prescribing Cascade

Polypharmacy is defined as the use of multiple medications from different medication classes for multiple indications. The problem is significant: over half of Medicare patients in the US take five or more medications [40]. Geriatric patients are more likely to be prescribed a greater number of medications due to multiple co-morbid and chronic medical disorders, as well as multiple prescribers. Medication adherence is poor or intermittent, risking medication toxicity. Causes of poor adherence include difficulty in tracking medication refills, doses, scheduling of multiple drugs, and cognitive deficits.

Prescribing cascade: Common elements of problematic, though common, *prescribing cascade* include [41]:

- An increased medication dosage to target symptoms, rather than assessing medication adherence, or absorption issues.
- A new medication for a new symptom that may actually be due to a side effect of another medication.
- A benzodiazepine for insomnia, causing cognitive impairment, which is then treated with a cholinesterase inhibitor, such as donepezil.
- An anticholinergic agent, which may cause overflow incontinence from urinary retention, as well as worsen cognitive function.

Collaboration with a pharmacist can improve the efficiency, accuracy, and safety of the medication reconciliation process. A study of an inpatient geriatric unit, newly staffed with a geriatric pharmacologist, identified 20% of patients had medical conditions with no medications prescribed, 15% of patients had actual and potential adverse drug reactions, 8% of patients had medications prescribed without any indications, and 5% had medications therapeutic duplication of medications [42]. Consultation with a colleague helps ensure that psychiatric and medical target symptoms are addressed with as few medications as possible.

Recommended practices: (1) Determine a specific indication (or target symptom) prior to starting a new medication. The target symptoms should ideally be measurable objectively such as amount of food consumed or hours slept. (2) Evaluate if the condition or target symptom can be treated by optimizing a medication already prescribed. (3) Determine if a symptom is due to a medication side effect. Reduce unnecessary medication whenever possible. (4) Prescribe a medication that can address more than one symptom while minimizing the risk of polypharmacy (Chap. 17: Medication Strategies for Switching, Tapering and Cross-Tapering Medications).

3.6.1 Laboratory and Imaging Work-Up

If possible, obtain a basic medical work-up prior to or at the time of admission to an inpatient unit. Physical examination and laboratory testing include a basic metabolic panel, liver function tests, complete blood count, thyroid stimulating hormone including free Thyroxin (T4) levels, urinary analysis, urinary toxicology, and an electrocardiogram. A computed tomography (CT) scan of the head is indicated for unexplained, new neurological findings, or altered mental status (Chap. 1: Essential Medical Work-up to Rule Out Medical Conditions).

Medications that need blood level monitoring require steady-state levels at initiation, change in dose, and addition or removal of another drug. Patients on medications that affect the liver, kidney, or thyroid functions should have these parameters monitored as well. Use cognitive screening assessments (MoCA or CAM) at admission, regularly if hospitalization is longer than 7 days, and at the time of discharge to determine if cognitive issues are chronic, new, and/or whether further work-up is warranted (Chap. 6: MNCD and Chap. 12: Delirium).

Optimum care of geriatric psychiatric patients may require more laboratory studies and extra vigilance to prevent medication-induced adverse events. In one study, geriatric patients who were staying on an inpatient geriatric psychiatry unit received complete medical work-ups, structured cognitive assessments, aging-sensitive aftercare, monitoring of psychopharmacological side effects, and blood levels of medications at a significantly higher rate compared to comparable patients on a general psychiatry unit [43].

3.7 Summary

The contribution of each of many variables to a medication-related problem is difficult to parse in the outpatient setting, prompting an inpatient unit admission. Medication reconciliation, as well as vigilance for potential side effects and drug-drug interactions, can facilitate the goal of decreasing polypharmacy. Pharmacokinetic and pharmacodynamic changes make the geriatric patient especially vulnerable to adverse drug reactions, and complicate the psychiatric/medical symptom presentation. The decrease in VOD, metabolism, renal excretion, and receptor densities can also impact the potential efficacy of medications. Principles of parsimonious prescribing, coupled with close monitoring in a controlled inpatient setting, can help identify adverse medication effects, as well as the effects of accompanying medical co-morbidities. At best, the inpatient setting can lead to a discharge plan with an improved medication regimen that will foster rational prescribing practices in the outpatient setting.

Take-Away

- Seek to reduce the number of medications.
- Search for one medication with two desired effects, rather than two medications.
- Avoid treatment of adverse effects by adding another medication; consider reduction of dose first.
- If the regimen includes multiple medications, consider the effect of medications on cognition and on psychiatric symptoms.
- Avoid starting a new medication when the patient is taking multiple different medications.
- Titration of medications should be slow, increasing or decreasing doses gradually, in tiny increments.
- Consider the patient's frailty, weight, intake, and hydration.
- Err on the side of under-dosing.
- Perform medication reconciliation in collaboration with the patient, family, caregiver, outpatient clinician(s), and pharmacist.
- Antidepressants, antipsychotics, and benzodiazepines increase the risk of falls.
- Benzodiazepines are not recommended in geriatric patients.

3.8 Prescribing Principle: CARES

C A R E S

Check
- baseline laboratory tests: chemistry, liver function, complete blood count, thyroid function test, electrocardiogram
- medication regimen and adherence
- cognitive function: baseline and ongoing

Avoid
- drug-drug interactions
- benzodiazepines and anti-cholinergic drugs
- polypharmacy

Rule-out
- worsening of medical conditions and medication side effects as cause of symptoms

Eliminate
- unnecessary medications

Start Low and Go **S**low
- Start medications at a lower dose and titrate slowly

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4 Interdisciplinary Roles and Interface

Debra Bakerjian

4.1 Introduction

Several intertwined acute and chronic problems complicate the delivery of comprehensive psychiatric inpatient treatment: medical conditions with complex medication regimens, cognitive deficits, psychosocial needs, and psychiatric symptoms/behavioral problems. Coordination between disciplines is crucial. The US Health Resources and Services Administration (HRSA) is a resource for understanding the types of clinicians in the mental health workforce in the United States. Table 4.1 lists these disciplines, roles, education, licensure requirements, and scope of practice. In the United States, states control the scope of practice; there is wide variation across states, which may differ in other nations. Figure 4.1 summarizes a recommended interdisciplinary practice during geriatric inpatient psychiatry hospitalization.

4.2 Vignette

A 72-year-old woman with a 20-year history of depression was referred to inpatient psychiatry from the nursing home (NH) for major neurocognitive disorder (MNCD) with behavioral disturbance. The patient was taken to the emergency department for agitation, constant pacing, insomnia, delusions, verbal aggression, and combativeness. Medical history included hypertension, type 2 diabetes mellitus, COPD, chronic kidney disease, and obesity. The NH report indicated that the patient had been eating sweets from other resident's trays, refusing insulin (Lantus 18 units daily), and refusing blood glucose draws. The patient said, "…they are trying to cut me up with a knife."

In the ED, she was irritable and agitated, said that staff stole her money ($25,000) and were trying to stab her to death. She refused to cooperate with a physical exam and threatened everyone, stating she was going to call the police if anyone touched her.

Medication history included antidepressants for at least 10 years, most recently Sertraline 100 mg daily for 3 years.

Upon admission to inpatient psychiatry service, members of the multidisciplinary team (psychologist, psychiatric mental health nurse practitioner, registered nurse, behavioral health specialist, psychiatrist and psychiatry fellow) reviewed the medical record and began treatment planning. Delirium was ruled out and the working diagnosis remained major neurocognitive disorder (MNCD) with behavioral disturbance.

Distribution of work was assigned: the acute psychiatric issues would be managed medically by the psychiatric fellow in consultation with the

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Table 4.1 Licensure requirements and scope of practice in the United States, by mental health provider type

Provider type	Degree	Supervised practice	Exam	Scope of practice
Requires doctoral level degree				
Psychiatrist	Medical Doctorate (MD) or Doctorate of Osteopathic Medicine (DO), both of which typically require 4 years to complete (including 2 years of clinical rotations). Coursework emphasizes physical medicine.	Generally requires 3 or 4 years of post-degree supervised clinical training (residency) in the specialty of psychiatry.	Generally requires a passing score on the United States Medical Licensing Examination (USMLE) for MDs or DOs. DOs can also elect to take the Comprehensive Osteopathic Medical Licensing Examination (COMLEX). *To become board certified,* an exam is administered by the American Board of Psychiatry and Neurology.	Diagnose psychiatric disorders. Provide psychosocial treatment for individuals, families, and groups. Can prescribe medication. Can diagnose and treat physical conditions; neuroimaging; perform procedures such as Transcranial magnetic stimulation (TMS) and Electroconvulsive Therapy (ECT)
Clinical Psychologist	Doctoral degree in psychology or a related field, which generally takes between 5 and 7 years to complete and requires academic coursework, clinical training, a dissertation, and an exam.	Generally requires 3000 hours of supervised clinical training, which takes approximately 2 years.	Generally requires a passing score on the Examination for Professional Practice in Psychology (EPPP).	Diagnose psychiatric disorders. Provide psychosocial treatment for individuals, families, and groups. Administer and interpret psychological tests. Generally cannot prescribe medication.
Requires Doctoral Level or Master's Degree				
Marriage and Family Therapist (MFT)	Master's degree (2–3 years), doctoral degree (3–5 years), or postgraduate clinical training (3–4 years) in marriage and family therapy or a related field. Coursework emphasizes the individual's mental health in the context of interpersonal relationships (e.g., family and peers).	Generally requires 2 years of post-degree supervised clinical training.	Generally requires a passing score on the Association of Marital and Family Therapy Regulatory Board's Examination in Marriage and Family or the equivalent California Exam.	Diagnose psychiatric disorders. Provide psychosocial treatment for individuals, families, and groups. Cannot prescribe medication.
Psychiatric Mental Health Nurse Practitioner (PMH-APRN)	Doctoral degree (DNP-practice focus or PhD-research focus) requiring 3–4 years of post-BSN training; DNP requires 1000 or more hours of supervised clinical practice OR Master of Science in nursing (MSN), generally requires 2 years of coursework and 500 or more clinical hours. Coursework and supervised clinical practice.	DNPs generally require 500 or more hours for Master's prepared nurses or 1000 or more hours for Bachelor's prepared nurses.	Requires a passing score on the national certification exam conducted by the American Nurses Credentialing Center for the APRN role; must be renewed every 5 years.	Diagnose psychiatric disorders. Provide psychosocial treatment for individuals, families, and groups. Can prescribe medication (dependent upon the scope of practice allowed in the state).
Requires Master's Degree				
Clinical Social Worker	Master of Social Work (MSW), which typically requires 2 years. Coursework emphasizes human and community well-being. Requires a supervised field practicum (internship).	Generally requires 3200–3400 post-degree supervised clinical hours, which take approximately 2 years.	Generally requires a passing score on the Clinical Exam of the Association of Social Work Boards.	Diagnose mental disorders. Provide psychosocial treatment for individuals, families, and groups. Cannot prescribe medication.

Table 4.1 (continued)

Provider type	Degree	Supervised practice	Exam	Scope of practice
Requires Bachelor's Degree or Associate Degree				
Psychiatric Mental Health Nurse (PMH-NP)	Requires preparation as a registered nurse that can be a 2-year program (Associate Degree Nurse) or 4-year program (Bachelor of Science Degree Nurse); plus 2 years of general nursing practice is required to specialize in psychiatric-mental health nursing.	2000 hours minimum and 30 hours of continuing education in psychiatric-mental health nursing within 3 years to sit for Board Certification.	Requires a passing score on the national certification exam conducted by the American Nurses Credentialing Center for Psychiatric-Mental Health Nursing; must be renewed every 5 years.	Specializes in providing nursing care for patients with psychiatric, mental health, and behavioral health problems. Assesses mental health needs of individuals, families, groups, and communities. Develops nursing diagnoses and care plans. Collaborates with other members of the team.

Adapted from The Mental Health Workforce: A Primer [1]
Sources: U.S. Department of Labor, Bureau of Labor Statistics; U.S. Department of Health and Human Services, Health Resources and Services Administration (HRSA); and various professional associations
Notes: The degree, supervised practice, and exam indicated in the table are those generally required to obtain a license for independent practice. Licensure requirements (defined by state boards) and scope of practice (defined by state laws) vary by state. Degree requirements may vary by program. In all cases, the information provided in the table reflects what is generally true in most U.S. states and programs. Elaborating the exceptions is beyond the scope of this report

attending psychiatrist; medical co-morbidities to be addressed by the nurse practitioner in consultation with the hospitalist team; behavioral health specialist and registered nurse to focus on admission and orientation to the unit environment. A dietician was engaged to help develop snacks that were more diabetic-compatible, with some snacks to be used as incentives to improve compliance. Psychologist and nursing staff proposed a non-pharmacological plan to address delusions (Chap. 18: Psychotherapies and Non-pharmacological Interventions).

Over the first 5 days of admission, sertraline was tapered and discontinued. Quetiapine 25–50 mg PRN twice daily and at bedtime was started to treat agitation, insomnia, and mood lability. In consultation with the hospitalist, the dietician encouraged small portions of blueberries and sliced apple as an incentive for blood draws. With gentle coaxing and rewards, within 3 days the patient began to cooperate with lab work ordered to rule out Cushing syndrome, hyperthyroidism, vitamin B12 deficiency, and to determine the status of diabetes. Oral medication was started for diabetes management; blood sugar management improved. Cognitive behavioral therapy attempted to provide an understanding of her diagnosis and need for treatment, but this was not successful. After 10 days of hospitalization, social worker and discharge planners (Chap. 21: Placement) met with NH staff to facilitate a warm hand-off.

4.3 The Mental Health Inpatient Workforce

According to Heisler and Bagalman (2015), no consensus has emerged as to which providers are essential to an inpatient mental healthcare team [1]. This may vary by each geriatric patient's specific needs. But geriatric inpatients with mental health issues often have several acute and chronic health conditions, prompting a range of specialty consultants, from speech-language pathologists (SLP), to neurologists, to physical therapists. These professionals may enter and exit the care team at various times within the hospitalization. The prevalence of medical co-morbidities in geriatric patients, in addition to acute psychiatric conditions, may require an even greater degree of case management and collaborative care [2] (Chap. 7: Acute Medical Events; Chap. 19: Medical Nursing Care and Communication Barriers). Such dynamics underscore the need for excellent communication.

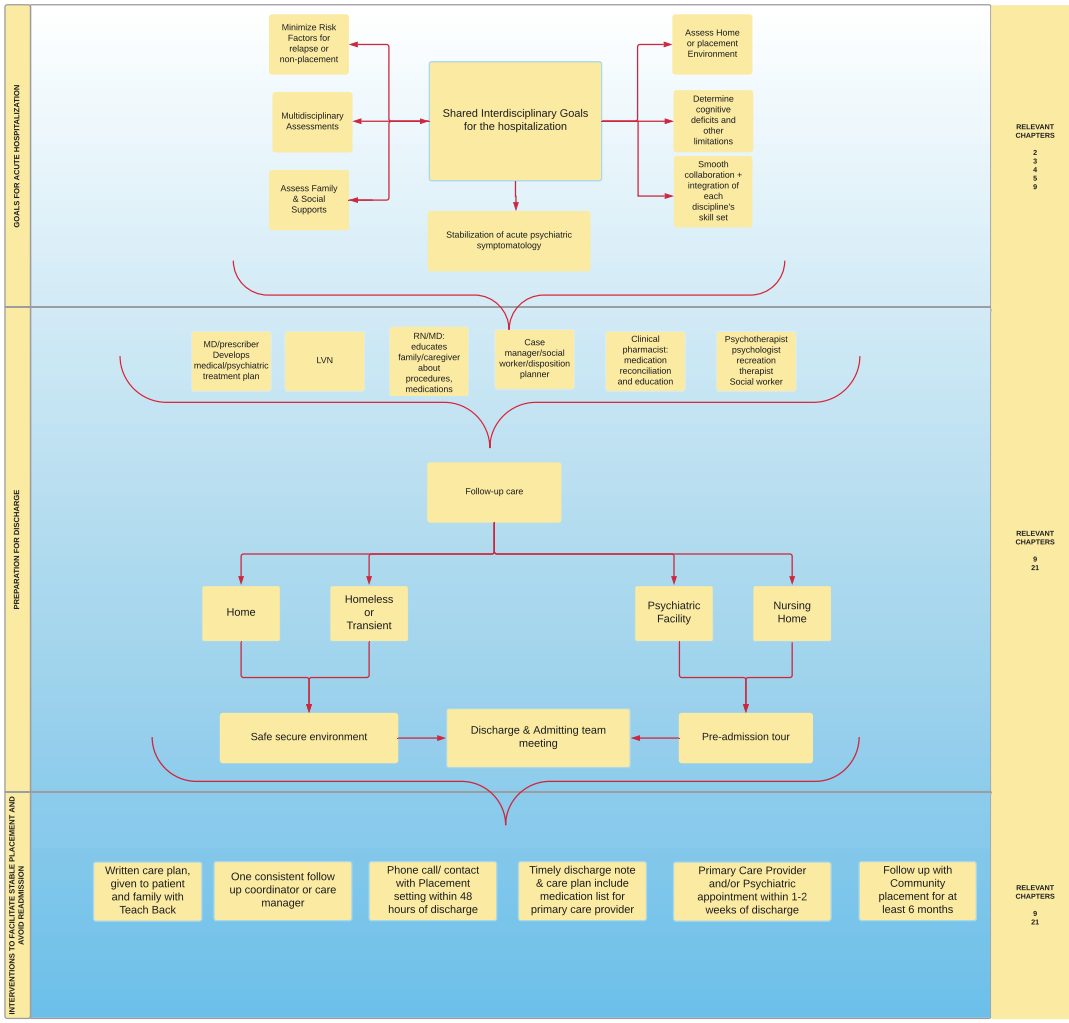

Fig. 4.1 Recommended interdisciplinary practice during geriatric inpatient psychiatry hospitalization

With fewer psychiatric hospitals and institutions, many patients with acute onset or exacerbations of psychiatric symptomatology remain in the emergency department (ED) until an acute inpatient bed is available [3]. The patient in the vignette was fortunate to be admitted within hours, which minimized the need for short-term providers of care.

As noted, several disciplines often participate in the inpatient care of a geriatric patient needing acute psychiatric care. The World Health Organization (WHO) provides a comprehensive summary and definitions of the mental healthcare workforce in various countries, in their Assessment Instrument for Mental Health Systems (WHO-AIMS). Additional professions, not listed in Table 4.1, include the following.

Primary health care worker Provides basic health services and links with other aspects of the healthcare system. This role may be filled by medical assistants, aide-level workers, multipurpose health workers, health assistants, and community health workers, among others. The training and functions vary across countries. Physicians, nurses, and other health professionals may supervise their work.

Nurse Has completed a formal training in nursing at a recognized, university-level school for a diploma or degree in nursing. Both registered nurses and licensed vocational nurses may work as part of the team.

Occupational therapist Has completed a formal training in occupational therapy at a recognized, university-level school for a diploma or degree in occupational therapy.

Primary healthcare physician A general practitioner, family physician, or other non-specialized medical doctor consulting to, or is based within, an inpatient psychiatric unit.

Primary healthcare nurse An RN working in the inpatient psychiatric unit.

Psychiatric clinical pharmacists Assist in pharmacological management in patients on multiple medications. Some clinical pharmacists have completed extra postgraduate training in psychopharmacology and are Board Certified Psychiatric Pharmacists (BCPP). A special expertise may be the ability to communicate information about medications in the inpatient geriatric population with psychiatric illnesses, which can enhance the informed consent process.

Case managers Coordinate transition to the community and arrange professional services for individuals with psychiatric conditions; they also monitor patient compliance and symptomatology, reporting information to the provider as well as providing support to families.

Mental health specialists Entry-level mental health professionals without a graduate degree who usually work under the direct supervision of a licensed professional providing crisis intervention, assisting with housing and employment, and arranging for placement and other support services.

Mental health recovery specialists Provide crisis care and support, assist in developing treatment plans for patients, conduct group therapy sessions, and other services. They differ from mental health specialists (described above) in that they have a minimum of a baccalaureate degree in social work, psychology, sociology, or behavioral science and can provide a higher level of care.

Psychiatric nursing assistants/attendants Often have high-school diplomas and have received nursing assistance training or on the job training to assist nursing staff.

Specialty therapists (activities, art, music, recreation) Specialists in recreational and similar activities who lead therapeutic activities and engage patients. On inpatient psychiatry, this role can support many other goals, such as treatment compliance, reduction of agitation, and improvement of nighttime sleep (Chap. 18: Psychotherapies and Non-pharmacological Interventions). An understanding of the specific patient's limitations and symptomatology is inherent in the success of this role.

Mental health teams vary in terms of structure, depending on the type of organization and the patient population. An effective team member understands other members' responsibilities, and facilitates effective communication and collaboration among staff, patients, and families.

4.4 Models of Collaborative Care

Psychiatric care benefits from a model of inpatient care delivery as well as an understanding as to how medical care will be delivered to the patient upon discharge. Conceptual models can translate research into the inpatient environment and provide a roadmap for the context of care, the specific resources available, and the needs of the patient and family.

4.4.1 Collaborative Care Models (CCMs)

An important goal of a geriatric inpatient hospitalization is to stabilize the patient so that

follow-up collaborative care models of care delivery (CCM) can take over upon discharge. Wagner's Chronic Disease Management Model has served as a foundation for most of the CCMs [4]. The model depicts the link between the community resources and the health system along with an informed activated patient and a well-prepared, knowledgeable, and proactive healthcare team. Adaptations of the chronic care model have improved the focus on population health [5] and incorporation of patient-centeredness, timely and efficient care, evidence-based/safe care, and care coordination.

Woltmann et al. (2012) [5] conducted a systematic review and meta-analysis comparing the effectiveness of collaborative chronic care models in mental health and found that the model improves both mental and physical outcomes across a variety of different care settings [6]. Use of CCM applies to the inpatient setting through the following:

1. Patient self-management with enhanced coaching and skill building by encouraging patients to participate in specific education about their illness, problem solving, and shared decision-making with the team;
2. Clinical systems (registries, reminders, decision support) to empower less expert clinicians by providing information about specific conditions;
3. Redefining roles for the various team members to share responsibility through team-based delivery redesign;
4. Experts to support less-experienced clinicians in decision-making, with telepsychiatry or Skype meetings;
5. Coordinating and linking community resources to patients and staff;
6. Providing organizational support for clinicians to receive appropriate levels of training.

The CCM model has been particularly effective for depression, bipolar, anxiety disorder outcomes, and quality of life [6].

4.4.2 Enhanced Primary Care

The enhanced primary care (EPC) model was developed in Great Britain in an attempt to better manage patients with severe and enduring psychiatric illnesses (SMI) [5]. The goal of EPC is to improve recovery and enhance safe discharge to the community. EPC teams consist of general practitioners, consultant psychiatrists, psychiatric nurses, psychologists, and social workers who work as a team to provide care. Liaison between outpatient EPC teams and an inpatient unit staff may provide the safety net to minimize re-admissions. Essential components of EPC are:

- Regular visits to the primary care provider (PCP)
- Enhanced support to PCP from psychiatrists and other mental health professionals
- Additional training and education of PCPs (including nurse practitioners) in how to manage SMI including psychopharmacology and therapeutic depot administration
- Integrating mental health teams (psychiatric registered nurses and behavioral health specialists) into primary care.

This model has been found to improve clinical outcomes, reduce hospital readmissions, and improve satisfaction of patients and clinicians [5] in Great Britain and has been replicated in a number of healthcare systems in the United States [7].

4.4.3 Stepped-Approach Models of Care

Another safety net to minimize re-admission to inpatient units involves a stepped approach to care, based on the concept that evidence-based, low-intensity treatments are the initial interventions and, if not effective, high-intensity treatments can be offered. Low intensity care may not involve healthcare professionals, rather it may involve self-help, including computer education

programs with minimal interaction with trained mental health personnel. In this model, patients have regularly scheduled reviews and are "stepped up" to the next level of treatment if they are not improving.

In the United Kingdom, the National Institute for Health and Care Excellence (NICE) Clinical Guidelines for mental health diseases such as depression and anxiety recommend this stepped approach to care and have had overall positive outcomes in improving access to mental healthcare. (Royal College of Psychiatrists – https://www.rcpsych.ac.uk/members/nccmh/niceclinicalguidelines.aspx). There are now several published studies of this model of care indicating varying levels of success that indicate that further studies are needed [8].

Ness and colleagues (2014), building upon the D'Amour and Oandasan model of Interprofessional Education for Collaborative Patient-Centered Practice (IECPEP), have proposed a model of collaborative practice for community-based mental healthcare [9]. They conceptualized collaborative practice in mental health as an approach to improve the effectiveness of mental health services to patients in community settings by involving collaboration among the professional providers, patients, and families. The model is comprised of the following four components: (1) the framework for service orientation, (2) two interconnected collaborative structures, (3) principles of collaboration, and (4) the processes of collaborative practice (see Fig. 4.2).

Collaborative practice integrates person-centered practice and recovery orientation to enhance mental healthcare in the community environment.

The *first* component of the framework embraces both person-centered practice and recovery-orientation perspectives together to

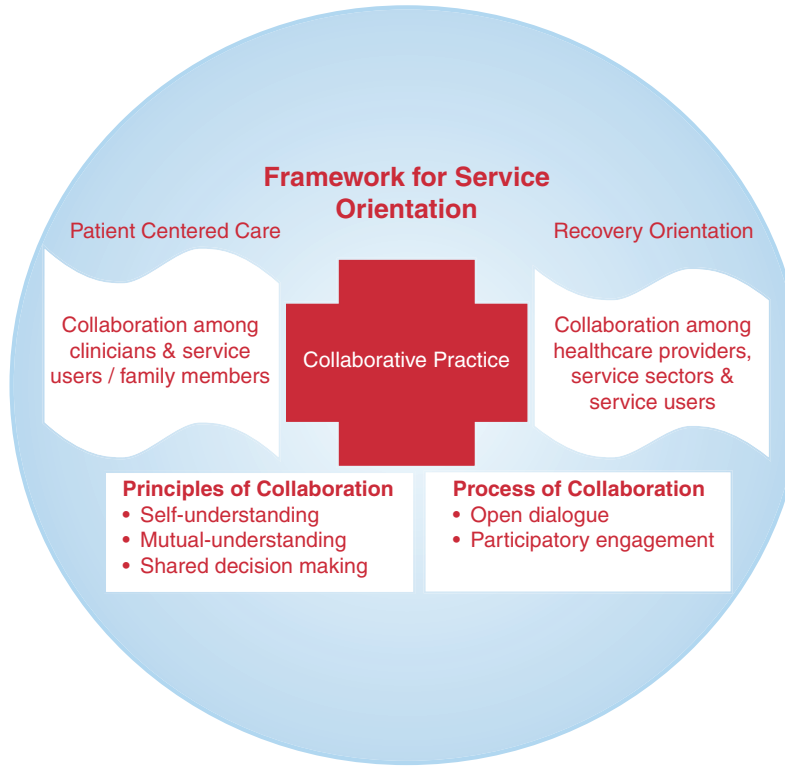

Fig. 4.2 Collaborative practice in community settings

inform the patient and family, placing them in the center of service delivery. Patients and families are equal partners in the planning, developing, and assessing mental healthcare to ensure it aligns with their goals. Person-centeredness, then, drives patients toward self-discovery and transformation and incorporates them into the decision-making processes related to mental healthcare. Recovery-orientation is conceptualized as an individual process but also as a social process that is affected by social conditions such as relationships, life conditions, services, and systems of care.

The *second* component of the framework is conceptualized as collaboration between two structures: the inpatient mental health team providing in-hospital service, and the mental healthcare system in the community. The model is easily adapted to any healthcare team and inpatient psychiatric settings. Coordination between both systems is essential to improve clinical outcomes and enhance the experience of care as well as to provide efficient services across patients, functions, activities, and settings.

The *third* component of collaboration includes three specific principles:

1. Self-understanding
2. Mutual understanding
3. Shared decision-making

Self-understanding requires each person to know her/his own perspectives, knowledge-base, motivations, and biases. Mutual understanding is about the relationship and the effort to communicate with the goal of understanding differences of opinion and focus on achieving truth. Shared decision-making is essential, with shared goals and accepting accountability.

The *fourth* component involves actual collaborative processes. It incorporates the practice of working together to meet the needs of patients and families, with partnering, a team approach, mutual trust, and respect. The authors describe two key processes: open dialogue and participatory engagement. Open dialogue requires team members to value uncertainty so that all are free to bring forth differing opinions, choices, interpretations, and courses of action. Participatory engagement is the willingness to share the group's work without constraints or prejudices, and with appreciation for each team member's strengths.

Inpatient psychiatric units can use this model effectively, as they discharge geriatric patients with residual or chronic psychiatric symptoms to the community. A patient-centered approach incorporates patients and families into the care planning and decision-making processes, and uses different clinicians to provide coordinated care.

Each of these models has theoretical significance and merit; outcomes are likely dependent upon the context of care and who is involved in the team. The principles of a multidisciplinary team with varying levels of knowledge and skill is important in the face of shortages of available resources. The models are based on the concept that a variety of different professions working in concert can provide more comprehensive, person-centered care, and achieve better outcomes.

4.5 Core Competencies for Collaborative Practice

Historically, there has been little attention paid to educating and training different professions and levels of healthcare workers to work as a team. But in 2009, several national health professions accrediting associations representing nursing, medicine, dentistry, osteopathic medicine, pharmacy, and public health formed a collaborative group that would promote and encourage their constituents to advance knowledge and skills in team-based care of patients and to improve population health outcomes. This group, the Inter-Professional Education Collaborative (IPEC), produced *Core Competencies for Interprofessional Collaborative Practice* published in 2011 [10]. It detailed four competencies necessary for successful teams and encouraged health profession schools to educate faculty to develop curriculum based on the competencies.

In 2016, several other health professional organizations joined the group, bringing the number up to 15. More than 60 other profes-

sions have participated in the process including Behavioral and Community Health, Occupational Therapy, Psychology, Rehabilitation Services, and Social Work. During the 2016 session, the core competency document was updated so that competencies would be organized under "Interprofessional Collaboration" with four core subdomain areas [11].

A complete list of core values and core competencies are listed in Tables 4.2 and 4.3. The goals are inherent: better health, better patient experience, and lower cost. The four core competencies are (1) values and ethics, (2) roles and responsibilities, (3) inter-professional communication, and (4) teams and teamwork.

- *Values and ethics*: Shared values and goals, and ways of working respectfully with each other. Issues related to confidentiality, ethical decision-making, equity inclusion, and cultural competence are important topics for the team to agree upon. Team members commit to maintaining high standards of patient-centered, culturally relevant care, acting with honest and integrity at all times, and to maintain their own professional competencies.
- *Roles and responsibilities*: Understanding one's own role and those of the team. Team members must communicate one's own roles and responsibilities within the team and acknowledge one's own limitations in knowledge and skills. It is equally important to fully understand other team members' capabilities, roles, and responsibilities as well and communicate those to patients, families, and other healthcare professionals. Knowledge of members' roles allows the team to use unique and complementary skills to optimize team care and facilitate positive patient outcomes.
- *Inter-professional communication*: Improvement of communication within the team and with patients, families, and other community members. Many patient healthcare-related safety errors result from poor communication skills. Communication must be clear, respectful, informative, timely, and without jargon. Active listening allows other opinions, thoughts, and concerns to be heard (Chap. 19: Medical Nursing Care and Communication Barriers).
- *Teams and teamwork*: Understanding principles of "team" and "teamwork," acknowledging all members of the team as important, and recognizing that leading within the team is context-specific. The team leader should be dependent upon the knowledge, skills, and abilities of the individual and not solely on the professional role. Each discipline at times may lead the team to develop aspects of the care plan. Teams must develop ways to manage disagreements in a professional, respectful, and constructive way. Effective teams share accountability for errors versus placing blame on individuals; think "team" first instead of "me" first.

Team leadership may help improve skills that incorporate various competencies by asking:

1. How does this discussion reflect any one of the competencies?
2. Are there any barriers to achieving this competence in the team?
3. How can we improve on this competence?

Team meetings ideally are open, transparent, and not threatening; a natural hierarchy exists in teams and may preclude some team members from participating. A mental health aide may feel inadequate to comment on a patient situation in the presence of the psychiatrist, even though he or she may be the team member who sees the patient most often. It is important that the team leader specifically asks for their input and reinforces their value as a team member.

A team culture that embraces *psychological safety* is optimal. This concept was originally explored by William Kahn (and further advanced by Amy Edmondson, a professor at the Harvard School of Business) [12]. Psychological safety means that all members of the team believe that it is safe to take interpersonal risks (such as suggesting a change in care) without fear of ridicule

or negative consequences. Edmondson advocates that high-performing teams need psychological safety to be able to admit mistakes, express gaps in knowledge, share concerns, and verbalize beliefs; this is critical in environments that are high risk and complex such as exists in healthcare. At the same time, individuals and teams must also be accountable for their actions. Teams that are both accountable and ensure psychological safety provide a learning culture in which teams can innovate and improve their team-based processes.

Figure 4.3 provides a summary of the Psychological Safety Framework [12]. The four quadrants range from low to high (left to right and bottom to top). On the horizontal axis, there is the pressure to be accountable and on the vertical axis is the degree that individuals feel psychologically safe in that environment. Individuals with low psychological safety and low accountability fall into the "apathy zone" during which the team member may simply do what needs to be done to get ahead but with relatively minimal effort. Individuals with low psychological safety but who feel highly accountable fall into the "anxiety zone" and typically feel anxious and stressed about their position on the team. Team members who experience high levels of psychological safety but demonstrate low accountability are in the "comfort zone." These team members are often complacent in their role and do not feel any pressure to do more than what is minimally expected. A highly functioning team balances between high psychological safety and high accountability, where the team members are in the "learning zone." Team members then feel safe to admit they do not know everything and are willing to innovate but also are willing to be responsible and accountable for their actions. Only in this zone can there be a high degree of organizational or team learning.

Edmondson and colleagues advocate for three building blocks essential for a learning organization: (1) supportive learning environments, (2) concrete learning practices and processes, and (3) leadership reinforcement of learning. Highly functioning and successful teams should strive to incorporate these structures and processes.

Tables 4.2, 4.3, 4.4, and 4.5 show core competencies for international collaboration, including ethics, roles and responsibilities, communication, and team work.

Values/ethics sub-competencies Work with individuals of other professions to maintain a climate of mutual respect and shared values. Table 4.2 lists values/ethics for inter-professional practice.

Roles/responsibilities sub-competencies Use the knowledge of one's own role and those of

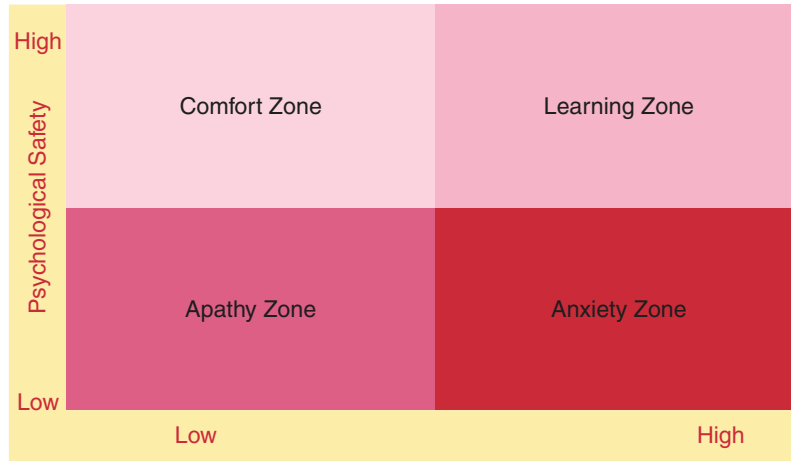

Fig. 4.3 Psychological safety framework [12]

Table 4.2 Values/ethics/sub-competencies to retain mutual respect between different professions

Values ethics/sub-competencies: work with individuals of other professions to maintain a climate of mutual respect and shared values (values/ethics for inter-professional practice)	
VE1	Place interests of patients and populations at center of inter-professional health care delivery and population health programs and policies, with the goal of promoting health and health equity across the life span
VE2	Respect the dignity and privacy of patients while maintaining confidentiality in the delivery of team-based care
VE3	Embrace the cultural diversity and individual differences that characterize patients, populations, and the health team
VE4	Respect the unique cultures, values, roles/responsibilities, and expertise of other health professions and the impact these factors can have on health outcomes
VE5	Work in cooperation with those who receive care, those who provide care, and others who contribute to or support the delivery of prevention and health services and programs
VE6	Develop a trusting relationship with patients, families, and other team members (CIHC, 2010)
VE7	Demonstrate high standards of ethical conduct and quality of care in contributions to team-based care
VE8	Manage ethical dilemmas specific to inter-professional patient-/population-centered care situations
VE9	Act with honesty and integrity in relationships with patients, families, communities, and other team members
VE10	Maintain competence in one's own profession appropriate to scope of practice

Table 4.3 Core competencies for international collaboration: roles and responsibilities

Roles/responsibilities sub-competencies: use the knowledge of one's own role and those of other professions to appropriately assess and address the health care needs of patients and to promote and advance the health of populations	
RR1	Communicate one's roles and responsibilities clearly to patients, families, community members, and other professionals
RR2	Recognize one's limitations in skills, knowledge, and abilities
RR3	Engage diverse professionals who complement one's own professional expertise, as well as associated resources, to develop strategies to meet specific health and healthcare needs of patients and populations
RR4	Explain the roles and responsibilities of other providers and how the team works together to provide care, promote health, and prevent disease
RR5	Use the full scope of knowledge, skills and abilities of professionals from health and other fields to provide care that is safe, timely, efficient, effective, and equitable
RR6	Communicate with team members to clarify each member's responsibility in executing components of a treatment plan or public health intervention
RR7	Forge interdependent relationships with other professions within and outside of the health system to improve care and advance learning
RR8	Engage in continuous professional and inter-professional development to enhance team performance and collaboration
RR9	Use unique and complementary abilities of all members of the team to optimize health and patient care
RR10	Describe how professionals in health and other fields can collaborate and integrate clinical care and public health interventions to optimize population health

Table 4.4 Core competencies for international collaboration: communication

Inter-professional communication sub-competencies: communicate with patients, families, communities, and professionals in health and other fields in a responsive and responsible manner that supports a team approach to the promotion and maintenance of health and the prevention and treatment of disease (interprofessional communication)	
CC1	Choose effective communication tools and techniques, including information systems and communication technologies, to facilitate discussions and interactions that enhance team function
CC2	Communicate information with patients, families, community members, and health team members in a form that is understandable, avoiding discipline-specific terminology when possible
CC3	Express one's knowledge and opinions to team members involved in patient care and population health improvement with confidence, clarity, and respect, working to ensure common understanding of information, treatment, care decisions, and population health programs and policies
CC4	Listen actively, and encourage ideas and opinions of other team members
CC5	Give timely, sensitive, instructive feedback to others about their performance on the team, responding respectfully as a team member to feedback from others
CC6	Use respectful language appropriate for a given difficult situation, crucial conversation, or conflict
CC7	Recognize how one's uniqueness (experience level, expertise, culture, power, and hierarchy within the health team) contributes to effective communication, conflict resolution, and positive inter-professional working relationships (University of Toronto, 2008)
CC8	Communicate the importance of teamwork in patient-centered care and population health programs and policies

Table 4.5 Core competencies for international collaboration: team work

Team and teamwork sub-competencies: apply relationship-building values and the principles of team dynamics to perform effectively in different team roles to plan, deliver, and evaluate patient-/population-centered care and population health programs and policies that are safe, timely, efficient, effective, and equitable (teams and teamwork)	
TT1	Describe the process of team development and the roles and practices of effective teams
TT2	Develop consensus on the ethical principles to guide all aspects of teamwork
TT3	Engage health and other professionals in shared patient-centered and population-focused problem solving
TT4	Integrate the knowledge and experience of health and other professions to inform health and care decisions, while respecting patient and community values and priorities/preferences for care
TT5	Apply leadership practices that support collaborative practice and team effectiveness
TT6	Engage self and others to constructively manage disagreements about values, roles, goals, and actions that arise among health and other professionals and with patients, families, and community members
TT7	Share accountability with other professions, patients, and communities for outcomes relevant to prevention and health care
TT8	Reflect on individual and team performance for individual, as well as team, performance improvement
TT9	Use process improvement to increase effectiveness of inter-professional teamwork and team-based services, programs, and policies
TT10	Use available evidence to inform effective teamwork and team-based practices
TT11	Perform effectively on teams and in different team roles in a variety of settings

other professions to appropriately assess and address the healthcare needs of patients and to promote and advance the health of populations. Table 4.3 lists roles/responsibilities.

Inter-professional communication sub-competencies Communicate with patients, families, communities, and professionals in health and other fields in a responsive and responsible

manner that supports a team approach to the promotion and maintenance of health and the prevention and treatment of disease. Table 4.4 lists core competencies for inter-professional communication.

Team and teamwork sub-competencies Apply relationship-building values and the principles of team dynamics to perform effectively in different team roles to plan, deliver, and evaluate patient/population-centered care and population health programs and policies that are safe, timely, efficient, effective, and equitable. Table 4.5 lists core competencies for team work.

4.6 Summary

The several clinical disciplines who comprise an inpatient psychiatry team collaborate best when roles are defined and respected and the ultimate goals of patient stabilization and safety are shared. The four competencies needed to work effectively in a high-quality collaborative team environment include: (1) values and ethics, (2) roles and responsibilities, (3) inter-professional communication, and (4) teams and teamwork. It is critical that team leaders promote these competencies by ensuring psychological safety so that the team can work within the learning zone. In that environment, team members can feel safe to ask for help, admit errors, and suggest innovations without feeling threatened or belittled. Teams that work within the learning zone are much more likely to ensure they are incorporating *all* valuable evidence into their care.

Understanding models of outpatient healthcare delivery can help prevent early readmission. The inpatient team can facilitate a smooth and lasting transition by anticipating the strengths and limitations of the outpatient teams, and adhering to good communication, which fosters collaboration. Each inpatient team member can support the overall team goals of patient stabilization and transition, by using her/his unique skills.

Take-Away

- Providing care to geriatric inpatients with acute psychiatric symptomatology and maladaptive behaviors, along with co-morbid medical conditions, requires an effective team of collaborating professionals from several disciplines.
- Each professional member of the team communicates and demonstrates her/his areas of expertise as well as limitations. This fosters respect and coordination.
- Each professional applies her/his expertise to support the goals for the patient, as well as engages the skills and expertise of other disciplines. This enhances an understanding among each discipline about what each can best offer, and it promotes effective collaboration.
- The healthcare delivery models practiced by an inpatient psychiatric team can foster a smooth transition to outpatient care.

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5 Legal Aspects of Inpatient Geriatric Psychiatry

Kelli Columbo, Mike Kelly, and Leah McGowan

5.1 Introduction

The number of geriatric patients living in the United States is expected to double – from 35 million in 2000 to 72 million in 2030 [1]. Currently, about 1 in 7 US residents is over the age of 65, and this ratio is expected to change to 1 in 5 by the year 2030 [2]. Geriatric patients have high rates of medical illness, cognitive impairment, and physical frailty. Fig. 5.1 summarizes the legal issues which can arise for geriatric psychiatry inpatients. These conditions require substantial healthcare decision-making that is amplified in the acute care setting. Decision-making is especially complex in the presence of acute psychiatric/behavioral symptomatology such as agitation, wandering, paranoid delusions, hallucinations, depressive symptoms, and cognitive deficits. Delirium, major neurocognitive disorder (MNCD), major depressive disorder, and anxiety disorders, among others, may increase the risk of impaired decision-making. As decision-making capacity declines, aging adults with cognitive and other psychiatric impairments are also at increased risk of elder abuse. These and related issues are discussed in the section below.

5.2 Vignette

An 81-year-old woman with major neurocognitive disorder and recurrent urinary tract infections (UTIs) was admitted from home to the inpatient psychiatry unit for an episode of major depressive disorder. Once on the inpatient unit, she developed greater urinary frequency and her urinalysis showed an acute UTI. The patient refused antibiotics. She stated her UTI symptoms were manageable and not worth the discomfort of the antibiotic side effects. The attending physician informed her of the risk of not taking the medications, including serious infection, and the patient repeated, "I know, I know, I'm still not taking that medication!" There was no family available and no other surrogate decision-maker.

On hospital day 7, the depressive symptoms remained significant, Geriatric Depression Scale (GDS) scored 12/15, and she continued to demonstrate UTI symptoms, now with fever and significant pyuria. She refused antibiotics

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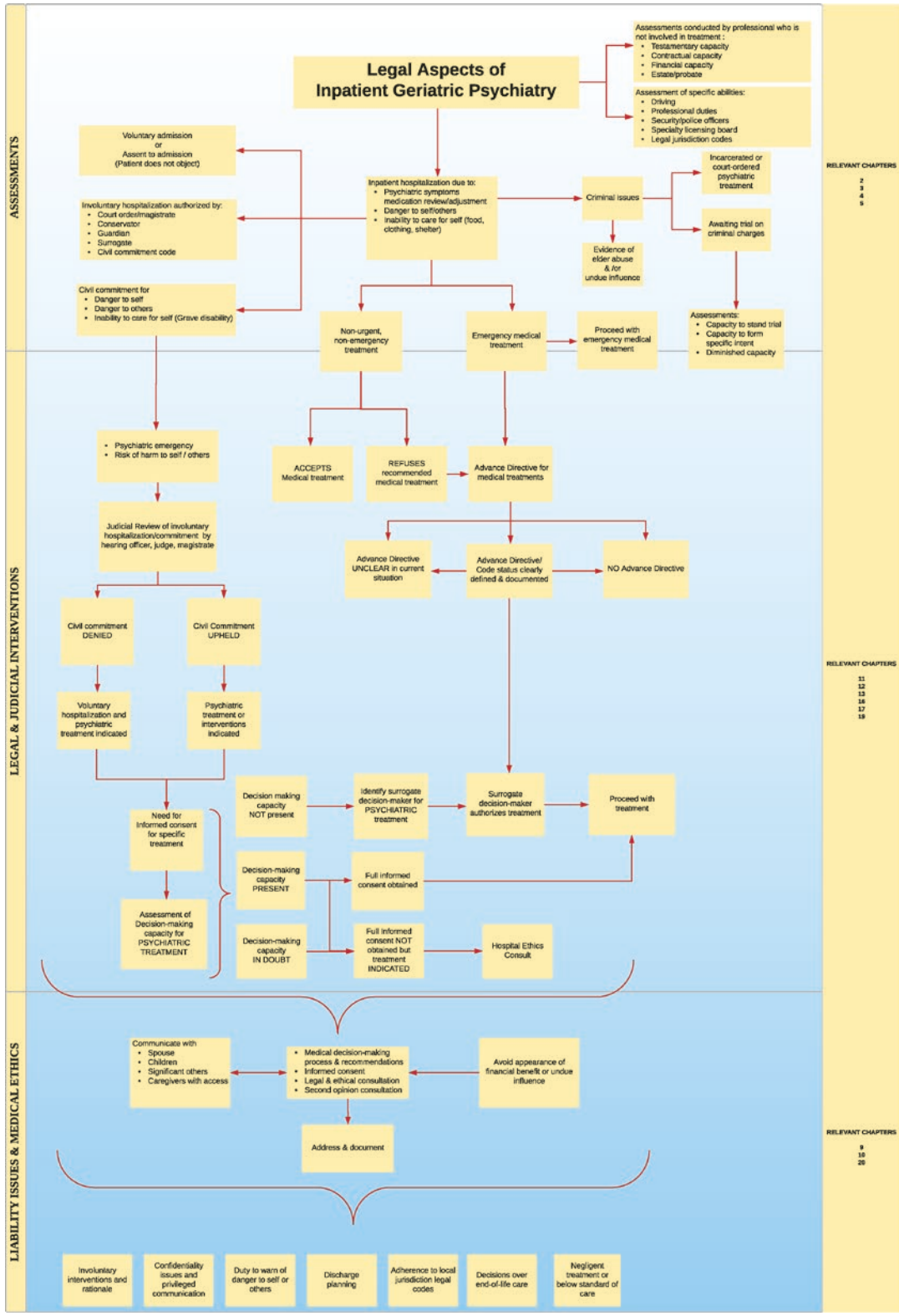

Fig. 5.1 Flow-chart of legal issues which can arise for geriatric psychiatry inpatients

again, and stated, "Don't use up your resources on me. I'm a little old lady and I don't want to waste people's time delaying the inevitable. I'm an old lady!" The patient also showed psychomotor retardation, lack of joy, a sense of worthlessness, and morbid ideation, criteria of a severe major depressive episode. Now she was not able to articulate a reasonable decision-making process to support her refusal of medication. The treatment team found her to lack capacity for decision-making about antibiotic treatment. A second physician's opinion confirmed a lack of decision-making capacity, which was documented. The medical opinions were based upon an evaluation of four principles underlying competence [3] and did not depend solely on the presence of the diagnoses of severe major depressive disorder and major neurocognitive disorder.

The hospital ethics committee convened by telephone within 1 day and endorsed the option of involuntary treatment with antibiotics, mixed surreptitiously into the morning pudding (Chap. 13: Involuntary Interventions). The UTI began to improve within 3 days of starting treatment. Depressive symptoms and neurocognitive deficits remained unchanged in severity, although the patient's cooperation with treatment improved; the patient was discharged within 2 weeks to a skilled nursing facility.

The patient was re-hospitalized to inpatient psychiatry 1 month later, through an involuntary commitment, after she became agitated at the skilled nursing facility. An acute UTI was discovered. The treatment team pursued a permanent guardianship, which the court upheld. The statue controlling guardianship in the hospital's jurisdiction allowed the guardian to have decision-making powers over all general medical and psychiatric treatment, including Electro-Convulsive Treatment (ECT) (Chap. 16: Neuromodulation Interventions). In consultation with the treatment team, a course of ECT was authorized, and after 10 treatments, the most severe depressive symptoms improved, although an episode of delirium complicated the course of treatment.

5.3 Decisional Capacity, Informed Consent, Advance Directives, and Testamentary Capacity

Geriatric patients in the hospital setting commonly lack decisional capacity, with estimates ranging from 20% to 50% [4, 5]. Aging adults face myriad healthcare decisions, and capacity evaluations are especially salient when medical issues are serious. Although all licensed physicians can make decisional capacity determinations, many lack formal training and tend to underutilize these assessments. When patients with limited decisional capacity make medical decisions, significant ethical and medicolegal concerns emerge. At a time when the geriatric population is expanding, it is essential that clinicians caring for aging adults understand and address the issue of capacity. Validation of the construct of decisional capacity, instrument development, and comparison of patient groups have contributed to the momentum of the field [6].

In the healthcare context, "decisional capacity" refers to a clinical judgment of one's ability to make a specific healthcare decision [6]. "Competency" is a judicial determination of legal status often used to refer to the ability to stand trial or make financial decisions. Despite the canonical distinction, the terms "competency" and "capacity" are often used interchangeably. For issues within the realm of civil law, and in keeping with the situation-specific nature of capacity, the terms "legal capacity" and "clinical capacity" have been used [7]. The clinician's assessment of capacity does not alter the patient's legal capacity but can inform the need to explore means for alternative decision-making. The clinician's documentation of the patient's cognitive status (and other psychiatric symptoms), and capacity for decision-making can also be used as evidence in legal and judicial contexts.

Most cases of suspected impaired capacity do not result in judicial review or determination. More often, the determination of the decision-making ability is a clinical one, focused on the

issue of informed consent for medical treatment. The issue of informed consent, therefore, is involved in almost every patient-physician encounter. The American Bar Association (ABA) and American Psychological Association (APA) co-authored a series of handbooks for use by attorneys, judges, and clinicians to promote the sound use of capacity assessment of older adults in the clinical setting (available for free download at http://www.apa.org/pi/aging/programs/assessment.index.aspx) [8].

The impact of neurocognitive disorders on decisional capacity is well studied. Aging is associated with the decline of multiple cognitive processes including memory, language, processing speed, and executive function. However, aging alone is not sufficient to cause incapacity. Comorbidities associated with aging, for example, major/mild neurocognitive disorders and psychiatric conditions, can lead to impaired judgment about healthcare decisions [9, 10]. Individuals with cognitive decline demonstrate greater impairment of capacity compared to healthy controls, even in the absence of comorbid psychiatric illness [11–13]. These impairments tend to be greatest in the domain of *understanding* choices, followed by *reasoning* and *appreciation* of the decision [7].

Medically ill or hospitalized older adults may also be at increased risk of incapacity [7, 14–18].

Psychiatric comorbidities can further impact reasoning and judgment, especially in older adults [19]. However, the relationship between psychiatric comorbidities and capacity is less straightforward. There is some data suggesting that depressed older adults are more likely to underestimate the benefits, overestimate the risks, and refuse life-sustaining treatment [20, 21]. Similar impairment may be found in psychotic disorders. Older adults with schizophrenia or schizoaffective disorder, when compared to those without, demonstrated impaired *understanding* of choice when assessed for capacity to consent to antipsychotic treatment [22]. Consistent with studies of younger adults with schizophrenia, specific cognitive domains, as opposed to the severity of psychopathology per se, correlated with capacity [23].

Decisional capacity is more commonly impaired in neurocognitive disorders when compared with mood disorders and psychosis [24]. In a study by Candilis et al. [25], direct comparison between groups with schizoaffective/schizophrenia, groups with medically ill subjects, and groups with non-ill subjects showed that impaired cognition and physical function predicted poor decision-making independent of psychiatric diagnosis [25]. Similarly, severely depressed patients consenting to ECT demonstrated no significant difference in decisional capacity when compared with controls. These data support the notion that even in the presence of significant psychiatric illness burden, capacity may be preserved, and should not be presumed absent on the basis of psychiatric illness per se. Given the variability in capacity across the psychiatric spectrum, and considering the extensive medical and legal ramifications of the determination of incapacity, inpatient psychiatrists, and especially those caring for aging adults, must routinely assess decisional capacity as a component of their treatment plans.

Decisional capacity assessment includes:

- History: A determination of the patient's history, baseline cognitive functioning, and psychiatric symptoms which may interfere with decision-making. Whether the patient's current presentation is a deviation from baseline is especially important in geriatric patients, who are prone to delirium. In patients who present with notable cognitive impairment and/or other psychiatric symptoms, the clinician should obtain history from caregivers, family and other providers.
- Clinical presentation: A general psychiatric assessment includes an assessment of current cognitive functioning. Use of a standardized cognitive rating instrument can help demonstrate whether there are symptoms and/or deficits which may affect decisional capacity (Chap. 2: Neuropsychological Assessment).
- Capacity instruments: A determination of capacity can be supported with a combination of clinical judgment and standardized capacity assessment tools, such as the MacArthur

Process of decisional capacity assessments:
- Use clinical judgment & standardized capacity assessment tools (MacCAT-T or ACE)
- Gather patient history
- Assess baseline cognitive and psychiatric functioning
- Level of deviation from baseline cognitive and psychiatric functioning
- Obtain collateral information from caregivers if needed
- Identify any psychiatric symptoms that may affect decisional capacity

Fig. 5.2 Assessment of decisional capacity

Competence Assessment Tool for Treatment (MacCAT-T) [19] or the Aid to Capacity Evaluation (ACE) [16], although the use of these decisional capacity instruments is more common in a research context and is not required to meet the standard of care. Figure 5.2, presents a summary of components in the assessment of decisional capacity.

5.4 Informed Consent

The formulation of informed consent dates to the case of *Schloendorff v. Society of New York Hospital* (1914), in which Justice Cardozo stated:

> Every human being of adult years and sound mind has a right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient's consent commits an assault for which he is liable in damages. This is true except in cases of emergency, where the patient is unconscious and where it is necessary to operate before consent can be obtained.

Capacity is the cornerstone of informed consent. To provide consent, the individual must have capacity to make the decision. *Informed* consent is the process by which an individual makes healthcare decisions based on an understanding of the implications of that decision and without any undue influence. Though ostensibly a legal issue, informed consent is premised on autonomy – specifically, that persons are entitled to make decisions based on their personal values. In the medical setting, the clinician determines capacity to consent based on the individual's apparent ability to understand the risks, benefits, and alternatives to a proposed intervention, and to communicate a choice about the intervention. Physicians are bound by legal and ethical obligation to disclose appropriate information to facilitate patients' ability to make a voluntary choice, prior to initiating treatment [26].

For informed consent to be valid, the patient must be deemed competent to make the *specific* decision at hand. All adults are presumed competent to make decisions until proven otherwise. Individuals whose neurocognitive or other neuropsychiatric illness causes a defect in reasoning or judgment that affects decision-making about a specific intervention are deemed to lack capacity to make that decision. Barriers to communication may also interfere with a patient's full understanding of the information provided and, therefore, to weigh the risks against the benefits. Barriers may include deficits in language reception, speech, and hearing (Chap. 20: Medical Nursing Issues and Communication Barriers). When capacity is absent, a substitute decision-maker must be identified prior to initiating treatment. If a surrogate decision-maker is not easily identified, the physician must institute formal legal consultation, including a petition for medical authority to treat this patient.

There are many pitfalls inherent in the assessment of capacity, especially with regard to the evaluation of aging adults with mental illness. Physicians may erroneously presume the incapacity of individuals within certain diagnostic groups, such as psychotic disorders or neurocognitive disorders. Alternatively, physicians may overlook incapacity and/or may not examine some essential components of capacity. Clinicians less familiar with capacity assessment may inappropriately permit the patient to refuse

treatment due to fear of a perceived adverse medical-legal consequence. Or clinicians may inadvertently exert undue influence on the patient to make a decision that aligns with their clinical recommendation.

Studies show wide variability in physician determination of decisional capacity. Standardized capacity assessments, however, are associated with improved inter-rater reliability [27]. The clinical interview may be inappropriately influenced by the clinician's personal values and ageism [27, 28]. In aging adults with physical or cognitive limitations, there is increased opportunity for use of visual cues and written presentation to supplement communication and overcome barriers (Chap. 20: Medical Nursing and Communication Barriers).

Capacity research has elucidated specific elements of capacity that tend to be compromised in various neuropsychiatric conditions. In patients with major neurocognitive disorder (NCD), the ability to understand and reason is more likely to be impaired than the ability to express a choice [12, 29]. This is important because while a patient with major NCD may adamantly express the wish to forgo a treatment, the clinician must accurately assess the patient's understanding of the circumstances before assuming the patient has capacity to make the decision. Importantly, however, capacity among patients with other chronic psychiatric illnesses such as schizophrenia is much more heterogenous [30]. Further, impaired capacity in patients with delirium or a major depressive disorder may be time-limited [8].

In many jurisdictions, any physician or psychologist familiar with the medical condition and treatment options for a patient can assess decisional capacity. However, in geriatric patients with psychiatric co-morbidities, who may be especially vulnerable to impaired capacity, psychiatrists, psychologists, and nurse practitioners may be the clinicians most familiar with aspects of impaired capacity.

Capacity in the setting of medical decision-making refers to the healthcare provider's determination of an individual's ability to understand the nature of the intervention, its risks, benefits, and alternatives, as well as the ability to communicate a clear choice (see Table 5.1). Higher risk procedures or interventions require a greater degree of decisional capacity than lower risk procedures. Therefore, the stringency of the capacity evaluation should vary directly with the *severity* of the anticipated consequences of the decision.

Also, if the patient's choice to *refuse* treatment carries a high risk, there is a *higher* burden of proof needed to show that she/he has the capacity for an informed choice. For example, a psychotic

Table 5.1 Criteria for decision-making capacity

Criterion	Questions for physician	Considerations	Example questions
Communicate a choice	Does the patient clearly and consistently indicate a choice?	Reversal of choice may suggest lack of capacity	Would you like to try taking antibiotics to treat your UTI?
Understand relevant information	Is the patient able to paraphrase information conveyed about the medical condition including recommended treatments, and the risks/benefits/alternatives?	Providers should discuss alternative treatments (including no treatment)	What do you understand about the treatment for a UTI and the other alternatives?
Appreciate the situation and its consequences	Is the patient able to acknowledge the medical condition (insight), the proposed treatment, and expected outcomes?	Patients who do not acknowledge illness cannot make valid decisions about treatment	Do you understand what might happen if you do not take the antibiotics for the infection?
Reason about treatment options	Is the patient able to compare treatment options and provide rationale for the decision?	Assess the process of arriving at the decision, not the decision itself	How did you decide on your decision to refrain from taking the antibiotics?

Adapted from Appelbaum [3]

patient who refuses antibiotics to treat sepsis would require a more stringent capacity evaluation than a healthy patient refusing an influenza vaccination.

An exception exists in an emergency situation, wherein informed consent need *not* be obtained, so that timely treatment proceeds to prevent death or avert catastrophic results (e.g., emergency interventions to minimize loss of brain tissue in a stroke) (Chap. 7: Acute Medical Events). Table 5.1 provides a summary of the criteria which determine a patient's ability to make decisions about medical treatment.

5.5 Advance Directives

In the United States, the Patient Self-Determination Act [31] requires that healthcare providers inform patients about the right to refuse or accept treatment as well as the right to create Advance Directives. Advance Directives are legal instruments designed to allow a patient to retain some choice in healthcare decision-making during periods of decisional incapacity. Patients may use Advance Directives to outline choices for healthcare treatment (Decision Directives), appoint surrogate decision-makers (Proxy Directives), and in patients with psychiatric illness, express preferences for psychiatric treatment (Psychiatric Advance Directives). If the patient is incapacitated, a surrogate may attempt to approximate the patient's wishes, based upon an Advance Directive. Advance Directives allow preferences to be respected.

Advance Directives can be created and executed without court order and may be changed to reflect a change in decision to accept or refuse treatment, at any time while the patient has decision-making capacity. But a patient who lacks capacity now may not change an Advance Directive which has been previously signed when the patient *had* capacity. If a patient who lacks capacity refuses a choice manifest in their Advance Directive, a guardian or court order may be necessary. Criteria required for a valid Advance Directive vary by jurisdiction. For example, some states legally recognize oral statements as Advance Directives if they are properly documented. Whether psychiatric care is included in the domain of a proxy decision maker also varies by jurisdiction.

The following are common types of Advance Directives:

- Decision directives (living wills): Patients can express choices for healthcare treatment in *decision directives*, or *living wills*, which become effective when the individual lacks capacity. Often, they address choices about cardiac resuscitation, ventilator treatment, artificial nutrition, invasive tests, and blood products. Decision Directives may only be revoked or altered by the patient, and this can only be done while the patient has capacity. Patients can express choices regarding acceptance or refusal of life-sustaining treatment in the event of terminal illness or impaired level of consciousness, which become valid in the event capacity is impaired. In these circumstances, physicians can honor a patient's previously designated choices about treatment by referring to Decision Directives. Despite the utility of Decision Directives, most US adults do not have a living will.
- Proxy Directives (Durable Power of Attorney): In Proxy Directives, patients authorize a surrogate decision-maker, or Durable Power of Attorney (DPOA), to make medical decisions on their behalf. Unless specified as a DPOA for healthcare, also called a healthcare proxy, the DPOA only makes financial decisions in the event of the patient's incapacity. Patients assign a DPOA while they still have capacity and may continue to express directives even after a proxy has been designated, in the same way that a decision directive can be changed during periods of capacity. The proxy assumes the decision-making role once the patient is deemed by a clinician to lack capacity. In these cases, physicians must obtain informed consent from the proxy as the surrogate decision-maker; however, if the patient has a living will in addition to a proxy, choices manifest in the living will generally supersede the proxy decision-maker.

- Psychiatric Advance Directives (PAD): Using PAD, patients with mental illness may retain choice over their psychiatric treatment during periods of compromised capacity due to mental illness [32]. In situations of conflict between the PAD and the standard of care or civil commitment law, clinicians may exercise professional judgment to override a PAD [33].

In the event of decisional incapacity when there is no Advance Directive, some jurisdictions have case law or statutes that authorize family members to make healthcare decisions according to a hierarchy, which is typically: (1) spouse; (2) adult children; (3) parents; (4) siblings; and (5) distant relatives [34]. Notably, long-term partners without legal standing are not recognized, which can lead to significant psychosocial distress.

5.6 Testamentary Capacity

Testamentary capacity is a legal term for the mental ability to understand the nature of the will, the extent of assets, and the claims of those included and excluded from the will. The issue most often arises in the outpatient setting, though families and other invested parties may raise it during an inpatient hospitalization.

Individuals must demonstrate testamentary capacity when creating, altering, or nullifying a Living Will [8]. The burden of proof often rests with anyone challenging a patient's testamentary capacity; an individual is assumed to retain testamentary capacity unless adjudicated to lack it. Courts interpret the requirement for testamentary capacity liberally; often, individuals with clear indications of diminished capacity in other domains are found to retain testamentary capacity.

Disputes regarding the applicability or validity of a will often require robust evidence of whether that person making the will (testator) had testamentary capacity at the time the will was created or changed.

Definitions of testamentary capacity vary by state. In general, one must demonstrate: (1) general understanding of the nature and extent of his or her assets; (2) an appreciation of who would be the natural heir to his or her assets; (3) understanding of the nature of the will and that he or she is signing a will; and (4) that he or she is free of delusion [34]. A court can also invalidate a will if the testator is deemed to be experiencing an *insane delusion* that affects the material of the will. Delusions are fixed false beliefs held despite conflicting evidence and are common in neurocognitive and psychotic disorders. In geriatric patients with psychiatric illnesses, the presence of delusions must be considered when assessing testamentary capacity.

Because testamentary capacity and contractual capacity often involve financial decisions regarding estate and probate matters, an appearance of a conflict of interest may arise if the evaluations are performed by treating clinicians. An argument could be raised that the treating clinician somehow stands to benefit from a determination of the patient's lack of capacity, especially if the evaluator has some alliance with those who *will* benefit from such an opinion. There are often other parties (e.g., partners, ex-spouses, half-children) who have an interest in the outcome of the evaluations, which can complicate the delivery of optimum care. For these reasons, it is recommended that an independent evaluator, who is not providing any treatment, perform evaluations of financial decision-making capacity. While this issue may not arise within the acute hospitalization, it is helpful for the inpatient psychiatric practitioner to have an understanding of these assessments and the process, in order to best advise families and patients.

Whether an individual has testamentary capacity is based on the assessment of capacity at the time the will is being executed, modified, or revoked. Formal evaluation and documentation of the presence of testamentary capacity each time a will is made or changed reduces the likelihood that the validity of the will is challenged [34].

Even if the testator has capacity, a will may be voided if that individual has been subjected to *undue influence.* Undue influence is the presence of an external influence that overcomes the testator's free will, due to his or her vulnerability, and

compromises the authenticity of the testator's stated wishes in the will. In cases of undue influence, the law recognizes that the will is invalid because it does not express the wishes of the testator [35]. To avoid any appearance of undue influence, any clinician involved in the treatment of the patient, and therefore with influence over the patient, should not participate in the assessment of capacity to make a will.

Factors associated with increased vulnerability to undue influence include older age, the presence of psychiatric or systemic medical illness, and the nature of the relationship between the testator and the individual alleged have exercised undue influence. Figure 5.3 shows other risk factors for undue influence.

In some jurisdictions, such as California, wills that benefit the person who drafted them, or people related to or associated with the person who drafted them, are automatically invalid, and the law presumes the invalidity of a will that gives a benefit to (1) a care custodian of the testator, (2) a person in a fiduciary relationship with the testator who transcribed the will or caused it to be transcribed, or (3) people related to, living with, or working with the people in (1) and (2) (CA Prob Code § 21380 [36]). This rule means that such will provisions are not valid unless the benefactor proves the absence of fraud or undue influence. Under certain circumstances, a testator may be subject to persuasion by a legitimate party, for example, a spouse. If the influence is

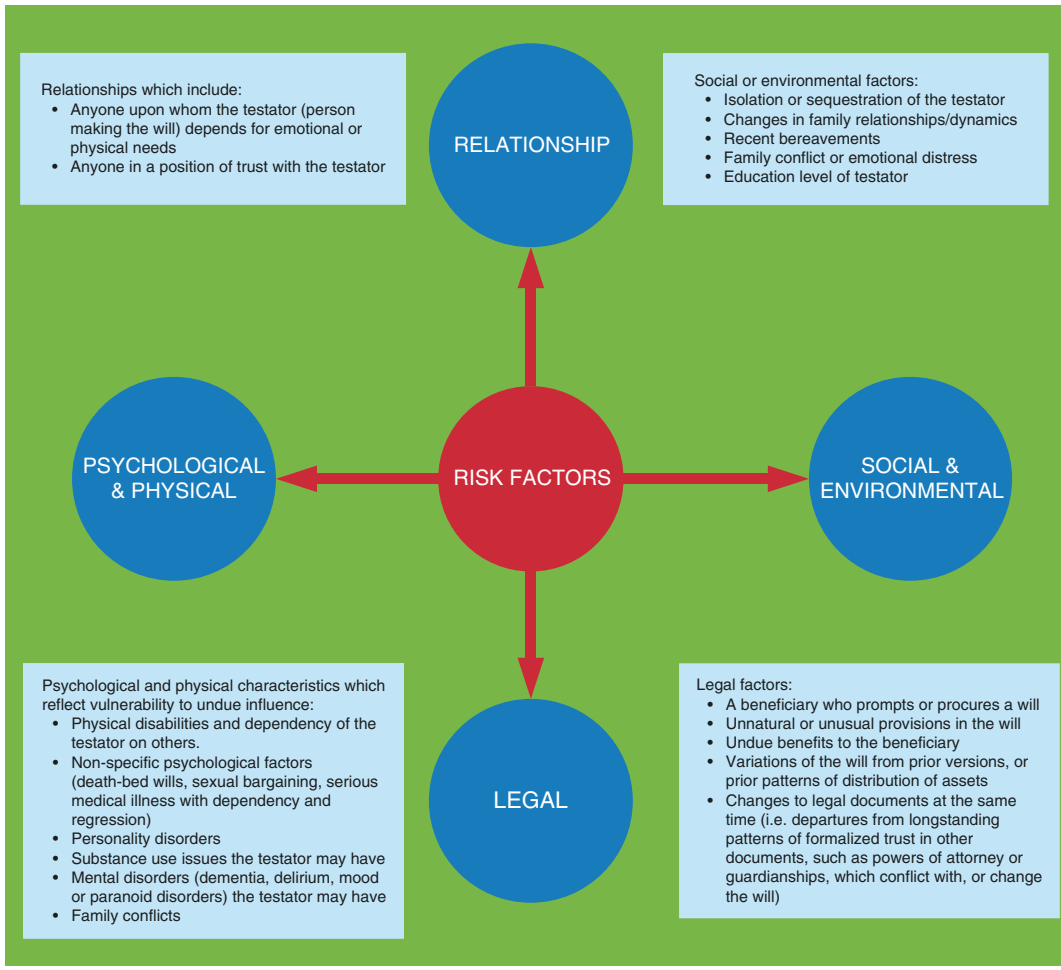

Fig. 5.3 Situations which may facilitate undue influence. (Adapted from Peisah et al. (2008))

reasonable, and not "undue," the will remains valid. Figure 5.3 lists some situations which may facilitate a relationship of undue influence.

5.7 Contractual Capacity

Aging patients with psychiatric impairments are vulnerable to impaired financial and contractual capacity. Financial capacity is considered an advanced activity of daily living [7]. There is no standardized assessment of financial capacity, but the assessment of actual functioning, rather than a psychiatric diagnosis, provides more valid information about one's capacity for financial decision-making. Patients unable to manage their finances may be assigned a guardian of estate or conservator (depending on the terminology of the jurisdiction) by the court. The determination of contractual capacity is based on the presence or absence of the individual's ability to understand the nature of the transaction [8].

5.8 Surrogate Decision Making

As many older adults grow increasingly dependent on external supports, including family members, friends, and caretakers, they more frequently involve others in their care. While there is implicit agreement that involvement of family members is ethically appropriate in the absence of objection by the patient, and especially when medically indicated, clinical judgment may be exercised to protect the patient's autonomy. If a clinician believes that it is in the patient's best interest to involve a family member, and a likelihood of negative outcome if the family member is *not* contacted, the clinician should assess the capacity of the patient who refuses.

In emergent situations, the clinician must determine the risk of harm, and, at times, override the patient's wishes to prevent that harm. In non-emergent settings, the clinician should maximize patients' autonomy, and respect wishes regarding the involvement of others, unless a medical or psychiatric condition impairs their capacity.

5.9 Guardianships and Conservatorships

Capacity assessments have implications far beyond the hospital setting. In guardianship/conservatorship proceedings, courts often seek expert opinions to guide capacity evaluations. A clinician who knows the underlying medical conditions *and* an individual's functional abilities (e.g., self-care, medical decision-making) can provide crucial information to courts.

The court may appoint a guardian, conservator, or surrogate decision-maker to act on behalf of a patient who lacks decision-making capacity in personal and financial affairs. This process is intended to ensure that vulnerable individuals who lack capacity are protected from exploitation and neglect. The Uniform Adult Guardianship and Protective Proceedings Jurisdiction Act (UAGPPJA) [37] details the procedures for appointment of guardians and conservators for incapacitated persons.

Although the definition of "incapacity" varies by jurisdiction, Section 102(5) of the UAGPPJA defines an "incapacitated person" as follows:

> … an individual who, for reasons other than being a minor, is unable to receive and evaluate information or make or communicate decisions to such an extent that the individual lacks the ability to meet essential requirements for physical health, safety, or self-care, even with appropriate technological assistance.

Some jurisdictions utilize a more functional definition of incapacity, which they define as the inability to provide for one's basic personal needs of food, clothing, and shelter. In these models, there is increased emphasis on the inability of persons to care for themselves or their property, rather than how they make decisions (Cal. Prob Code S 1801 (a). ***) As of 2017, 45 states have the UAGPPJA.

A *guardian* is a person who is qualified pursuant to court appointment and has custody of the individual lacking capacity. Typically, guardianships of the person involve the appointment of someone to make decisions that are not financial in nature, such as healthcare decisions and housing [34]. Guardianships of the estate may

be appointed to manage financial affairs. Certain jurisdictions use the term "guardianship" to signify both financial and non-financial decision-making power, while other states refer to this as a "conservatorship." Guardianship essentially denies a person lacking capacity the legal right to engage in decision-making. When a guardian is appointed, the individual lacking capacity may not enter contracts, manage funds, file lawsuits, vote, or consent to surgery.

A *conservator* is a person appointed by a court to manage the business affairs and estate of the individual lacking capacity. The law assumes that, unless proven otherwise, individuals with appointed guardians or conservators, *also* lack *testamentary* capacity, the capacity to make a will.

The person who is subject to limitations on her/his self-determination may experience lowered self-esteem, passivity, and feelings of inadequacy and incompetency. Because of this potential for adverse psychological effects, it behooves the court to seek less restrictive alternatives which may minimize the impact on an individual's autonomy and legal rights. Limited guardianships may be effective for persons who lack decision-making capacity in one domain (e.g., healthcare decision-making) but retain decision-making capacity in another (e.g., finances). This type of guardianship allows the incapacitated individual to remain protected under the law within the domain of incapacity, while retaining the authority and autonomy to make decisions within other functional domains where decision-making remains intact.

Guardians and conservators make decisions on behalf of the individual lacking capacity according to two standards:

1. The "best interest standard" by which the appointed person acts in accord with the best interest of the incapacitated person.
2. The "substituted judgment standard" whereby the appointed person must make choices based on what they think the incapacitated person would have chosen prior to becoming incapacitated. Advance Directives can provide evidence for the incapacitated persons' wishes.

Alternatives to guardianships include the use of *advance directives*, appointment of a payee or custodian for funds or income (e.g., for Social Security benefits), or a *healthcare power of attorney*. In 2016, the ABA published the PRACTICAL tool, a 28-page guide to identifying and implementing less restrictive alternatives to guardianship in decision-making for persons with impaired capacity. This can be found at: http://www.supporteddecisionmaking.org/sites/default/files/PRACTICALGuide.pdf

5.10 Liability Issues

5.10.1 Negligence

Negligence claims are commonly referred to as malpractice claims. The leading cause of malpractice claims against psychiatrists is patient suicide [38]. Claims of negligence are brought in civil court, as opposed to criminal court. Negligence claims involve four components: duty of care, breach of the duty of care, causation, and damages. Clinicians have a duty of care to their patients (e.g., the doctor-patient relationship). Psychiatrists have a duty of care that includes, among other duties, a duty to provide reasonable care upon discharge. In some jurisdictions, psychiatrists have a duty of care to third parties that includes a duty to warn them about harm from their patients (see below). Clinicians may breach that duty of care if they deviate from the accepted standard of care.

For causation of an injury to be found, the deviation from the accepted standard of care must have been a *direct* cause of an injury. The damage may be a personal injury, psychic harm, or death. These components must line up. For example, a plaintiff might claim that the defendant owed him a duty of care because the defendant was his psychiatrist, that the psychiatrist breached the duty of care by putting the patient on a new medication with severe side effects and then left town without providing coverage. The plaintiff would then have to prove that this breach caused damages – for example, that the patient became acutely suicidal because of the new medicine,

was unable to get help because of the lack of coverage, and sustained physical injuries from an ensuing suicide attempt.

5.10.2 Confidentiality

Aging adults often present to medical settings accompanied by family members or caretakers. This dependence on others for cognitive and physical support may predispose the patient to compromised confidentiality [39]. Paternalistic tendencies within medicine can lead to more liberal exchanges of information with older patients' family members, and an assumption of impaired capacity. In a study by Perez-Carceles et al., over one-third of family doctors disclosed information to others without prior consent [39]. Clinicians should be mindful of this tendency.

5.10.3 Duty to Warn

In some jurisdictions, such as California, psychiatric treaters have a duty to warn third parties about potential harm from patients. The obligation to warn others may be greatest during inpatient psychiatric treatment. The presumption is that, opposed to other treatment settings, the inpatient setting affords more time and resources to assess danger to self or others, inability to care for self, and to arrange appropriate outpatient follow-up. While the issue of duty to warn may not appear urgent during the bulk of an inpatient's stay, the issue deserves attention as soon as discharge planning begins, which should be early in the hospitalization.

The duty to warn is balanced against the duty to maintain a patient's confidentiality. The contours of the duty to warn have developed through "duty to warn" or "duty to protect" statutes and case law, and vary across jurisdictions. The most famous and first "duty to warn" case is detailed below.

In 1976, the Supreme Court of California decided the early landmark case of *Tarasoff v. Regents of the University of California* (1976). In this case the patient, Mr. Prosenjit Poddar, studied naval architecture at UC Berkeley. Mr. Poddar met a fellow student, Ms. Tatiana Tarasoff, at a folk dancing class in 1968. By all accounts, Mr. Poddar and Ms. Tarasoff started off as friendly acquaintances and shared a New Year's Eve kiss. Unfortunately for Ms. Tarasoff, Mr. Poddar become obsessed and stalked her. Mr. Poddar began seeing a therapist at the student mental health clinic, Dr. Lawrence Moore. Mr. Poddar eventually told Dr. Moore that he planned to kill Ms. Tarasoff. Dr. Moore notified the campus police and recommended that Mr. Poddar be civilly committed; however, Dr. Moore's supervisor disagreed. Ms. Poddar was not civilly committed and Ms. Tarasoff was never provided a warning about Mr. Poddar's plans.

Mr. Poddar tracked down Ms. Tarasoff at her parents' home and stabbed her to death. Ms. Tarasoff's parents sued UC Berkeley and Mr. Poddar's therapist, Dr. Moore. After a series of lengthy legal battles the California Supreme Court ruled that "when a therapist determines, or pursuant to the standards of his profession should determine, that his patient presents a serious danger of violence to another, he incurs an obligation to use reasonable care to protect the intended victim against such danger." The discharge of this duty depends on the nature of the case, and may require the therapist to "warn the intended victim or others likely to apprise the victim danger, to notify the police, or to take whatever other steps are reasonably necessary under the circumstances" (*Tarasoff v. Regents of the University of California* (1976), 17 Cal. 3d 425, 431). As for the competing duty to maintain a client's confidentiality, the court stated that "[t]he protective privilege ends where the public peril begins."

In the 40 plus years since the *Tarasoff* decision, psychiatrists' duty to protect has expanded based on case law from a variety of jurisdictions. For example, in the case of *Lipari v. Sears* (1980) a Nebraska court expanded *Tarasoff* by ruling that potential victims of violence did not need to be readily identifiable as long as it could be "reasonably foreseen" that a "class of persons" was in harm's way (*Lipari v. Sears, Roebuck & Co.*, 497 F. Supp. 185, 195 (D. Neb. 1980)). In *Naidu v. Laird* (1988), the Supreme Court of Delaware

affirmed the trial court's ruling that a psychiatrist who discharged a patient during a voluntary state hospital admission 5 months prior was liable for a man's death in a motor vehicle caused by the former patient. The court affirmed the trial court's imposition of a duty owed by the mental health professional to the decedent. It held that the "special relationship" between mental health professionals and a patient created a duty not just between the professional and the patient but also between the mental health professional and third parties. The psychiatrist breached that duty by failing to properly review the patient's extensive history of psychiatric hospitalizations and medication noncompliance prior to discharge, and that this negligence was the "proximate cause" of the motor vehicle accident victim's death (*Naidu v. Laird*, 539 A.2d 1064, 1075–76 (1988)).

In *Ewing v. Goldstein*, the California Court of Appeals expanded the duty to warn, holding that psychotherapists have a duty to warn of threatened violent behavior of a patient not just when they learn about the threatened violent behavior from the patient, but when they learn about the threat from the patient's family members or other individuals (*Ewing v. Goldstein* (2004), 120 Cal. App. 4th 807, 814). The Ewing case applies to practitioners in the state of California and has significant ramifications for those who provide care for elder adults. In the context of elderly care, caregivers often receive information from family members. Practitioners need to become familiar with the duty of care and duty to protect statutes and case law in the jurisdiction where they practice. The Ewing Court stated: "… this holding broadens the instances in which a therapist has a duty to warn, imposing a duty based not only on threats made by the patient but also on communications from third parties made about the patient's alleged threats."

The authors offer the opinion that a treating clinician working *on an inpatient setting* may have an even greater obligation to warn others of a patient's future danger than from other treatment settings. We assume that a court would consider that on an inpatient unit, as opposed to other treatment settings, more time and resources are available to assess dangerousness, and to arrange appropriate outpatient interventions, including appropriate warnings. We recommend that warnings, if appropriate, be provided early as soon as discharge planning begins, which should be early in the hospitalization (Chap. 23: Placement).

5.11 Criminal Issues

5.11.1 Elder Abuse

Elder abuse has been described in the literature since the 1970s [40]. Yet it remains highly underreported [41]. Over the past decade, growing research interest in the topic of elder abuse has led to a consensus about the inclusion of five major types in the definition of elder abuse: physical abuse, psychological or verbal abuse, sexual abuse, financial exploitation, and neglect [42]. Considered together, these types of abuse occur in approximately 10% of older adults over a 12-month period. One in four older adults dependent on a caregiver report significant psychological abuse and one in five reported neglect [41]. Among the type of reports made to Adult Protective Services, most (70%) are for neglect.

Inpatient geriatric psychiatrists face several challenges to screening for elder abuse. Victims of abuse may not be forthcoming about their experience. Suspected victims should be interviewed alone to provide a safe space, and indirect questions can be used to make the interview less threatening (e.g., "Do you feel safe at home?").

Older adults with psychiatric conditions may have difficulty reporting their experiences due to an underlying cognitive or mood disorder, or other communication barriers and all should be assessed as part of the evaluation for elder abuse (Chap. 20: Medical Nursing and Communication Barriers). Elder abuse may be especially difficult to detect among individuals with multiple medical comorbidities. Clinicians may overlook signs of abuse (e.g., bruising misattributed to fall) or misattribute spontaneous findings to abuse (e.g., burn misattributed to physical abuse). Notably, cases of elder abuse do not occur exclusively outside of the hospital. In *Bergman v. Chin* (1999), a physician was charged with elder abuse for

Fig. 5.4 Factors which can contribute to elder mistreatment

insufficient pain control in an 85-year-old male hospitalized for compression fractures and suspected lung cancer [43]. Keeping these factors in mind, care providers should remain aware of risk factors for elder abuse [44]. Figure 5.4 summarizes some factors which may predispose to elder mistreatment.

Reports of elder abuse can be made by contacting an adult protective services agency. In many jurisdictions, clinicians are Mandated Reporters, which means they are obligated to report suspected elder abuse. Mandated Reporters are not typically held liable for reports of abuse made in good faith; failure to report or interfering with a report of elder abuse is a misdemeanor in many states, however. Adult Protective Services agencies help older adults who are victims of abuse by providing investigatory, protective, and supportive services through state or county funding. Family members and the elder victims may be willing to report abuse when queried. But mental health clinicians must also guard against the impulse to adopt the role of a detective, gathering evidence against a suspect, at the expense of clinical care.

5.12 Civil Commitment

Civil commitment is the process by which a person with mental illness is required to accept psychiatric inpatient hospitalization or psychiatric treatment. It is generally initiated with a clinical judgment that is followed shortly thereafter with a legal decision by a judge. Standards vary by jurisdiction. The typical basis for civil commitment is that the person represents a danger to self or others, or is unable to provide for basic needs of food, clothing, and shelter due to a mental illness. Legal status concerning capacity to make decisions, including the capacity to refuse medications, is a separate issue and not directly impacted by civil commitment. Similarly, the appointment of a guardian does not necessarily follow civil commitment [8].

The decision about whether or not to institute a civil commitment is particularly challenging in geriatric psychiatry. Caregivers and family may request admission, in part based upon the overwhelming demands of caregiving, but in the absence of meeting adequate standards for involuntary commitment. The situation may be compounded by a patient who may have limited capacity to understand the situation or articulate a perspective, but still not meet criteria for civil commitment.

5.13 End of Life Care

End of life care involves the appropriate balance of intervention and palliation [45]. In an effort to improve end of life care with Advance Directives, federal law requires that hospitals provide patients with information about goals of care upon admission [31].

Inpatient geriatric psychiatrists should encourage deliberate attention to end of life care direc-

tives. Many aging adults have not made end of life care wishes explicit, and face the risk of developing a medical or psychiatric condition that compromises their capacity to make decisions.

The US Supreme Court makes the following distinction between ending one's life (e.g., physician-assisted death) and refusing lifesaving treatment:

> … when a patient refuses life sustaining medical treatment, he dies from an underlying fatal disease or pathology; but if a patient ingests lethal medication prescribed by a physician, he is killed by that medication [46, 47].

Withholding treatment is an act of *omission*, often seen as abstaining from subjecting a patient to a burdensome intervention. Withdrawing treatment is an act of *commission*, whereby the clinician may feel a sense of responsibility for performing the act.

Some US jurisdictions have statutes authorizing physician-assisted death. These statutes typically allow individuals to obtain a prescription for a self-administering lethal medication if (1) they have capacity to make healthcare decisions and (2) they have been diagnosed with a terminal illness with death anticipated within 6 months. The statutes, commonly referred to as "death with dignity" statutes, can only be implemented after the individual has denied treatment that satisfies the standard of care (i.e., comprehensive palliative care). Individuals requesting physician-assisted suicide who demonstrate cognitive or psychiatric conditions that could impair their judgment are required to undergo psychological evaluation to determine their decisional capacity. "Death with dignity" is legal in five states (California, Colorado, Oregon, Washington, and Vermont) and the District of Columbia.

"Do not resuscitate" (DNR) is an order given by a physician that prohibits resuscitative protocol of a patient in cardiopulmonary distress. It can be written only after the physician has discussed the decision with the patient or, in cases of impaired capacity, a surrogate. Though DNR orders do not imply "do not treat," studies suggest that patients who are made explicitly "DNR" are less likely to get aggressive treatment, despite Presidential Directives dictating otherwise.

5.14 Summary

Legal issues often arise in the treatment of the geriatric patient, especially on inpatient units wherein treatment of many medical and psychiatric problems are intertwined. In this setting, clinicians often need to assess whether patients have the capacity to make treatment decisions. Capacity is a unique construct with clinical, ethical, and legal referents. It is important to assess specific decision-making capacities in the geriatric patient, especially when the patient may initially appear to lack such capacity. While the presence of a psychiatric condition does not necessarily confirm incapacity, it may increase its likelihood.

Many other medical-legal issues can also emerge during an inpatient stay. For example, psychiatric clinicians who treat geriatric patients should routinely screen for elder abuse, which is often underreported. Advance Directives may not be in place prior to an inpatient hospitalization, prompting the need for such discussions. Dangerousness issues, such as duty to warn others, and suicidality, also arise during inpatient stays, and present risks of medical negligence.

In sum, medical-legal issues are common among geriatric patients during an inpatient psychiatric hospitalization, and the provider can help ensure that the care conforms to legal and ethical standards.

Take-Away

- The presence of a psychiatric condition does not imply, nor, by itself, prove, impaired decisional capacity. A psychiatric condition and impaired decisional capacity are not mutually exclusive.
- Consult hospital legal counsel and policies where there are questions concerning medical-legal issues and involuntary treatment.
- Consult the Guidelines of the American Psychiatric Association (APA), or other consensus guidelines from subspecialty

professional organizations, if there is uncertainty about proposed treatment or its risks. Document references which describe the standard of care.

- Assess the decision-making capacity of geriatric patients who *consent* to recommended interventions, as well as those who *refuse* recommended interventions. The standard is *informed* consent, especially for interventions which carry considerable risk.
- It is possible to incorrectly assume that a geriatric patient *lacks* capacity for decision-making, simply because of communication barriers, neurocognitive deficits, and/or psychiatric symptomatology. These barriers should not prevent an attempt to assess a specific decision-making capacity.
- Aging patients should be encouraged to preserve their choices, while they are in a state of capacity, by instituting advance directives to dictate future healthcare and financial decisions.
- Alliances with family and surrogates are crucial when treatment recommendations and alternatives are proposed. Document such discussions, especially when there is disagreement among family members, or when the treatment has serious risks.
- Aging adults are at risk of neglect and abuse, at home, at an institutional facility, and in hospital settings; assess for possible abuse.
- Assess dangerousness to self or others, and document information and treatment plans.
- End of life care is an important part of healthcare advance directives.
- As a treatment provider, avoid participation in the assessment of decision-making capacity over financial or estate matters. These issues are best addressed by professionals who do not have responsibility for treating the patient.
- Advance directives may need clarification, especially if the patient no longer has capacity to provide informed consent. Document whether or not the Advance Directive for a specific intervention is the guiding document.

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Part II

Prevalent Problems in Inpatient Geriatric Psychiatry

6 Major Neurocognitive Disorder with Behavioral Disturbance (Behavioral and Psychological Symptoms of Dementia—BPSD)

Christopher O'Connell, Howard H. Fenn, and Rita Hitching

6.1 Introduction

Major neurocognitive disorder (MNCD) with behavioral disturbance, also known as behavioral and psychological symptoms of dementia (BPSD), consists of behaviors and psychiatric symptomatology which are not readily assessed by standard neuropsychological testing batteries, nor do the symptoms always present as manifestations of cognitive deficits. It has been estimated that 70% of residents in nursing homes in the VA Health Care System display some form of disruptive behavior, such as wandering, agitation, irritability, aggression, or aberrant motor behavior [1]. Figure 6.1 illustrates one approach to the evaluation of MNCD with behavioral disturbance.

The phenomenology of behavioral and psychological symptoms of Alzheimer's disease, the most prevalent MNCD, has been studied extensively, but their underlying pathophysiology has not been fully delineated. Cluster analyses have organized the diverse BPSD into at least three symptom complexes [2]:

- Behavioral disturbance with agitation and aggression
- Psychosis with delusions and hallucinations
- Mood disturbances, such as depression, apathy, or elation

BPSD can also be divided, with some overlap, into two major categories:

1. Non-cognitive symptoms (NCS), which include disruptive or inappropriate behaviors.
2. Neuropsychiatric symptoms (NPS), comprising psychiatric symptoms which are included within other DSM-5 psychiatric disorders.

6.2 Vignettes

6.2.1 Vignette 1

An 87-year-old widowed woman, diagnosed with major neurocognitive disorder (MNCD) secondary to mixed Alzheimer's disease (AD) and vascular disease, was admitted from her nursing home to inpatient psychiatry for six months of agitation, wandering, and delusional symptoms. The patient was preoccupied with her deceased husband, who passed away 1 year earlier.

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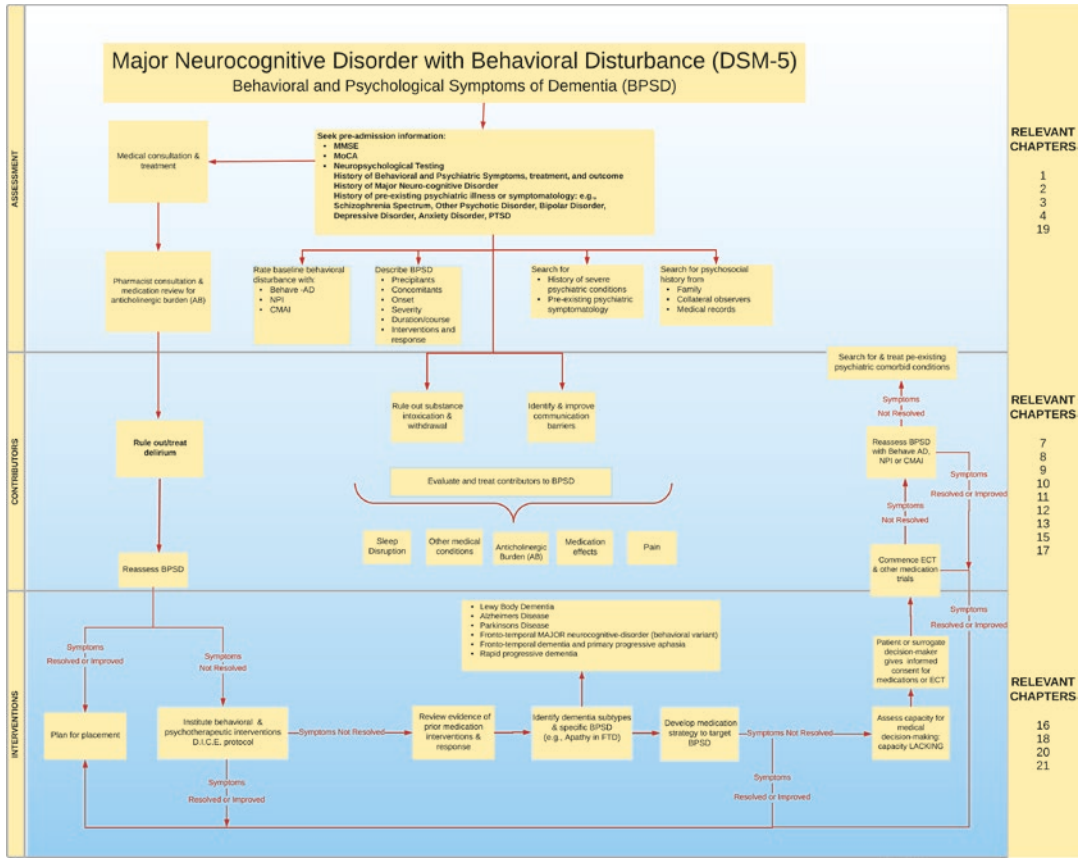


Fig. 6.1 Evaluation of major neurocognitive disorder (MNCD) with behavioral disturbance

Table 6.1 Non-cognitive (NCS) and neuropsychiatric symptoms (NPS) of major neurocognitive disorder (MNCD)

Non-cognitive symptoms (NCS)	Neuropsychiatric symptoms (NPS)
Disruptive behavior and verbalizations	Depressive symptoms: psychomotor retardation, morbid thoughts, suicidality, worthlessness, lack of joy, reduced energy
Intrusiveness	Manic symptoms, hypomanic symptoms, flight of ideas, pressured speech, grandiosity
Assaultive behavior/aggression: scratching, hitting	Anxiety symptoms and/or panic episodes
Socially inappropriate behavior	Somatic preoccupation, somatization
Hyperorality	Delusions, disorganized thoughts, paranoid ideation
Inappropriate sexual behavior	Visual hallucinations (most common in Lewy body related disorders)
Wandering	Obsessive-compulsive symptoms
Motoric pacing, trying doors	Hyperactivity: goal-directed/speeded
Agitation	Obsessive-compulsive symptoms
	Apathy
	Euphoria

On admission, she could perform all basic activities of daily living (ADLs) and some instrumental activities of daily living (IADLs). Her Montreal Cognitive Assessment (MoCA) score was 18/30, and she was disoriented to time and place. Her level of arousal was adequate and she followed simple directions, but she forgot new information within 5 minutes. No attentional or concentration deficits were noted, nor were there any laboratory findings or medications which would support a diagnosis of delirium. Medications included donepezil 10 mg daily and memantine 10 mg twice daily. Her cognition had been without significant decline over the prior six months.

On the second day of psychiatric hospitalization she walked off the unit into the street three times, and opened doors to others' rooms about every hour. She said her husband was in distress, was calling her, that he had been kidnapped, and that she was not being allowed to see him. Staff gently redirected her, but she pulled her arm away, made a fist, and scratched them. She approached and touched other residents. She awoke every night, thrashing and/or screaming.

Staff offered reminders, distraction, massage, music, aromatherapy, and redirection. A sitter was assigned for several hours each night. Sleep improved slightly as the wandering at night became less frequent, but episodes of agitation continued during the day. A daughter who lived nearby held Durable Power of Attorney for Health Care (DPAHC) and gave informed consent for a trial of citalopram 10 mg/day, which was increased to 20 mg daily after one week. A second daughter from out-of-state opposed the use of any medications, and staff mediated a tense family meeting via videoconference. The decision of the local daughter with Durable Power was honored and citalopram was continued. After 6 weeks, the patient had scratched two other patients during that period, and the citalopram, now at 30 mg/day, was deemed not effective. Complaints from other patients' families were lodged. Hospital administration considered a discharge to another facility, but no other living situation would accept the patient.

A dose of 1.0 mg of risperidone at bedtime was authorized by the local daughter/surrogate. The patient began to sleep better, did not hear her husband's voice as often, wandered less often, and she no longer scratched others. But her walking now became stooped, slow, unsteady, and she developed a resting tremor (Chap. 11: Psychiatric Symptoms Co-morbid with Neurological Syndromes) The risperidone dose was reduced to 0.5 mg. The second daughter filed a complaint with the hospital director and threatened the attending physicians with a lawsuit claiming negligence for not preventing or monitoring the extrapyramidal adverse effects.

The patient remained convinced that her husband was being held next door, but complained less about hearing her husband's voice. She continued to be disruptive at night, and one night she left her room and returned to lie down in her roommate's bed. The roommate pushed the patient, who fell to the floor. The on-call clinician discovered a painful, swollen, non-weight-bearing ankle. The patient was confined to bed, which further reduced wandering, but she began screaming at night from pain. She was transferred to a skilled nursing facility (SNF) to rehabilitate. The behavioral disturbance symptoms continued in the SNF. After 3 months, she was re-admitted to inpatient psychiatry on an emergency basis for agitation.

Discussion This patient developed auditory hallucinations and a paranoid delusion in the context of complicated bereavement and MNCD. Her psychotic symptoms did not resolve with interpersonal interventions, and symptoms only partially improved with the medication. Her family disagreed over the medication management.

Agitation resulted in an orthopedic injury (Chap. 7: Acute Medical Events).

6.2.2 Vignette 2

A 75-year-old man, with a 4-year history of MNCD and a 30-year history of schizophrenia, was admitted from a Board and Care (B&C) home where he had lived for the past 6 months. He had been stable up until 2 months earlier when he stopped taking his daily dose of 5 mg olanzapine, after which his chronic auditory hallucinations increased. He became disorganized and agitated.

On admission his affect was intense, bizarre, and labile. He accused staff and residents of "working for Satan" and poisoning his food. Auditory deficits caused him to misinterpret verbal communications, which he perceived as threatening. He said, "… there is going to be a World War III," and "…a computer made in Russia" was controlling him and forcing him to have a lobotomy. He was convinced that other residents of the boarding house would kill him because he owed money for cigarettes. He denied suicidal or dangerous intent (Chap. 19: Medical Nursing and Barriers to Communication). The B&C operator barred his return until he was more stable.

Medications at admission included divalproex 500 mg twice daily and olanzapine 5 mg at bedtime, and these were continued. Delirium and adverse medication effects were ruled out: laboratory findings showed no evidence of hyperammonemia, nor elevated liver function tests, nor subtherapeutic or toxic valproate levels. CT of the brain did not show new findings. The patient was too agitated to cooperate with cognitive screening.

In the hospital, he slept only 2–3 h per night and left his room at least three times every night. Thought processes were loose, and speech was pressured. The working diagnosis included MNCD with behavioral disturbance and schizophrenia, paranoid type, in acute exacerbation. Olanzapine dose was increased to 10 mg q HS, and he received "as-needed" doses of up to 30 mg daily for agitation. Hydroxyzine 25 mg and 2 mg benztropine twice daily were added to prevent extrapyramidal symptoms (EPS).

Three days after admission the patient complained that he wanted to "end it all." The on-call clinician prescribed amitriptyline 50 mg at bedtime for depression and insomnia. Within 5 days the patient became even more agitated, less cooperative, mentioned visual hallucinations (which he had never had before), and was preoccupied with somatic complaints. He pointed to his lower abdomen for pain "down there." Physical examination revealed dry hot skin and dry mucous membranes. A bladder scan showed urinary retention, and urinary catheterization produced a post-void residual (PVR) of 600 cc. A diagnosis of anticholinergic toxicity was offered. All medications with anticholinergic properties were held, including amitriptyline, benztropine, olanzapine, and valproate. Voiding was encouraged prior to bedtime, and a sitter assigned to help him return to bed at night. A dose of aripiprazole 2 mg at bedtime was started and the patient was rewarded with a snack when he took his medications.

Over the next 5 days he became more cooperative with unit routine and he complained less often. The paranoid delusions and auditory hallucinations continued, but he was less agitated, and the B&C operator accepted him back after three weeks in hospital (Chap. 7: Acute Medical Events).

Discussion Psychotic symptoms and agitation, consistent with an exacerbation of schizophrenia, were initially interpreted as symptoms of MNCD with behavioral disturbance. In an effort to ameliorate the behavioral disruption, the dose of antipsychotic medication olanzapine was increased. The patient's subsequent distress and dysphoria was interpreted as the onset of a depressive disorder, which was treated with a highly anticholinergic antidepressant. The new medication regimen increased the anticholinergic burden (AB), precipitating visual hallucinations and increase in urinary retention. The patient's disorganization and poor communication skills interfered with an accurate diagnosis.

6.3 Assessment of MNCD with Behavioral Disturbance

6.3.1 Pre-admission Screening

Days in hospital are a precious commodity due to rising health care costs and uncertainty over insurance coverage. Yet an inpatient psychiatric hospitalization can facilitate the thorough, efficient evaluation of presenting symptoms in order to institute rational and effective management strategies. Pertinent clinical information, such as onset, quality, duration, severity, precipitants, and concomitants of behavioral or psychiatric symptoms, available *before* admission, can speed evaluation and treatment time while in hospital. Patients transferred from stable living situations, especially a Medicare-certified nursing home, skilled nursing facility, or similar setting, may already have detailed documentation of neuropsychiatric and behavioral symptoms. In the US, such data is required under the Omnibus Budget Reconciliation Act (OBRA) which requires a Minimum Data Set (MDS) using the Resident Assessment Instrument Version 3.0 from October 2014 [3].

Partial hospitalization, and other alternatives to full inpatient hospitalization, such as short-term centers for surgical procedures, are becoming more prevalent. It is not clear whether these models of care delivery can provide the equivalent to intensive inpatient assessment and treatment for the geriatric patient with psychiatric symptoms. However, the efficiency of an inpatient hospitalization may be improving through the use of information technologies which facilitate gathering accurate data from outpatient settings and transferring it to the inpatient setting [4]. Cognitive assessment applications on digital devices, such as iPad, for example, may allow the patient, family, or caregiver to enter information about behavioral and cognitive functioning directly into mobile applications. This data can then be accessed by inpatient staff prior to admission [5] (Chap. 20: Telemedicine and Information Technology). Figure 6.2 summarizes pre-hospitalization history which can inform inpatient treatment plans for BPSD.

6.3.2 Baseline Assessments of Behavioral Disturbance Signs and Symptoms

A consensus definition of agitation states that "Inappropriate verbal, vocal, or motor activity that is not explained by needs…." [6, 7]. Wandering, intrusiveness, agitation, and other disruptive behaviors increase the risk of injury and/or assault, for all inpatients, not just the instigator [8]. Administration of behavioral rating scales upon admission and at regular intervals during the hospital stay can help determine the effectiveness of medical and non-medical interventions. Widely used scales in North America include the Neuropsychiatric Inventory (NPI), the Cohen-Mansfield Agitation Inventory (CMAI), and the Behavioral Pathology in Alzheimer's Disease (Behave-AD) (Table 6.2). All provide a standardized way to document the symptoms, signs, severity, and frequency of prevalent behavioral disturbance. Staff who are trained to administer

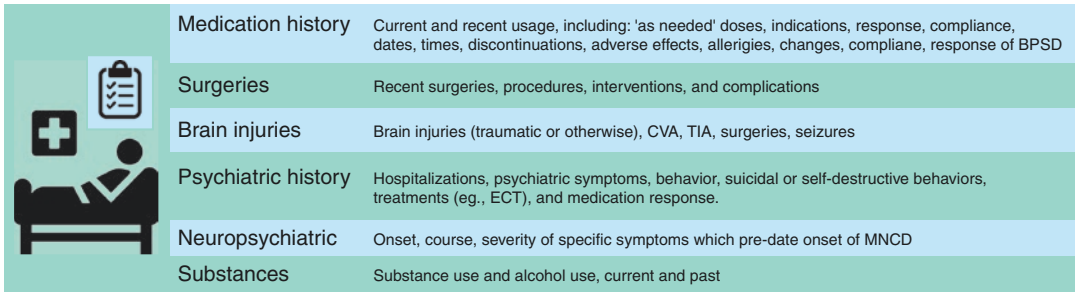


Fig. 6.2 Information relevant to behavioral disturbance

these instruments can improve their own observational skills, can enhance inter-rater reliability, and can better implement behavioral interventions [10–12] (Chap. 2: Neuropsychological Assessment). Table 6.2 shows items in Behavioral Disturbance Assessment Instruments.

6.3.3 Essential Rule-Outs and Considerations

An evaluation of the problematic behavior which led to hospital admission should start by ruling out delirium and by identifying the presenting

Table 6.2 Behavioral disturbance assessment instruments

Neuropsychiatric inventory (NPI) 12 domains [11]	Cohen-Mansfield agitation inventory short form (CMAI) 14 domains [10]	Behavioral pathology in Alzheimer's disease (Behave-AD) 7 domains [12]
Delusions: believes others are stealing, or wanting to harm them	Cursing, verbal aggression	Paranoid/delusional ideation: abandonment, impostor, infidelity, suspiciousness
Hallucinations: auditory, visual, speaking to no one there	Hitting, kicking, pushing, biting, scratching, spitting	Hallucinations: visual, auditory, olfactory, haptic (touch), vague, or nonspecific
Agitation/aggression: stubborn, resistive to help	Grabbing, throwing things, destroying property	Activity disturbances: wandering, purposeless activity, inappropriate activity
Depression/dysphoria: sad, in low spirits, tearful, crying	Aggressive (toward others): falling intentionally, verbal or physical sexual advances, eating inappropriate things, hurting self	Aggressiveness: verbal outbursts, physical threats and/or violence, agitation (other than above)
Anxiety: upset when separated, nervous, sighing, shortness of breath, tense	Pacing, aimless wandering	Diurnal rhythm disturbances: day/night disturbance: repetitive wakening during night for purpose other than toileting, less than 50% of former sleep cycle at night
Elation/euphoria: appears to feel too good, excessively happy	General restlessness, tapping, strange movements	Affective disturbances: tearfulness, depressed mood
Apathy/indifference: less interested in activities, plans	Inappropriate dress/disrobing	Anxieties/phobias: regarding upcoming events, money, future, health, memory, fear of being left alone, other phobias (crowds, travel, darkness, strangers, bathing, etc.)
Disinhibition: acts impulsively, says things that may hurt other's feelings	Handling things inappropriately	
Irritability/lability: impatient, cranky, difficulty coping with delays or waiting	Constant requests for attention or help	
Motor disturbances: repetitive activities, pacing/doing things repeatedly	Repetitive sentences, calls, questions, words	
Nighttime behaviors: awakening at night, up too early or excessive napping	Complaining, negativism, refusal to follow directions	
Appetite/eating: increase or decrease in weight, change in food likes/dislikes	Strange noises, weird laughter or crying	
	Hiding things, hoarding things	
	Screaming	

symptoms of BPSD, their phenomenology, and any precipitants or contributors.

6.3.3.1 Delirium

Due to its fluctuating course and abrupt onset, delirium can be considered in the differential diagnosis of any geriatric patient who presents with MNCD with Behavioral Disturbance *even if* delirium has been ruled out in *prior* work-ups. The rapid onset of delirium and its multifactorial nature, as well as the vulnerability of the geriatric population, all support this approach. The exclusion of delirium cannot rely upon one factor, such as a negative laboratory workup, and it may re-emerge due to a new combination of medications, a change in pathophysiology, or the onset of acute medical conditions, such as sepsis.

The possibility of a pharmacological contribution to delirium in the geriatric patient should prompt a review of the medication regimen, especially appropriate dosing, drug-drug interactions, duration, responses, and adverse effects. Over-the-counter remedies and supplements should be included in the review. Drug-drug interactions can contribute to the onset of delirium due to interference with hepatic metabolism, thereby raising serum levels. Aging can contribute to increased adverse effects due to a reduction in renal clearance. Dehydration, substance/alcohol withdrawal, abrupt discontinuation of medications, and increased anticoagulation (with increased risk of intracranial bleeding), can also precipitate the onset of delirium. Less obvious contributors include occult head trauma, falls, or surreptitious substance use [13].

"Hypoactive," "prolonged," or "mixed" specifiers of delirium may be difficult to identify due to overlapping symptomatology with other conditions, and a delay in recognition following precipitating factors. Delirium may also occur in the context of other psychiatric syndromes, exacerbating the behavioral/psychological symptoms and complicating the assessment. The hypoactive delirium subtype can be misidentified as depression [14] or apathy. Even if ruled out on admission, delirium may remain a possibility during an inpatient hospitalization. The development of newer typologies of delirium offers the clinician a more refined understanding of its pathophysiology and facilitates its early identification [15].

The following screening instruments can help identify the emergence of delirium (Chap. 12: Delirium).

(a) The Montreal Cognitive Assessment (MoCA), which takes 15–20 minutes to administer, detects mild cognitive impairment and has greater sensitivity than the Mini-Mental State Examination (MMSE) for executive and language dysfunction [16]. The typical cutoff score for normal performance is 26 (i.e., 25 and below are considered abnormal—http://www.mocatest.org). If the MoCA is 26 or above, it does not reflect cognitive impairment beyond normal aging. Executive function tests, such as (Trail-Making A&B), Draw-a-Clock, divided attention (Digit Span from WAIS-III) show that geriatric patients are slightly impaired relative to younger populations.
(b) The Confusion Assessment Method (CAM) [17] is a widely used screening tool to identify delirium [18]. It prompts the provider to look for the following features: (1) acute onset or fluctuating course, (2) inattention, (3) disorganized thinking, and (4) altered level of consciousness. The diagnosis of delirium by CAM then requires Features 1 and 2, along with either 3 or 4.
(c) The "A" Test for vigilance and attention is a bedside screening tool used to quickly assess the need of further evaluation for delirium [19].

In DSM-5, subtypes of delirium include: substance intoxication delirium and substance withdrawal delirium, medication-induced delirium, delirium due to another medical condition, and delirium due to multiple etiologies (Chap. 10: Alcohol and Substance Use Disorders: Intoxication and Withdrawal). Specifiers include: (1) Duration: Acute (a few hours or days) versus Persistent (weeks or months) and (2) Activity level: Hyperactive, hypoactive, or mixed. An insidious subtype which can easily be overlooked

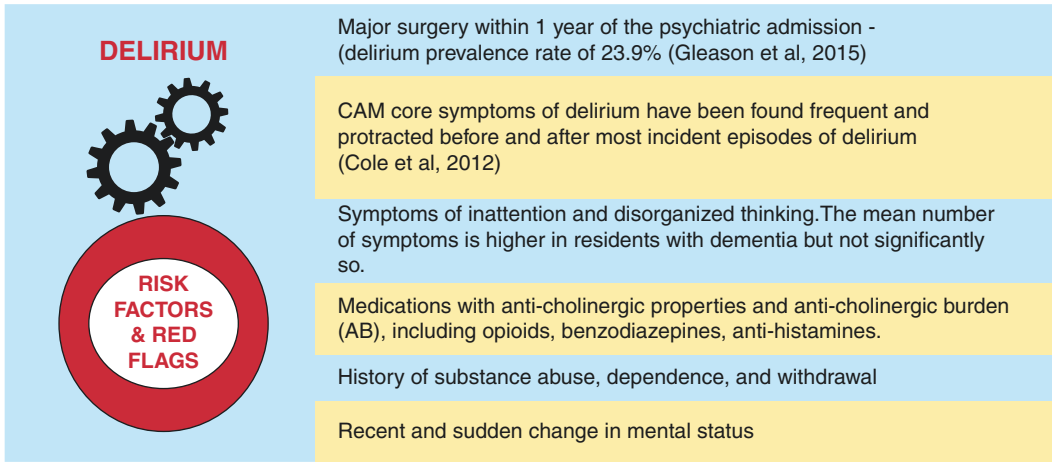


Fig. 6.3 Lists risk factors and red flags to help diagnose delirium

is a delirium with a persistent course, little disruptive behavior, and hypoactive motor activity.

The Confusion Assessment Method (CAM), described above, can detect subtle, or hypoactive, delirium and identify delirium before the full syndrome appears [14, 18]. A study by Cole et al. [18] found that CAM core symptoms of delirium *preceded* 38 (92.7%) episodes of delirium for many weeks and CAM core symptoms *followed* 37 (90.2%) episodes for many weeks. Cole et al. [18] also found that signs of inattention and disorganized thinking were the most commonly seen, with the mean number of symptoms higher in long-term care residents with MNCD, though not significantly. As measured with the CAM, delirium has been associated with prolonged length of stay, discharge to another higher level of care (institutional discharge), and re-admission within 30 days [20, 21]. Delirium also has been correlated with increased incidence of future MNCD [22]. Figure 6.3 lists the risk factors and red flags to help diagnose delirium [23].

6.3.3.2 Pre-Existing Major Mental Illness (PMMI)

The incidence of pre-existing major mental illnesses (PMMI) such as schizophrenia and bipolar disorder is higher between the ages of 18 and 30 than after age 65. The chronic and episodic symptoms of these syndromes which emerge in early life do not usually resolve entirely, and disabling psychiatric symptoms continue into old age. Symptoms of PMMI have become more prevalent in institutional settings for geriatric patients. For example, the prevalence of schizophrenia in nursing home residents has increased from 7.3% to 8.7% from 2011 to the end of 2015. This constitutes a rise of 19% [3].

Frailty, cognitive deficits, and limitations in communication may obscure the recognition of PMMI, especially in the presence of MNCD with behavioral disturbance. Acute mania, for example, may be interpreted as part of the MNCD, instead of a pre-existing bipolar disorder I, resulting in a delay in recommended mood stabilizer treatment [18] (Chap. 3: Pharmacological Overview in Geriatrics). Clues such as a history of psychiatric medications, hospitalizations, disruptions of relationships, homelessness, as well as inability to complete military service, education/training, or other career benchmarks, can provide evidence that disabling psychiatric symptoms may have started early in life, prior to the current presentation.

6.3.3.3 Substance or Alcohol Withdrawal Syndromes

Substance intoxication or withdrawal must be considered, especially for geriatric patients who have been living alone or in unsupervised settings, and for patients who have been prescribed benzodiazepines or opioids. These patients may

have been taking medications inappropriately. Upon hospitalization, subtle or overt withdrawal syndromes and/or delirium can result. The abrupt emergence of perceptual distortions, visual hallucinations, or paranoid delusional symptoms should prompt consideration of a substance withdrawal syndrome or delirium (Chap. 10: Alcohol and Substance Use Disorders: Intoxication and Withdrawal).

6.4 Contributors to Behavioral Disturbance of MNCD

6.4.1 Pain

A cross-sectional study of a large sample of frail, cognitively impaired nursing home seniors found that pain was associated with behavioral and psychiatric symptoms (i.e., verbal agitation, complaining, negativism, repeating sentences, cursing, and verbal aggression) [24]. Researchers studied 352 residents over the age of 65 with a DSM-IV diagnosis of dementia and divided patients into two treatment groups: (1) stepwise treatment of pain vs. (2) usual management. Patients treated for pain effectively showed an average reduction of agitation on the CMAI at weeks 2, 4, and 8. When pain treatment was withdrawn at week 8 in the same intervention group, CMAI scores on agitation worsened. Treatment of pain also significantly improved the overall severity of behavioral disturbances seen in MNCD [25]. Of interest, a separate study found that the specific behavioral symptoms of *agitation and wandering* were *inversely* correlated with pain and *did not correlate with greater severity of pain* [26]. One explanation might be that the increased activity and movements of agitation exacerbate pain, and may result in a tendency of the patient to not move as much.

The assessment of pain should be pursued even in patients who *do not complain* or have limited communication skills. Observational-behavioral pain assessment instruments have been found reliable and valid in patients with MNCD [27]; the Pain Assessment In Elderly Adults with Dementia (PAINAD) Scale is an example of such an instrument [28]. Pain assessment instruments have also been found reliable in determining *non-verbal* expressions of pain. One study found that laypeople and nurses were able to effectively differentiate painful from non-painful situations using these standardized tools; they were able to identify and quantify pain in patients with limited verbal communication ability [14]. Another study found that the assessment of pain by laypeople (under the guidance of health professionals) offered the potential of earlier detection and treatment of pain in older adults with MNCD who live in community settings [29, 30]. Husebo et al. [30] reported that it was possible to discriminate among gradations of pain (i.e., no pain, mild, moderate, severe) within comparable time as other usual clinical observations. Pain behavior and pain intensity in older persons with severe MNCD can also be reliably assessed with the Mobilization-Observation-Behavior-Intensity-Dementia (MOBID and MOBID-2) Pain Scales [31, 32] (Chap. 14: Pain).

6.4.2 Communication Barriers

Perceptual, sensory, speech, and language deficits can impair communication and exacerbate behavioral disturbance in patients with MNCD. Communication barriers include aphasias [33], speech apraxia, dysphasias, dysarthria, and auditory reception impairment. Simple auditory deficits can result in the misidentification of stimuli. Deficits in processing language and expressing language can result in behaviors which are disruptive at best, and self-destructive at worst (Chap. 19: Medical Nursing and Communication Barriers).

6.4.3 Sleep Disturbances

6.4.3.1 Insomnia

Insomnia disorder affects 30–50% of aging adults [34], and an even higher proportion of adults with MNCD, compared with age-matched controls. More than 80% of psychiatric inpatients suffer from some form of chronic insomnia [35, 36]

(Chap. 9: Sleep Disorders, Parasomnias, and Environmental Contributions). Consequences of chronic insomnia may include mood disturbances, fatigue, decline in cognition, decreased quality of life, increased pain, and an increased risk of falls. The quality and quantity of sleep, and behavior *during* sleep can be assessed in an inpatient stay, but at the risk of disrupting the very phenomenon under study. Often one must rely upon polysomnography, caregivers, and/or family for assessments of sleep prior to admission [37].

The Epworth Sleepiness Scale (EPS) can help quantify the daytime impact of loss of sleep but this may need to be gleaned from caregivers [38]. In the inpatient setting, it is important to minimize environmental disruptions of sleep and prevent alterations of the sleep-wake cycle. Figure 6.4 shows some parameters of sleep quality, which can be assessed during hospitalization.

6.4.3.2 Parasomnias

Agitation and/or wandering at night have been associated with probable restless legs syndrome (RLS), which can interrupt sleep and contribute to cognitive deficits. Rose and colleagues studied the impact of parasomnias on BPSD in 59 geriatric patients with a mean age of 79.1 and mean MMSE score of 20.1 [35]. Subjects were assessed by polysomnography for 2 nights; severe cognitive impairment, low apnea-hypopnea index (AHI), and probable RLS were associated with nocturnal agitation behaviors.

Fig. 6.4 Parameters to assess quality of sleep

RLS was identified as the parasomnia most associated with MNCD. In the same study, obstructive sleep apnea (OSA) and periodic limb movements of sleep (PLMS) were *not* associated with agitation at night [35]. Further investigation is warranted to determine if treatment of RLS impacts nocturnal agitation behaviors in patients with MNCD.

Of note, RLS may be difficult to diagnose in this population due to the patient's impaired ability to recall specific symptoms and behaviors. Parasomnias and related disturbances during sleep are also difficult to accurately describe and quantify without direct observation. The CMAI, modified for observers, has been used to identify nocturnal behaviors in patients with MNCD, in addition to pacing, screaming, biting, and restlessness [35]. In the same study of 59 Alzheimer's disease (AD) subjects, a probable diagnosis of RLS was found in 24% of subjects, higher than the 4–11% reported prevalence in the geriatric population [39]. Figure 6.5 summarizes environmental factors and medical conditions which may disrupt restful sleep during an inpatient stay.

6.4.4 Normative Aging

Even without delirium or another neurocognitive disorder, normal cognitive decline of aging is a stressor to be considered in the evaluation of a geriatric patient with behavioral disturbance (Chap. 2: Neuropsychological Assessment). Figure 6.6 summarizes declines in specific cognitive domains that can contribute to frustration and mood disorders, leading to behavioral disturbance [1, 6, 7, 40, 41].

6.5 Treatment Interventions

6.5.1 Non-Pharmacological Interventions

Non-pharmacological approaches are the first line of intervention for MNCD with behavioral disturbance. Several protocols are available but their respective efficacies have not been assessed side-

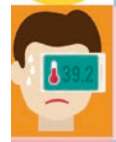
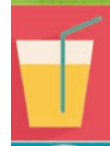
	Contributing Factor	Specific Symptoms and Related Etiology
	Pain	Chronic pain of osteoarthritis, peripheral neuropathy
	Sleep	Parasomnias, (PLMS, RLS), obstructive sleep apnea
	Cardiac	CHF, hypoperfusion, orthopnea
	GI & Urology	Impaction constipation, bowel obstruction, reduced bowl motility, urinary frequency, urinary retention
	Infection	Any infection, UTI
	Blood Glucose	Blood glucose fluctuations & poor oral intake
	Orthostatic changes, autonomic dysfunction	Hypotension when arising, light-headedness
	Injury	Head trauma
	Airway	COPD & poor oxygenation, aspiration due to late meals
	Medication	Drug-drug interactions, discontinuation syndromes, toxic adverse effects
	Substance	Withdrawal syndromes, opioids, alcohol, benzodiazepines
	Fluids	Dehydration, electrolytes, hyponatremia, SIADH
	Vascular	Stroke -Transient Ischemic Attack (TIA)

Fig. 6.5 Environmental and other factors associated with interrupted sleep

by-side, and the choice of which protocol to use often depends upon resources available. [41] With the help of nursing staff, the treating physician or provider may develop a treatment plan based upon caregivers, staff, and family availability/skills. Kales et al. described a systematic approach (D-I-C-E) for first-line, non-pharmacological interventions in steps listed below [42]. Variables which influence the choice of interventions include: the milieu, other patients on the unit, availability of sitters, physical environment, severity of the problematic behavior, support of family, and outcome of prior non-medical interventions (Chap. 18: Psychotherapies and Non-pharmacological Interventions; Chap. 4: Interdisciplinary Roles).

Specific behavioral interventions for caregivers can also be found at the website for the Alzheimer's Association (www.alz.org). Figure 6.7 summarizes the D-I-C-E and the steps in development of non-pharmacological interventions for MNCD with behavioral disturbance.

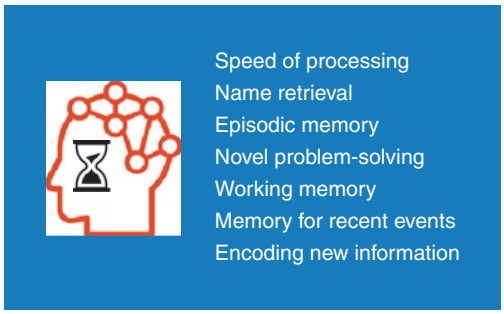


Fig. 6.6 Cognitive domains which can decline with normal aging

If the D-I-C-E protocol or other non-pharmacological approaches and interventions are unsuccessful, the patient with MNCD with behavioral disturbance may continue to disrupt medical care, constitute a risk to self or others, enhance suffering, and diminish her/his quality of life. Under these conditions, psychopharmacological approaches should be considered.

6.6 Pharmacological Interventions

No medications have been approved by the United States Food and Drug Administration (FDA) to treat behavioral disturbance of MNCD, although many have been used. Kales and colleagues remind us to address expectations of patients and their families by emphasizing that certain disruptive behaviors may not respond to medications. Such symptoms include poor attention, rejection/refusal of care, unfriendliness, assaultiveness, repetitive questions or verbalizations, shadowing, and wandering [42].

The patient's capacity to provide informed consent for medications should be assessed. If the patient is found to lack this capacity, informed consent for medications can be pursued from family or surrogates. The process of providing information and the results should be documented (Chap. 5: Legal Aspects). In addition, the risks due to the patient's behavioral disturbance if she or he is *not* treated pharmacologically should *also* be explained to the patient and surrogates [16]. Sometimes, the results of *not* intervening

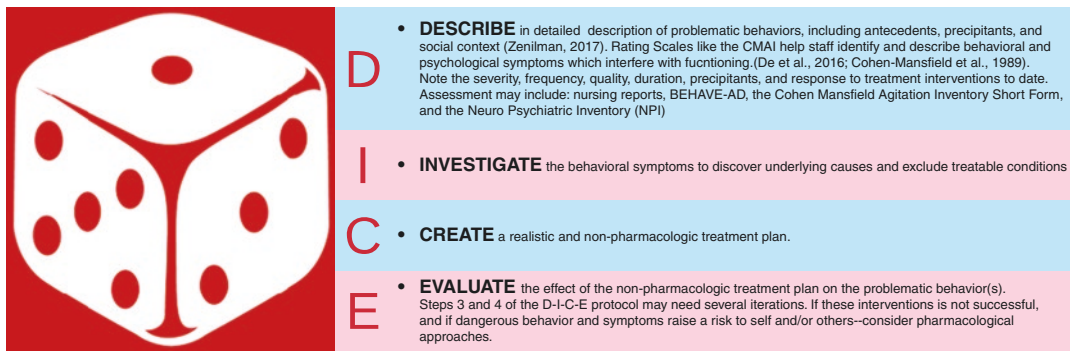


Fig. 6.7 D-I-C-E: Non-pharmacological interventions for MNCD with behavioral disturbance. (Kales et al. [42])

with medications to manage behavioral disturbances can be at least as adverse as the risk from adverse medication effects. If informed consent for medication treatment is not received, and the patient and/or surrogate refuses such treatment, the *informed refusal* should be documented.

6.6.1 Prescribing Principles for MNCD with Behavioral Disturbance

The risks of medications in the geriatric population must be kept in mind, especially in the old-old, those over age 85, and in the context of MNCD and frailty. Physiologic changes with age as well as multiple medications can contribute to serious adverse effects (Chap. 3: Pharmacological Overview). Figure 6.8 illustrates the pitfalls of prescribing in the context of MNCD.

Pesiah et al. [9] have described the development of a tool with which to identify suboptimal prescribing practices for geriatric patients with MNCD. The quality use of medications in dementia (QUM-D) is summarized in Table 6.3; it identifies ten practices to avoid [9].

6.6.2 Step-Wise Strategy of Medication Recommendations for BPSD

Medication recommendations in Table 6.4 are derived primarily from studies of behavioral disturbance in the context of Alzheimer's disease. Table 6.4 shows one step-wise approach for the treatment of MNCD with behavioral disturbance. A review of the literature of prescribing specific medication categories is reviewed in the sections which follow.

6.7 Antipsychotics (AP): General Guidelines

In 2016, the *American Psychiatric Association* (*APA*) published guidelines for the use of antipsychotic agents to treat agitation and psychosis in

Fig. 6.8 Pitfalls of prescribing for the geriatric patient with MNCD

Table 6.3 Recommendations for quality use of medications in dementia (QUM-D)

Quality parameter	Avoid prescribing
Alternatives	If there is no evidence of psychotherapeutic trials or nonpharmacological interventions
Indication	A drug without clear target symptom, or for symptoms not considered targets for psychotropic use by consensual opinion, e.g., wandering, screaming, repeating
Choice of drug	A drug with unsupported evidence base or lacking general consensus in this context, e.g., sodium valproate, chlorpromazine, trifluoperazine, phenobarbitone Use of a drug otherwise contraindicated, e.g., medication with high anticholinergic burden (AB) in a patient with retention/prostatism, or use of medication contraindicated in a patient with high insulin resistance
Consent	If there is no evidence of consent documented from either person themselves or proxy as per hierarchy of NSW Guardianship Act 1987 (i.e., guardian, persons responsible etc.)
Dosage	Doses[a] in excess of best practice guidelines, e.g., Haloperidol > 2 mg [a] Olanzapine > 10 mg [a] Risperidone > 2 mg [a] Quetiapine > 200 mg [a] Use of doses FAR in excess of best practice guidelines, e.g., Haloperidol ≥ 5 mg daily Olanzapine ≥ 20 mg daily Risperidone ≥ 4 mg daily Quetiapine ≥ 300 mg daily
Mode of administration	Depot antipsychotics or long-acting injectable APs
Titration	Rapid upward titration (e.g., usually no faster than every 3 days) – unless emergency Unreviewed – review must be for side effects (frequently, e.g., every 3 days) and efficacy (after a week, or two/three for antidepressants with dose adjustment)
Polypharmacy	>2 Psychotropics simultaneously, including PRN medications if given, and including: sedative/hypnotics such as temazepam, nitrazepam or flunitrazepam, oxazepam, clonazepam, and opioids Use of > 4 psychotropics Use of > 2 psychotropics from same drug class, e.g., × 2 antipsychotics
Toxicity	If there is evidence of adverse effects at a persistent or unacceptable level without review, e.g., drowsiness, falls, EPSE, swallowing difficulties, QT prolongation
Review	Unreviewed continuous use of same drug for >3 months (review must constitute medical review including symptom review +/– trial of discontinuation) or continuous use of drug despite specialist advice or recommendation

Adapted from [9]
EPSE extrapyramidal side effects
Key: [a]Frail elderly specifier. In frail elderly, with several co-morbid medical conditions, lower cap doses may be required and lower doses may be considered "burdensome." For these patients, consider rating "2" for: haloperidol >1 mg; risperidone >1 mg, olanzapine >5 mg, quetiapine >50 mg

MNCD. Neither first-generation antipsychotics (FGA) nor second-generation antipsychotic (SGA) agents have an indication for BPSD, and all carry a black box warning for increased mortality for their use in geriatric patients with dementia. The guidelines recommend that clinicians identify contributing conditions (pain, delirium, etc.) and that the etiology of the MNCD may influence treatment approaches [43] (APA Guidelines) [44]. There is a caution that antipsychotics (APs) should only be implemented when agitation and/or psychosis "…is severe, dangerous, and/or causing significant distress to the patient" [43, 44]. *APA* guidelines furthermore recommend that long-acting injectable antipsychotic medications should not be used unless otherwise indicated for a co-occurring chronic psychotic disorder.

Table 6.5 lists seven recommendations for the use of antipsychotics (APs) to treat agitation or psychosis in patients with MNCD. These recom-

Table 6.4 Stepwise management of MNCD with behavioral disturbance

Steps	Recommendations for treatment of major NCD with behavioral disturbance
Step 1	Assess, rule out/treat delirium Assess for reversible etiology of behavioral disturbance, e.g., pain, constipation, medical illness, sleep deprivation Review medication regimen for anticholinergic burden (AB) Use D.I.C.E. or another method to develop a nonpharmacologic treatment plan
Step 2	If no improvement or minimal improvement, consider addition of cholinesterase inhibitor (donepezil, rivastigmie, galatimine) and/or memantine
Step 3	If no improvement or minimal improvement, consider addition of citalopram titrated to efficacy at no higher dose than 30 mg
Step 4	If no improvement or minimal improvement, add a low dose of a second-generation antipsychotic (SGA), such as risperidone or olanzapine, and titrate to efficacy. Use quetiapine 25–50 mg in LBD or PDD
Step 5	If no improvement or minimal improvement, consider other augmentation strategies (carbamazepine, buspirone, trazodone, etc.)
Step 6	In refractory cases, consider ECT or dextromethorphan-quinidine
Step 7	When behavioral symptoms have stabilized, conduct an inventory of psychopharmacologic agents; consider tapering antipsychotic medications after several weeks of stability

Prior to medication trials: perform and document an assessment of capacity to give informed consent for specific medications. If capacity is *present*, document results and discuss with family, caregivers, other significant others. If capacity is absent, institute a procedure to obtain informed consent from surrogate decision makers

Table 6.5 JAMA clinical guidelines for antipsychotic use for agitation or psychosis

Outside of situations involving an imminent threat to patient or others, APs should be used for agitation or psychosis only when symptoms are severe, dangerous, or cause significant distress to the patient
A clinical response to nonpharmacologic interventions should be reviewed before any nonemergency use of an AP
The AP should be started at a low dose and titrated up to the minimum effective dose tolerated
After a 4-week trial of an adequate dose of an AP, if no clinically significant response is evident, the AP should be tapered and withdrawn
For patients who *do* show an adequate response to AP, an attempt to taper should made within 4 months of drug initiation—unless a recurrence of the target symptoms during the previous taper process
During tapering of an AP, symptoms should be assessed at least monthly, for at least 4 months after medication discontinuation, to seek any sign of recurrence; a risk-benefit analysis should review need for treatment
In the absence of delirium, if nonemergency AP treatment is needed, haloperidol should not be used first line

mendations were also published in a JAMA Clinical Guidelines Synopsis [45].

6.7.1 First-Generation Antipsychotics (FGA)

A systematic review of FGA found little evidence to support FGAs in general as effective in ameliorating BPSD, and no statistical differences have been noted among agents [8]. Specifically, haloperidol has been found useful in the treatment of delirium or aggression in emergent situations. Four clinical RCTs of BPSD have shown haloperidol to have benefit for aggressive behavior and to show some improvement for psychosis, but little improvement in agitation [46]. But haloperidol is not recommended as a first-line, routine agent for agitation or psychosis related to MNCD, according to the *APA* guidelines [43, 44].

6.7.1.1 Adverse Effects of First-Generation Antipsychotics (FGA)

Haloperidol has been associated with the highest mortality risk of antipsychotics in MNCD with behavioral disturbance, in two large retrospective cohort studies [42, 47, 48]. This finding was repeated by Huybrechts and colleagues' cohort study of 75, 445 older adults living in nursing homes, wherein haloperidol demonstrated a hazard ratio of 2.07 (95% CI: 1.89–2.26) compared to risperidone [49]. Given its higher mortality risk, haloperidol is specifically not recommended for BPSD; its use should be reserved for situations in which other medications have not been effective and the patient's behavior is dangerous to self and/or others. However, some authors

have found no reason to believe that other FGAs would be safer than haloperidol [45]. Still, FGAs may play a role in short-term use in the management of delirium [44, 45].

6.7.2 Second-Generation Antipsychotics (SGA)

SGAs are often favored given their lower risk of extrapyramidal symptoms (EPS) and tardive dyskinesia [50]. Large reviews cite that risperidone, aripiprazole (technically a 3rd-Generation Antipsychotic [51], and olanzapine offer a small, but statistically significant, efficacy over placebo for BPSD [8, 52, 53]. The authors also note that the minimum clinically important difference is not known

In the Clinical Antipsychotic Trials of Intervention Effectiveness Alzheimer's Disease (CATIE-AD) trial, 421 *outpatients* with BPSD, across 42 sites, were randomized to risperidone, olanzapine, quetiapine, or placebo [54]. This multicenter trial reported that risperidone and olanzapine demonstrated modestly greater improvement over placebo in measures of anger, suspiciousness, and hostility, while quetiapine did not differ from placebo. The investigators postulated that the latter finding for quetiapine may have been the result of the relatively small doses prescribed (mean quetiapine dose 56.5 mg/day) [54] Sultzer and colleagues found that antipsychotics did not offer significant improvements over placebo in cognition, functioning, care needs, or quality of life; and olanzapine appeared to worsen functioning and withdrawn depression scores [54].

In spite of some evidence in uncontrolled trials, quetiapine does not appear to have significant efficacy in randomized controlled trials (RCT) [55]. One exception, Zhong et al., reported that in a RCT, quetiapine, dosed twice-daily at a total of 200 mg/day was more effective than placebo for agitation, while doses of 100 mg/day were not [56]. Of note, mortality rates were numerically higher in quetiapine groups with relative risk of death of 2.08 (95% CI: 0.61–7.16), but the difference was not statistically significant [56].

6.7.3 Other Adverse Events of Antipsychotics

APs also pose an elevated risk of stroke [8, 57, 58], sudden cardiac death [59, 60], community-acquired pneumonia [61], falls/fractures [62], and a potential for worsening cognition [63]. All factors should be considered in the risk vs. benefit analysis of using APs in this population.

6.7.4 Risk of Death with Antipsychotics

FGA and SGA both increase the risk of death in geriatric patients with MNCD, and it appears that APs have a greater risk of mortality than non-antipsychotic psychotropics [47]. As mentioned, the FDA has issued a black box warning for increased mortality in the elderly with MNCD who take neuroleptics [64]. The mortality risk may vary by the AP. Maust and colleagues conducted a retrospective case-control study of 90,786 veterans older than 65 years with MNCD and reported the following odds ratios of increased mortality and number needed to harm (NNH) of various antipsychotics for users vs. non-users, haloperidol, OR = 3.8%, NNH 26; risperidone, OR = 3.7%, NNH 27; olanzapine, OR = 2.5%, NNH 40; and quetiapine OR = 2.0%, NNH 50, after controlling for relevant risk factors [48]. Quetiapine has been associated with the *lowest* mortality risk among antipsychotics [48], with little to no risk [65] and, in some studies, has been associated with decreased mortality risk [49, 66]. Mortality risks were higher for all antipsychotics included in the study (haloperidol, risperidone, olanzapine, quetiapine) compared to antidepressants (tricyclics and monoamine oxidase inhibitors were excluded) [48].

Providers should use the lowest effective doses of antipsychotics and taper off if there is no response at an adequate dose after a 4-week trial [43] as higher doses are associated with higher mortality risks [48, 60, 66] and there may be a sustained risk of mortality in patients who remain on antipsychotics vs. those who discontinue them [67].

6.7.5 Discontinuation of Antipsychotics

The aforementioned *APA* practice guidelines provide recommendations for the duration of antipsychotic (AP) treatment for MNCD patients who have demonstrated an adequate response: tapering attempts should occur within 4 months of commencement unless the patient has a history of symptom recurrence during previous tapering trials. Patients should be assessed at least monthly during tapering and for four months following antipsychotic discontinuation [43]. Guidelines recommend the lowest effective doses of antipsychotics, and to taper off if there is no response at an adequate dose after a 4-week trial [43, 44].

The Dementia Antipsychotic Withdrawal Trial-Alzheimer's Disease (DART-AD) reported that 165 older adults treated with a neuroleptic, who were randomized to continue or discontinue (given placebo) their antipsychotic, did not demonstrate significant differences in behavioral measures over a 1 year follow-up period [67].

However, in some patients who have demonstrated a response to antipsychotic treatment, tapering may lead to recurrence of BPSD. The Antipsychotic Discontinuation in Alzheimer's Disease (ADAD) Trial reported that patients initially responding to risperidone over 16 weeks of open-label treatment demonstrated two-to-four times greater risk of reemergence of symptoms at 16–32 weeks after risperidone discontinuation vs. risperidone continuation [68]. Patel and colleagues recently published a post hoc analysis of the ADAD Trial, investigating whether particular symptoms from the 12 NPI domains could predict risk of relapse [69]. The study found that patients with baseline hallucinations (particularly auditory) who were randomized to discontinue risperidone demonstrated a higher rate of relapse (76.5%) vs. those who continued risperidone (38.5%) (RR = 1.99, 95% CI = 1.14–3.46, $p < 0.03$), and those with severe baseline irritability/lability who *discontinued* risperidone had a *higher* risk of relapse than those who continued risperidone treatment (HR = 7.78, 95% CI = 2.32–26.07, $p < 0.01$) [69].

Although patients need lower doses of antipsychotics to achieve symptom control and those with lower baseline severity of BPSD may have better success after drug discontinuation [70], patients with severe baseline irritability/lability or auditory hallucinations may have a higher risk of relapse [69]. And although reevaluations of antipsychotic use in nursing homes, combined with social interaction, can help reduce antipsychotic use and mortality, discontinuation of antipsychotics *without* alternative interventions (social interventions, exercise, etc.) may *worsen* BPSD [71].

6.8 Antidepressants

A systematic review of double-blind, placebo-controlled, randomized clinical trials and meta-analyses by Sink and colleagues found that only citalopram was effective in addressing neuropsychiatric symptoms (other than depression) associated with MNCD [8]. In a Cochrane Review of 9 antidepressant trials, sertraline and citalopram were found to improve agitation modestly, compared to placebo in two studies. When compared to haloperidol or placebo, citalopram showed few statistically significant differences in adverse effects [72].

The Citalopram for Agitation in Alzheimer Disease (CitAD) Study was an RCT of citalopram for agitation in patients with probable Alzheimer's disease [73]. The trial was conducted in multiple sites, following *outpatients* over 9 weeks. It found that patients taking citalopram (commenced at 10 mg/day with planned titration to 30 mg/day over 3 weeks) had statistically significant improvement in agitation on validated behavioral scales compared to placebo (psychosocial intervention). The treatment group's caregivers also had decreased distress. The citalopram group had a mean decrease of 1 point on the Mini-Mental State Examination (MMSE) and experienced higher rates of QTc prolongation [73]. In a secondary analysis of the CitAD trial, Leonpacher and colleagues evaluated the efficacy of citalopram on the symptom domains of the NPI. Citalopram (30 mg/day) appeared to decrease delusions (OR = 0.40), anxiety (OR = 0.43), irritability/lability (OR = 0.38),

and demonstrated better control of hallucinations compared to placebo at 9 weeks [74]. But sleep/nighttime behavior disorders were increased in the citalopram group [74].

An *inpatient* RCT to treat agitation in 85 patients with varied forms of MNCD compared citalopram (mean dose of 20 mg/day), or perphenazine (mean 6.5 mg/day), to placebo. Although both perphenazine and citalopram demonstrated improvements on several factors of the Neurobehavioral Rating Scale (NRS), only citalopram demonstrated a statistically significant reduction on the total NRS [75]. Citalopram also improved agitation/aggression and lability/tension factors, although dropout rates (54%) were high often due to lack of efficacy [8, 76].

6.8.1 Cardiac Adverse Effects of Antidepressants

Despite evidence for potential benefits of antidepressants, some antidepressants, including citalopram, prolong QTc length in a dose-dependent manner and can increase the risk of fatal arrhythmias such as Torsades de Pointes (TdP). The FDA issued a black box warning for citalopram and lowered the recommended maximum dose to 20 mg daily for individuals older than 60 years and for those with hepatic impairment or who are poor CYP2C19 metabolizers or taking CYP2C19 inhibitors [77].

Evidence suggests citalopram induces QTc prolongation around 10–20 ms; however the actual risk of TdP may not be clinically significant [78], as a large cohort study by Zivin et al. reported that citalopram doses >40 mg daily did not bear great risk of arrhythmia or mortality compared to doses <20 mg daily [79]. Nonetheless, it is still recommended to discontinue citalopram in individuals with persistent QTc intervals greater than 500 ms and to not prescribe citalopram in patients with hypomagnesemia, hypokalemia, recent myocardial infarctions, or decompensated heart failure, as well as in those with a history of congenital long QT syndrome (FDA website). FDA-cited risks must therefore be weighed with the modest benefits citalopram can offer in this population. The same FDA warnings have not been extended to other SSRIs, including escitalopram [78].

Several antidepressants increase the risk of hyponatremia in the elderly, particularly the SSRIs [80–82]. Incidence rates of hyponatremia may range as high as 15–20% depending on which agent is used and what parameters are used to define hyponatremia [81]. Hyponatremia is most likely to occur in the first 2 weeks of therapy [81, 82]. The proposed mechanism of antidepressant-induced low sodium is thought to be syndrome of inappropriate antidiuretic hormone secretion (SIADH) [81, 82]. Physicians and other providers are advised to monitor serum sodium levels, particularly early in the course of antidepressant treatment [81, 82]. Risk factors for hyponatremia in patients taking antidepressants include older age, female, concomitant diuretic use, low body mass index (BMI), and baseline low-normal plasma sodium levels [80].

Earlier studies found evidence that antidepressants help to improve depression in individuals with dementia [83], but more recent meta-analyses and systematic reviews suggest inadequate statistically significant evidence [84–88]. Sepehry and colleagues encourage more studies, and point out previous trials have heterogeneous criteria for depression and outcome measures [140]. Nonetheless, some guidelines recommend a trial of an antidepressant to target depressive symptoms in the context of dementia—after a careful risk-benefit assessment [89]. In a 2011 editorial, Lenze offers guidance to clinicians in treating depressive symptoms in those with dementia [90].

6.8.2 Fall Risk Related to Antidepressants

Falls are the fifth leading cause of death among older adults and may contribute to fractures, head trauma, and institutionalization [91]. Antidepressants appear to increase fall risk in the elderly [92] with odds ratios of 1.6–1.9 depending on the study [90]. Whether secondary to orthostasis, sedation, or decreased postural con-

trol [92], antidepressant-related falls are of particular concern in the elderly, given that around 10–15% of falls cause major injuries in this population [93]. In a retrospective study comparing outcomes of post fall proximal femoral fractures, fractures acquired during *inpatient* stay settings had worse outcomes compared to outpatient [94]. Beyond psychotropic medications, other risk factors for falls include gait instability, agitated confusion, urinary incontinence and frequency, and a history of falls [94, 95].

6.8.3 Discontinuation of SSRIs

The risk of discontinuation of SSRIs has also been noted. Bergh and colleagues reported in their randomized clinical trial of 128 individuals with Alzheimer or vascular MNCD that discontinuation of SSRI antidepressants was found to precipitate depressive symptoms even in individuals without a pre-existing depressive disorder compared to those who continue therapy [96].

6.9 Mood Stabilizers

Evidence of benefit for antiepileptic drugs (AEDs) in BPSD is sparse. In 2013, Seitz and colleagues published a systematic review of pharmacological interventions for BPSD of MNCD, including four RCTs investigating anticonvulsants (one study on carbamazepine, two studies on valproic acid derivatives, one study on oxcarbazepine). Only carbamazepine (mean dose 300 mg/day) demonstrated modest, but statistically significant, improvements on neuropsychiatric scales compared to placebo in the long-term care setting [97], which was also suggested in another small trial [98].

Valproic acid has demonstrated little efficacy as a monotherapy in RCTs investigating the treatment of BPSD of MNCD [99, 100], although uncontrolled trials suggest that some individuals had decreased agitation with serum levels between 40 and 60 mcg/mL and relatively low doses (i.e., 7–12 mg/kg per day) [101]. Dolder et al. suggested that valproic acid may help augment other agents in the management of BPSD of MNCD. A multicenter, RCT of flexible dose valproic acid (target dose of 10–12 mg/kg) in 313 patients with moderate Alzheimer's disease who had not yet experienced agitation or psychosis found that valproic acid did not appear to prevent the emergence of agitation/aggression and psychosis compared to placebo, had significant side effects, and was associated with greater brain atrophy [100].

Although small case series have shown modest benefit of gabapentin and there is modest evidence for topiramate and lamotrigine, more RCTs are needed to investigate their benefit in this population [102, 103].

In a case series of six patients with MNCD (three AD, three FTD), lithium improved agitation/aggression at doses of 300–600 mg daily, and the authors suggest that lithium may also play a role in augmenting other agents when the latter are either ineffective or unable to be increased due to side effects [104]. Currently, lithium is being investigated for the treatment of BPSD in Alzheimer's disease and frontotemporal dementia (Clinical Trials.gov; [102]).

The majority of mood stabilizing agents require frequent laboratory monitoring, a disadvantage to their use in the geriatric population. Hyponatremia and agranulocytosis with carbamazepine, thrombocytopenia and hepatotoxicity with valproate, and renal, thyroid and cardiac functions with lithium, all need attention in the geriatric population [105].

6.10 Cholinesterase Inhibitors and Memantine

There is modest evidence that cholinesterase inhibitors can delay of onset of BPSD [106, 107]. Cholinesterase inhibitors and memantine do not show robust evidence of benefit for agitation associated with MNCD [108].

Data are mixed regarding the efficacy of cholinesterase inhibitors for other neuropsychiatric symptoms. Howard and colleagues reported that donepezil 10 mg daily demonstrated no statistically significant improvement in a 12-week RCT

of 272 patients with Alzheimer's MNCD compared to placebo [107]. In an RCT from three centers in England, Burns et al. found no differences in Alzheimer's disease-related agitation between aromatherapy (massaged into hands, arms twice daily), donepezil (started at 5 mg daily, increased to 10 mg daily after 1 month), and placebo at 4 and 12 weeks. Of note, the placebo group and donepezil group each had therapeutic hand massage with placebo lotion perhaps accounting for the 18% and 37% improvement in Pittsburgh Agitation Scale (PAS) and NPI respectively across all groups, supporting the therapeutic benefits of hand massage and touch that all groups received [109].

In 2015, Wang and colleagues published a systematic review and meta-analysis of RCTs with several pharmacological agents (including cholinesterase inhibitors) vs. placebo effects on individuals with Alzheimer's disease, using NPI inventories as outcome measures [110]. A subgroup analysis excluding trials with estimated SDs demonstrated that only galantamine (−0.13, 95% CI: −0.22 to −0.03) had a modest but significant improvement in NPI scores, while donepezil did not (−0.09, 95% CI: −0.27 to 0.08). Of note cholinesterase inhibitors increased risk of adverse effect-related dropouts (2.25 95% CI: 1.53–3.26) [110].

Rodda and colleagues reported on the efficacy of donepezil, rivastigmine, and galantine compared to placebo in their systematic review of 14 RCTs for BPSD in possible/probable Alzheimer's disease [111]. Three trials found small but statistically significant benefits in neuropsychiatric measures compared to placebo (two of nine donepezil trials, one of three galantamine trials, and no rivastigmine trials); however authors recognize that the interpretation of these results may be limited by methodological considerations [111]. Trinh and colleagues, in a systematic review of 29 RCTs looking at the efficacy in cholinesterase inhibitors, found an improvement of 1.72 on the NPI (95% CI 0.87–2.57) in six trials and 0.03 points on the ADAS-non-cognitive scale (95% CI 0–0.05 points) in ten trials [112].

Other authors have noted that the positive findings for cholinesterase inhibitors may not be clinically significant, given only modest improvements in ADL/IADL measures [113]. Klein and Newton recommend that it is reasonable to consider a trial of a cholinesterase inhibitor after weighing the risks and benefits tailored to an individual patient [113]. Of note: overstimulation of muscarinic and nicotinic receptors can lead to bradycardia and precipitate syncope, worsening heart block, as well as increased nausea, vomiting, exacerbations of peptic ulcer disease, and risk of bronchoconstriction in reactive airway disease. Other serious side effects may include lowered seizure threshold and, paradoxically, neuropsychiatric symptoms [114].

Sink and colleagues identified two RCTs of memantine (titrated to 20 mg daily), over 24–28 weeks, for the treatment of BPSD in moderate to severe Alzheimer's disease, with conflicting results [8]. Maidment and colleagues found a very small, but significant, improvement of 1.99 on the NPI scale (95% CI –0.08 to –3.91: $p = 0.041$), with memantine treatment, in a 2008 meta-analysis of Alzheimer's disease patients [115].

Cummings and colleagues published a post hoc analysis comparing memantine vs. placebo for BPSD of Alzheimer's disease in patients already taking donepezil. The authors found memantine reduced agitation/aggression, irritability, and appetite/eating disturbances on the NPI, compared to placebo. Patients with higher agitation/aggression at baseline demonstrated significant reductions when treated with memantine over placebo and, interestingly, those with little baseline agitation/aggression had significantly lower emergence of these symptoms if treated with memantine [106]. In a post hoc analysis of three RCTs of moderate to severe Alzheimer's disease patients with baseline agitation/aggression or psychosis, memantine demonstrated significant improvement in the neuropsychiatric symptom cluster of NPI scores over placebo at all time points and particularly in agitation/aggression (55.3% vs. 43.1% at week 12, $p = 0.011$; 61.0% vs. 45.0% at week 24/28, $p < .001$) [116]. A more recent systematic review and meta-analysis (including eight RCTs of 20 mg/day of memantine) of pharmacological treatment of Alzheimer's disease did not show statistically significant improvements of NPI scores with memantine over placebo (95% CI −0.27 to

0.03) [110]. McShane and colleagues' [117]. Cochrane review reported that memantine therapy appears to lower the likelihood of developing agitation in dementia, albeit with a small effect size Number Needed to Treat (NNT) of 17 and may not effectively treat neuropsychiatric symptoms which are already present [117].

6.11 Other Agents

6.11.1 Buspirone

A dearth of randomized clinical trial data is available about the use of buspirone in MNCD. However, given its relatively benign side effect profile, the drug has been used as either as a monotherapy or augmenter. A case report discussed the success of buspirone (15 mg BID) in augmentation of trazodone 300 mg daily, citalopram 40 mg daily, olanzapine 10 mg QHS in improving severe anxiety and related agitation in a 69-year-old woman with Alzheimer's MNCD [118]. Holzer and colleagues used buspirone (titrated to 10 mg BID) successfully as a monotherapy for aggression in an 85-year-old man with mixed Alzheimer's and vascular MNCD [119]. Tiller et al. reported buspirone's benefit (dosed 5 mg TID) on agitation, disinhibition, jealousy in an 80-year-old man with vascular dementia [120]. Buspirone offers the advantage of having little risk of EPS and sedation compared to other agents [119].

6.11.2 Trazodone

Martinón-Torres et al. published a Cochrane review of two trials of trazodone for agitation in MNCD. One study used a parallel-group study of individuals with Alzheimer's disease and the other used a cross-over design for patients with frontotemporal MNCD. The studies were of 16 and 6 weeks duration respectively and comprised 104 patients, using trazodone from 50 mg to 300 mg daily. Trazodone did not differ from placebo in terms of statistically significant benefits for behavioral symptoms of MNCD nor clinical impression of change, and did not differ in side effects [121]. In a Brazilian open-label, retrospective study of trazodone for sleep disorders in MNCD (64% with Alzheimer type), trazodone (50–100 mg QHS) demonstrated 65.7% effectiveness, assessed on the NPI (improving sleep complaints and reduction in the exhaustion of the caregivers) [122]. Trazodone improved NPI scores compared to placebo ($p = 0.028$) in a French randomized, double-blind, placebo-controlled cross-over study of 26 patients with frontotemporal MNCD [123]. Sultzer and colleagues compared haloperidol (mean dose 2.5 mg/day) and trazodone (mean dose 218 mg/day) in a trial, finding that each improved overall behavioral scales in MNCD, although trazodone was better tolerated. The authors noted that heterogenicity of MNCD diagnoses and lack of a placebo arm were weakness of the study [124].

6.11.3 Methylphenidate

Apathy, a common symptom of MNCD, can have significant implications on caregiver burden, activities of daily living, as well as mortality, and appears to be one of the most lingering behavioral disturbances of MNCD [125, 126]. Many experts recommend cholinesterase inhibitors first, particularly if they have not been tried, as they have shown benefit for apathy in several studies [127]. However, a review by Harrison and colleagues notes that previously reported benefits of cholinesterase inhibitors and memantine have not been replicated in more recent studies [128]. Short duration trials have demonstrated modest benefit of methylphenidate on some, but not all, apathy scales for individuals with Alzheimer MNCD. Padala and colleagues recently published a 12-week trial with male, community dwelling veterans (mean age 77 years), demonstrating that methylphenidate titrated to 10 mg BID showed significant improvement over placebo for apathy as well as cognition, depression, functional status, and caregiver burden [126]. Clinicians should monitor for sleep disturbances, appetite suppression, and weight loss. Caution must be exercised in patients with significant cardiovascular disease or history of arrhythmias.

6.11.4 Prazosin and Propranolol

Medications that modulate the adrenergic system such as propranolol and prazosin [70] may be of benefit in some patients, as some researchers propose there is noradrenergic dysfunction in Alzheimer's disease [129]. Case reports and case series have demonstrated the merits of propranolol (from 10 mg to 20 mg to several hundred milligrams in divided doses) as an alternative or adjunct to more conventional therapies [130–132]. One small randomized trial using adjunctive propranolol (mean 106 mg/d ± 38 mg/d) in nursing home patients with probable or possible Alzheimer's disease demonstrated statistically significant reductions in agitation/aggression and anxiety on the NPI compared to placebo [133]. Summers provides a thoughtful dose titration schedule for propranolol [131]. Prazosin (mean dose 5.7 mg/day ± 0.9 mg/day) outperformed placebo on NPI, BPRS, and CGIC scales in a study of nursing home and community dwelling individuals with Alzheimer's disease [134]. In addition to caution in asthmatic/reactive airway disease and diabetic patients with propranolol, heart rate and blood pressure must be monitored, especially in frail patients with advanced age or orthostatic hypotension [130, 131, 133].

6.12 Novel Treatments and Future Directions

There are several ongoing trials investigating the efficacy of cannabinoids, lithium, and analgesics for neuropsychiatric symptoms associated with Alzheimer's disease. Case reports, retrospective cohorts, and small trials show promise that dronabinol may decrease scores on validated behavioral scores [102], and currently an RCT of nabilone, cannabinoid receptor type 1 (CB1), and cannabinoid receptor type 2 (CB2) agonist is being investigated (Clinical Trials.gov).

Dextromethorphan-quinidine, approved for the treatment of pseudobulbar affect, demonstrated statistically significant reductions in NPI scores in a 10-week, multicenter, double-blind RCT for agitation in Alzheimer's disease with reduced caregiver burden, compared to placebo. The medication was tolerated relatively well and the treatment group had low rates of dizziness and diarrhea, findings consistent with those observed in trials for pseudobulbar affect. The medication combination did not appear to have resultant cognitive impairment, sedation, or worrisome QTc prolongation [6, 7]. Of note, stable dosages of antidepressants, antipsychotics, hypnotics, and cognitive enhancers were allowed in the trial, and future study may clarify if dextromethorphan-quinidine has benefit as an augmentation to these agents, or if it is effective as a monotherapy. Costs may be prohibitive at this time and more RCTs of longer duration are needed to confirm these findings and clearly establish clinical benefit.

Pimavanserin is an antipsychotic agent that was FDA approved in 2016 to treat psychotic symptoms associated with Parkinson's disease. Pimavanserin acts as a 5HT2A inverse agonist [135, 136]. Its minimal dopamine (D2) receptor antagonism appears to minimize the extrapyramidal side effects typical of other antipsychotic agents. There is little evidence of this agent for the treatment of MNCD with Lewy bodies. Currently, pimavanserin and brexpiprazole are being investigated in their role in treating BPSD of Alzheimer's disease [102] (ClinicalTrials.gov). Ballard and collegues, in an RCT of pimavanserin vs. placebo in nursing-home dwelling Alzheimer's patients, showed initial benefit on the NPI-nursing home (NPI-NH) psychosis scores at week 6. Their early improvements were no longer significant at week 12 [137].

6.13 Neuromodulation

Transcranial magnetic stimulation (TMS) may aid the cognition of those with Alzheimer's disease [138]; however there is little evidence of this intervention for BPSD. Electroconvulsive therapy (ECT) may hold promise in treatment-refractory BPSD [139]. Burton and colleagues recently published a small pilot study of nine patients (six received ECT) with BPSD, demonstrating efficacy and safety of ECT, but without statistically significant improvement over the

non-treatment group, though the authors contend the latter finding may be secondary to a small sample size [140]. A recent systematic review of 17 studies of ECT for BPSD (no RCTs) by van den Berg and colleagues reported that clinically significant improvements could be attained in treatment-refractory agitation and aggression in MNCD. The authors recommended more substantiation via prospective trials [141].

6.14 Etiological Specifiers of Neurocognitive Disorder Subtypes and Potential Interventions

Newer neuroimaging technologies have made progress in delineating relevant neural circuits and networks which may be associated with subtypes of MNCD, but research has not yet translated its findings into medication recommendations which can be relied upon to improve specific behavioral disturbances of MNCD [108]. Although diagnosing etiological specifiers, or subtypes, of MNCD is challenging in clinical practice, the process can nonetheless improve observational skills, and may aid in the future development of more individualized interventions. A large number of RCTs focus upon treatment of BPSD in Alzheimer disease, yet future studies of specified MNCD subtypes may help clinicians choose medications with a greater chance of success and/or reduced risk of harm [142].

6.14.1 Vascular Disease (VD)

In one multicenter trial, BPSD were reported in 92% of vascular MNCD patients, with apathy (65%) being most prevalent, followed by depressive symptoms (45%), irritability (42%), and agitation/aggression (40%) [143]. The same study reported that patients with *small-vessel* (as determined by neuroimaging) vascular MNCD had more apathy, aberrant motor behavior, and hallucinations than patients with large-vessel MNCD [143]. Most guidelines do not recommend cholinesterase inhibitors or memantine in vascular MNCD as these agents do not carry consistent evidence in the literature [108]. In addition, benefits of these agents on global functioning, ADLs, and behavior are inconsistent at best [144]. One small, open-label, prospective study looked at 75 patients with vascular or mixed-type MNCD with associated BPSD, treated with risperidone (1.69 mg/day ± 0.7 mg at end-point) for up to 6 months. Subjects demonstrated improvements on mean NPI scores compared to baseline, with a favorable side effect profile [145]. There is a dearth of literature on pharmacological treatments of BPSD in vascular MNCD, and treatment approaches targeting the associated neuropsychiatric profiles of vascular lesions (or patterns of insults) remain to be elucidated in further research [143]

6.14.2 Lewy Body Disease (LBD) and Parkinson's Disease

Lewy body-related MNCD has a significant deficit in acetylcholine neurotransmission; therefore it is theorized that cholinesterase inhibitors may be effective in targeting cognitive and behavioral symptoms, as demonstrated in several trials of cholinesterase inhibitors for Parkinson's disease dementia (PDD) [146]. Rivastigmine (dosed up to 12 mg/day) and donepezil (dosed at 5–10 mg daily) have demonstrated statistically significant improvement in behavioral symptoms of LBD in two trials [147, 148].

Although theoretically these agents could worsen Parkinsonism, this is not common in clinical practice, and the agents are generally tolerated [149]. Patients may experience worsening of tremor with these agents and have similar side effects to the Alzheimer population. A meta-analysis by Matsunaga and colleagues of 17 RCTs ($n = 1798$) reported statistically significant improvements of cognitive function, behavioral disturbances, activities of daily living, and global functioning in patients with Lewy body disorders (such as Parkinson's disease, Parkinson's disease dementia, and dementia with Lewy bodies), treated with cholinesterase inhibitors over con-

trols [150, 151]. Of note, adverse events which led to discontinuation of cholinesterase inhibitors was higher in the treatment group (RR = 1.59, NNH = 20) [150, 151]. Clinicians should favor daytime dosing of these medications as they may increase vivid dreams [149]. Although memantine showed superiority over placebo in clinical global impression of change, it did not show statistically significant effects on MMSE scores, NPI scores, or ADL scores in a systematic review and meta-analysis [150, 151].

For severe psychotic symptoms that are not responsive to dose reductions of dopaminergic agents or cholinesterase inhibitors, judicious use of second-generation antipsychotics (SGAs) may benefit. While quetiapine appears to lack statistically significant efficacy in PDD, it may have benefits in patients with LBD, as seen in a case series [152]. A small RCT investigating quetiapine in LBD, PDD, and Alzheimer's disease with parkinsonian features failed to show significant improvements in psychosis and agitation; however authors admit that an unexpectedly large placebo effect, inadequate dosage (mean 120 mg/day), and inadequate power may have contributed to lack of demonstrable benefit [153]. These findings are supported by a systematic review of seven RCTs (n = 241) that reported no statistically significant benefit of quetiapine (mean dose 103.0 mg) [154]. Importantly, Desmarais and colleagues note that there was no significant worsening of motor symptoms and that heterogeneity was high due to inclusion criteria. In an RCT, neither citalopram nor risperidone demonstrated significant improvement in LBD with behavioral symptoms [155].

Clozapine may decrease agitation in PDD [156] and has been found effective for targeting drug-induced hallucinations and delusions in PD [157]. But there is a dearth of clinical trials investigating clozapine in LBD [152]. In addition to monitoring for neutropenia and constipation in LBD patients on clozapine, physicians must monitor for the severe neuroleptic sensitivity reactions [158].

In 2016 the 5-HT2A inverse agonist pimavanserin was FDA approved for Parkinson's disease psychosis and has demonstrated efficacy for hallucinations and delusions over placebo without adverse effects on motor symptoms or orthostasis [159]. Future investigation for the use of pimavanserin in LBD is needed [156].

6.14.3 Frontotemporal Lobar Dementia

The mainstay of treatment for FTD involves behavioral strategies; however SSRIs have modest evidence in small trials of lowering levels of stereotypical behaviors, impulsivity, disinhibition, and disordered eating associated with FTD [160, 161]. In a small randomized, double-blind, placebo-controlled cross-over study, Lebert and colleagues reported that trazodone was statistically superior to placebo in lowering NPI scores in FTD [123]. Risperidone, olanzapine, and aripiprazole have shown some positive results and may play a role in certain clinical situations; however evidence from controlled studies is lacking [161]. The cognitive enhancers—cholinesterase inhibitors and memantine—have not shown consistent benefit [160, 161], and the former may actually worsen behavioral symptoms [162]. Novel treatments, such as intranasal oxytocin, may show promise to address behavioral symptoms and emotional recognition deficits associated with FTD, but require further study [163, 164].

Sexually inappropriate behaviors (SIB) are challenging symptoms to target, and affect individuals with FTD, as well as other subtypes of MNCD. In addition to behavioral interventions, medications may be needed. Unfortunately there are no practice guidelines, and few, if any, randomized clinical trials for the treatment of SIB [165]. Clinicians, therefore, are left to use a trial-and-error pharmacological approach.

Case studies and series have reported some benefit from antidepressants and SSRIs, which are often considered first-line [166]. Various other agents have been used in published case reports, including anticonvulsants, neuroleptics, and hormonal agents [167]. Certain medications in the latter category may increase risk of venous thromboembolism, in addition to raising ethical concerns; some argue they are tantamount to

"chemical castration" [166]. Thus, communication with patients and their families regarding the risks, benefits, alternatives, and rationale for such agents is imperative [166]. Researchers are working on a standardized definition of SIB, which may allow for future trials that target specific symptoms of SIB [168].

6.15 Summary

Behavioral disturbances and psychiatric symptoms of MNCD are prevalent within the growing geriatric population, and are even more disruptive to patients and caregivers than the cognitive deficits alone. Non-pharmacological approaches are first-line, with pharmacological treatment as the second-line. But medications for these behaviors and symptoms cannot be recommended with any certainty of success over time, nor without any risk of adverse effects. Although various guidelines are available, no consensus has yet emerged regarding specific evidence-based strategies, medication combinations, or an approach to the treatment for MNCD with behavioral disturbance [169].

Because BPSD may lead to inpatient hospitalization, and effective treatment cannot be assured, the inpatient evaluation should include an attempt to identify, rule out, and treat conditions or etiologies which may contribute to the behavioral and psychiatric symptoms. In addition, A step-wise, rational, strategy of non-pharmacological interventions should be developed. If medications are needed, prescribing practices should incorporate principles which minimize known risks of prescribing for geriatric patients, such as anticholinergic burden (AB).

An advantage of inpatient hospitalization is that laboratory and other studies, subspecialty consultation, and the basic components of a complete work-up, can be obtained efficiently. In addition, the controlled inpatient environment, along with trained staff, can observe the patient 24 hours a day, to glean accurate information about precipitants, interventions, and other details of the disturbed behavior. Individualized treatment interventions can then be developed. Essential aspects of BPSD inpatient management include involvement of family and caregivers in decision-making, development of non-pharmacological interventions, and informed consent. If medication interventions are needed, the inpatient unit can foster an alliance around potential benefits as well as the risks, so that treatment can proceed with a realistic understanding of the limitations.

Take-Away

- Determine the course, onset, precipitants, and context of the behavioral disturbance and psychiatric symptoms.
- Document baseline and follow-up ratings of behavioral disturbances with a reliable instrument.
- Rule out and treat delirium.
- Obtain history of prior interventions and outcomes.
- Review/revise medication regimens which have anticholinergic burden (AB), adverse effects, and drug-drug interactions.
- Identify and address conditions which may contribute to behavioral disturbance, such as pain, constipation, urinary retention, and sleep disruption.
- Identify and treat exacerbations of pre-existing major mental illnesses (PMMI) and acute substance intoxication/withdrawal.
- Develop non-pharmacological intervention strategies based upon the D-I-C-E model or a similar approach.
- Assess the patient's capacity to give informed consent.
- Document informed consent for antipsychotics, antidepressants, and other psychotropic medications—from the patient, if capacity is present.
- Document informed consent for antipsychotics, antidepressants, and other medications from surrogate decision-makers and family members—*whether or not* the patient has been formally adjudicated to lack capacity.

- Adhere to the seven APA recommendations for the use of antipsychotics in the treatment of agitation or psychosis.
- Consider prescribing principles of the quality use of medications in dementia (QUM-D) tool.
- Avoid medications with little evidence of benefit, especially those with significant risk, such as benzodiazepines.

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7 Acute Medical Events: Falls, Seizures, CVAs, Urinary Retention, Cardiac Events, Hypotension, SIADH, Dehydration, and Infection

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7.1 Introduction

Systemic illness and psychiatric morbidity have a significant impact on each other and overlap often in geriatric populations [1, 2]. Geriatric psychiatry inpatients have on average 5.6 medical diagnoses [3]. A high prevalence of systemic comorbid complexity and frailty in this population is multifactorial, partially related to increased life expectancy. Often these comorbidities are stable on admission; but geriatric patients may decompensate during their inpatient stay for several reasons. Patients with psychiatric illness are at higher risk of systemic comorbidity and avoidable mortality than the general population [4]. They have greater cardiovascular risk, due in part to higher rates of smoking, obesity, and hypertension, and have poorer access to primary care, diabetes care, and cardiovascular procedures [4]. There is an additional increased risk of disease-drug and drug-drug interactions, due to age-related changes in pharmacokinetics and pharmacodynamics, and greater numbers of baseline medications. Systemic illness is also associated with increased risk of mental health issues due to factors related to the underlying mechanism of the illness (e.g., Parkinson's disease), psychotropic effect of medical therapies (e.g., corticosteroids), as well as the psychosocial burden of the systemic condition [5].

System-of-care issues include pressures to shorten length of stay in acute medical settings, limited availability of step-down convalescence units, and limited access to psychiatry consultation/liaison services. As a result, the case mix of patients admitted to inpatient geriatric psychiatry units is becoming more medically complex and requires more intensive management of systemic issues [1]. Other factors that impede the overall health of patients with psychiatric illness include self-neglect, poor adherence to medical treatment and to modification of risk factors, impaired communication and social skills, denial of physical illness, lack of integration of medical and psychiatric care, and stigmatization of mental illness among other healthcare providers [5, 6]. All of these factors contribute to the treatment gap between those with, and those without, psychiatric illness [7].

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The increasing frailty and medical complexity of the geriatric population underscore the need for healthcare providers to *anticipate* common acute medical scenarios. The following case vignettes illustrate the need for urgent assessment of the patient's medical stability in the context of medical history, risk factor profile, and medication history—with attention to changes that could precipitate morbidity. The level of medical acuity accepted and managed in psychiatry units varies widely [1]. Hence, understanding the policies and procedures on any inpatient unit, and appropriate referral pathways, is always helpful.

The three case vignettes below highlight typical presentations of acute medical events, and include comorbidities and complications prevalent in a geriatric population, such as falls, polypharmacy, urinary retention, stroke, seizures, and cardiac events. Other common acute medical events among geriatric patients, though not fully discussed here, include airway obstruction, acute infection, deep vein thrombophlebitis, and pulmonary embolism.

Specific recommendations for management of medical issues are beyond the scope of this text. What follows instead are guidelines to initial steps in the medical assessment of geriatric psychiatry inpatients, using examples of common acute medical events. Figure 7.1 summarizes the assessment of sudden adverse medical events in geriatric inpatient.

7.2 Falls and Related Issues

7.2.1 Vignette 1

An 85-year-old female, with a history of severe depression, was admitted to inpatient psychiatry after her family expressed concerns about excessive sleeping, minimal intake, and self-neglect. Past medical history included hypertension, diabetes mellitus, osteoporosis, overactive bladder, and myopia. She lived alone in an apartment and was previously independent of all her ADLs (Activities of Daily Living). Her daughter helped with IADLs (Instrumental Activities of Daily Living), including finances and shopping. The patient managed her own current medications: doxazocin 6 mg daily, hydrochlorothiazide 25 mg daily, aspirin 81 mg daily, atorvastatin 40 mg at night, glyburide 5 mg daily, venlafaxine 37.5 mg daily, trazodone 50 mg at night, tolteradine 2 mg twice daily, alendronate 70 mg weekly, and vitamin D 2000 iu daily.

Three days after admission, the patient became disorientated and was wandering. Over the next 2 weeks she fell three times, sustaining bruises to her left hip and both knees. A nurse witnessed one fall in which the patient tripped over a walker at the foot of her bed. There was no loss of consciousness. The family complained of negligence to the hospital administration and retained a prominent law firm. The attending psychiatrist was subpoenaed to testify at deposition (Chap. 5: Legal Aspects).

Physical examination after her first fall: heart rate was 55 bpm, blood pressure 95/50 mm Hg which fell to 70/40 mm Hg after standing for 3 min, temperature 37.0 C (98.6 F), respiratory rate 18/min, and oxygen saturations 97% on room air. Her weight was 47 kg (103.6 lb) with a body mass index of 18 kg/m^2. Cardiovascular and respiratory examinations were unremarkable. Abdominal exam revealed a palpable bladder. Bladder scan: a post-void residual of 800 ml. Neurological examination was not localizing except for proximal weakness in all four limbs. She struggled to stand from a chair without using her arms to push up. She was unable to stand on one leg or tandem stand for longer than 5 s. With her walker, she had a slow and steady gait with a Timed Up and Go (TUG) time of 20 s.

7.2.2 Discussion

While about a third of seniors fall annually [8], the incidence of falls is three to four per 1000 patient days in the hospital setting. Fall risk is estimated to be higher in inpatient geriatric psychiatry units [9]. In one study 84 out of 144 inpatients fell (over 58%); significant predictors included cardiovascular disease, psychosis, and use of antipsychotic medications. These falls

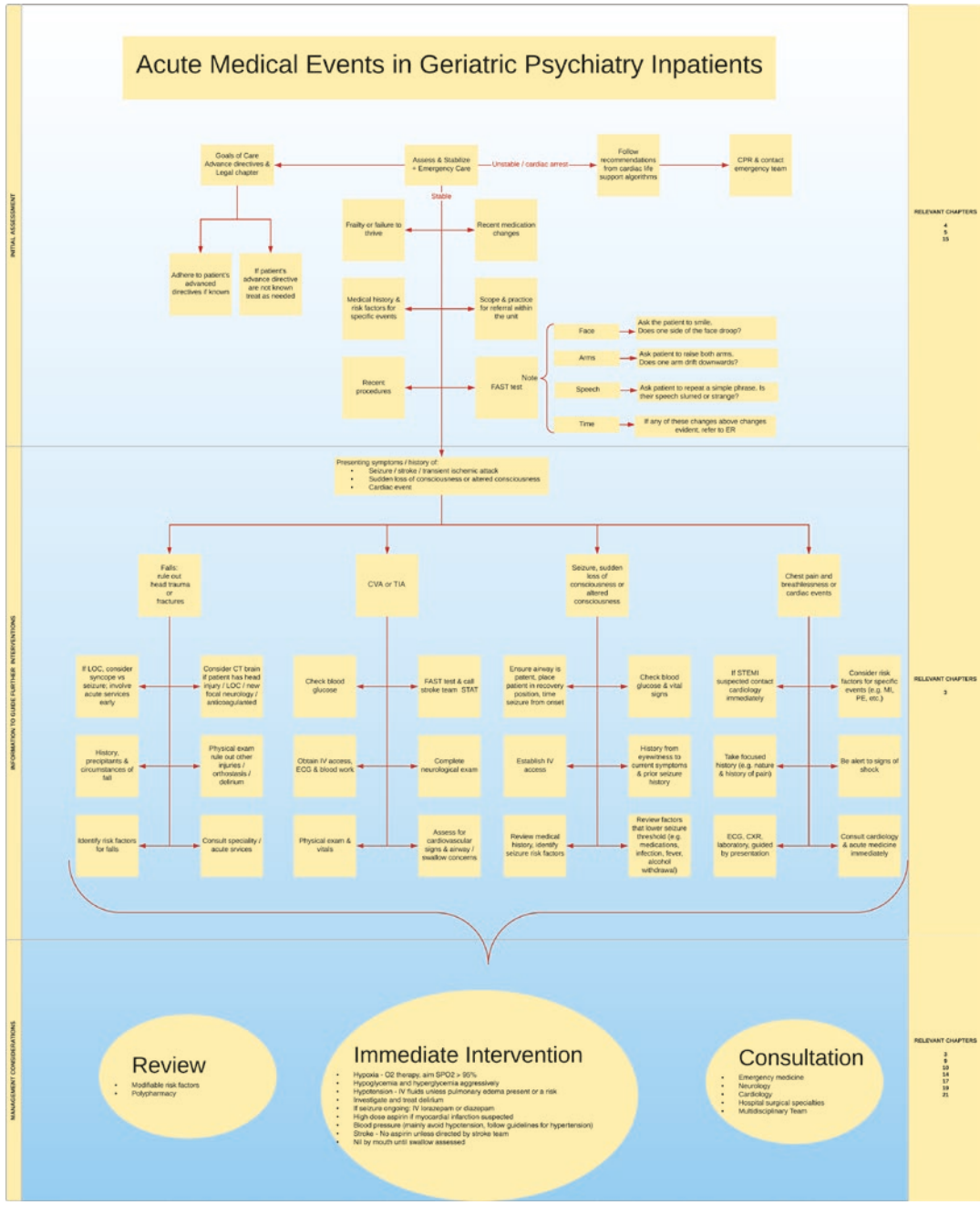

Fig. 7.1 Flowchart assessment of sudden adverse medical events in geriatric inpatient psychiatric centers

tended to happen more in the evening in several locations including bedroom, bathroom, and hallway where they could go unwitnessed [10].

Injurious falls are more common in the geriatric population due to the higher prevalence of systemic disease, such as osteoporosis, as well as increased levels of frailty, which is defined as an increase in vulnerability to poor outcomes when exposed to a stressor [11]. Factors that contribute to frailty include cognitive disorders, depressive

disorder, polypharmacy, multi-morbidity, and poor nutrition. Around 40–60% of falls lead to injury. Of these, 30–50% result in minor trauma, 10–15% in serious injuries, and 5–10% result in serious fracture including 1–2% of these being hip fracture [8]. Given the prevalence and potentially serious consequence of falls, an approach to the identification of falls risk factors and the management of falls is needed to optimize patient safety.

7.2.3 Risk Factors and Prevention

The cause of falls in the aged is often multifactorial, caused by the interaction of multiple contributory intrinsic and extrinsic risk factors. Common intrinsic and extrinsic contributors are illustrated in Fig. 7.2.

7.2.4 Medication

Multiple medications and polypharmacy have been shown to increase the risk of falls [12]. Tinetti et al. [13] demonstrated that community dwelling older people taking sedative medication had an odds ratio (OR) of 28.3 for falls. In a study of falls on an inpatient geriatric psychiatry population, Blair and Gruman [14] also found that medication was the strongest predictor of fall episodes, regardless of psychiatric diagnosis, with high-dose antipsychotics given at any time having the highest correlation to falls. Other classes of drugs associated with increased falls risk include benzodiazepines, antihypertensives, antidepressants, anticholinergics, and diuretics.

Medication reconciliation and review should be part of any falls risk assessment with drug streamlining/rationalization and either discontinuation or dose minimization of drugs associated with falls.

7.2.5 Syncope

Syncope is defined as a temporary loss of consciousness due to a fall in blood pressure. There is significant overlap between syncope and falls—the majority of patients with syncope suffer a fall. Causes of syncope and pre-syncope (near loss of consciousness) include arrhythmias (including bradycardia or tachycardia), orthostatic hypotension, neurocardiogenic (vasovagal) syndrome, valvular heart disease, and carotid sinus syndrome. The Canadian Cardiology Society has provided a useful framework for risk stratification and investigation of syncope [15] including cardiovascular investigations.

7.2.6 Orthostatic Hypotension

Orthostatic hypotension is the sustained reduction of blood pressure within 3 min of standing [16]. It is common in the elderly and can be exacerbated by factors including dehydration, medication, and prolonged bed rest. Measuring orthostatic blood pressure (BP) has been shown to be the most cost-effective test in the investigation of falls [17]. Performing the test involves measuring the patient's heart rate (HR) and blood pressure (BP) after being in a supine position for 5 min. The patient is then asked to stand up and HR and BP measurement is taken at 1 min and 3 min of standing. The test is deemed positive if there is a drop in systolic BP of 20 mm Hg and in diastolic BP of 10 mm Hg or a rise in heart rate of 30 beats per minute. Always correlate patient symptoms with the results as it is possible for patients to experience asymptomatic orthostatic hypotension. Conducting orthostatic vitals should be avoided if the patient has supine hypotension or if the patient is not mobile enough to get out of bed.

7.2.7 Urinary Symptoms

Urinary symptoms are common in geriatric patients and are an independent risk factor for falls [18], particularly when patients rush to the bathroom with orthostatic hypotension, poor balance, unstable gait, limited walking aids, poor footwear, and poor lighting at night. Urinary retention (UR) is the inability to partially or com-

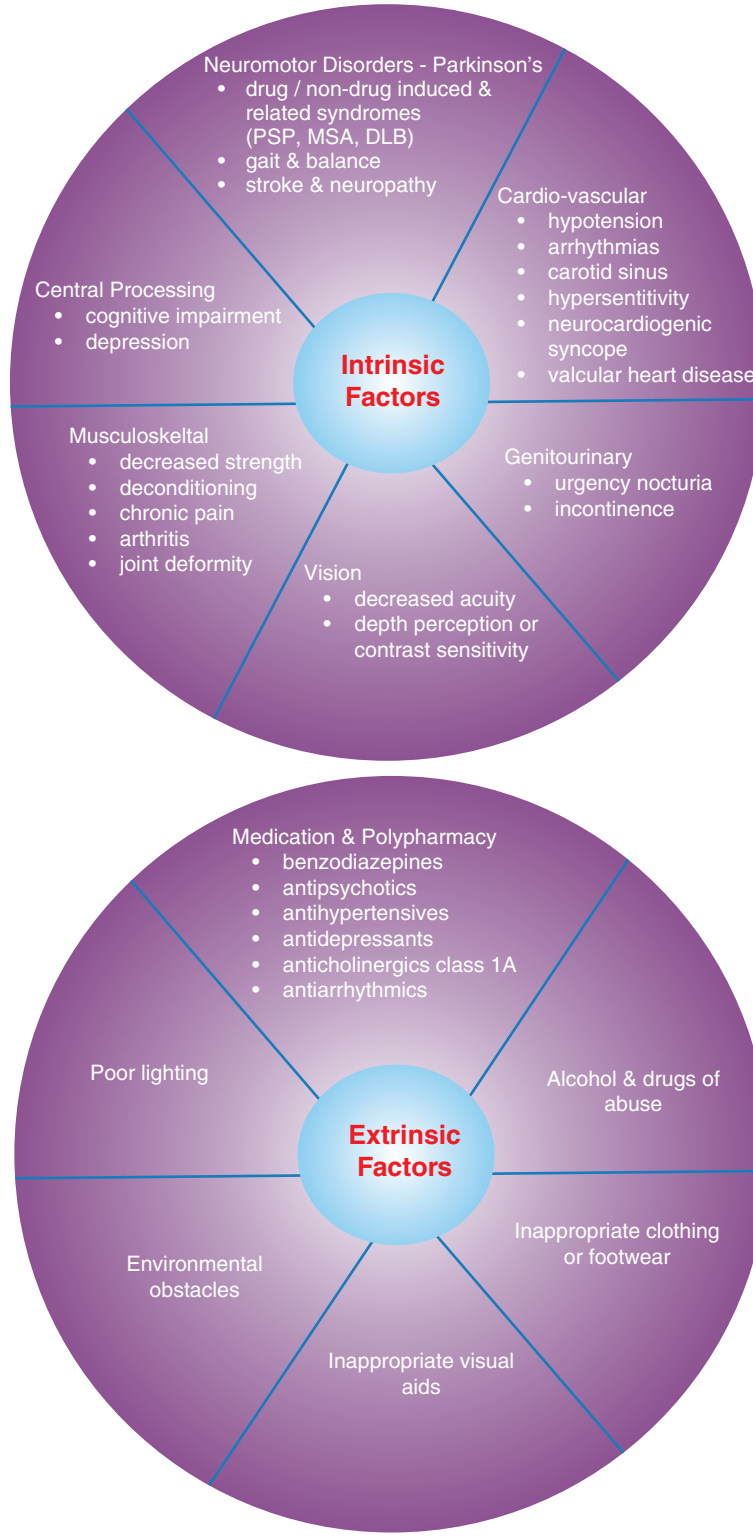

Fig. 7.2 Common intrinsic and extrinsic risk factors for falls

pletely empty the bladder leading to a spectrum of symptoms from urinary urgency and incontinence to suprapubic discomfort and anuria. UR has shown an association with poorer outcomes such as urinary tract infection, distended bladder, and higher mortality rates [19, 20]. It can also cause renal failure secondary to obstructive uropathy, and can lead to delirium [21]. It is estimated that acute urinary retention occurs in one in three men over the age of 80 [22].

Caution: Lower abdominal pain associated with UR may not always be appreciated due to concomitant use of analgesics and cognitive impairment (Chap. 6: MNCD with Behavioral Disturbance).

Risk factors for UR include male sex [23] due to an enlarged prostate, constipation, long-standing diabetes for ≥ 15 years, and anticholinergic medication use [24]. Patients with a fall history should have an evaluation of urinary function, urinary frequency, urgency, dysuria, nocturia, history of incontinence, and assessment for risk factors mentioned above. A medication review will assess for contributory drugs such as diuretics or anticholinergic medication.

If UR is suspected, lower abdominal palpation may reveal a palpable suprapubic mass that is dull to percussion. Palpation may give the patient a sensation of needing to void. The volume of urine retained can be confirmed using a bladder scanner immediately after the patient has voided, giving a post-void residual (PVR) volume. If a bladder scanner is not available, in and out catheterization can be performed. In younger adults, a post-void residual urinary volume of <50 ml is normal; however in older adults up to 150 ml PVR is considered acceptable due to decreased contractility of the detrusor muscle with aging. Once UR has been diagnosed, immediate treatment is to decompress the bladder, either through catheter insertion or in-and-out catheterization, followed by identification and treatment of underlying risk factors. Catheterization may not be needed in all patients with PVRs over 150 ml; a recent study suggested it may be appropriate to conservatively manage aging men with incidentally found PVRs of < 400 ml [25]. There is no reason to believe that women should be managed differently, however women tend to have more symptoms of urgency [26] and hence, often require catheterization at lower volumes. If a patient is symptomatic (e.g., suprapubic discomfort, urgency), and the PVR is >150 ml, then at least twice daily PVRs should be measured and the patient intermittently catheterized in the first instance. Indwelling urinary catheters are sometimes required; though they are a major risk factor for delirium and falls, and should be discontinued as soon as possible.

7.2.8 Approach to a Patient Who Has Fallen

The UK NICE guidelines on "Falls in Older People" (QS86) [27] provide a comprehensive evidence-based approach to assessment and management of falls. Check to see if a local "post-fall protocol" is available.

1. Initial evaluation of patient who has fallen:
   - Priority: determine whether the patient is stable or unstable. Check vital signs and blood glucose. Then assess for evidence of injury, including spinal injury, fractures, skin tears, or soft tissue injuries. Evidence of head injury in the context of anticoagulation may require a CT head to rule out intracranial hemorrhage.
2. Investigate the etiology of the fall(s): Once the patient is stabilized, investigate contributory intrinsic and extrinsic risk factors. Develop a problem list and management plan to address reversible factors:
   - Evidence of an acute illness that may have precipitated the fall, e.g., stroke, acute myocardial infarction, sepsis and commence investigation (e.g., ECG, CXR, metabolic/hepatic panel, CBC, urinalysis if indicated), and appropriate management.
   - Polypharmacy can be a major contributor to falls. A thorough medication review should be part of every falls assessment to scrutinize rationale and doses, particularly for medications that may increase fall risk.
   - Orthostatic vitals in everyone who has fallen.

3. Multidisciplinary (MDT) management (shown in Fig. 7.3):
   - Manage contributory medical issues, e.g., delirium, urinary retention, polypharmacy.
   - The multifactorial/multicomponent approach to falls assessment and prevention is supported by a significant body of evidence including two meta-analyses by the Cochrane Collaborative [28] and by Chang et al. [29]. Refer the patient to physiotherapy for assessment, as they may benefit from gait, strength, and balance training.
   - Ensure environmental factors are assessed. These are simple to optimize and can significantly reduce a patient's risk of falls. Refer to occupational therapy for environmental and function review.
   - The patient may benefit from review by other members of the MDT team; e.g., if poor nutritional intake is contributing to sarcopenia (decline of skeletal muscle with age), a dietician review would be appropriate.

Key Points

- Falls are often multifactorial and require a comprehensive multidisciplinary approach to assessment and management.
- When reviewing a patient who has fallen, the first priority is to establish medical stability, followed by assessment of injuries sustained.
- Once stabilized, use a systemized approach to explore the contributory intrinsic and extrinsic risk factors, involving the multidisciplinary team early.

7.3 Acute Neurological Events

7.3.1 Vignette 2

An 80-year-old man, a retired truck driver, was admitted to inpatient psychiatry with progressive Alzheimer's MNCD (major neurocognitive disorder, or dementia) and behavioral disturbance. In recent months, he was agitated and physically aggressive at home toward his wife. He was paranoid of intruders in the basement attempting to kidnap him. Past medical history included a left hemispheric stroke 5 years earlier with mild, residual right-sided weakness, hypertension, degenerative disk disease, diabetes mellitus type 2, hypercholesterolemia, peripheral vascular disease, and chronic obstructive pulmonary disease (COPD). Medications included aspirin 81 mg daily, ramipril 5 mg twice daily, metoprolol 25 mg twice daily, donepezil 10 mg daily, atorvastatin 20 mg at night, acetaminophen 650 mg four times daily, citalopram 20 mg daily, inhaled corticosteroids and beta agonists, and

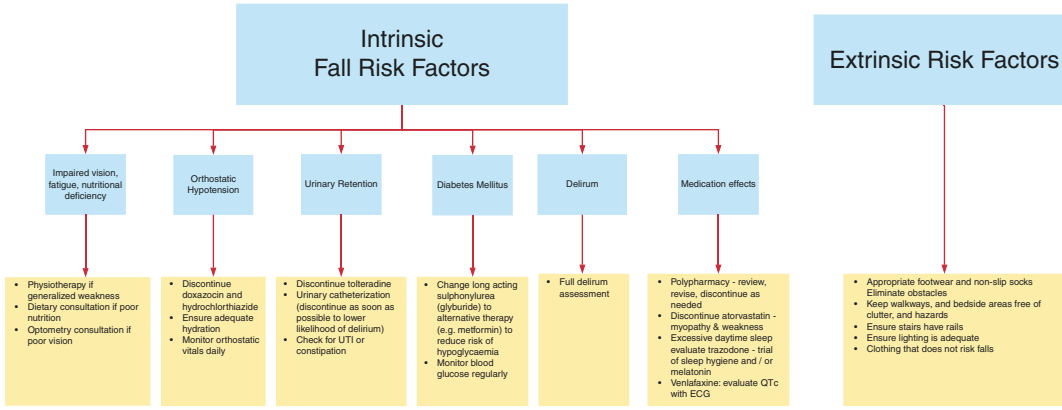

Fig. 7.3 Fall risk reduction (Vignette 1)

ciprofloxacin for a recent urinary tract infection. He smoked one pack of cigarettes per day from the age of 15 up to 5 years ago. There was no history of significant drug or alcohol abuse. He needed increasing help from his wife with ADLs such as washing and dressing. He could ambulate independently with a rollator walker. His speech and swallow were normal and food intake adequate.

At 10 A.M. the day following admission, he was found in bed less responsive than usual, unable to speak. He could not move the right side of his body and did not respond to any stimuli when approached from the right. The nurses reported that he was fine at breakfast time (8:00 A.M.) and needed only minor assistance with his ADLs. During a physical examination, his right arm started jerking, he lost consciousness and then had a generalized tonic-clonic seizure for 1 min. He was immediately drowsy following the episode. His right side continued to be weak.

7.3.2 Discussion: Stroke

The World Health Organization (WHO) defines stroke as a syndrome of "rapidly developing clinical signs of focal (and at times global) disturbance of cerebral function, lasting more than 24 h or leading to death with no apparent cause other than that of vascular origin" [30]. In the United States, approximately 795,000 patients present with acute stroke every year, nearly three-quarters occurring in patients over the age of 65. Approximately 610,000 of these are first or new strokes and the remainder occur in individuals with a previous stroke history [31]. Approximately 87% of strokes are ischemic in etiology, with the remainder hemorrhagic [31]. Strokes lead to significant morbidity and mortality, but advances in the last two decades have revolutionized stroke care, which is now considered a preventable and treatable condition [32].

A patient with acute neurological signs is a medical emergency. The onset of right-sided weakness with possible right-sided visual defect or neglect indicates an acute neurological deficit that follows a vascular distribution. The findings in the vignette are consistent with an acute stroke using *FAST* (Face Arm Speech Test); see Fig. 7.4 [33]; however hypoglycemia should be ruled out, as sudden onset focal neurology may be caused by low blood glucose in the context of diabetes. If blood glucose is low (< 3.2 mmol/L or < 60 mg/dL), the patient should receive 50 ml of 50% dextrose intravenously with or without glucagon (1 mg IM/SC), depending on whether or not IV access is achieved. Vomiting is a common side effect of glucagon, so it is important to take precautions to prevent aspiration. If blood sugar is normal or high, hypoglycemia has been ruled out and the patient had an acute stroke until proven otherwise. If available, a thrombolysis team should be paged immediately. If not, emergency services should transfer the patient to the closest acute stroke service for interventions such as thrombolysis and stroke unit care.

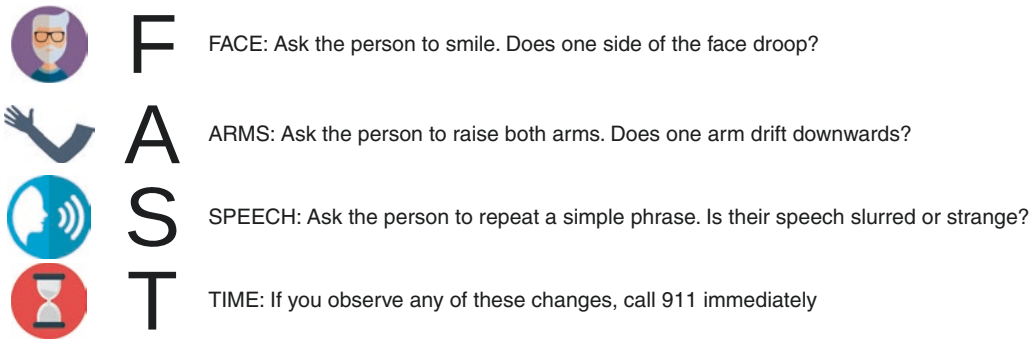

Fig. 7.4 FAST – face arm speech test

No workup should delay the stroke intervention. The *FAST* test can be done in 30 s. The most important piece of information for the stroke team is the time of onset of symptoms or when the patient was last seen "normal." Reporting the patient's risk factors will also be beneficial. This vignette patient was at high risk for a vascular event, given his history of previous stroke, diabetes, hypertension, hypercholesterolemia, and peripheral vascular disease. Though an antipsychotic was discussed for the treatment of agitation, luckily, none was prescribed. Second-generation antipsychotic (SGA) can increase the risk of stroke [34, 35]. Figure 7.4 represents the FAST – face arm speech test – stroke assessment approach.

The onset of the stroke was less than 2 h previously, so the vignette patient was well within the time window for IV thrombolysis (stroke onset within 4.5 h) [32]. The time window for thrombectomy is up to 6 h. Recent evidence suggests that thrombectomy up to 24 h after stroke onset—in those who have severe clinical deficits, which are disproportionate to the radiological findings—has significantly better functional outcomes at 90 days compared to usual care [36]. Hence, now even patients who wake up with an acute neurological deficit with an unknown time of onset should also be referred urgently to acute stroke services.

A seizure (an absolute contraindication to thrombolysis) should not deter paging the stroke team, because even if deemed ineligible for thrombolysis, the patient requires urgent specialist assessment and brain imaging and may indeed be suitable for interventional radiological procedures such as thrombectomy. Regardless of suitability for these acute interventions, outcome from acute stroke will be significantly improved with management on a stroke unit [37]. Outcomes are better the earlier thrombolysis is administered, so even though there is a time window of 4.5 h from symptom onset for administering thrombolysis, 1.9 million neurons are lost per minute following an acute stroke [38], hence the "time is brain" notion.

While awaiting transfer of the patient or for the stroke team to arrive, ensure the patient is stabilized and that cerebral blood flow and oxygenation is optimized:

- Ensure the airway is patent (see seizure management below), check vital signs and insert an intravenous cannula.
- Monitor oxygen saturations continuously and administer supplemental oxygen therapy if oxygen saturations drop below 95%.
- Higher blood pressures (BP) are preferred in the case of acute stroke. Therefore, antihypertensives are not given except in the case of hypertensive emergency with concomitant medical issues, for example, hypertensive encephalopathy, or if the systolic BP is $\geq$ 220 mm Hg. In candidates for thrombolysis (following discussion with acute stroke services), blood pressure reduction to 185/110 mm Hg may be considered. In hypotensive patients [39], that is, BP significantly lower than their baseline, or systolic <120 mm Hg, one should lie the head of the bed flat and consider administering an isotonic saline infusion aiming for euvolemia in order to improve cerebral perfusion (dextrose-containing fluids may worsen the cerebral insult unless the patient is hypoglycemic).
- Hypoglycemia should be treated urgently, as outlined above. Maintain blood glucose levels between 4 and 11 mg/dl using local guidelines.
- For appropriate glycemic control, intravenous insulin sliding scale should be used in all Type 1 diabetics with suspected acute stroke.
- It may be beneficial to send urgent bloodwork to help facilitate workup for potential thrombolysis (CBC, glucose, electrolytes, renal function, PT, INR, aPTT, cardiac markers) [39]. An ECG may identify new atrial fibrillation and should be completed if time allows.
- Keep the patient nil by mouth (NPO) until swallow is assessed to reduce the risk of aspiration.

Caution: Prior to the advent of thrombolysis, a dose of aspirin 300 mg was recommended even before brain imaging because the benefits outweighed the risks; **this is no longer recommended**

because antiplatelet agents as well as anticoagulants may increase the risk of bleeding complications post-thrombolysis.

A transient ischemic attack (TIA) is defined as a stroke presentation that resolves within 24 h [30], although most TIAs resolve within 1 h or before any chance to examine the patient. Sometimes, patients with deficits lasting less than 24 h will subsequently be found to have an associated acute infarct on MRI and are considered as having non-disabling stroke despite the duration of symptoms. Amaurosis fugax or sudden onset monocular blindness due to retinal ischemia, although not included in the WHO definition of stroke or TIA, is caused by similar embolic phenomena within the same vascular bed and requires the same assessment and management as a stroke or TIA.

A TIA is a clear marker of threat to the brain. In the setting of a TIA, the ABCD2 score (see Table 7.1) is used to help stratify the patient's risk of subsequent stroke [40]. The NICE guidance on stroke [32] advises that a patient at moderate to high risk (ABCD2 score ≥ 4) should be started immediately on aspirin 300 mg, and discussed with neurology so that urgent (within 24 h) assessment and investigation (e.g., neuroimaging, carotid imaging) can be initiated. Other high-risk patients who should be discussed urgently with neurology include those with two or more TIAs in 1 week (crescendo TIAs) even if their ABCD2 score is less than 3, as well as those on anticoagulation.

Do not start aspirin in those on anticoagulation, without specialist recommendation. In those at low risk of stroke (ABCD2 score 0–3), start aspirin 300mg daily and refer to specialist services for assessment as soon as possible and definitely within 1 week of symptom onset. In high-risk cases, urgent brain imaging is indicated, preferably MRI with diffusion-weighted sequences although this should not delay contacting neurology services urgently. In low-risk cases, brain imaging is also indicated and can be arranged while awaiting neurology review (within 1 week). Table 7.1 summarizes the stroke risk stratification following a TIA: ABCD2 score (https://www.mdcalc.com/abcd2-score-tia).

7.3.3 Seizures

Approximately 10% of people will have at least one seizure during their lifetime. The distribution of seizure incidence is bimodal with the first peak

Table 7.1 Stroke risk stratification following a TIA: ABCD2 score (https://www.mdcalc.com/abcd2-score-tia)

A	Age≥60 years	No = 0	Yes = +1
B	Blood pressure≥140/90 *(initial BP systolic≥140 and/or diastolic≥90)*	No = 0	Yes = +1
C	Clinical features of TIA	Unilateral weakness = +2	
		Speech disturbance without weakness = +1	
		Other symptoms = 0	
D1	Duration of symptoms	< 10 min = 0	
		10–59 min = +1	
		≥ 60 min = +2	
D2	History of diabetes	No = 0	Yes = +1
Scoring and risk stratification			
	Low risk = 0–3 points	Moderate risk = 4–5 points	High risk = 6–7 points
2-day stroke risk	1.0%	4.1%	8.1%
7-day stroke risk	1.2%	5.9%	11.7%
90-day stroke risk	3.1%	9.8%	17.8%

in early childhood and a second and higher peak in people over the age of 65 years [41]. The causes of seizure in geriatric patients can be similar to that in younger patients (e.g., alcohol withdrawal), however more often seizures in this age bracket are related to age-related etiological pathologies, for example, strokes. Treating seizures in this age bracket can be challenging due to the risk of drug toxicity and interactions with other medications.

Over one-quarter of all first acute seizures occur in older age. In older adults, important causes include metabolic abnormalities (e.g., hypoglycemia, hyponatremia, uremia, hypocalcemia), acute stroke or intracranial hemorrhage, CNS infections (e.g., viral encephalitis), trauma, and drug withdrawal (e.g., abrupt discontinuation of sedative and anxiolytic drugs) [41]. Importantly, almost one-third of aging patients with a first acute seizure present in status epilepticus, double the rate in the general population [42], and hence require emergency management. Often status epilepticus, particularly non-convulsive status, is misdiagnosed and the resultant delay in appropriate treatment carries high morbidity and mortality [42]. Another important cause of acute seizures in older patients are medications that can lower seizure threshold, many of which are commonly used in geriatric patients, particularly those with psychiatric illness (e.g., bupropion, phenothiazines, tricyclic antidepressants, quinolone antibiotics, meperidine, tramadol) [41, 43].

Recurrent unprovoked seizures (epilepsy) also peaks in incidence in aging adults, and can be due to underlying vulnerability secondary to previous stroke or brain trauma, major neurocognitive disorder, or primary or secondary brain malignancy, however the cause remains unknown in almost half of cases. Patients commonly present with partial seizures, the focus often located within the frontal or parietal lobes rather than the temporal lobes, which modifies pre-ictal and ictal manifestation. Patients who present with pre-ictal dizziness, paresthesias, posturing, and other nonspecific symptoms make the diagnosis challenging. When there is loss of consciousness or loss of awareness, collateral history is extremely important, however is not always available. Furthermore, other conditions can be mistaken for seizures, such as syncope, transient ischemic attacks, vertigo, and transient global amnesia.

Seizure evaluation includes a thorough history from the patient, family, and an eyewitness (crucial), noting a history of head trauma, medication history with any recent changes, and other seizure risk factors; full neurological examination; blood work (CBC, electrolytes, renal and liver function tests); brain imaging; ECG; and electroencephalography.

Status epilepticus requires emergency management. The following recommendations are from the American Epilepsy Society [44]. As with any acute presentation, first stabilize the patient, ensuring the airway is patent (may need to use an oropharyngeal or nasopharyngeal airway). Check vital signs (heart rate, BP, oxygen saturations) and administer oxygen via nasal cannula or facemask. If the airway is compromised, intubation may be required, which may necessitate transferring the patient to acute services depending on the setting. Time the seizure from its onset. Conduct a brief general neurological exam, especially for loss of motor function. Check blood glucose and treat blood sugars less than 60 mg/dL (3.3 mmol/L) with 100 mg IV thiamine, followed by 50 ml of 50% Dextrose. Establish IV access if possible and send blood for CBC, electrolytes and, depending on the scenario, levels of anti-epileptic medications and/or a toxicology screen.

If the seizure continues beyond this initial assessment, medication to stop the seizure should be initiated. If the patient has IV access, then give either intravenous (IV) lorazepam (0.1 mg/kg/dose, maximum 10 mg/dose, maximum two doses), or IV diazepam (0.15–0.2 mg/kg/dose, maximum 10 mg/dose, maximum two doses). A second line IV choice is IV phenobarbital (15 mg/kg/dose, only once); however, intramuscular midazolam (10 mg for those > 40 kg and 5 mg for those < 40 kg, only once) is preferred over phenobarbital. If the patient has no IV access, the

first-line choice is intramuscular midazolam; however if this is not available, rectal diazepam (0.2–0.5 mg/kg, maximum 20 mg/dose, only once) or buccal or intranasal midazolam may be used. Only one of the above medications should be used during this phase. At this point, you should call local internal medicine or neurology services for further assistance depending on local pathways, however the patient needs to be supported until specialty services can be accessed.

Further management is usually done by the internal medicine or neurology service, including other lines of treatment to abort persistent status epileptics. Once the patient has stopped seizing, care is directed toward supporting airway, breathing, and circulation as well as preventing complications.

The vignette patient had a partial seizure with secondary generalization that resolved spontaneously within 1 min. Hence, no immediate anti-epileptic medication was required. This seizure was in the setting of acute focal neurology on a background history of previous stroke (affecting the same side) and no history of seizure. This seizure may have been due to an acute stroke or indeed his focal neurology may be a Todd paresis secondary to an earlier unwitnessed seizure. It is important to obtain as much eyewitness account of his condition throughout the morning, consider any recent medication additions that may have lowered the patient's seizure threshold (e.g., ciprofloxacin). Even though this seizure resolved without requiring intervention, regardless of the presence or absence of focal neurological signs, the local neurological team should be contacted, as the patient is likely to require urgent workup and management to prevent further seizures.

As with all acute events in geriatric or chronically ill inpatients, the goals of care must be considered before initiating emergency investigation and management. It is important to have this discussion proactively with the patient and/or their next-of-kin or power of attorney on admission, so that decision making in the event of acute medical deterioration is facilitated and in line with the wishes of the patient and his/her family [Chapter on Legal Aspects].

Key Points

- A sudden, possible acute neurological event in a patient with diabetes: first check blood glucose and treat hypoglycemia per hospital protocol.
- If no hypoglycemia, and FAST test is positive, immediately contact local stroke services.
- Stabilize the patient, ensure airway is patent, keep oxygen saturations above 94% with supplemental oxygen, and treat hypotension with IV fluids. Do not treat hypertension unless systolic BP is >220 mg Hg.
- In the setting of status epilepticus, initiate anti-epileptic medication as per local guidelines and contact neurology services.
- Consider the patients' baseline frailty and goals of care, as well as their risk factor profile for the acute event.

7.4 Cardiovascular Events

7.4.1 Vignette 3

An 85-year-old female, an inpatient for treatment of Bipolar disorder, depressive episode, was responding well and became psychiatrically stable. She was due for discharge within one week. Past medical history included coronary artery disease with a myocardial infarction 15 years ago, diabetes, hypertension, hypercholesterolemia, hypothyroidism, osteoarthritis, gout, COPD, and Meniere disease. Medications included aspirin 81 mg daily, ramipril 5 mg daily, atorvastatin 40 mg at night, levothyroxine 100mcg daily, acetaminophen 500 mg four times daily, naproxen 500 mg daily prn, allopurinol 300 mg daily, betahistine 16 mg three times daily, citalopram 10 mg daily, and inhaled corticosteroids and beta agonists.

While watching TV, she complained of epigastric burning pain. She thought it was indigestion and took some antacid for relief. Despite this, her pain worsened and began to radiate

down her left arm. She became pale, sweaty and unwell. Her breathing became more labored. Vital signs showed a heart rate of 120 beats per minute, a blood pressure of 90/40 mm Hg, respiratory rate of 40/min, and oxygen saturations of 92% on room air. The on-call psychiatrist arrived as the patient, now unresponsive, was being wheeled out. She was pronounced dead in the Emergency Department.

7.4.2 Discussion

7.4.2.1 Chest Pain and Breathlessness

Chest pain and breathlessness are relatively common complaints encountered among geriatric inpatients. They can occur individually or concurrently, the cause of which may be serious or relatively benign. Serious causes of chest pain are more prevalent in the elderly than in younger age groups [45]. Underlying mental health disorders can contribute to symptomatology. Anxiety is a common cause of both chest pain and shortness of breath; e.g., 30–50% of patients with recurrent chest pain and normal coronary angiograms meet criteria for panic disorder [46]. Conversely, depression is known to be common in patients with coronary artery disease and is independently associated with increased cardiovascular morbidity and mortality [47]. The natural default position should be to exclude serious systemic medical issues first. As such, it is important to have an approach to the assessment of chest pain and breathlessness in order to assess and manage patients safely, and to identify those who require urgent referral to specialist services for further investigation. Geriatric patients often present atypically so the assessing physician should consider past medical history, risk factor profile, and other elements from the history and examination to help inform differential diagnosis and decision-making in terms of further investigations and treatment. Chest pain and breathlessness can originate from many different organ systems. Figure 7.5 summarizes the most common etiologies for chest pain and breathlessness.

The most serious and potentially life-threatening conditions in this chapter include:

- Acute myocardial infarction
- Aortic dissection
- Pulmonary edema
- Pulmonary embolism
- Pneumonia

The patient's past medical history, risk factor profile, and presentation will influence the probability for each condition which presents with acute chest pain. Significant risk factors for chest pain are outlined in Table 7.2.

As aging patients do not always present typically, even with serious pathology, it is important to be vigilant. Examples include (1) infectious disease such as pneumonia without mounting a fever, even if the patient is not taking acetaminophen on a regular basis, and (2) myocardial infarction with epigastric rather than central chest pain, or without any reported pain at all, due to an altered sensory perception associated with aging or the presence of cognitive impairment.

All acute illness in geriatric patients can present with acute confusion and delirium and/or other geriatric frailty syndromes such as falls or functional impairment.

7.4.3 Physical Examination in Patients in Cardiovascular Distress

Critically ill patients need to be rapidly identified so that appropriate management can be initiated and, if necessary, safe transfer to an acute site for definitive assessment and management can be expedited. Firstly, is the patient alert and responsive or is their level of consciousness reduced? If he/she is unresponsive, the practitioner must check for airway and breathing and for a carotid pulse to immediately ascertain if the patient is in cardiorespiratory arrest. If there is no definite pulse palpable or the patient is not breathing, the practitioner must immediately start resuscitation efforts unless a do not resuscitate (DNR) status is confirmed, and follow the cardiac arrest protocol in your institution. Do not wait for the arrest trolley or the arrest team to arrive—check the airway, start effective chest compressions, and provide

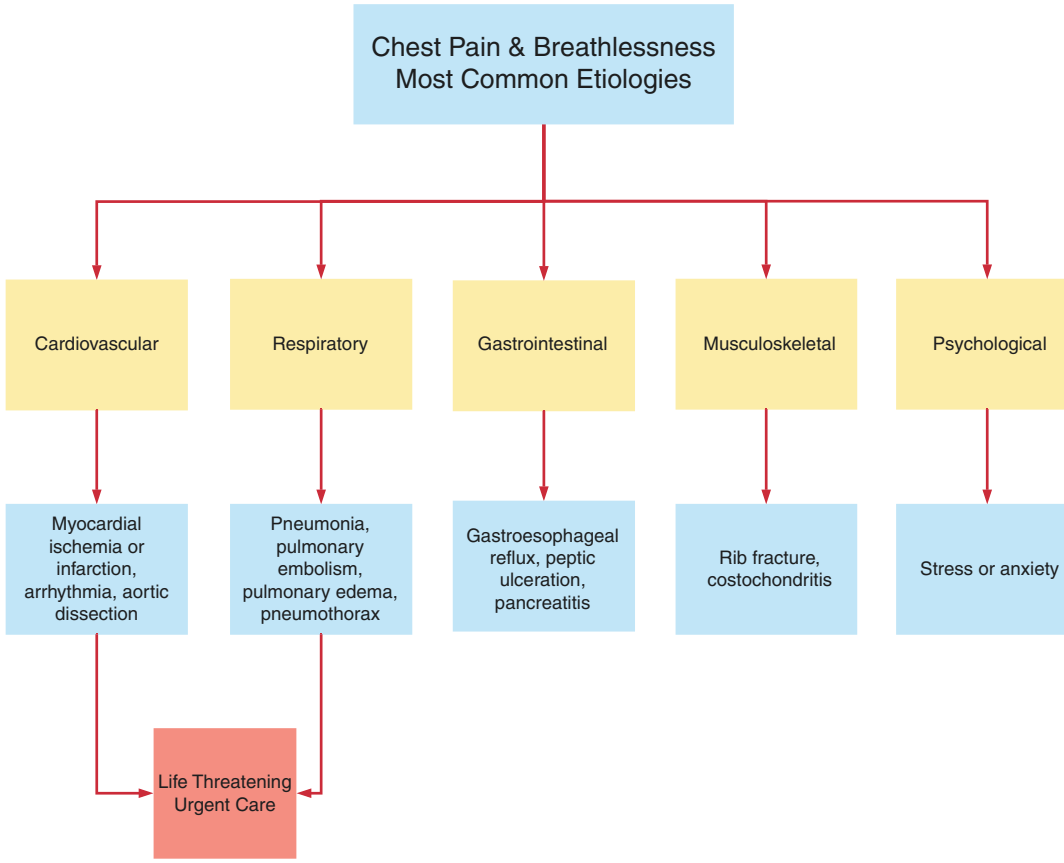

Fig. 7.5 Chest pain and breathlessness – most common etiologies

breaths (30 chest compressions followed by two breaths in continuous cycles) until the defibrillator and/or help arrives.

If the patient is not in a cardiorespiratory arrest, the practitioner needs to consider if the patient looks well or unwell on general inspection. Physical signs that suggest critical illness include pallor, reduced perfusion with cold extremities, diaphoresis, increased respiratory rate (tachypnea), and altered level of consciousness. A complete set of vital signs should be urgently obtained as part of the initial assessment. This includes heart rate, blood pressure, temperature, respiratory rate, and oxygen saturations. If aortic dissection is considered (e.g., due to presence of risk factors laid out in Table 7.2), blood pressures should be obtained in both arms. A discrepancy of 20 mmHg in the context of chest pain radiating into the back is suggestive of dissection [48]. Bradycardia (HR < 60 bpm) or tachycardia (HR > 100 bpm) are potentially disconcerting. A change in rhythm (e.g., new irregular rhythm) may suggest an arrhythmia such as new onset atrial fibrillation.

Low blood pressure can be of greater concern than high blood pressure. Although a blood pressure reading of < 90 mm Hg systolic and/or < 60 mm Hg diastolic is generally considered low, it is important to interpret this in the context of the patient's previous readings. A drop in BP from baseline may be accompanied by lightheadedness and even fainting, fatigue, blurred vision, nausea, and/or poor concentration. If the drop in BP is significant, it may be indicative that the patient is in shock; i.e., poor perfusion of vital organs and tissues which may quickly lead

Table 7.2 Risk factors and typical presentation: common serious causes of chest pain and breathlessness

	Risk factors	Typical presentation
Myocardial infarction	Known coronary artery disease	Central crushing chest pain
	Positive family history for ischemic heart disease	Radiates to jaw and/or left arm
	Smoking	Nausea
	Diabetes mellitus	Diaphoresis
	Male sex	Pallor
	Hypercholesterolemia	Can be accompanied by shortness of breath secondary to pulmonary edema
	Hypertension	
Thoracic aortic dissection	Known thoracic aortic aneurysm	Severe central chest pain radiating into the back
	Recent aortic valve manipulation (surgical or transcatheter)	Stabbing or tearing pain
	History of connective tissue disorder (Marfan's syndrome, Ehlers–Danlos syndrome)	BP differential of ≥ 20 mm Hg between arms is suggestive of dissection
		A new murmur
Pulmonary edema	History of heart failure (systolic or diastolic)	Sudden-onset shortness of breath
		Raised JVP and peripheral edema (if right heart failure)
		Tachycardia is often present
		Tachypnea
		Respiratory crackles on auscultation
Pulmonary embolism*	History of venous thromboembolism	Sudden-onset shortness of breath
	Immobility	Pain is stabbing in nature
	Recent trauma	
	Recent surgery	Accompanied by shortness of breath, tachycardia, and hypoxia
	Malignancy	May have concurrent symptoms suggestive of deep vein thrombosis
	Hormone replacement therapy	
	Hypercoagulable states: anti-phospholipid syndrome, Protein C or Protein S deficiency	
Pneumonia	Underlying lung disease; e.g., COPD	Increased shortness of breath with or without chest pain
	Immunosuppression (e.g., long-term steroid therapy, immuno-suppressant therapy)	Chest pain may be pleuritic in nature
	Immobility	Pyrexia
	Poor swallow (leading to aspiration pneumonia)	Hypoxia

*Wells criteria can be accessed from https://reference.medscape.com/calculator/wells-score-pe

to end-organ damage. The patient may be confused, cold, and clammy with rapid shallow breathing and a weak and rapid pulse. Shock can be caused by several mechanisms including hypovolemia (e.g., due to bleeding); sepsis (e.g., due to pneumonia); cardiogenic shock (e.g., pump failure due to acute myocardial infarction); anaphylaxis (e.g., medication allergy); and neurogenic shock (e.g., disruption of autonomic pathways due to spinal cord injury). In the situation of acute cardiac or respiratory conditions, the most common types of shock encountered are septic shock and cardiogenic shock, however it is important to be aware of the other mechanisms.

If the patient looks acutely unwell and is clinically unstable (tachycardic, hypotensive, low oxygen saturations), resuscitative efforts need to be initiated (see below), and expert medical advice needs to be pursued urgently.

7.4.4 Investigations for Acute Chest Pain and Cardiovascular Distress

7.4.4.1 Electrocardiogram (EKG/ECG)

A 12 lead ECG should be performed as soon as possible in order to assess the cardiac rhythm and to look for evidence of myocardial ischemia or infarction. Although many ECG machines report an automatic interpretation of the ECG patterns, this cannot be relied upon, and hence, in emergency situations all doctors need to be able to recognize critical pathology on the ECG. ST segment elevation (see Fig. 7.6) suggests an acutely occluded coronary artery and is considered the most important pattern to recognize. ST (segment) elevation myocardial infarction (STEMI) requires *IMMEDIATE* medical attention, as early intervention can prevent irreversible myocardial damage. ST segment depression or new-onset T wave inversion (see Fig. 7.7a, b) is suggestive of cardiac ischemia and requires urgent further assessment. If a pulmonary embolism (PE) is present, the most common ECG finding is sinus tachycardia, which is nonspecific and may be due to several other etiologies, e.g., sepsis and hypovolemia. Some ECG changes, also seen frequently in PE (e.g., Right Bundle Branch Block, right axis deviation, ST and T wave changes, atrial fibrillation), are associated with negative outcomes [49]. The classical, though less prevalent finding of S1Q3T3 pattern (S wave in lead I and a Q wave with an inverted T wave in lead III), is also a poor prognostic indicator [49].

7.4.4.2 Chest X-Ray (CXR)

A CXR is invaluable in the assessment of a patient with chest pain and/or breathlessness. If the patient is unstable, a portable x-ray should be requested so that the patient is not moved unnecessarily. In most centers, an urgently requested CXR will be reported by a radiologist reasonably quickly; however in emergency situations, practitioners may need to act quickly, and having a basic knowledge of CXR interpretation can help guide management. Table 7.3 summarizes the common findings in the setting of chest pain and/or breathlessness. Figure 7.8a represents a chest x-ray depicting an aortic dissection, Fig. 7.8b represents a chest x-ray showing a pulmonary edema, and Fig. 7.8c demonstrates a chest x-ray representing consolidation.

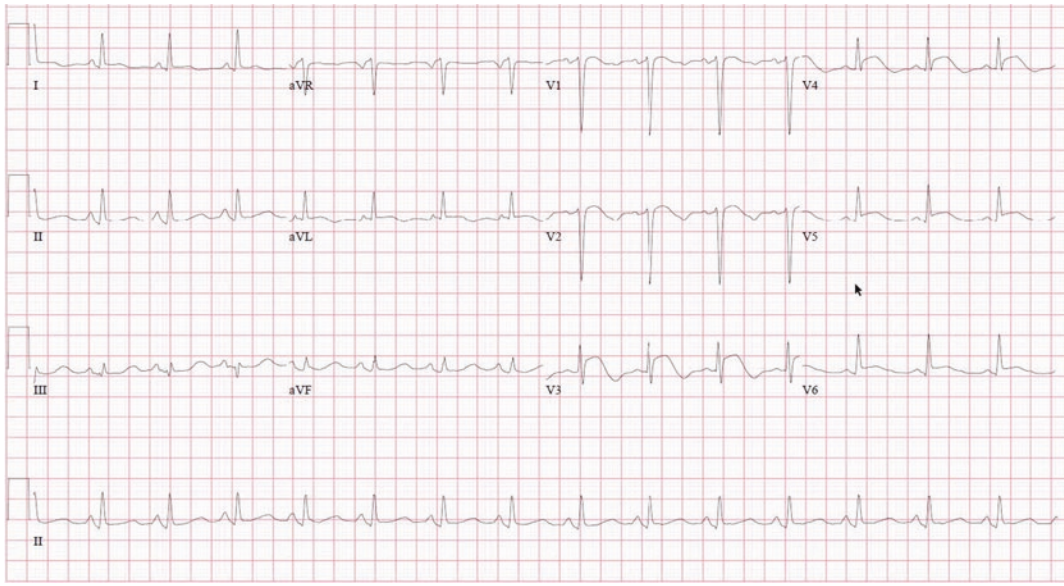

Fig. 7.6 Anterior STEMI

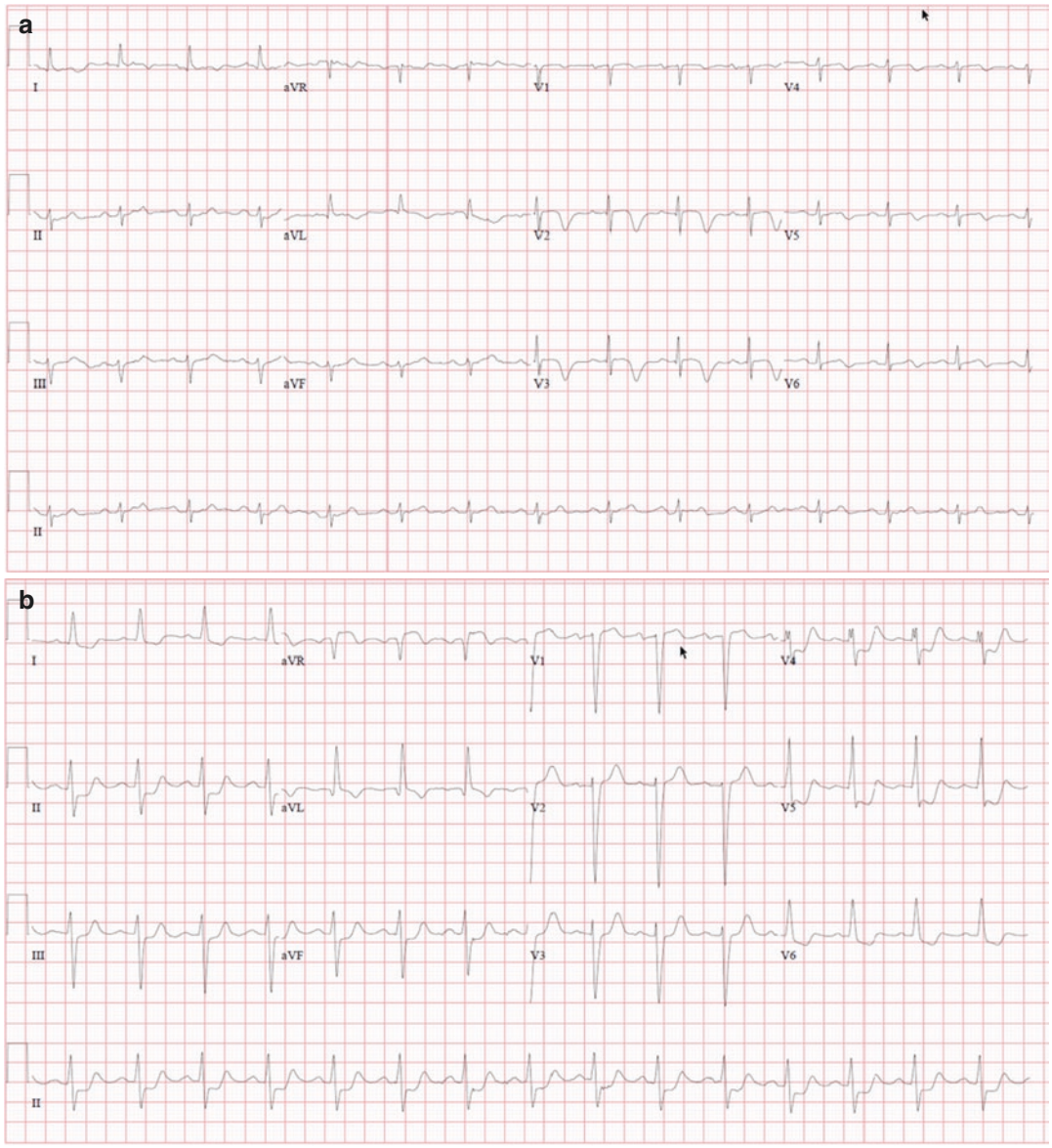

Fig. 7.7 (**a**) NSTEMI anterior T wave inversion, (**b**) NSTEMI with ST depression throughout

7.4.5 Laboratory Studies for Acute Chest Pain and Cardiovascular Distress

Laboratory studies may help differentiate between the various pathologies discussed above. All acutely ill patients should have blood sent for CBC to investigate for anemia or leukocytosis, as well as urea, creatinine, and electrolytes. Other tests should be guided by the specific condition suspected:

- If myocardial ischemia/infarction is considered, serial serum troponins should be checked. Timing of this differs between institutions, as it will depend on the local assays used. It is important to be familiar with local guidelines.
- If pulmonary embolism (PE) is considered, a validated clinical prediction tool should be used (e.g., Wells criteria [53]). If the patient is deemed at moderate or high risk of PE, further investigation is warranted such as a CT

Table 7.3 Chest X-ray findings in chest pain and/or breathlessness

Condition	Chest X-ray findings
Myocardial infarction	No specific CXR findings in acute myocardial infarction unless co-existent pulmonary edema
Thoracic aortic dissection	The most notable CXR finding in aortic dissection, and reasonably easy for non-radiologists to identify, is widening of the mediastinum to greater than 8 cm and the presence of abnormal aortic contour (see Fig. 7.8a). Other abnormalities may be identified by a radiologist [45]; however, up to 12.4% of patients can have an entirely normal CXR [50]
Pulmonary edema	Common features of heart failure on CXR are (see Fig. 7.8b): Cardiomegaly or a cardiothoracic ratio of > 0.55 (if the film is taken AP as opposed to PA; this sign is not reliable as the heart appears larger on AP films) Upper lobe venous diversion (a sign of increased pulmonary venous pressure) Kerley B lines (interstitial edema)
Pulmonary embolism	CXR changes do not always occur in acute pulmonary embolism, although in patients aged 70 or older changes can be seen in up to 82%. The most common findings include cardiomegaly and pulmonary venous congestion [51]. Importantly, these findings are non-specific and poor predictors of pulmonary embolism. The main value of a CXR in this situation is to exclude other pathology that may mimic pulmonary embolism; e.g., pneumonia [52]
Pneumonia	Airspace opacification, lobar consolidation, or interstitial opacities without volume loss are all features of pneumonia (see Fig. 7.8c). Air bronchograms, which are air-filled bronchi made visible by surrounding opacification due to consolidation, are also characteristic. These appear as straight dark lines through an area of opacification. Consolidation at the right or middle lower lobe increases suspicion for aspiration pneumonia

pulmonary angiogram or ventilation perfusion scan. If the patient is deemed low risk, a high sensitivity d-dimer level should be checked. If it is normal (<500 ng/mL), the probability of PE is low, and there is no indication for further imaging for PE. If it is positive, further imaging may be warranted although it is important to recognize that D-dimers can be raised in multiple other conditions, including renal failure, malignancy, and infection [54]. D-dimers should not be conducted in patients with a high pre-test probability as it does not aid decision-making. These patients require further investigation regardless of the D-dimer result.

7.4.6 Management of Acute Chest Pain and Cardiovascular Distress

Management will be guided by the diagnosis after a thorough history and clinical examination together with the results of relevant investigations. The goals of care are to respect a patient's wishes regarding the intensity and invasiveness of proposed management. General advice in principle, but not exhaustive:

- Correct hypoxia with oxygen therapy in acutely breathless patients or if O_2 saturations are < 94%. Caution is required in patients with COPD as they may be dependent on their hypoxic drive for respiration. In these patients, check their baseline O_2 saturations and for any previous history of hypercapnic respiratory failure. If at risk of hypercapnia, use O_2 therapy to keep saturations between 88% and 92%.
- If acute cardiac ischemia is suspected, high-dose aspirin (300–325 mg) needs to be administered without delay, as long as there is no contraindication. Nitrate spray or transdermal patch may help with chest pain while awaiting specialist assessment, although monitoring for hypotension is needed. Nitrates must be avoided

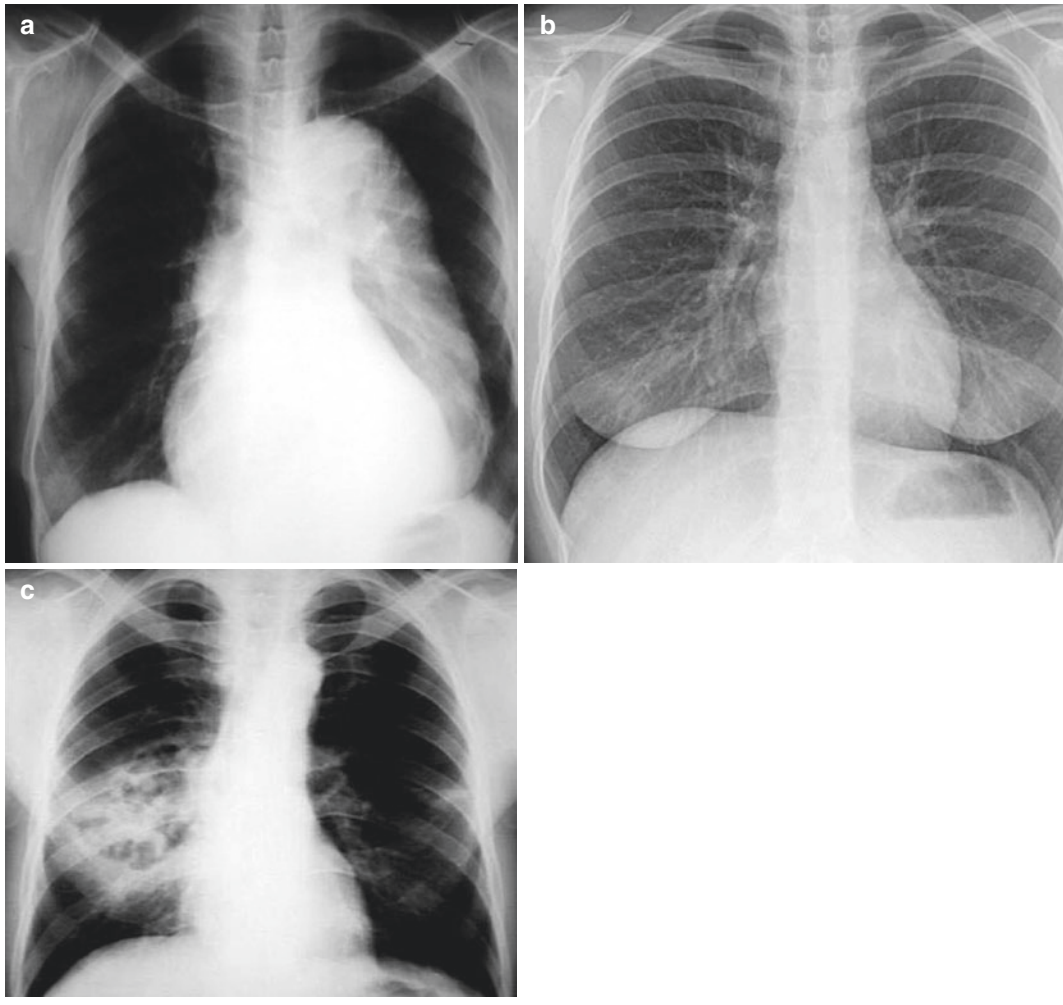

Fig. 7.8 (**a**) Aortic dissection, (**b**) Pulmonary edema, (**c**) Consolidation

in those who have taken a phosphodiesterase inhibitor in the previous 24 h (e.g., sildenafil). If there is no relief with nitrates, then IV morphine should be considered to treat pain.

- Referral to specialist services (emergency department, cardiology) should be made immediately to facilitate appropriate therapy including revascularization if indicated.
- If a thoracic aortic dissection is suspected, immediate cardiothoracic surgery involvement is required.
- If acute pulmonary edema is suspected, urgent medical review is often required to investigate and treat the cause, as well as to treat the heart failure episode itself. Treatment options such as intravenous furosemide may be considered as long as the patient's BP allows.
- If PE is confirmed, anticoagulation will be required and investigation of provoking factors would be important. Type and duration of anticoagulation (e.g., novel oral anticoagulant vs warfarin) will depend on local guidelines. Consideration of concurrent medications is required to assess risk of bleeding.
- If there is evidence of pneumonia, antibiotics are indicated either orally or intravenously depending on severity and the patient's ability to swallow.

- In patients with recurrent pneumonia, the possibility of aspiration should be considered.
- Red flags for aspiration include witnessed coughing or choking while eating or drinking and/or the presence of predominantly right-sided infiltrates on CXR. In these scenarios, expeditious swallow assessment should be sought in order to review aspiration risk and make recommendations as to appropriate food and fluid consistencies to minimize that risk.
- Risk factors for aspiration include reduced level of consciousness (e.g., post-ictal state, medications), neurological deficits (e.g., Parkinson's disease, stroke, dementia), and disorders of the upper gastrointestinal tract (e.g., reflux). Furthermore, recent evidence suggests that in addition to these factors, aspiration is more common in older patients and in those with congestive cardiac failure and chronic liver disease [55]. Hence, a low threshold for swallow assessment is warranted in the multi-morbid older population.
- Consider patient comfort and if chest pain is severe, the use of intravenous opioid medication is indicated.
- If the patient is hypotensive and shocked, supporting BP is essential but consider the potential mechanism and risk of fluid overload (e.g., intravenous fluid bolus could exacerbate acute pulmonary edema in cardiogenic shock, however would be an essential component of treatment to support BP for all other causes of shock).

7.4.7 Vignette 3

Further discussion: The patient had coronary artery disease with a previous myocardial infarction, and risk factors for ischemic heart disease such as diabetes, hypertension and hyperlipidemia. Her vital signs suggested that she was unstable with tachycardia, hypotension, and a raised respiratory rate. The history of her chest pain was typical for ischemic chest pain and was progressively worsening together with increasing shortness of breath. Urgent attention by emergency medicine, internal medicine, and/or cardiology (depending on the site) was necessitated; however, unfortunately she deteriorated rapidly and died during transfer. A high-dose aspirin should be given immediately and emergency assistance sought. In this patient's case, it may have been unsafe to give nitrates for her chest pain due to her low BP. To alleviate pain, IV morphine 2.5–5 mg may be trialed while closely monitoring vital signs. An urgent ECG and portable CXR should be arranged and definitive treatment decided with the results of these investigations.

Key Points

- With shortness of breath or chest pain, serious pathology needs to be ruled out.
- Rapid assessment is required to determine whether the patient is stable or unstable through observation, physical examination, and vital signs. If a patient is unstable, expert advice needs to be sought immediately from acute services.
- Past medical history, medications, and risk factor profile for the various presentations must be considered when assessing a patient with chest pain or shortness of breath.
- ECG and CXR are helpful tests to differentiate different types of pathology.
- If an acute ST elevation myocardial infarction is suspected, the patient needs to be transferred urgently for specialist cardiology input.

7.5 Summary

The three-case vignettes illustrate several acute systemic medical events common in the geriatric population, highlighting the complexity of comorbidity and frailty which is prevalent in this patient population. A psychiatrist is not expected to be an expert in acute internal medicine; however, it is

important to know how to approach the assessment of an acutely unwell patient, to initiate investigation, management, and specialist referral should the need arise.

Take-Away

- Assessment of patient stability and recognition of the critically ill patient is crucial in order to reduce the risk of morbidity and mortality.
- Expedite transfer of the critically ill patient to acute care for definitive management.
- Consider past medical history, current and prior medications used, and any recent changes.
- Consider the potential complications of recent pharmacological and non-pharmacological interventions.
- Involve specialty services as appropriate.
- Involve family members in decision-making as early as possible.

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8 Suicide in the Geriatric Population: Risk Factors, Identification, and Management

Manisha Shenava, Rita Hitching, and Laura B. Dunn

8.1 Introduction

According to World Health Organization [1], individuals who are age 70 years and older have the highest rates of death by suicide in most areas in the world. The lethality of suicide attempts in the geriatric population is higher than all other age groups. The proportion of *completed* suicides among suicide *attempts* is greater in aging individuals than in younger age groups [2]. It is estimated that in the general population, 10 to 20 suicide attempts occur for every 1 death. In the geriatric population, the estimate is *four suicide attempts for every one death* by suicide [3].

Suicide rates among all age groups in the United States have increased between 2000 and 2011, up to 12.7/100,000/year in 2011 (Centers for Disease Control and Prevention, National Center for Injury Prevention). Within the ages 50–74, deaths from suicide almost doubled from 2000 to 2011 [4]. It has been hypothesized that geriatric individuals are more successful in attempting suicide than younger adults, due to comorbid medical conditions, limited physiological reserve, and greater determination. In 2011, white males 85 years or older had a suicide rate of 47.3/100,000/year [2]. It has also been reported that geriatric individuals are methodical, plan their suicides, and may need decisive interventions to prevent suicidal behavior [5, 6]. In sum, it is reasonable to assume that over the next 20 years, geriatric patients with risk factors for suicide will comprise an increasing proportion of the psychiatric inpatient population. The flowchart in Fig. 8.1 offers a stepwise process for decision-making in the assessment of geriatric suicide risk and management.

8.2 Clinical Vignettes

8.2.1 Vignette 1

A 92-year-old widowed woman was found in her senior care unit with a cord tied around her neck and onto her bedpost. The patient was barely conscious. 911 was called, and after a brief emergency department (ED) evaluation, the patient was admitted to a medical unit to rule out injuries and/or any medical sequelae. Superficial abrasions on her neck were found, and she was treated

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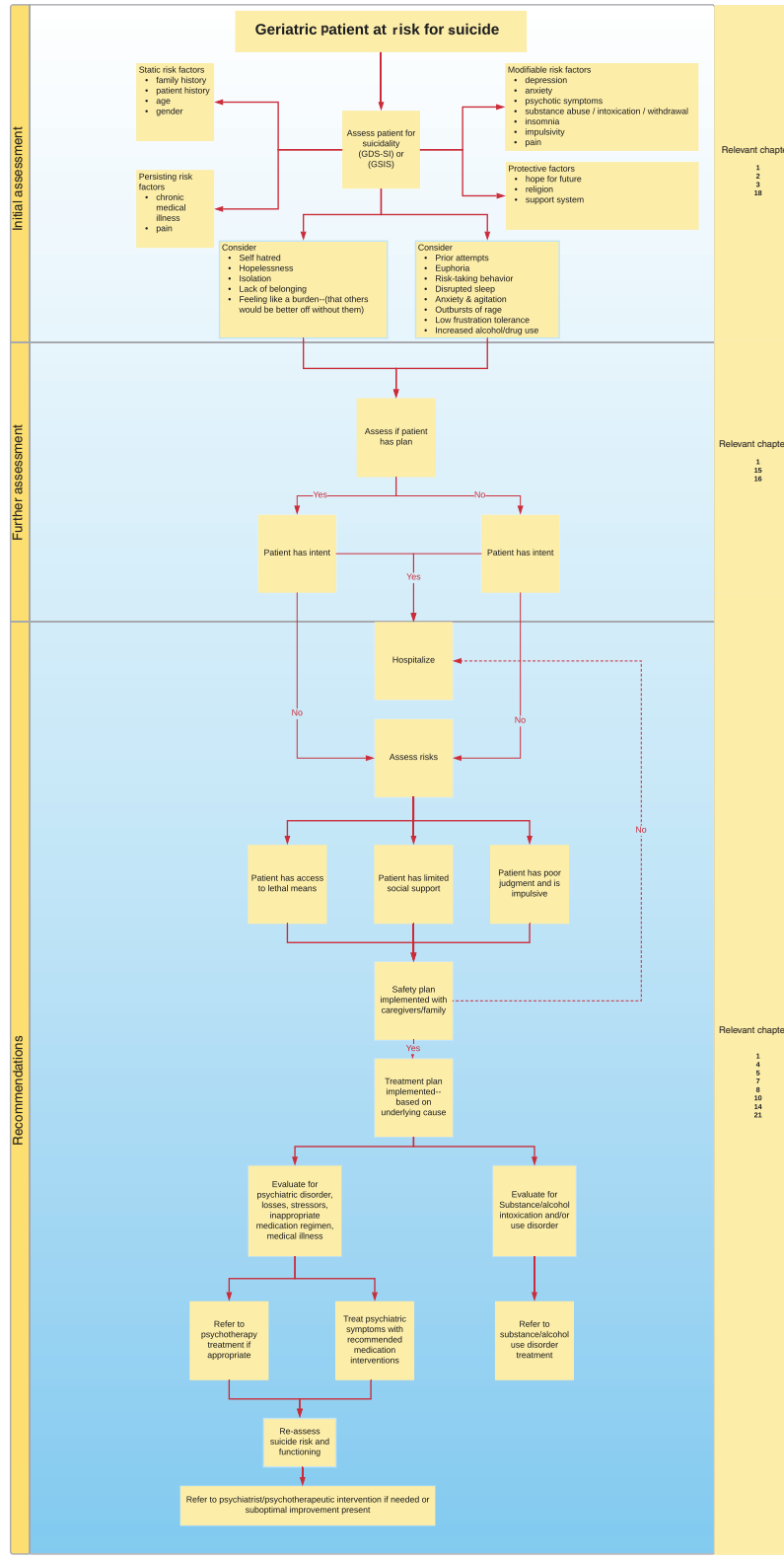

Fig. 8.1 Flowchart: geriatric suicide risk assessment

for uncontrolled hypertension. After 2 days of observation, the patient was transferred to the psychiatric inpatient unit.

There was no known psychiatric history, no history of alcohol/substance abuse, and no prescription of psychotropic medications. Notes from the senior care unit revealed that 3 months earlier the patient expressed difficulty initiating sleep, restlessness, and worry about abandonment. Four weeks prior to admission, the primary care physician prescribed 1.0 mg of lorazepam regularly at bedtime and another 1.0 mg PRN for awakening at night. Within 2 days of starting lorazepam, the patient became less anxious at bedtime and had less difficulty initiating sleep.

After lorazepam was added, a caregiver noted that the patient had less energy and ate less. The patient was still preoccupied with abandonment and was increasingly concerned that her daughter, who called every Sunday, did not call or care about her anymore. The patient's daughter, age 68, however, had continued the regular Sunday telephone calls, but the patient no longer recalled these contacts. The onset of anterograde amnesia due to the benzodiazepine lorazepam was considered.

Over the first 4 days of psychiatric hospitalization, lorazepam was tapered and discontinued. The insomnia and restlessness at bedtime returned within 2 days of the discontinuation. The patient was sullen, withdrawn, and looked more anxious and frightened. Mirtazapine 7.5 mg at bedtime was started for insomnia and poor appetite. The patient talked to daughter by telephone once within the first 4 days.

On psychiatric hospital day #4, the patient was found in the morning with her bedsheet tied around her neck. There was no loss of consciousness and no strangulation. After this event, the environment was scrutinized and further means of suicide removed. The hospital chaplain, social worker, and inpatient psychiatrist each visited the patient daily. It emerged that, since the first day of hospitalization, the patient had become convinced that admission to the psychiatry unit meant permanent placement in a psychiatric facility, accompanied by electroconvulsive treatment (ECT). Indeed, the patient had overheard a discussion of ECT among the team during one of the morning attending rounds.

By psychiatric hospitalization day #6, social workers facilitated contact via Skype with her daughter twice a day, in order to reassure the patient that she was not abandoned. The patient became more verbal. She tolerated the mirtazapine well, which was increased to 15 mg at bedtime, and she began to eat more. Daughter visited on psychiatric hospitalization day #8, and after another 14 days, the patient was discharged to a residential care unit closer to daughter. There were no more suicide attempts, and the patient passed away from pneumonia 3 years later.

8.2.2 Vignette 2

A 66-year-old man with a prostate-specific antigen (PSA) of 4.6 ng/mL was referred by his primary care physician to urology. The patient refused the referral, and within 1 year, his PSA had risen to 14.7 ng/mL. The patient then agreed to see a urologist. A biopsy was not scheduled. Six months later the patient followed up with the urologist, and the PSA was essentially unchanged at 14.8 ng/mL. After another 6 months, the primary care physician obtained a PSA which had risen now to 23.8 ng/mL. A biopsy revealed prostate cancer with a Gleason score of 8, which represented an advanced tumor and a grave prognosis.

A week after the biopsy, the patient developed acute hematuria, and, because he was taking warfarin anticoagulation for chronic atrial fibrillation, he was admitted to a medical unit, where his international normalized ratio (INR) was 4.6. A bone scan found metastases to the spine, iliac bones, and pubic ramus. Hormone therapy was started but was not effective. The warfarin dose was re-adjusted, and his INR stabilized over the next 2 weeks to 3.2. After the first dose of chemotherapy, the patient expressed vague suicidal ideation and was transferred to inpatient psychiatry on an involuntary civil commitment for 72 hours.

Once on the psychiatry unit, the patient was cooperative, though quiet, and showed no obvious signs of major depressive disorder (MDD);

when asked directly, he denied any suicidal intent. He acknowledged that he was preoccupied with his cancer and insisted he was eager to go home to "…finish things up…." The inpatient team could not find sufficient grounds for an extension of the involuntary civil commitment, and he was discharged on the second day of psychiatric hospitalization. At home, he was compliant with the chemotherapy, though he complained that it was intolerable. His wife noted that the patient appeared demoralized. One week after discharge, he killed himself with a gunshot to the head.

The patient's widow sued the urologist for not *initially* recommending a prostate biopsy after the first elevated PSA, nor documenting the patient's "informed refusal" of the biopsy. The widow considered filing a malpractice suit against the inpatient attending psychiatrist for negligence in discharging the patient from psychiatry inpatient unit. Wife's counsel dissuaded her from this legal action due to lack of evidence of negligence; the inpatient notes showed daily assessments for suicidal intent and a risk assessment within 24 hours of discharge.

8.3 Discussion of Vignettes

The vignettes illustrate features common to suicidality within the geriatric population, including:

- Inpatient medical hospitalization can be a stressor in itself and a risk factor for suicide.
- Psychiatric hospitalization can contribute to a sense of abandonment and loss, especially if the patient does not understand the rationale for the admission.
- Discharge from psychiatric hospitalization raises the possibility of a tort claim if there is an untoward outcome, such as a suicide attempt.
- Appropriate documentation of the process of suicide assessment at discharge, and a plan for suicide prevention, can mitigate the risk of a malpractice claim.
- Psychiatric inpatient care should include scrutiny of the environment and adequate monitoring.
- Discharge of the potentially suicidal patient should include a risk analysis and a follow-up plan developed with family/caregivers.
- Medication regimens which have adverse effects on memory, mood, energy, and level of arousal can contribute to suicidality.
- In-depth psychiatric interviewing can supplement screening instruments to provide insights to a patient's motivation.
- White males in the oldest-old age group (age 85 and above) carry a high risk for a lethal suicide attempt, especially in the presence of systemic medical illness, due to their preference for firearms and their reticence.
- Approximately 75% of aging adults who commit suicide have never made a prior attempt [7].
- Loss of spouse is a risk factor for suicidal behavior [8].
- 45% of patients who commit suicide have seen a primary care physician within the previous month [9].

8.4 Risk Factors

8.4.1 Psychiatric Illness and Prior Suicide Attempts

The prevalence of psychiatric disorders [4, 10–13] is high among geriatric patients who die by suicide; however, the *absence* of a psychiatric history does not necessarily moderate the risk of suicide in the aging individual. Many aging adults who commit suicide never sought mental health treatment [9]. In addition, approximately 75% of aging adults who commit suicide have *never* made a prior attempt ([14]; NCIPC 2018). A thorough psychiatric history should be sought in order to provide appropriate treatment; according to several studies, up to 97% of suicide attempts have been linked to a psychiatric illness [15]. Major psychiatric illnesses were found in 71–95% of suicides, with depressive disorders the most common conditions, seen in 54–87% of

Fig. 8.2 Suicide risk factors

suicides [7, 16, 17]. Among depressive disorders, major depressive disorder has been associated with the highest likelihood of suicide. Figure 8.2 illustrates the broad categories of suicide risk factors.

8.4.2 Screening for Suicidal Ideation

In spite of the high prevalence of lethality, routine screening for suicidality among the geriatric population in emergency departments (EDs) may have declined, according to a study by Betz et al. [18]. The authors reviewed 142,534 visits to 8 EDs across the United States. Approximately 23.3% visits were of individuals aged 60 and older [18]. Screening for self-harm (SH), suicidal ideation (SI), or suicide attempts (SA) was documented. The percentage of those patients screened as suicidal was lower with greater age, from about 81% in younger age groups to a low of 68% in those aged 85 and older. *Positive* screens for SH, SI, or SA *also* declined with age of the patient, from a prevalence in young and middle age (9%) to the lowest point: after the age of 75 (1.2%) [18].

In light of the high prevalence of *completed* suicides in a burgeoning geriatric population, screening for suicide risk may need to be given a higher priority [19]. Because aging adults often do not report suicidal thoughts spontaneously, it is helpful to use a screening instrument as a first step. Heisel et al. [20] administered the 15-item Geriatric Depression Scale (GDS) to a cross-sectional cohort of 626 primary care patients (235 men, 391 women), 65 years of age and older. The GDS includes a five-item GDS subscale (GDS-SI) designed to identify suicide ideation. The Hamilton Rating Scale for Depression and the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), were used to validate suicidal ideation. The authors found that the GDS and GDS-SI accurately identified aging patients with suicide ideation. Patients who expressed suicide ideation ($n = 69$) scored higher on the GDS and GDS-SI than those who did not ($n = 557$). Using a GDS cut score of 4 maximized sensitivity (0.754) and specificity (0.815), producing an area under the curve of 0.844 ($P < 0.001$) and positive and negative predictive values of 0.335 and 0.964, respectively. Optimal cut scores were 5 for men and 3 for women. A GDS-SI cut score of 1 was optimal for the total sample and for both men and women. Figure 8.3 lists the 5 screening questions from the GDS-SI scale which indicate suicidal ideation.

The Geriatric Suicidal Ideation Scale (GSIS), another suicide screening instrument, has also been validated in geriatric outpatients [20, 21]. In addition, in a systematic review of several suicidal assessment scales, Batterham et al. [22] found the Beck Scale for Suicide Ideation (BSSI) and the Adult Suicidal Ideation Questionnaire (ASIQ) both showed significant psychometric robustness, although these have not been validated in a geriatric population. Figure 8.4 lists recommended stages of suicide assessment.

8.4.2.1 Physical Illness and Risk

Erlangsen et al. reviewed medical records of 1,849,100 adults, age 65 and above, living in Denmark between 1990 and 2009. Of this group, 4792 died by suicide. An association was found between suicide and 39 systemic medical diseases, with a higher risk of suicide *immediately after discharge* from the hospital, as well as shortly after a diagnosis of cancer [23]. A systematic review of

Fig. 8.3 Geriatric Depression Scale-Suicide Ideation Screening Items (Scoring for the Geriatric Depression Scale (GDS) items involves assigning a response of "yes" or "no" to each item. These items were drawn from the GDS)

GDS-SI

GDS Item	Question
3	Do you feel your life is empty?
7	Do you feel happy most of the time?
11	Do you think it is wonderful to be alive?
12	Do you feel pretty worthless the way you are now?
14	Do you feel that your situation is hopeless?

Suicide assessment process

Stages	Assessment	Tools
Stage 1	Evaluate risk Protective factors & warning	Question, Persuade Refer, (QPR) Recognizing & Responding to Suicide Risk
Stage 2	In-depth clinical interview Assess suicidal ideation Behavior, planning & intent	Chronological Assessment of Suicide Events (CASE) Columbia Suicide Severity Rating Scale (C-SSRS) Beck Suicide Scale (BSC) Depressive Symptom Inventory (DSI) Patient Health Questionnaire (PHQ-9)
Stage 3	Clinical risk formulation	Suicide Risk Assessment (SRA) Suicide Risk Formulation (SRF) Suicide Contract Planning (Safety Plan Template)

Fig. 8.4 Process of suicide assessment

65 articles found suicidal behavior among individuals aged 65 or above was associated with functional disability and specific medical conditions, including malignant disease, neurological disorder, pain, COPD, liver disease, male genital disorders, and arthritis/arthrosis [24]. Serious systemic illness in any organ category (odds ratio 6.4) is an independent risk factor for suicide [25]. Figure 8.5 summarizes the odds ratios for three medical illnesses found highly associated with increased risk of suicide: visual impairment (odds ratio 7.0, 95% confidence interval 2.3 to 21.4), neurological disorders (3.8, 1.5 to 9.4), and malignant disease (3.4, 1.2 to 9.8) [26].

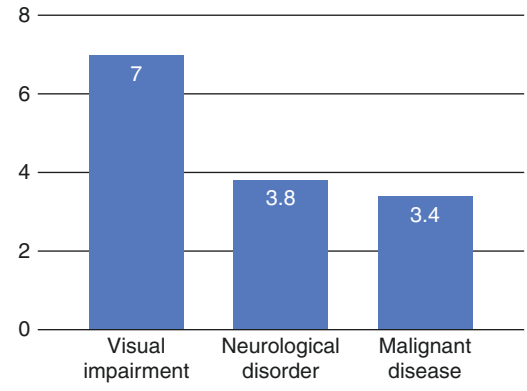

Fig. 8.5 Odds ratios for medical illness and suicide

8.4.3 Medications and Risk of Suicide

Voaklander et al. [27] studied a population-based case-control study of 602 individuals with a mean age of 76 who suicided, matched with 2999 controls. The authors found, in the 30-day period of time prior to the date of suicide, a correlation with the use of *non-recommended* benzodiazepines. The correlation between prior benzodiazepine use and suicide was exhibited even after poisoning-related suicides were excluded from the analysis. The non-recommended benzodiazepines included medications with long half-lives and/or excessive doses: chlordiazepoxide, diazepam, quazepam, halazepam, clorazepate, flurazepam, lorazepam >3 mg, oxazepam >60 mg, alprazolam >2 mg, temazepam >15 mg, and triazolam >0.25 mg. The explanation offered was that *inappropriate* benzodiazepines are those which exceed dose recommendations and/or are medications which persist too long in the geriatric patient, contributing to sedation and depressive symptoms.

Opioid analgesics, such as fentanyl, hydromorphone, and morphine, were also correlated with suicide, and individuals with severe pain were found to be at increased risk of suicide. Table 8.1 compares the odds ratios of benzodiazepines and opioid analgesics at recommended versus *non-recommended* doses. Non-recommended doses of these categories of medications were found to correlate with suicide. Prior prescriptions of these medications, even at *recommended* doses, also correlated with subsequent suicide.

Table 8.1 Odds ratios of recommended vs. non-recommended benzodiazepines and opioid analgesics and suicide

	All suicides ($N = 602$) adjusted odds ratio	Non-poisoning suicides ($N = 474$) adjusted odds ratio
Benzodiazepines		
None	1.0	1.0
Recommended	3.78	3.10
Not recommended	5.85	7.82
Opioid analgesics		
None	1.0	1.0
Recommended	2.10	1.72
Not recommended	6.30	7.56

8.4.4 Gender and Suicide in Geriatrics

Among people who committed suicide, serious illness or disability has been found to be more common in men than in women (65% vs. 44%, $P = 0.05$), and physical illness was associated with a fourfold increase in the suicide rate in men [28]. In this case/control study, consecutive medical records of 210,703 people aged 65 years and over were reviewed. The prevalence of medical illness in those who committed suicide and had undergone forensic examination (46 men, 39 women) was determined from interviews with relatives of the deceased people. This data from the suicide group was compared with medical records of living control participants from the tax register (84 men, 69 women). Physical illness was rated in 13 organ systems according to the cumulative illness rating scale. When the sexes were analyzed separately, serious physical illness was associated with suicide in men (4.2, 95% confidence interval 1.8 to 9.5), as was a high burden of physical illness (2.8, 1.2 to 6.5). No such associations were found in women, possibly because of the small sample size. This finding was consistent with a previous study which showed a similar association of serious physical illness in elderly men who committed suicide (55%) versus women (31%) [29]. In sum, these studies suggest that physical illness may be a stronger risk factor for suicide in men than in women.

White males age 85 and older have been found consistently, among all demographic groups, to have the highest risk for suicide (In 2011: 47.3/100,000/year) [12]. Systemic medical illnesses, pain, disability, hopelessness, and

Fig. 8.6 Rates of suicide in men vs. women geriatric patients [27]

decreased social connectedness have been noted to contribute to suicidality among this group [4, 30–34]. Figure 8.6 demonstrates the rates of suicide in men vs. women geriatric patients [27].

8.4.5 Primary Care Visits and Subsequent Suicide

Ahmedani et al. [35] noted that aging adults who die by suicide have often consulted their physicians within weeks of their death, and >2/3 of older adults who suicided had been seen in primary care within the prior 30 days [36].

8.5 Means of Suicide

Recognition of the various methods of suicide and their prevalence among seniors can help alert caregivers and family of available dangers in the environment. Psychiatric inpatient units can anticipate behaviors and develop policies to restrict access to methods of suicide. One study of 602 seniors who died of suicide between 1993 and 2002, with an annual suicide rate 13.2 per 100,000/year, found high prevalence of use of firearms, hanging, and poisoning [27]. The most common methods used. Figure 8.7 summarizes the percentage of the various methods of suicide in among geriatric patients.

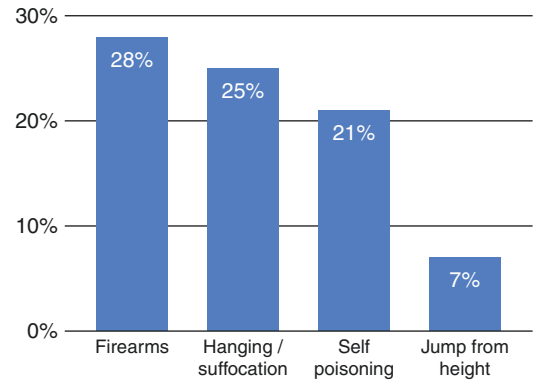

Fig. 8.7 Methods of suicide in geriatric patients

8.6 Major Neurocognitive Disorder and Suicide

Suicidal behavior may also pose a risk factor for the development of major neurocognitive disorders. Using a database in Taiwan, Tu et al. [37] studied 1189 patients aged 65 years and older who attempted suicide, compared to 4756 controls. Geriatric patients who attempted suicide had an increased risk of developing major neurocognitive disorders later in life, independent of major depressive disorder and medical comorbidities [37]. It is also helpful to recognize the means of suicide preferred among patients with major neurocognitive disorder. Seyfried et al. studied patients with major neurocognitive disorder and found that 81:100,000 US veterans with major neurocognitive disorder died by suicide; the three most frequent methods included firearms, hanging, and self-poisoning [38]. Figure 8.8 represents death by suicide and the most common means in US veterans.

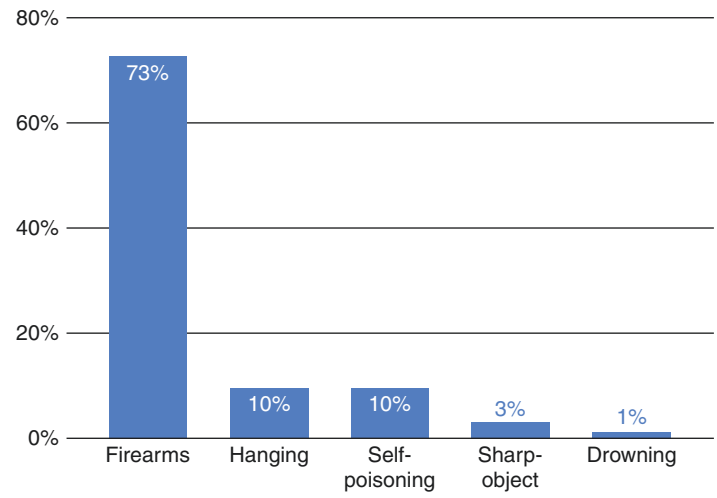

Fig. 8.8 Methods of suicide of US veterans with neurocognitive disorders

8.7 Explanatory Theories

The interpersonal theory of suicide postulates two constructs which are often present together in the suicidal geriatric patient: *thwarted belongingness* and *perceived burdensomeness* [39]. The theory proposes that the various manifestations of social isolation associated with suicide are low social support, loneliness, and living alone. Figure 8.9 summarizes the interpersonal theory of suicide.

Thwarted Belongingness a psychologically painful mental state that results when the fundamental need for connectedness is unmet (seen in Vignette #1). These states of mind are associated with suicide across the lifespan because they are indicators that the *need to belong* has been thwarted [39]. Endorsers of *thwarted belongingness* are more likely to use more immediate lethal means for their suicide attempt and more likely to re-attempt during follow-up.

Perceived Burdensomeness a mental state characterized by thoughts that others would be better off if the person is dead [39] (possibly present in Vignette #2). The interpersonal theory proposes that functional impairment, family discord, and unemployment are associated with suicide across the lifespan because these factors are likely to produce a perception of burdensomeness on others.

Evaluation of the unmet needs of aging individuals may help address these important suicide risk factors. Efforts during an inpatient hospitalization to gather more support (e.g., prior to hospital discharge) can help the aging patient identify sources for greater connectedness and hopefully minimize presence of both constructs. Figure 8.9 illustrates key components of the interpersonal theory of suicide.

8.7.1 Self-Harm Themes

Behavior that does not have the explicit intention of suicide, but remains dangerous, has not been extensively studied in the geriatric population. Wand et al. conducted and studied in-depth interviews of geriatric patients over the age of 80 from two teaching hospitals and community settings, who had engaged in self-harm behavior, in the previous month. The following common psychological themes emerged: "enough is enough"; "loneliness"; "disintegration of self"; "being a burden"; "cumulative adversity"; "hopelessness and endless suffering"; "helplessness with rejection"; and "the untenable situation." Themes for the consequences of self-harm were "becoming engaged with or dis-

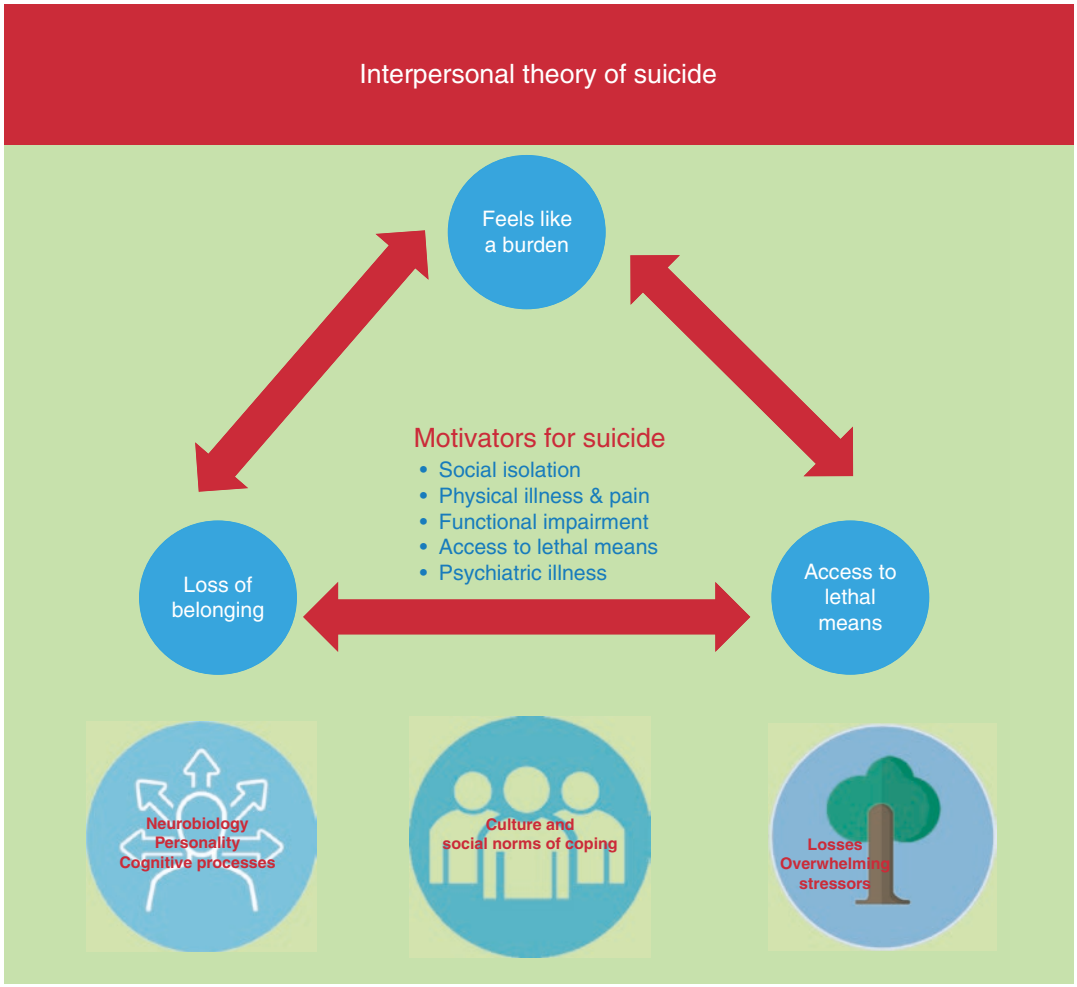

Fig. 8.9 Interpersonal theory of suicide

tanced from family"; "the problem was solved"; "gaining control"; "I'm worse off now"; "rejection by health professionals"; and "tension in the role of the inpatient clinical environment" [40].

8.8 Assessment of Suicide Potential

A suicide risk assessment involves more than informal surveillance or structured screening. A comprehensive, three-stage process can include (1) an evaluation of risk and protective factors as well as warning signs; (2) an in-depth clinical interview to elicit suicidal ideation, behavior, planning, and intent; and (3) a clinical risk formulation [41]. Table 8.2 summarizes some factors which indicate risk and can help inform prevention interventions [42].

8.9 CASE Approach

The Chronological Assessment of Suicide Events (CASE) approach [41] addresses the critical second stage of suicide assessment

Table 8.2 Summary of suicide risk categories correct typo: illness

Social risks	Medical risks	Intrinsic factors
Social isolation	Recent hospitalization for medical illness, especially in the oldest-old (≥ 85)	
Thwarted belongingness		
	Visit to primary care within the prior month	
Burdensomeness	Medical illnesses	Functional impairment
Access to lethal means, especially firearms	Inappropriate prescribing of benzodiazepines, opioids	Male sex
		Caucasian race
		Age 85 or over
	Limited physiological resilience	
	Terminal, intractable, or worsening physical illness	
Widowed		

(clinical interviewing skills) and explores the nature and extent of the patient's suicidal thoughts, feelings, and specific plans. The interview can uncover obstacles (e.g., shame) that may be interfering with understanding the suicidal behavior. Suicide is addressed in an unassuming manner, normalizing any suicidal feelings, to encourage sharing. The use of "shame attenuation" allows the patient's own feelings and words to discuss suicide. The CASE approach explores four regions of the patient's life from the distant past to the present:

- Presenting suicide events (past 48 hours).
- Recent suicide events (over the preceding 2 months).
- Past suicide events (from 2 months ago back in time).
- Immediate suicide events (suicidal feelings, ideation, and intent that arise during the interview itself).

8.10 Interventions

8.10.1 Prevention Plans

Interpersonal interventions: Significant others and caregivers may be asked to participate in the development of suicide prevention plans. The plans vary, depending upon the patient's condition, stressors, living environment, resources, and risks. At the very least, with patient's permission, the risk of self-destructive behavior and clinical severity should be communicated to caregivers, healthcare providers, and family prior to discharge from hospital. Caregivers themselves are also at risk of suicide [43]. Safety contracts have not been demonstrated to provide consistent prevention of suicide [44]. Explanations of the meaning of medical illnesses, identification of support groups, and development of social networks can help mitigate risk [24, 26].

8.10.2 Environmental Precautions

Risk management strategies on the inpatient unit should also focus on establishing an immediate safety plan that incorporates the restriction of access to potentially lethal means (e.g., special rooms/safe rooms, medications). Where potentially dangerous equipment is necessary for patient treatment, the clinician should follow suicide risk management (SRM) protocols [45], as well as policies specific to each facility to minimize possible access to lethal means. These tools can provide a framework on the inpatient unit for determination of severity, and guidance on risk management strategies, based in part upon the lethal means the patient has alluded to [46]. Table 8.3 lists some precautions to minimize the risk of suicide in the environment.

Table 8.3 Environmental precautions

	Fixtures	Environmental
Suggested modifications	Installation of safety glass	Restriction or removal of glass and sharp objects
	Restriction of window openings	Nail polish and removers
	Blocking stairwells	Crochet and knitting needles
	Break-away shower rods and clothing hooks	Matches and lighters
	Covering of exposed pipes, electrical outlets, grills, porch screens, and railings	Cords of all types including blind and drape cords
		Pop cans
		Hangers
		Plastic bags

8.10.3 Pharmacological Interventions

Medications should be used judiciously in the geriatric population, especially in the oldest-old (Chap. 3: Pharmacological Overview). Consideration must be given to the effect that an added medication may have on the entire medication regimen and the impact on the patient's cognition, energy, feeling of well-being, and mobility. Evidence of potential efficacy in the treatment of depression, suicidality, and impulsivity must be weighed against the risk of adverse effects (Chap. 17: Medication Strategies: Switching, Tapering, Cross-Over, Overmedication, Drug-Drug Interactions, Discontinuation Syndromes). Comments are offered below about lithium and antidepressants.

8.10.3.1 Lithium

In a meta-analysis of 48 published medication trials between 1968 and 2013 [47], lithium was compared to placebo, antidepressants, antipsychotics, and mood stabilizers, in the treatment of 6674 patients with diagnoses of unipolar depression (major depressive disorder), bipolar disorder, and schizoaffective disorder. Lithium was associated with reduced risk of suicide compared with placebo. The authors offered the explanation that perhaps lithium reduced recurrence of depressive episodes, as well as mitigated impulsivity. Although this study was not focused upon the geriatric population, an inpatient hospitalization is an ideal location in which to initiate a safe lithium trial, especially for a geriatric patient. An appropriate diagnosis must be present and medical comorbidities/adverse effects considered [47].

8.10.3.2 Antidepressants

The addition of an antidepressant to treat depressive symptoms in the geriatric patient must be weighed against the risk of adverse effects, which can contribute to discouragement and depressive symptoms. One study suggested that the duration of depressive illness should also be taken into account. Nelson et al. [48] reviewed ten placebo-controlled trials of second-generation antidepressants in outpatients with major depressive disorder who were age 60 or older. Those patients with an illness duration of greater than 10 years and a Hamilton Depression Rating Scale score ≥ 21 showed relatively robust drug-placebo difference in their response rates. In patients with a *shorter duration* of depressive illness, drug-placebo differences were *not* robust. If depressive symptoms are of relatively short duration, alternative interventions may need to be considered first.

8.10.4 ECT and TMS

Electroconvulsive treatment (ECT) remains an effective intervention for bipolar and unipolar depression and resulting suicidal impulses. Current ECT administration techniques are designed to minimize adverse sequelae, especially cognitive effects. The benefit of TMS on suicidality awaits further study (Chap. 16: Neuromodulation Interventions: ECT, rTMS).

8.11 Summary

Although the prevalence of attempted suicides is lower in the geriatric population than in younger age groups, a greater proportion of suicide attempts among geriatric individuals are success-

ful [26, 28]. An in-depth evaluation for potential suicidal behavior should be part of the treatment plan of hospitalized geriatric patients and particularly those who are admitted for a psychiatric indication.

Because more time and resources are available on inpatient units than in many other treatment settings, patients, families, and referral sources have a reasonable expectation that a thorough suicide evaluation will be conducted during

Take-Away

In the context of a burgeoning geriatric population, and a relatively high prevalence of suicide in the geriatric age group, screening for suicide risk factors is warranted for geriatric inpatients.

The following general recommendations may reduce suicide risk on the inpatient unit:

- Evaluate for suicide potential regularly and *document* the assessment.
- Ensure medication and treatment compliance.
- Scrutinize the medication regimen for appropriate use of opioids and benzodiazepines.
- Design medication delivery to prevent hoarding and ensure compliance.
- Ensure environmental safety.
- Target and treat symptoms of the psychiatric Axis I conditions.
- Rule out underlying causes of suicidal behavior.
- Record specific suicidal behavior, precursors, and correlates.
- Implement a plan for monitoring, reassessment, prevention, and follow-up after discharge.
- Note that physical illness may be a stronger risk factor for suicide in men than in women.

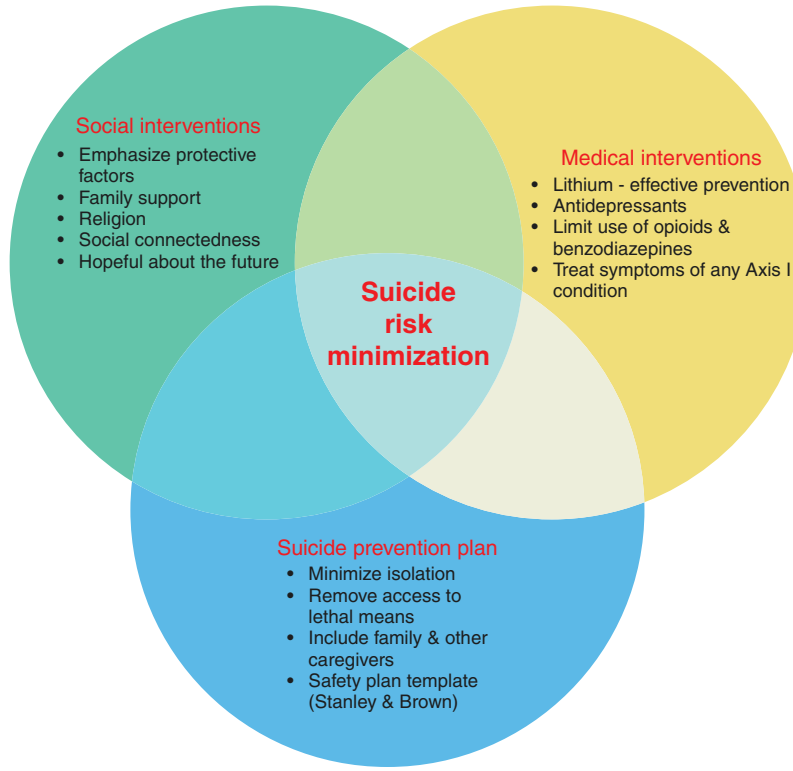

Fig. 8.10 Interventions to minimize the risk of suicide

the hospitalization. Cursory checklists or similar screening instruments alone cannot be relied upon to discern a patient's attitudes and impulses. Persistent interviewing can uncover underlying perceptions, such as thwarted belongingness and burdensomeness, as well as suicide plans. Prevention of suicidal behavior and its management in geriatric patients must include a recognition of risk factors for suicide, management of undertreated psychiatric conditions, elimination of environmental hazards, detailed follow-up plans, and collaboration with family and caregivers.

One must keep in mind that if suicidal behavior occurs during hospitalization, or shortly after discharge, medical records may be scrutinized for documentation of appropriate suicide assessments and prevention plans. If the psychiatric/medical evaluation and suicide prevention planning are not within the standard of care, the risk of a tort claim is increased. Figure 8.10 illustrates some strategies to minimize suicide risk.

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9 Sleep in Geriatric Psychiatry Inpatients

Kim A. Hardin and Zachary C. Ryder

9.1 Introduction

It is estimated that 40–70% of aging adults experience chronic sleep problems, which are often unrecognized and can lead to chronic sleep deprivation [1, 2]. Sleep deprivation is known to negatively affect cognitive and physiological functioning [3, 4]. Systemic medical and psychiatric illnesses (including neurocognitive disorders), pain, and medications can contribute to sleep disruption, as well as the presence of a primary sleep disorder. Hospitalization further causes sleep disruption and exacerbates underlying sleep problems, which can contribute to cognitive dysfunction, pain, delirium, and delayed recovery time [3]. Figure 9.1 summarizes the sleep problems and treatment of disrupted sleep in hospitalized geriatric patients.

We review changes in sleep physiology that occur with aging, disorders that can impact sleep in this population, and how these can affect daytime and nighttime function. Knowledge of these factors enhances diagnosis and treatment strategies tailored to the geriatric patient, particularly in the hospital setting (Fig. 9.2).

9.2 Vignettes

9.2.1 Vignette 1

A 78-year-old woman with anxiety, hypertension, and coronary artery disease was admitted to the inpatient psychiatric unit for progressive paranoia. During the daytime, she was confused and tired. She refused to take her medications, which included venlafaxine, sertraline, metoprolol, aspirin, and vitamin D. She was started on citalopram 10 mg daily for depression and paranoia. She would awaken at night and sit on the side of her bed. She was sweaty but denied other complaints. She continued to be tired and frequently was found dozing. She would jump awake with a startled expression.

Sleep medicine was consulted. A portable, limited channel sleep study was conducted to evaluate for sleep apnea. The test was positive, and she was started on positive airway pressure (PAP) therapy. She had some difficulty using it initially. Over the next week, she had improved daytime cognition and energy and no longer had startle episodes and stated that she slept better at night.

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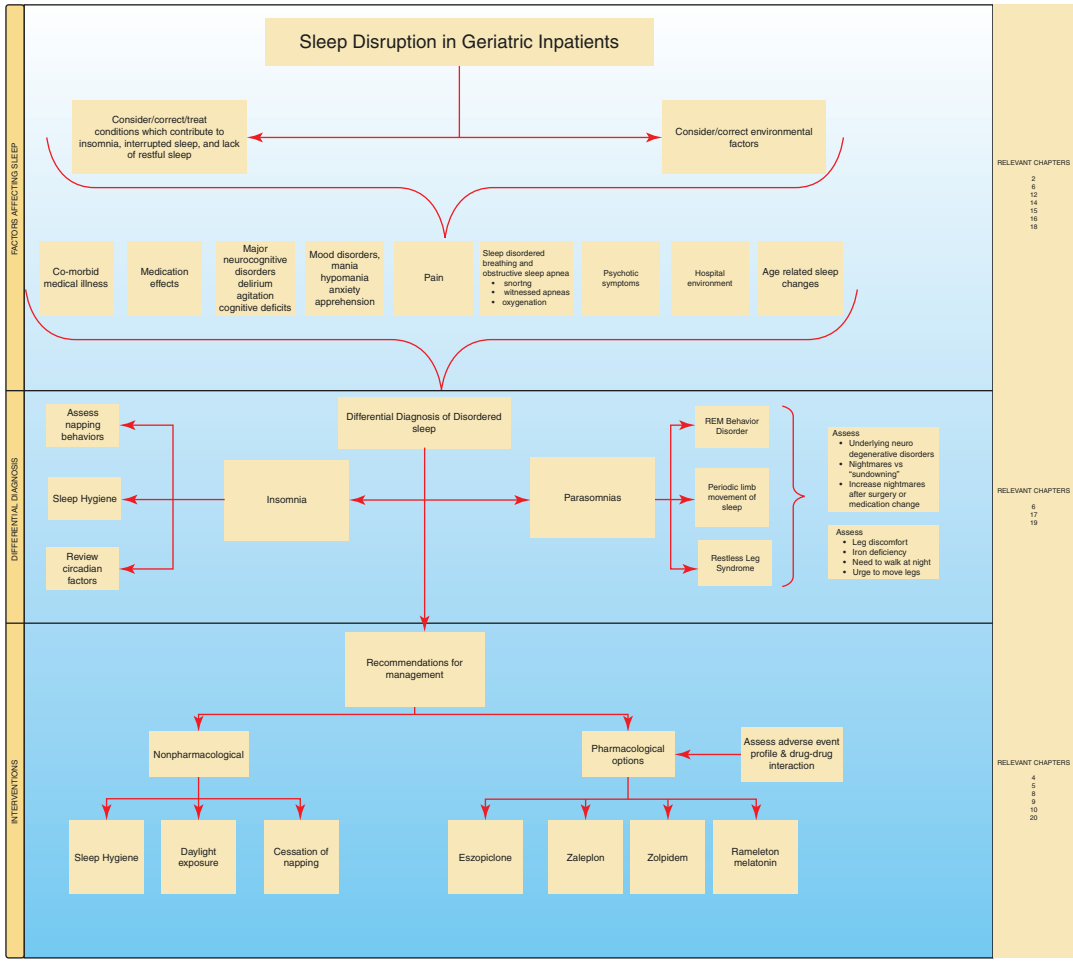


Fig. 9.1 Flowchart of sleep problems and treatment of sleep in hospitalized geriatric patients

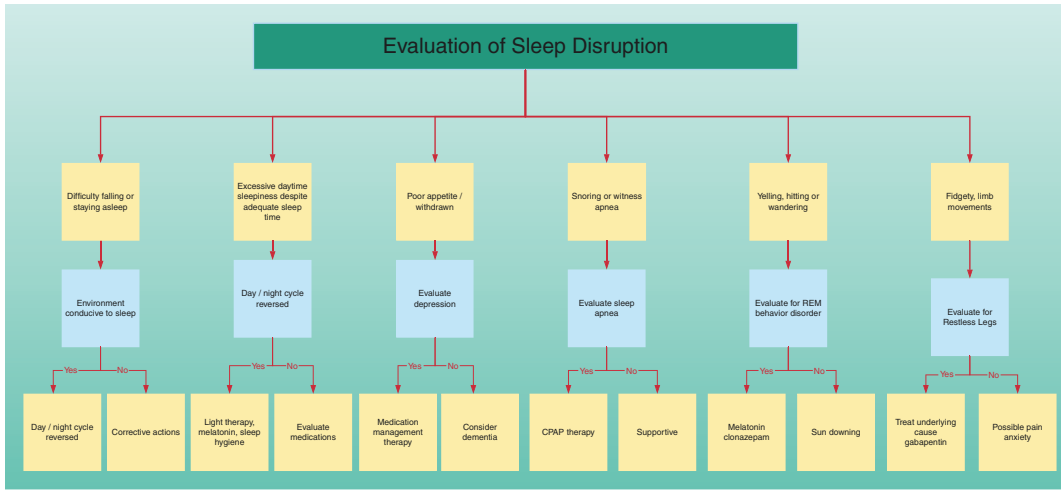


Fig. 9.2 Evaluation of sleep impairment

9.2.2 Discussion

Individuals with sleep apnea have fragmented sleep, which results in daytime sleepiness, lethargy, and decreased cognitive function. Chronic sleep deprivation may make depressive symptoms worse or may prompt a misdiagnosis of depression. Ongoing sleep loss, particularly in older individuals, can result in increased confusion. Symptoms may include choking and gasping, sweating, need to sit up or open windows for air, and need for daytime naps. Women complain of fatigue and poorer daytime function, with less snoring, and are diagnosed with depression more frequently compared with men of similar age and severity of sleep apnea. A high level of suspicion is necessary in this population. Portable, limited channel sleep studies measure only cardiorespiratory parameters and cannot infer information regarding sleep architecture. They are useful in situations wherein there is a suspicion suggested by the *symptoms*. Additionally, comprehensive in-lab sleep studies are usually not attainable while patients are in the acute hospital or inpatient psychiatric setting. When the need for diagnosis of sleep apnea may impact patient management, a *portable limited channel study* can be useful.

9.2.3 Vignette 2

A 70-year-old man with Parkinson's disease and restless leg syndrome (RLS) was treated with carbidopa/levodopa and ropinirole. He would cry and tell his wife that he was afraid to go to sleep. He became more withdrawn and his wife could no longer care for him. He was admitted to a facility for patients with major neurocognitive disorder (MNCD) and treated for depression. However, he became more restless at night, thrashing about in bed, kicking his legs and arms. His ropinirole was subsequently increased for presumed worsening RLS. However, he became even more agitated and fearful, wandered, and had hallucinations. He was started on clonazepam at bedtime. He continued to have hallucinations, anxiety, and increasing paranoia. Ropinirole was discontinued and carbidopa/levodopa dosage was reduced. His nighttime thrashing and restlessness improved, but hallucinations continued, and he was subsequently started on pimavanserin.

9.2.4 Discussion

Individuals with Parkinson's disease are at increased risk of RLS and REM behavior disorder (RBD), as well as depression and neurocognitive dysfunction. Initial medication management of Parkinson's disease, which also treats RLS and RBD, is with dopaminergic agents. However, as major neurocognitive disorder (MNCD) increases, excessive dopamine may worsen the hallucinations and aggravate psychosis. Decreasing the ropinirole and carbidopa/levodopa, or stopping it altogether, is the first line of therapy. Clonazepam can be used to treat RLS, RBD, and agitation, thereby promoting sleep at night and improved circadian regulation. Clonazepam may have had no effect on hallucinations, which persisted in this case. Pimavanserin (a selective serotonin reverse agonist), can be used to treat refractory hallucinations or psychosis, but only after the dopaminergic agents would be discontinued or reduced (Chap. 11: Psychiatric Symptoms Co-morbid with Neurological Syndromes).

9.3 Sleep Architecture and Changes with Aging

9.3.1 Normal Physiology

Sleep is comprised of two phases: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM sleep is further divided into light sleep (stages N1 and N2) and deep or slow-wave sleep (SWS or stage N3) [5]. REM sleep follows N3, and each sleep cycle is approximately 90–120 min. SWS has longer periods in the first half of the night, whereas REM sleep has longer periods in the later part of the night. SWS is an anabolic phase of sleep

associated with bodily restoration and repair. REM sleep is hypothesized to be necessary for memory consolidation and cognitive and emotional well-being.

The National Sleep Foundation (NSF) recommends that adults aged 26–64 sleep 7–9 h per night and adults aged 65 and older sleep 7–8 h per night. Healthy sleepers can experience up to ten short arousals (i.e., awakenings on the order of just 3 s long) per sleep hour [6].

The sleep/wake cycle is a 24-hour intrinsic rhythm that is controlled by the master clock located in the suprachiasmatic nuclei (SCN) of the hypothalamus and regulated by several genes [7]. Various extrinsic stimuli influence the expression of this clock, with *light* as the strongest stimulus for wakefulness. The suprachiasmatic nucleus regulates melatonin secretion by the pineal gland. Melatonin modifies circadian rhythm and signals day-night transitions. Melatonin levels in the pineal gland are low during the daytime and increase after the onset of darkness, reaching peak levels when it is darkest.

Sleep is also controlled by a homeostatic drive to sleep. The greater the sleep loss, the greater the drive to sleep until the ability to maintain wakefulness is overcome and sleep ensues regardless of time of day or location. Thus, the homeostatic system promotes the amount of sleep needed, whereas the circadian system optimizes the best timing to sleep. Disturbance of these systems can result in abnormal sleep patterns, fragmented sleep, and sleep deprivation, causing psychological and physiological sequelae [8, 9].

9.3.2 Specific Sleep Changes with Age

Sleep architecture varies across the age span. Changes in sleep patterns occur due to changes in circadian regulation, response to circadian signals, and environmental stimuli. Degeneration of cells in the SCN has been shown to occur in aging adults, particularly those with MNCD [10–12]. There also seems to be a reduced response to light entrainment to facilitate wakefulness. Light transmission requires specific retinal photoreceptors to transmit light input to the SCN clock.

Many aging adults have cataracts or retinal deterioration that can reduce blue light transmission [12]. This can create shifting of sleep onset to an earlier time, known as advanced phase syndrome. This often leads to unwanted early morning awakenings. Some studies have also demonstrated a reduction in circadian core body temperature amplitude, and melatonin levels, which influence sleep onset. Melatonin is released from the pineal gland, and its output is regulated by the SCN [13]. A lower level of melatonin at night may contribute to difficulty maintaining sleep and earlier wake time. Adenosine levels, which promote sleep, increase with age, suggesting an increased sleep drive; yet aging also appears to be associated with adenosine resistance, which might reflect reduced sleep ability.

Geriatric individuals tend to be less active, with decreased social interaction or exposure to daylight. This can create interference with staying awake in evening and can lead to further social isolation. Unwanted early morning awakenings follow, which may cause an inability to carry out necessary daytime activities [14]. Studies have consistently shown that *healthy* older adults experience decreased total sleep time (TST), sleep efficiency (SE), deep sleep (N3 or SWS), and *more frequent awakenings and arousals*, with greater difficulty initiating and maintaining sleep independent of comorbid medical conditions. However, many of these studies had methodological limitations [15–17]. A large meta-analysis of 3577 subjects ages 5–102 years was conducted and confirmed that TST, SE, percentage of SWS, and REM sleep all decreased with age [18]. Sleep latency (SL) increased with age, but this change was very subtle. Moraes et al. [19] then conducted the first large population-based study with a sample size > 1000 participants, which included both genders, various ethnicities, and ages ranging from 20 to 80 years. Subjects with psychiatric and/or systemic medical conditions that limited independence, shift workers, and pregnant women were all excluded. Polysomnography (PSG) was performed on each individual. The results

Table 9.1 Sleep architecture across the age span

Age->	20–24	25–34	35–49	50–69	70–75	75–80
TST (min)	375	365	338	320	292	291
SE (%)	87	86	80–82	75–79	72	65
SL	13.8	14.8	17	20	28	35
% N1	3.8	4.3	4.5–4.8	5.4[a]	4.9	6.4
% N2	54	53	54	54.5–57	55.4	58
% N3	23.6	21.8–23.2	20.9–23[b]	21.3	23	18.3
% REM	18.4	20.3	19.6	17.8	16.5	17

Data derived from: Moraes et al. [19]
Key: *TST* total sleep time, *SE* sleep efficiency, *SL* sleep latency, *N1* stage N1 sleep, *N2* stage N2 sleep, *N3* stage N3 sleep, *REM* rapid eye movement sleep
[a]at age 65, a decrease started at 4.9%
[b]larger variability in SWS

Table 9.2 Indicators of poor sleep

	SL	WASO	Aw	N1	N2	N3	REM
	> 60 min	> 51 min	> 5 min	> 21 min	> 81%	< 5%	< 21% or > 41%

Key: Seep latency (SL)/wake time after sleep onset (WASO)/rapid eye movement (REM)/stages of sleep (N1-N3)

verified Ohayon's meta-analysis [18] that older adults had decreased TST, SWS, and REM sleep but also more difficulty with sleep onset (Table 9.1).

Sleep fragmentation was also assessed by the number of arousals per hour causing disruption of sleep. The average arousal index (AI) was 8.8/hour in the 20–24-year-old group and demonstrated a positive correlation with increasing age. Most of the arousals were secondary to respiratory-related events. Periodic limb movements (PLMs) were also shown to increase with age with a mean of 0.7/h at age 20–10/h at age 75; whether they were associated with arousals or awakening was not reported. Gender differences also occurred with women having slightly more SWS than men for the same age, but less REM sleep. It has been previously shown that women tend to have better sleep quality, as well as quantity of deep sleep compared to men of similar age, but have significantly more complaints of daytime fatigue and poorer daytime function [19]. Decreased sleep has also been associated with negative outcomes in the nursing home setting, including decreased survival [20]. Table 9.1 shows sleep architecture across the age span from 20 to 80.

The National Sleep Foundation (NSF) formed an expert panel to formulate guidelines in sleep quality parameters [21]. Over 277 studies were included in the analysis, with ages ranging from infancy to geriatric population. Consensus was difficult to establish in some parameters, particularly in the older adult population. Results in Table 9.2 indicates >80% consensus among the panel regarding sleep continuity variables and what would be considered poor sleep. The following parameters were determined: SL > 60 min indicates poor sleep quality for all ages. For older adults, adequate SL can be up to 60 min, and less than 30 min is considered good quality. For all ages, > 4–5 awakenings are considered as poor sleep. Wake time after sleep onset (WASO) should be < 20 min, and in all age groups, > 51 min indicates poor quality of sleep. REM < 21% was considered poor. In addition, REM > 41% in any adult population indicates poor sleep or an abnormality in sleep. Poor sleep is also indicated if N1 is > 21%, N2 > 81%, or N3 < 5% in older adults.

Napping behavior was also evaluated as a sleep parameter in the NSF analysis [21]. There was less than 80% consensus in whether daytime napping is appropriate in middle- and older-age adults. Napping more than once in a 24-h period or for >100 min/nap was definitely considered an indication of poor sleep. Another community-based study in older women suggested that short sleep at night, poor sleep efficiency (SE), and increased napping during the day are all associated with increased risk of falls, as well as increased risk of shorter survival [20].

Despite multiple studies illustrating sleep quality is worse in older adults and its negative consequences, expert consensus requires severe deprivation of deep sleep or excessive delay in sleep onset and increased wake time to consider sleep quality as poor. Given the above evidence, more stringent standards for deficient sleep may be considered. Further prospective studies are needed to determine if such poor sleep correlates with poor health outcomes.

9.4 Factors that Disrupt Sleep in the Hospitalized Aging Population

When daytime sleepiness or sleep problems are present in geriatric patients, it is essential to assess whether sleep duration, quality, and timing are adequate, as well as hospital-related and medical factors that may impact sleep quality. Sleep impairment may be multi-factorial and ongoing; repeated evaluation is required as therapies are instituted and the patient's condition changes (Fig. 9.3).

There are many challenges and barriers to assessing sleep and management in the inpatient setting, including hospital environments, patient-related issues, ability to measure and track patient sleep, as well as instituting good practices to promote sleep. The next sections discuss these barriers and suggestions for promoting better sleep during hospitalization.

9.4.1 Environment

Normal age-related changes that occur with neurologic regulation and environment predispose the older community to advanced phase circadian disorder, i.e., going to bed early and waking up early. However, environment is a significant contributor, particularly for individuals living in residential facilities and hospitals. Even in the geriatric community-dwelling population, contemporary western society has created an environment that leads to sleep disruption. Computer use, smartphone use, Internet activity, and TV viewing late at night are present almost everywhere.

Many studies have demonstrated sleep disruption and fragmentation in the hospital setting [3, 4]. While present in all hospital units, this is even more severe in nursing homes. There is a decrease in deep sleep and REM sleep. Because of frequent disruptions, in order to attain a total sleep time of > 5 h, sleep occurs over the 24-hour period rather than being consolidated to the nighttime hours. This can disrupt the individual's normal pattern.

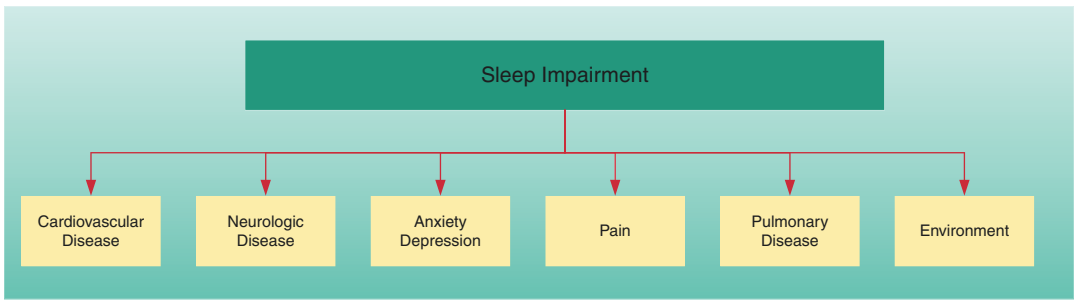


Fig. 9.3 Factors that may impact sleep quality

Studies measuring sleep in the hospital setting have been conducted using polysomnography, which is cumbersome and not practical for daily use. Therefore, staff observation is the means of verifying a person's sleep state. With the current technology, there are many inexpensive over-the-counter devices that are actigraphy-based measure movement and assume non-movement to be sleeping and may be an alternative means of tracking sleep behaviors in patients who are complaining of poor sleep or in those noted to be restless. Since electroencephalography is not performed, an absolute quantification or qualification of the stage of sleep cannot be made.

Noise, light, cold/hot temperature, uncomfortable bedding, and nursing and medical interventions all create constant disruption throughout the night- and daytime period. These environmental factors can be altered, and it has now become standard in acute care hospitals to institute dedicated "sleep time" at night with stricter measurement of noise and light, as well as dedicated "rest or sleep time" in the early afternoon. Although sleep is a necessary component for healing, napping multiple times throughout the daytime period may hamper consolidated nighttime sleep and contribute to restlessness, confusion, and delirium at night. Patients who are more restless and disoriented at night often receive more sleeping medications, which have negative consequences. Because patients are ill and often sleep-deprived, fixed nap times earlier in the day may facilitate optimum recovery and functional status. Table 9.3 lists common environmental

Table 9.3 Sleep barriers in hospital

Sleep	Factors	Sleep optimization strategy
Nighttime		
	Overnight monitoring	Evaluate orders to decrease frequency vital signs, labs, glucose monitoring
	Non-urgent clinical care tasks	Hold bathing, dressing changes, I/O monitoring
	Medications	Change medication regimens to promote sleep (e.g., nighttime diuretics); give medications before 9:00 P.M.
	Minimize administration benzodiazepines for sleep	
	Sleep hygiene	Maintain routine
		No napping in late afternoon
Noise		
	Reminders	Daily hospital announcement/floor announcement of quiet hours
		Signs indicating quiet hour times and reminder for quiet conversations
	Visitors	Limit number of visitors, enforce visiting hours
		Post signs to minimize conversations at or near bedside
	Patients	Offer ear plugs
		Offer headphones for television; limit the use
		Close door to patient rooms
		Turn down phone ringer volume
Nocturnal		
	Reflux	Head of bed greater than 30 degrees; limit PO intake after 8:00 P.M. antacid therapy if appropriate
	Pain	Optimize pain management regimen/strategies during
		Remove Foley catheters
		Keep room at a comfortable temperature
Light		
	Nighttime	Offer sleeping masks
		Limit continuous nighttime light as much as possible
	Daytime	Promote daytime wakefulness with bright lights on, window curtains pulled open to allow natural light, promote physical activities, no late afternoon napping

barriers that can disrupt inpatient's sleep and improvement strategies [22]. Change in inpatient culture, staff behavior, and elimination of unnecessary activities, such as routine nighttime vital signs, have all facilitated patient-centered practices that promote sleep.

9.4.2 Comorbid Systemic Medical Illness

Many geriatric inpatients, especially the very old, have several systemic medical problems with symptoms that prevent quality sleep. Cardiopulmonary disorders, such as congestive heart failure (CHF) and chronic lung disease, produce shortness of breath (SOB), nighttime cough, and nocturia. These symptoms can be exacerbated by the normal physiology that occurs during sleep itself.

Normally during REM sleep, there is muscle atonia; i.e., all muscles are paralyzed except the eye muscles and diaphragm, which creates an erratic respiratory pattern, with increased work of breathing and lower oxygenation. Patients who rely on accessory muscles of breathing may have more pronounced symptoms and may awaken dyspneic, with feelings of suffocation and fear of dying. This results in chronic sleep deprivation with daytime symptoms of fatigue, excessive sleepiness and diminished cognitive function, impaired ability to carry out daytime tasks, and lower quality of life (QoL).

Although discussion of each factor is beyond the scope of this chapter, comorbid illnesses, such as vascular disease, cancer-related illness, pulmonary disease, metabolic disease, nocturia, pain, and psychiatric disorders, such as depressive and anxiety disorders, are common contributing etiologies. Table 9.4 lists common medical diseases, associated sleep-related issues, and management suggestions. Effects on both daytime functioning and nighttime sleep quality are included.

Table 9.4 Medical problems, implications, and strategies to improve sleep

Disease	Clinical implications	Strategies to improve sleep
Asthma	Exacerbations and symptoms may worse at night	Optimize asthma medication treatment
	GERD is worse lying flat and increases the risk of aspiration	Allergen free bedding
COPD	Persistent nocturnal hypoxemia/ REM-associated oxygen desaturations	Oxygen supplementation to maintain Pa02 55-60 mmHg
		If hypercapnia, evaluation for NIV
ESRD	Increased risk of restless leg-walking	Treat iron deficiency
	Dyspnea	Medical management with gabapentin or dopaminergic agent, e.g., ropinirole
	Pruritus	
Stroke	Focal neurologic deficits; e.g., dysphagia, weakness, paralysis	Elevate head of bed (HOB); maintain sleep-wake schedules
	Assessment for SDB	
Pain	Increased immobility	Use of orthotic devices; position management for comfort
	Frequent awakenings	Medical management with scheduled acetaminophen, gabapentin
CHF	Orthopnea, PND, CSR, CSA, nocturia	Elevate HOB
		Diuretic therapy adjustment for daytime diuresis

Abbreviations: *CHF* congestive heart failure, *COPD* chronic obstructive pulmonary disease, *HOB* head of bead, *GERD* gastroesophageal reflux disease, *NIV* noninvasive ventilation, *CSA* central sleep apnea, *CSR* Cheyne-stoke respiration, *HOB* head of bed, *LABA* long-acting bronchodilator agent, *LAMA* long-acting muscarinic agent, *PND* paroxysmal nocturnal dyspnea, *SDB* sleep disordered breathing

9.4.3 Medications Which Disrupt Sleep

Medications for a variety of conditions may also disrupt sleep in inpatient settings. These include antidepressants, anti-hypertensives (e.g., beta-blocker, alpha-blocker), antiparkinsonian drugs (e.g., levodopa), bronchodilators (e.g., theophylline), steroids, antihistamines (e.g., H1 and H2 blockers), and diuretics. Table 9.5 lists commonly used medications and their effect on sleep [23].

Table 9.5 Medication sleep-related side effects

Medication category	Examples	Sleep-related side effects	Administration of medication
Cardiovascular drugs			
Lipophilic	Propranolol	Suppress melatonin secretion	Scheduled dosing 0800 and 1800
B-blockers	Pindolol	Fatigue nightmares	
		Metoprolol	Atenolol is nonlipophilic and less associated with sleep disturbance
		Timolol	
Ca channel blockers	Amlodipine decreased esophageal sphincter tone >> GERD >> sleep disturbance	Scheduled for morning dosing and avoid nighttime dosing	
	Verapamil	Exacerbates RLS/PLMD	
	Nifedipine		
Diuretics	HCTZ	Nighttime diuresis >> frequent nighttime awakenings	Schedule dosing for A.M. and early evening such as 6:00 P.M.
	Furosemide		
CNS Drugs			
Bupropion		Exacerbate insomnia and headaches	Consider in those with EDS and depression
		Exacerbate narcolepsy/cataplexy as increased REM	
SNRI	Venlafaxine	Can either be activating or sedating in some patients	Venlafaxine effective for insomnia, depression
	Duloxetine	May cause vivid dreams or nightmares	
SSRI	Sertraline	Paradoxical effect in some patients causing activation rather than sedation	Stop if history of Parkinsons or RBD
	Fluoxetine	May worsen RLS/PLMD	
	Citalopram	Unmask REM sleep behavior disorder (RBD)	
	Escitalopram		

Modified from Duong et al. [23]
Abbreviations: *CNS* central nervous system, *EDS* excessive daytime sleepiness, *HCTZ* hydrochlorothiazide, *PLMD* periodic limb movement disorder, *RLS* restless legs syndrome

9.5 Sleep, Cognition, and Major Neurocognitive Disorder (MNCD)

Sleep deprivation affects cognitive function, but conclusive evidence has not yet shown whether or not sleep is a risk factor for neurologic injury and/or major neurocognitive disorder (MNCD). Psychiatric disorders may also be exacerbated by MNCD, poor sleep, and medications used to treat these illnesses. Several studies have evaluated sleep disturbance in older adults and found inconsistent results regarding which factors may be related to change in cognition [24–26]. Subjective excessive daytime sleepiness (EDS) seems to be a more predictive measure associated with cognitive change. In the Cognitive Function and Aging Study conducted in community-living adults (age > 65 years), short sleep duration (< 6.5 h/night) and EDS at baseline were 105% and 121%, respectively, more likely to have cognitive impairment at 10 years, whereas baseline napping was associated with a 62% reduction in risk of cognitive decline. Napping was not related to prior sleep duration, and those who napped for > 1 h maintained cognitive protection at 10-year follow-up [26]. But, since actual sleep was not measured, sleep quality cannot be substantiated. Interestingly,

this seems to contradict the expert consensus statement that napping is reflective of poor sleep. Further investigation is warranted whether modification of sleep behaviors is neuroprotective.

Alzheimer's disease (AD) is the most common cause of MNCD in western countries. AD is associated with the degeneration of the cholinergic neurons in the nucleus basalis of Meynert, the pedunculopontine tegmental and laterodorsal tegmental nuclei, and noradrenergic neurons of the brainstem that may induce reduction of REM sleep in Alzheimer's disease (Fig. 9.3) [27]. Degeneration of the brainstem respiratory neurons and the supra-medullary respiratory pathways may place the patient at risk of sleep-disordered breathing (SDB).

Patients with Alzheimer's disease and other MNCDs are more prone to the circadian rhythm disruption known as "sundowning." Sundowning is associated with increased confusion and behavior problems in the evening or night and may have an underlying neurochemical basis due to loss of acetylcholine [28]. It is a common cause of institutionalization in patients with Alzheimer's disease and often leads to the empirical off-label use for anti-antipsychotic medications for sedation and agitation management. [Chapter 6: Major neurocognitive disorder with behavioral disturbance]. Use of bright light in the morning and strict sleep hygiene and environmental control measures helps to maintain circadian regulation. Insomnia may also occur and is addressed later in this chapter.

Current pharmacotherapy for cognitive decline in Alzheimer's disease involves the use of cholinesterase inhibitors (e.g., donepezil, galantamine, rivastigmine). However, these medications can increase REM sleep density, exacerbate insomnia, and induce vivid dreams causing more sleep disturbance, which results in greater subsequent treatment with sedative-hypnotics or other psychotropic medications. Melatonin supplementation may facilitate restoration of circadian rhythm and improve sleep. Melatonin levels diminish with aging, and patients with Alzheimer's disease have a more profound reduction in this hormone. Data from clinical trials indicate that melatonin supplementation improves sleep and slows down the progression of cognitive impairment in Alzheimer's disease. Melatonin has antioxidant properties and protects neuronal cells by arresting the formation of amyloid fibrils [29].

9.6 Primary Sleep Disorders in the Hospitalized Geriatric Population

Sleep disturbances may be related to many factors in geriatric patients. Disruption of sleep can result in excessive daytime sleepiness. Habitual snoring and witnessed apnea during sleep are signs of obstructive sleep apnea (OSA). Insomnia can be due to a variety of etiologies. Sleep-initiation and/or maintenance problems that are accompanied by restlessness of the lower extremities should prompt evaluation for restless leg syndrome (RLS). REM sleep behavior disorder (RBD) should be suspected when nocturnal vocalization, sleep talking, and abnormal movements or behavior related to dream content are witnessed by a bed partner or a nursing staff member. Sleep disorders commonly observed in older adults as well as their causes and treatment are reviewed.

9.6.1 Insomnia

Insomnia is the inability to initiate or maintain sleep (despite adequate time and environment), which results in daytime sleepiness and fatigue. Insomnia is reported to occur in >40% of individuals greater than 60 years of age, making it the number one sleep disturbance in this population [30]. Although sources differ in terms of specific criteria, any underlying factor, such as comorbid systemic illness, pain, other sleep disorders, and psychiatric illness, should be effectively remedied before the diagnosis of primary insomnia can be established [31, 32]. Other factors that may cause insomnia in the older adult include

delirium (whether or not associated with comorbid MNCD), anxiety disorders, depressive disorders, alcohol use disorder, psychological factors, and maladaptive response to major life events (e.g., loneliness following the death of a partner/spouse, adjustment to hospitalization). Although quality of sleep declines with age, older adults tend to complain less than younger individuals [5]. This may be due to acceptance or tolerance to poorer health or unconscious adjustments in daily living.

Significant adverse consequences of insomnia (non-restorative sleep) have been documented [33]. Stone et al. investigated the risk of falls in 2978 community-living women greater than 70 years of age and noted that sleep duration < 5 h/night and sleep fragmentation (frequent awakenings) were associated with increased risk of falls (odds ratio, 1.52; 95% CI, 1.03–2.24), independent of medications or other conditions [34]. Falls in aging persons can result in hip fractures, which are well known to result in a serious decline in health and QoL. Thus, recognition of insomnia and related factors is imperative in preventing injury and maintaining optimum functionality.

Insomnia also co-exists in approximately 40% of individuals with psychiatric illness. Insomnia predicts the later development of depressive disorder within 1–3 years and precedes anxiety disorders 38% of the time. Older patients with insomnia have a 23% increased risk of developing depressive symptoms compared to those without insomnia [35]. The IMPACT study showed that a majority of patients with persistent insomnia had higher prevalence of continued depressive symptoms as compared to those without insomnia [36]. Loss of appetite and interest in addition to insomnia may suggest depressive disorder. In patients without a prior psychiatric history, insomnia in the preceding year—compared to controls who did not have insomnia—is correlated with an increased risk of developing an initial panic disorder, major depressive disorder, or alcohol abuse [37]. Treating comorbid psychiatric illness may improve or resolve the insomnia.

9.6.2 Inpatient Treatment of Insomnia

Both non-pharmacological and pharmacological treatment strategies for insomnia can be applied in the inpatient setting. Sleep hygiene measures should be used initially, along with promotion of the sleep environment. Assessment of medications and their appropriateness in the acute hospital setting, as well as adjustment of the timing of administration, can also facilitate sleep (Fig. 9.4) (Tables 9.3, 9.4 and 9.5). As previously discussed, use of light therapy in the morning may help with circadian balance and decrease daytime sleepiness. Decreasing stimuli and facilitating darkness and a cool environment promote sleep onset and deep sleep [29, 38]. Hospitals are usually inadequately lit, but many are starting to use light therapy within hospital settings to treat various disorders including depressive disorder and non-motor manifestations of Parkinson's disease [39]. For example, a randomized clinical trial of patients with Parkinson's disease found that those exposed to bright light therapy slept better at night and were less sleepy and more active in the day, compared with patients given a dim-red light therapy as a control [39]. Additionally, other primary sleep disorders can also exacerbate insomnia and are discussed in the next section. When non-pharmacological strategies do not work well, pharmacological treatments should be considered. Supplemental melatonin may be tried as the initial pharmacotherapy. Hypnotic drugs should be used in the lowest effective dose, with short-term use, and tailored to sleep-onset or sleep-maintenance insomnia (Table 9.6).

Both benzodiazepine and non-benzodiazepine GABA-active medications (e.g., eszopiclone, zopiclone, zolpidem) bind to $GABA_A$ (gamma amino butyric acid) receptors in the brain, exerting hypnotic effects (Fig. 9.6). Benzodiazepine hypnotics bind equivalently to all $GABA_A$ receptors (α1-, α2-, α3-, and α5-containing subtypes). In contrast, non-benzodiazepine GABA-active hypnotics (zolpidem, zopiclone, eszopiclone) preferentially bind α1-containing subtypes, and the shorter half-life of the effects of binding to these receptors lowers the risk of fall and residual

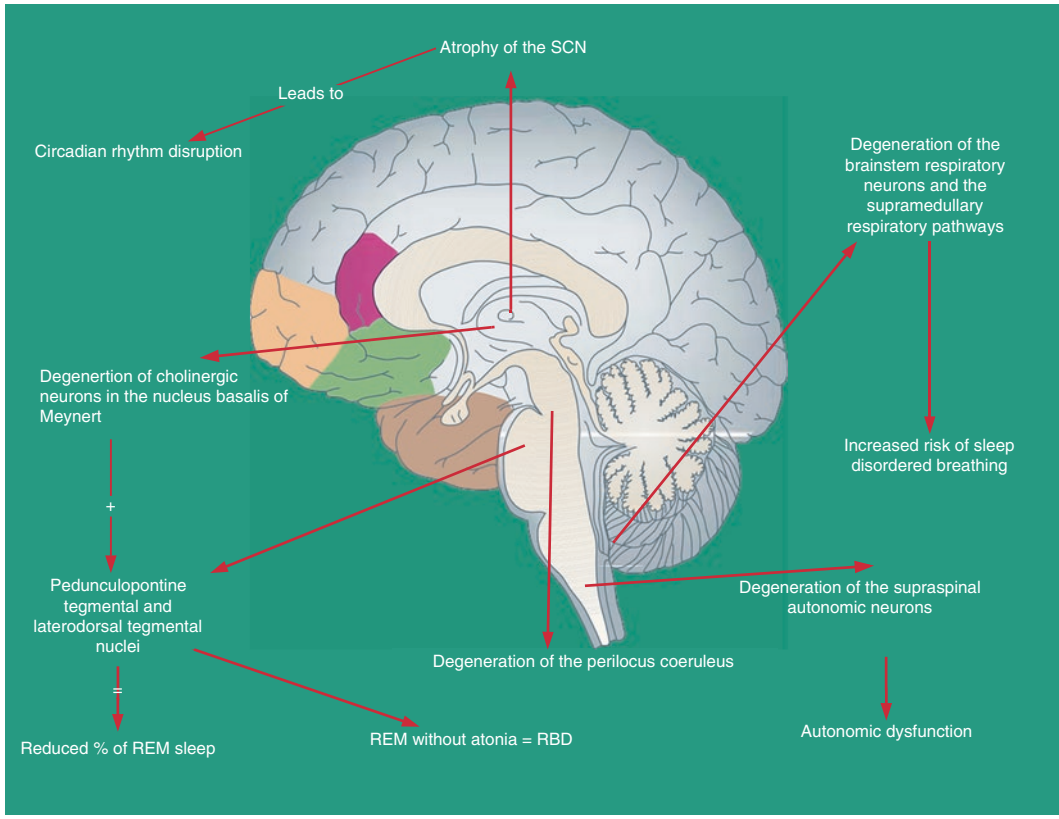


Fig. 9.4 Neurodegenerative mechanism of sleep. (Modified from Avidan [27])

sleepiness [23]. Eszopiclone has been reported to be efficacious in treating patients with insomnia with comorbid diseases such as depressive disorder and Parkinson's disease. Compared to non-benzodiazepine medications, benzodiazepine medications have more anxiolytic effects, which may be helpful in patients with anxiety disorders. But withdrawal syndromes, rebound, tolerance, and dependence pose risks with GABA-active medications. In prospective studies that included geriatric patients, long-term use of benzodiazepines was related to an increased risk of MNCD (odds ratio, 1.6–3.5) [40].

Ramelteon is a selective agonist of MT1 and MT2 melatonin receptors located in the SCN in the hypothalamus [23]. Activation of MT1 receptors decreases the alerting signal in the evening, and activation of MT2 receptors can shift the phase of the circadian system. Compared with benzodiazepine hypnotics, ramelteon is associated with lower risk of falling, withdrawal, and tolerance. Additionally, the administration of 8 mg/day ramelteon for 7 days to older patients admitted for acute care protected against the occurrence of delirium in a multicenter, rater-blinded, randomized, placebo-controlled setting [41].

Suvorexant, a dual orexin receptor antagonist, is a new class of hypnotic that works by selectively blocking the binding of the wakefulness-promoting neuropeptides orexin A and B. Suvorexant has a half-life of approximately 12 hours and is useful in subjects with sleep-onset as well as sleep-maintenance insomnia. Suvorexant has been used in patients with mild to moderate chronic obstructive pulmonary disease without negative effect on oxygenation [42].

Table 9.6 Medications FDA-approved as sleep aids

Medication category	Drugs/dose (mg)	Half-life (h)	Onset (min)	Major effects/clinical comments
Benzodiazepine∞	Lorazepam 0.5–1.0 mg	12	30	Caution use in elderly patients
				Side effects: daytime sedation, anterograde amnesia, cognitive impairment
	Estazolam 1–4 mg	10–24	60	Frequent arousals and early awakening. Short-term use (7–10 d)
				Avoid in OSA patients
	Flurazepam 10–30 mg	47–100	15–20	In general avoid in hospitalized medical patients
	Quazepam 15-30 mg	25–114		In general avoid in hospitalized medical patients
	Temazepam 15–30 mg	6–16	15–20	Sleep onset and maintenance. Short-term use (7–10 d)
				Doses ≥ 30 mg/d: morning grogginess, headache, nausea, vivid dreaming
	Triazolam 0.125–0.25 mg	1.5–5.5	15–30	Short-term use (7–10 d). Should be in bed when taking medication
				Contraindicated: certain antifungals and HIV protease inhibitors
Non-BzRAs°				
	Eszopiclone 1–2 mg	5–7	Rapid	Difficulty falling asleep in elderly: initially 1 mg, maximum 2 mg
				Difficulty staying asleep: 2 mg
	Zaleplon 5 mg	1	Rapid	Initiation of sleep and/or requiring next day wakefulness
				Short-term use (7–10 d)
	Zopiclone 7.5–10 mg	3.8–6.5 (5–10 in elderly)	30	Short-term use (7–10 d)
				Contraindicated: liver disease and depression
	Zolpidem 2.5–5.0 mg	1.4–4.5	30	Sleep onset and maintenance. Short term (7–10 d)
				Rapid onset; should be in bed when taking medication
Melatonin agonist				
	Ramelteon 8 mg	1–2	30	Use for Sleep onset insomnia
				Use for circadian rhythm disturbances
Tricyclic antidepressant				
	Doxepin 6–10 mg	8–24	30	Sleep onset and maintenance
				For comorbid depression, consider 75–100 mg daily
Dual orexin receptor antagonist				
	Suvorexant 15–20 mg	12	30–60	Sleep onset and maintenance insomnia
				Relatively contraindicated in: narcoleptics, possibility of cataplexy, sleep

9.6.3 Treatment for Sleep-Disordered Breathing (SDB)

Among the forms of sleep-disordered breathing (SDB), obstructive sleep apnea (OSA) is more common, including in the older population. When central sleep apnea or Cheyne-Stokes respiration is identified, screening for vascular disease, cardiac, congestive heart failure, and cerebrovascular disease should be performed.

OSA is defined as intermittent upper airway obstruction occurring during sleep that results in alteration in airflow. This is measured by the presence of episodes of apnea and/or hypopnea lasting a minimum of 10 seconds and accompanied by a measurable drop in the oxygen saturation and/or cortical arousals. The apnea-hypopnea index (AHI) reflects the severity of SDB and is calculated by measuring the number of events per hour. OSA is associated with metabolic syndrome, diabetes mellitus, hypertension, and cardiovascular events [43]. Patients with moderate-to-severe OSA have been shown to have an independently increased risk for all-cause mortality and stroke [43]. Importantly, treatment with positive airway pressure is shown to improve these outcomes.

Aging has a direct effect on risk of developing obstructive breathing due to the normal changes in tone of the pharyngeal muscles [19]. AHI and the percentage of time with oxygen saturations below 90% are shown to be more severe with aging. The AHI ranged from 1.4 in individuals age 20–24 to 20.9 in individuals > 75 years, even when corrected for co-variables. This suggests that the development of a degree of OSA may be a part of the normal aging process. In the classic Sleep Heart Health Study of 5615 community-dwelling men and women between 40 and 98 years of age, OSA was found to be common in subjects aged 60 years or older (approximately 50% had an AHI of 5–14, and approximately 20% had an AHI ≥ 15) [44]. Additionally, in older adults, an AHI > 15 was associated with lower daytime functioning and longer psychomotor reaction times [33].

OSA can increase agitation in patients with Alzheimer's disease. Neuropsychological analyses revealed that in patients with OSA who were treated with continuous positive airway pressure (CPAP) therapy, cognitive flexibility, attention, vigilance, memory, and executive functioning improved [45]. Additionally, studies have found that patients with underlying psychotic disorders have higher rates of OSA and poor sleep. Poor sleep is associated with higher odds of psychotic, depressive, and manic symptoms. Use of atypical antipsychotic medications decreased symptoms and improved sleep [46, 47]. Table 9.7 shows the various alternative medications per drug class that may be used in disordered sleep treatment.

9.6.4 Parasomnias

Parasomnias are a group of sleep disorders defined as undesirable physical and/or experiential events that occur within entry into sleep, during sleep, or during arousals from sleep. This section focuses on parasomnias that are most common in the geriatric population and have connections to neuropsychiatric illness.

9.6.5 REM Behavior Disorder (RBD)

Many parasomnias occur more often in childhood, such as sleepwalking, sleep talking, and nightmares, and are usually benign. However, REM behavior disorder (RBD) begins in adulthood and is characterized by unpleasant dreams or nightmares with loss of normal atonia during REM sleep, thus allowing dream enactment behavior [31]. The behaviors may be simple to complex with yelling, screaming, punching, and kicking, predisposing the patient and/or bed partner to risk for injury. The eyes are closed and the patient is unresponsive to the surrounding environment. When awakened, the person rapidly achieves full alertness and has a recollection of the dream content. He/she may also display mild cognitive, motor, and autonomic impairments similar to that seen in early neurodegeneration patients. On polysomnography (PSG), there is increased electromyographic activity.

Table 9.7 Alternative drugs for dual psychiatric symptoms and sleep disorder

Medication class	Drugs	Pertinent side effects	Clinical comments
Antidepressants			
	Mirtazapine	Somnolence	May be beneficial for comorbid depression and insomnia
		Increased appetite	Lower doses (≤ 15 mg) increase sedation, but not an effective antidepressant
		Weight gain	Avoid RLS
		Dry mouth	
	Trazodone	Residual daytime sedation	May be beneficial for comorbid depression and insomnia
		Headache	Not recommended as first-line agent for insomnia
	TCAs	Delirium	Avoid in hospitalized patients due to side effects
		Decreased cognition	May be beneficial for comorbid depression and insomnia
Antihistamines			
	Diphenhydramine	Residual daytime sedation	Better than placebo to treat insomnia, but not definitively indicated for insomnia
		Delirium	
	Hydroxyzine	Drowsiness	Avoid in patients > 60 years old, closed angle glaucoma, prostatic hypertrophy, severe asthma, and COPD
		Dry mouth	
Antipsychotics			
	Quetiapine	Sedation	Used as sleep aid. Most sedating of the atypical antipsychotics
		Orthostatic hypotension	
	Olanzapine	Sedation	Insomnia
		Hyperglycemia	Avoid in elderly for indication of sleep, all atypical
	Risperdal	Sedation	Extrapyramidal side effects
		Weight gain	Give low dose of this typical antipsychotic

RBD may be idiopathic or secondary to neurodegenerative diseases, including Parkinson's disease (PD), MNCD with Lewy bodies, and multiple system atrophy (MSA) [48]. The prevalence of idiopathic RBD is 0.5%, and approximately 30–50% of patients will develop PD within a decade and 50–90% ultimately succumb to the associated neurodegenerative disease. RBD is more common in geriatric men than women. The presence of RBD in PD predicts a more severe, aggressive course. The neurodegenerative mechanism of sleep is demonstrated in Fig. 9.4.

Previously, it was thought that only the dopaminergic pathway was affected in PD and thus the high association of RBD with PD. However, it is now known that dopamine deficiency alone is not the cause of RBD. The etiology of RBD is complex and associated with destruction of the REM-related sleep centers in the brain (locus subcoeruleus, amygdala, limbic system) involving several neurotransmitter pathways, including GABA, glutamate, and monoaminergic (dopamine) systems that control REM and wakefulness [48]. Neuroimaging studies have shown dopaminergic, cholinergic, and cortical abnormalities in RBD similar to patients with alpha-synucleinopathies, and it is now suggested that idiopathic RBD may actually be another, milder form of neurodegenerative disease (Fig. 9.3). Additionally, RBD may occur with other disorders, albeit rare, that damage the brain

areas associated with REM control, such as stroke, tauopathies, and amyloidopathies (e.g., Alzheimer's disease).

RBD or pseudo-RBD may be present in other sleep disorders, such as untreated OSA, PLMD, or RLS, insufficient sleep disorder, and medications. These disorders should be treated first before establishing the diagnosis of idiopathic RBD. Importantly, medications which alter cholinergic, serotoninergic, and monoamine neurotransmitters may be associated with RBD. In a study by Postuma et al., patients on antidepressants and dream enactment behavior also demonstrated other symptoms associated with the alpha-synucleinopathy disorders, including loss of smell, constipation, and mild motor impairments. Whether the antidepressants cause or unmask RBD in individuals predisposed to develop neurodegenerative disorders is unknown [49]. Antidepressants are frequently employed to treat numerous disorders, and if patients develop clinically problematic alteration in dream content or change in behavior, these medications should be discontinued. Behavioral treatments for insomnia are highlighted in Fig. 9.5 [38].

Treatment of RBD is limited to protective measures during sleep to avoid injury and medications if there is regular occurrence of dangerous behaviors. Previously, dopaminergic agents were used, as it was speculated that RBD was related to dopamine deficiency. However, these agents did not eliminate the dream-related motor behavior. In fact, increased doses of dopamine may precipitate or increase hallucinations. Medications that have been effective in RBD are high-dose melatonin (6–15 mg) taken 30 min prior to bedtime and low-dose clonazepam (0.5–2 mg). Melatonin may help to reset the circadian clock and suppress the REM frequency and has little side effects. Clonazepam acts on the GABA receptor and may decrease the acetylcholine activation of REM. Because of the sedating effect of clonazepam, it should be taken immediately prior to bedtime; older patients may be at particularly high risk for falls [50]. Figure 9.6 summarizes the GABA receptor subtypes [51].

9.6.6 Restless Leg Syndrome

Restless leg syndrome (RLS) is a sleep-related movement disorder characterized by the following criteria: the uncontrollable urge to move the legs, unusual sensations in the legs, and symptoms that occur in a circadian pattern with increased symptoms in the nighttime and worsen with inactivity resulting in difficulty initiating and at times maintaining sleep. In older adults with major NCD or other illnesses associated

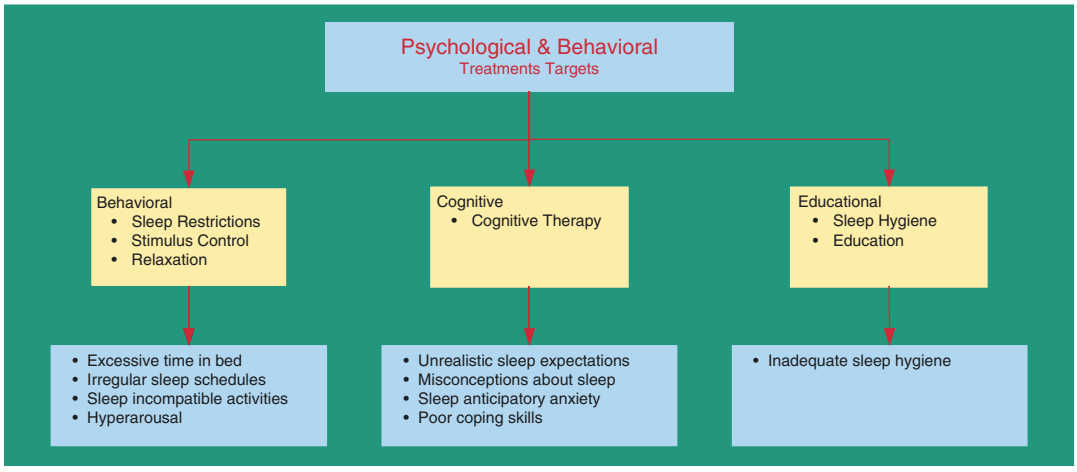
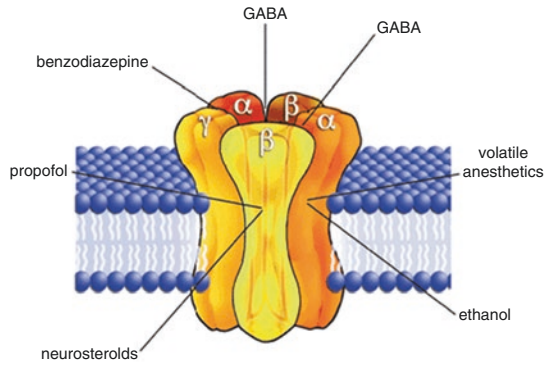


Fig. 9.5 Behavioral treatments for insomnia. (Modified from Jacobs et al. [38])

GABA A receptors are made up of 2 alpha-subunits.

With further sub-specific sites: 1-6.

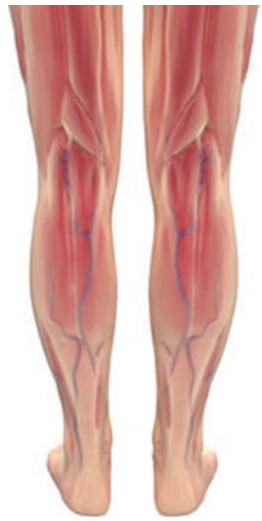
Benzodizepines are known to bind non-selectively to alpha units.



- Non-benzodiazepine BzRA have a binding affinity to α1 (or α1,2,3 in the case of eszopiclone / zopiclone).
- The specificity to certain GABA A receptor subtypes causes specific hypnotic actions without anxiolytic & anti-convulsant activity.

Fig. 9.6 $GABA_A$ receptor subtypes. (Modified form Jacobs et al. [51])

Table 9.8 Evidence supporting restless leg syndrome



Responsive to a dopaminergic medication
First-degree relative with RLS
History of prior symptoms
Observed limb movements
Periodic limb movements noted on PSG
Sleep-onset difficulty
Better sleep quality in daytime than at night
Restless in the night with increased movement
Low serum ferritin level
Co-existing renal disease, neuropathy, or diabetes

with cognitive dysfunction, RLS must be distinguished from other disorders that may disrupt sleep. Table 9.8 illustrates supporting criteria that confirm a high suspicion of RLS.

9.7 Summary

Nighttime sleep disruption and daytime sleepiness are common in geriatric patients. Sleep disturbance in the geriatric *inpatient* is often caused by a multitude of factors, including systemic medical and psychiatric illness, medications, circadian rhythm abnormalities, sleep-disordered breathing, and other primary sleep disorders, environmental factors, and lifestyle habits.

The inpatient environment can contribute to sleep disruption. Improvements in complex hospital milieus should be addressed, toward allowing a normal circadian rhythm and dedicated uninterrupted periods for sleep consolidation. These improvements can facilitate daytime optimum alertness and functioning.

Nonpharmacological therapies should be attempted first to promote sleep in this population, especially in inpatient settings.

Pharmacotherapy may be needed for patients with comorbid psychiatric illness, primary sleep disorders, or other ailments that may contribute to sleep disruption. Specific medications with sleep-promoting properties should be chosen for treatment of underlying comorbid medical and psychiatric conditions, as well as to avoid potential adverse effects. It is crucial to be cautious about medications, such as benzodiazepines, which may provide apparent benefit early in treatment but can produce significant adverse effects over time.

Take-Away

- Assessment and management of sleep disruption are important goals of inpatient geropsychiatric care.
- Depressive disorders, anxiety disorders, and behavioral disturbance due to MNCDs, common in hospitalized geriatric patients, are likely to include sleep difficulties.
- Psychiatric syndromes (including neurocognitive disorders), systemic medical comorbidities, and medications all contribute to the etiology of sleep disruption in the hospitalized geriatric inpatient, making accurate diagnosis challenging. Medication regimens should be reviewed to minimize any contribution to sleep disruption.
- Environmental factors which interrupt sleep should be recognized and addressed.
- Nonpharmacological approaches, including behavioral treatment and good sleep hygiene, are first steps in the treatment of acute or chronic insomnia.
- Principles of safe pharmacological intervention for insomnia are crucial for the prevention of untoward effects.

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10 Alcohol and Substance Use Disorders in the Geriatric Psychiatry Inpatient: Acute Treatment, Detoxification, and Withdrawal

Karin Kerfoot and Amer M. Burhan

10.1 Introduction: Substance Use Disorders in Geriatrics

The population cohort born between 1946 and 1964 is expected to show a steady increase in substance use disorder presentations [1–3]. In comparison to previous generations, this cohort has had greater exposure to, and acceptance of, psychoactive substance use and has a relatively long life expectancy. In addition, alcohol use disorders have been estimated to be present in up to 30% of older inpatients in general medicine settings and in up to 50% of older inpatients on psychiatric units [4, 5]. Though less data is available for benzodiazepines and opioids, these medications pose risks in the geriatric population, including the potential for overdose and death [6, 7]. Substance use disorders with alcohol, benzodiazepines, and opioids, are often under-detected and sub-optimally treated, especially within inpatient settings where life-threatening issues, including active withdrawal, arise. Benzodiazepine and opioid prescribing for older adults has recently been a source of societal concern because of escalating use and associated adverse consequences [8, 9].

Geriatric adults can develop substance use issues for the first time in old age, particularly following significant adjustments and losses, including retirement, death of a partner, pain, and/or medical illness (Chap. 14: Pain). These issues occur frequently in men and women alike. The majority of substances are increasingly hazardous with advancing age because of physiological changes with aging (Chap. 3: Pharmacology Overview); increased comorbidities, including neurocognitive conditions (Chap. 6: Major Neurocognitive Disorder with Behavioral Disturbance); and frequent polypharmacy [10]. The interplay between substance use and these factors has been shown to contribute to dangerous outcomes such as excessive sedation, ataxia, cognitive "clouding," falls, and impaired driving (Chap. 7: Acute Medical Events). Polypharmacy of opioids and benzodiazepines in geriatrics continues to rise [11]. Tobacco use is also prevalent and the use of nicotine replacement therapy may be helpful, since it has been shown to be effective in aging adults [12].

Features of substance withdrawal can include discomfort, delirium, and, for alcohol and benzodiazepines, autonomic hyperactivity, seizures, and death [13, 14]. Alcohol, benzodiazepines,

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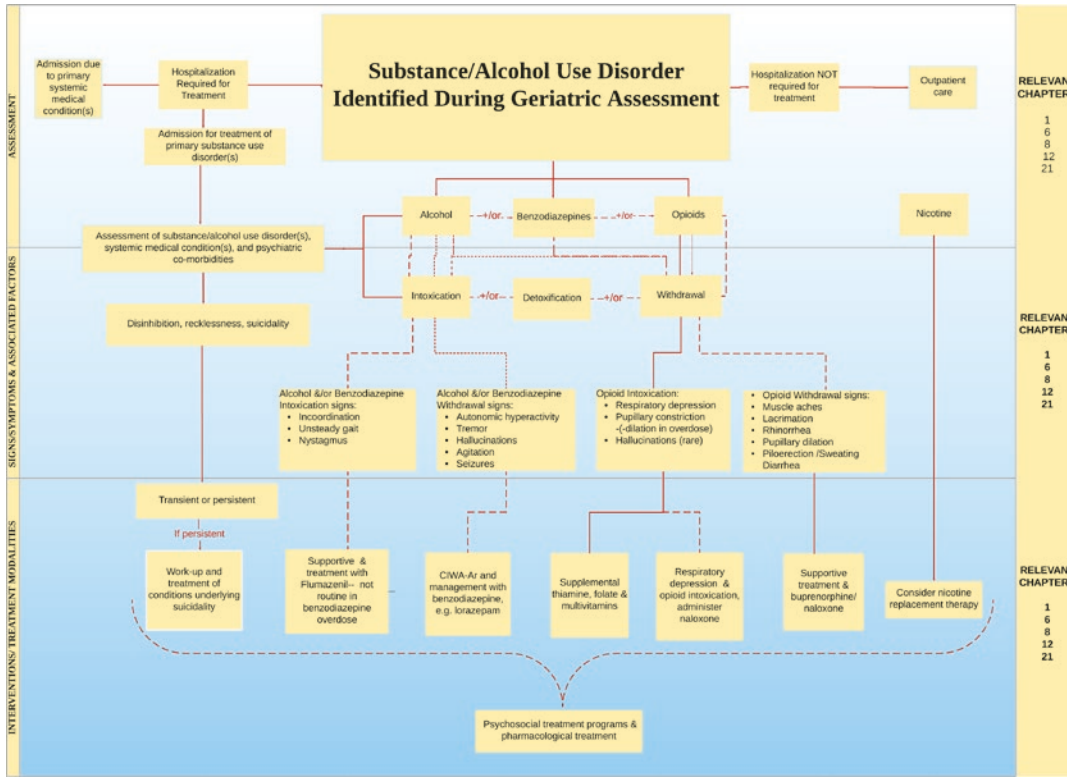

Fig. 10.1 Flowchart—identification of substance use disorder during comprehensive assessment

and opioids can be fatal in overdose, alone, in combination with each other, with other centrally-acting medications, or with central nervous system conditions. A heightened risk of overdose follows sudden discontinuation and re-starting of opioids. Overuse of depressant substances, such as alcohol and benzodiazepines, can also impact mood, and contribute to disinhibition, dysphoria, and suicidality. In sum, several factors make geriatric patients more vulnerable to the effects of substance abuse, intoxication, dependence, and withdrawal. The inpatient setting is the most appropriate and efficient place to recognize and treat these conditions. Figure 10.1 presents a guideline to the identification of substance use disorder during comprehensive clinical assessment.

10.2 Clinical Vignettes

10.2.1 Vignette 1

A 68-year-old practicing attorney was brought to the emergency department by a friend after the patient expressed concrete plans to kill herself. Stressors over the preceding year included the loss of her driver's license for driving while intoxicated, the deaths of her husband and dog, and estrangement from her daughter and newborn granddaughter.

She had a 40-year history of excessive consumption of alcohol and had used benzodiazepines regularly for at least 20 years, primarily for the treatment of anxiety associated with post-traumatic stress disorder (PTSD). She was vague

regarding the details of her recent alcohol use. She mentioned that she recently received an additional lorazepam prescription from a walk-in clinic after she lost her regular supply. It was later revealed that she had a pattern of losing her lorazepam and other benzodiazepines, for which was always prescribed early refills.

She was emotionally labile. She continued to express an intent to end her life, because she had "lost everything," with the final straw being her driver's license. She implied that her alcohol intoxication was the cause for her loss of license but angrily denied this when asked directly. Her speech was slurred and she had difficulty maintaining her balance when ambulating. She was unable to provide details and did not appear to recall that her friend brought her to the emergency department. She complained of anxiety in the very chaotic emergency department setting and was given lorazepam 1 mg SL.

Over the next 2 days, she continued to express suicidal intent. For her safety as well as detoxification, she was admitted to the inpatient psychiatry unit. She insisted that she had cut down alcohol consumption over recent weeks and had never previously experienced alcohol withdrawal. Her benzodiazepines were discontinued, and vital signs were monitored every 8 hours.

Approximately 48 hours following her initial presentation to the emergency department, her heart rate rose to 130 bpm, her blood pressure was 180/100 mm Hg, and she was diaphoretic, agitated, and anxious. The on-call team ordered a revised Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar) protocol including PRN lorazepam in a sign/symptom-triggered fashion [15]. With PRN lorazepam administration, her vital signs stabilized and she felt more comfortable. She became forthcoming about her alcohol and benzodiazepine use prior to admission, which was of greater magnitude than previously disclosed. She was switched to regular dosing of a longer-acting benzodiazepine, clonazepam, which was gradually tapered over the next 5 days. She engaged with treatment planning and was discharged to a residential rehabilitation center.

10.2.1.1 Discussion

The abuse of several substances, including alcohol and prescription medications, is frequent in geriatrics. Suicidality in the context of psychosocial losses and depressant substance use is a safety issue and, together with the risk of significant withdrawal, prompted an inpatient admission. Patients may express transient suicidal ideation in the context of intoxication, only to reverse this when sober. It is critical to determine whether suicidality is transient and secondary to intoxication, or persistent and symptomatic of an underlying mood disorder. The aging patient may minimize substance use, highlighting the importance of attention to proactive management for the risk of substance-related withdrawal.

10.2.2 Vignette 2

A 74-year-old retired truck driver was brought to the emergency department by his wife for weakness and shortness of breath. His medical comorbidities included severe rheumatoid arthritis, chronic pain, three transient ischemic attacks (TIAs), and a right middle cerebral artery (MCA) distribution stroke. Depressive symptoms had been diagnosed at various times as major depressive disorder (MDD), persistent depressive disorder (dysthymia), substance−/medication-induced depressive disorder, depressive disorder due to another medical condition (stroke), and unspecified depressive disorder. One provider offered the diagnosis of somatic symptom disorder with pain. Trials of several antidepressant medications did not improve his symptoms, but he did benefit slightly from six sessions of electroconvulsive therapy (ECT), which was discontinued midcourse due to post-procedure supraventricular tachycardia.

The patient had used oral opioids (OxyNeo and oxycodone immediate release) to manage

pain for 9 years. He was transitioning from oral opioids to transdermal fentanyl because of concerns about his alternating opioid intoxication and withdrawal. He was morbidly obese (over 270 lbs), weak (effectively bedridden), short of breath, and required oxygen supplementation. His level of arousal was compromised and he appeared drowsy. He was diagnosed with community-acquired pneumonia and admitted to hospital for IV antibiotics. Several medications, including his oral opioids and fentanyl patch, were held because of possible contribution to respiratory suppression.

On his second day at the hospital, he reported inability to sleep overnight. His pain had increased and he could not eat due to nausea, abdominal distress, and diarrhea. He was perspiring and his temperature was 39 degrees Centigrade, 102.2 degrees Farenheit. A consulting psychiatrist recommended maintaining his duloxetine 90 mg daily while resuming fentanyl to address opioid withdrawal symptoms and pain. Intravenous fluids and antibiotics based on blood cultures and sensitivities were started, and the fentanyl patch was resumed at a lower dose.

The patient continued to complain of pain, and his acute respiratory and opioid withdrawal symptoms resolved; he was transferred to the inpatient geriatric rehabilitation program. The fentanyl patch was maintained at the lower dose previously established in hospital and PRN oxycodone was prescribed for breakthrough pain. He made modest gains during his rehab stay and after 10 more days was discharged home with comprehensive outpatient support. At home, he experienced intolerable pain and self-administered a second fentanyl patch. He expired within 2 hours after application of the second fentanyl patch. The cause of death was determined to be respiratory arrest secondary to opioid depression of the respiratory drive and possible aspiration.

10.2.2.1 Discussion

Significant complexity and risk accompany the treatment of opioid dependence, chronic pain, and medical comorbidities in the geriatric patient. Careful monitoring in the inpatient setting and proactive management can head off the risk of opioid withdrawal symptoms. The stroke and white matter brain changes of this patient resulted in added disability and depressive disorder, which interacted in a complex reciprocal fashion with his pain. His tolerance of pain and judgment were compromised. His excessive fatigue, low level of activity, and morbid obesity also led to worsening of disability and chronic pain. He relied upon added doses of opioids to treat his intractable pain—which was aggravated by depression—and the added dose of fentanyl resulted in respiratory depression, aspiration, and death.

10.3 Review of Literature and Evidence for Interventions

10.3.1 Definitions and Diagnoses

DSM-5 describes that a substance use disorder is characterized by the problematic use of a substance, leading to clinically significant impairment or distress, with at least two of several criteria, including (see Fig. 10.2: DSM-5 Substance Use Criteria):

- Excessive use
- Desire/unsuccessful attempts to cut down
- Time investment to acquire or use the substance
- Craving
- Failure to fulfill major role obligations
- Continued use in spite of significant social or interpersonal problems
- Neglect of important activities
- Use in physically hazardous situations
- Continued use despite persistent physical or psychological problems
- Tolerance, i.e. need for markedly increased amount to achieve intoxication/desired effect or diminished effect with use of the same amount
- Withdrawal [16]

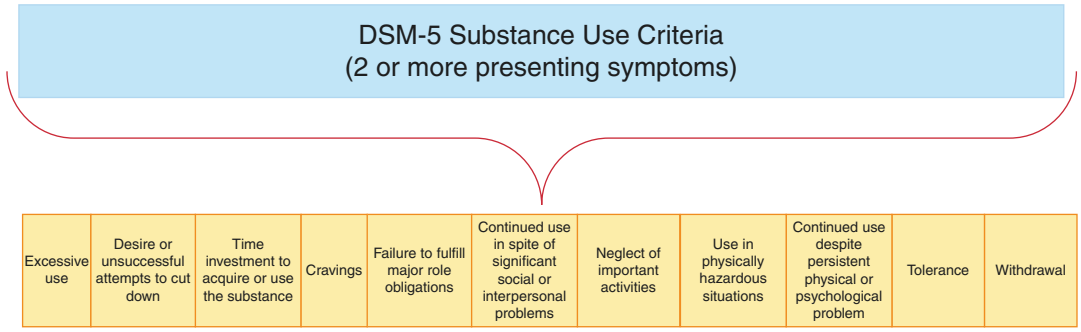

Fig. 10.2 DSM-5 substance use criteria

10.3.2 Specific Substance Use Disorders and Intoxication Syndromes in Geriatrics

Substance use disorders can be easily missed or incorrectly diagnosed in the aging adult due to overlap with comorbid systemic medical symptoms. Light-headedness, altered arousal, depressive symptoms, disorientation, falls, seizures, and sleep disturbance, for example, can have numerous potential causes, including substance intoxication or withdrawal phenomena. Substance use issues should be included within the differential diagnosis.

Though it may seem unlikely that substance intoxication will present in geriatric patients within the inpatient setting, it is important that intoxication is not overlooked. When given off-unit passes and/or with visitors, geriatric patients may gain access to substances. With regard to prescribed medications, the geriatric patient may use her/his own outpatient supply of benzodiazepines and opioids while in hospital, especially if these medications are accessible on the unit. Geriatric patients may develop suicidal ideation within the hospital setting for several different reasons, including during acute intoxication. Suicidality due to substance intoxication can be temporary, abating following resolution of the intoxicated state [17]. But suicide attempts are more likely to be lethal in the geriatric population (Chap. 8: Suicide). It is important to determine whether suicidal ideation in the intoxicated older patient is transient or persistent beyond the resolution of intoxication [17].

10.3.3 Alcohol Intoxication

Alcohol intoxication follows the recent consumption of alcohol and is marked by problematic behavioral or psychological/behavior changes such as inappropriate sexual/aggressive behavior, mood lability, and impaired judgment [16]. Loss of social inhibition is a marker. Alcohol intoxication is also demonstrated by one or more of several signs and symptoms including slurred speech, incoordination, gait unsteadiness, nystagmus, impaired attention/memory, and stupor/coma.

10.3.4 Benzodiazepine Intoxication

Benzodiazepine intoxication shares the same signs and symptoms as alcohol intoxication.

10.3.5 Opioid Intoxication

Opioid intoxication occurs if there has been an excessive dose of the opioid, beyond the patient's daily, regular dose, and is accompanied by neuropsychological or behavioral changes. There may be impaired judgment, diminished arousal, psychomotor agitation/retardation, or mood changes

Table 10.1 Intoxication: management considerations in alcohol, benzodiazepine, and opioid intoxication

	Changes from baseline behavior of intoxication	Signs and symptoms of intoxication	Management considerations for intoxication/overdose (depending on severity)
Alcohol and benzodiazepines	Inappropriate sexual/aggressive behavior, mood lability, impaired judgment	Slurred speech, incoordination, unsteady gait, nystagmus, impaired attention/memory, stupor/coma	Airway protection, oxygen support, IV fluids, behavioral redirection (rarely flumazenil for benzodiazepines)
Opioids	Initial euphoria, subsequent apathy, dysphoria, psychomotor agitation/retardation, impaired judgment	Respiratory depression, pupillary constriction (or dilation in severe overdose), drowsiness/coma, slurred speech, impaired attention/memory, hallucinations (rare)	Respiratory support, naloxone (initial dose 0.4 mg), with subsequent empiric dosing

such as dysphoria or initial euphoria followed by apathy [16]. Opioid intoxication is also characterized by constriction of the pupils (or dilation in cases of anoxia secondary to severe overdose), and one or more symptoms: drowsiness/coma, slurred speech, and impaired attention/memory. Hallucinations may also be present during opioid intoxication.

10.3.6 Reversal of Severe Intoxication

Management of intoxication depends upon the particular substance and characteristics/severity of the accompanying signs and symptoms. Table 10.1 details the accompanying behavioral and psychological changes, physical signs/symptoms, and priorities in the management of alcohol, benzodiazepine, and opioid intoxication. Though management of intoxication is generally supportive in mild-moderate cases, severe intoxication can be fatal with each substance, particularly opioids, and especially when there is a combination of substances. Opioid intoxication can often be fatal because of respiratory depression, and the administration of naloxone can be life-saving [18]. Flumazenil, a selective benzodiazepine receptor antagonist, is sometimes used to treat benzodiazepine overdose, though its use is not routinely recommended in cases of mild-moderate benzodiazepine intoxication due to risks with pharmacologically-precipitated withdrawal (which carries its own potential dangers). The use of flumazenil is complicated from a monitoring and administration perspective, as many benzodiazepines have longer half-lives than flumazenil.

10.4 Withdrawal Symptoms

10.4.1 Substance Withdrawal

Withdrawal symptoms are prevalent when consumption is decreased or abruptly discontinued in patients who have a pattern of regular, moderate-heavy use. Geriatric patients are vulnerable when health-care providers are unaware that there is pre-existing, regular use of a substance, abuse of a substance, or dependence. Alcohol, benzodiazepine, and opioid withdrawal symptoms can range from negligible and mild to life-threatening and severe. Aging adults are more likely to experience adverse functional and cognitive effects, as well as a longer duration of withdrawal symptoms, in comparison to the general adult population [19]. Because of comorbid medical and psychiatric conditions, geriatric patients have a heightened risk of complications (Chap. 14: Pain). Management of substance-related withdrawal in the geriatric patient should be determined by the particular substance and the likelihood for the development of serious withdrawal signs and adverse consequences. Table 10.2 summarizes circumstances, signs, and symptoms which accompany alcohol, benzodiazepine, and/or opioid withdrawal syndromes. Proactive withdrawal management strategies are recommended.

Table 10.2 Withdrawal: circumstances, signs, symptoms, and management in alcohol, benzodiazepine, and opioid withdrawal

	Circumstances of withdrawal development	Possible signs and symptoms of withdrawal	Monitoring and management of withdrawal
Alcohol	Within 6–96 h after stoppage or reduction of heavy and prolonged alcohol use	6–48 h: Autonomic hyperactivity, tremor, insomnia, agitation, nausea/vomiting, anxiety, generalized tonic-clonic seizures	High index of suspicion, vigilant vital sign monitoring, use regular or symptom-triggered (CIWA-Ar) dosing of benzodiazepines; e.g., lorazepam or oxazepam
		12–48 h: Hallucinations	
		48–96 h: Delirium tremens	
Benzodiazepines	Within several hours to 2–4 days (depending on half-life of particular benzodiazepine) after stoppage or reduction of prolonged benzodiazepine use	Tremor, insomnia, nausea/vomiting, dysperceptions, tinnitus, agitation, anxiety, irritability, panic, impaired concentration. Abrupt cessation of high doses: hypertension, seizures, delirium	Regular monitoring of symptoms and vital signs, taper gradually with patient's current benzodiazepine; switch to longer-acting benzodiazepine with caution: monitor for sedation and falls
Opioids	Within 6–8 h after last dose. Begin slowly, peak at 48–72 h, gradually taper over next 4–7 days. Develop within minutes after administration of an opioid antagonist	Agitation, anxiety, dysphoric mood, nausea/vomiting, lacrimation/rhinorrhea, diarrhea, fever, insomnia, yawning, piloerection/sweating, tachycardia, pupillary mydriasis, 8–12 h after last dose, increased blood pressure and respiratory rate. At peak, pronounced anxiety, tremors, smooth muscle and skeletal muscle cramping, joint and deep bone pain	Regular monitoring of symptoms, consider use of COWS (Clinical Opiate Withdrawal Scale), taper opioids gradually, consider buprenorphine/naloxone while in acute withdrawal

Because of a dearth of age-specific evidence, few guidelines are available for the management of substance withdrawal in geriatric patients. Therefore, an understanding of the specific patient's substance use history, previous episodes of withdrawal and associated complications, the extent of recent use, medical comorbidities, and polypharmacy must guide current treatment. Table 10.3 (including CIWA-Ar) offers details of standard withdrawal protocols for each substance.

The ultimate abstinence and complete elimination of a substance may be the goal, but this should be considered in the context of the geriatric patient's history, need for pain management, treatment of psychiatric symptoms, and safety, as well as mitigation of overdose potential. In many cases of benzodiazepine use disorders, patients have a long-term history of use, and consideration should be given to gradual tapering: a 25% dose reduction every 1–2 weeks may minimize the most severe withdrawal symptoms.

Though the majority of substance use treatment is delivered on an outpatient basis, there are important indications for inpatient admission for detoxification and treatment [19, 20]. In the clinical vignette 1, admission for detoxification and treatment was indicated for patient's suicidality and serious comorbid psychiatric illness, as well as her heightened potential for dangerous withdrawal symptoms.

Indications for inpatient hospitalization for substance withdrawal include:

- A high potential for dangerous abstinence symptoms (e.g., delirium, seizures).
- Suicidality or reckless, dangerous behavior.
- Uncontrolled comorbid systemic illness, and/or serious comorbid psychiatric illness.

Table 10.3 Recommendations: alcohol, benzodiazepines, and opioids withdrawal protocols for geriatric patients

	Alcohol – clinical institute withdrawal	Benzodiazepines – gradual tapering	Opioids – gradual tapering, buprenorphine/naloxone
Recommended monitoring	Check symptoms every 4 h and 1 h after each medication dose (for continuing symptoms or excessive sedation)	Regular assessment of withdrawal symptoms, including vital signs; inquire also about benefits of tapering (e.g., increased alertness, less fatigue, better mood)	Administer COWS to assess opioid withdrawal, prior to buprenorphine/naloxone administration, and serially to track symptoms
Medication administration	Administer Lorazepam or Oxazepam with CIWA-Ar score greater than 8–10	Taper slowly with patient's current benzodiazepine. If switching to longer-acting benzodiazepine, monitor closely for sedation and falls	Taper opioids slowly. For acute withdrawal (COWS > 25, no opioids > 12–24 h) consider buprenorphine/naloxone
Medication dosing	Lorazepam 1–2 mg PO/IV/IM or Oxazepam 30–60 mg PO, given hourly when symptomatic (CIWA-Ar >8–10)	Attempt 25% reduction of dosing every 14 days. Longer tapering schedule may be done as outpatient over 4–5 months. Updosing (returning to higher dose) with withdrawal symptoms not recommended	Initial dose of buprenorphine 2 mg SL. Administer additional 2 mg SL in 2 h if necessary. Maximum 4 mg on first day. May taper over several days-weeks or maintain dose (4–16 mg daily)
Geriatric considerations	Uncertain reliability/validity of CIWA-Ar in older adults; Less specific with co-morbidities (e.g., infection, autonomic neuropathy) and medications (e.g., beta-blockers) that mimic or blunt withdrawal; not proven in patients with seizure history	Common practice of tapering with longer-acting benzodiazepine may cause sedation and falls in older adults; second-generation antipsychotics (SGAs) to assist with withdrawal-associated sleep disturbance due to increased risk of arrhythmias and death in older adults	Buprenorphine safer than methadone in older adults; initial buprenorphine administration will precipitate opioid withdrawal (advise > 12 h last immediate release dose, > 24 h last controlled release dose); reassess daily for sedation and falls

10.4.2 Alcohol and Benzodiazepine Withdrawal: Specifics

Withdrawal from alcohol can develop within hours to a few days following cessation or reduction of alcohol, and includes two or more signs or symptoms, including: autonomic hyperactivity (increased blood pressure, heart rate, and temperature), tremor, insomnia, nausea/vomiting, transient hallucinations/illusions, psychomotor agitation, anxiety, and seizures. Severe withdrawal can include hallucinations, delirium, and coma. Benzodiazepine withdrawal shares the same signs and symptoms as withdrawal from alcohol.

In the management of alcohol and/or benzodiazepine withdrawal, a shorter-acting benzodiazepine such as lorazepam is recommended and is not dependent on hydroxylation by the liver [21–23]. Dosing can be scheduled at regular, predetermined intervals or on an as-needed basis. Symptom-triggered tools such as the CIWA-Ar have been used successfully with geriatric patients and can facilitate the regular monitoring of symptoms within the inpatient setting [15, 24]. The need for supplemental doses of thiamine, folate, and multivitamins while treating aging adults for alcohol withdrawal should be considered.

Recommendations have suggested that the initial dose of a medication prescribed for the suppression and management of withdrawal symptoms in geriatric patients should be *one-third to one-half* the usual dose, continued for 1–2 days with frequent monitoring, and then gradually tapered with continued monitoring and dependent upon the treatment goals [19]. Due to the potential for accumulation and toxicity in aging adults, it is inadvisable for patients to be discharged on high doses of long-acting benzodiazepines, such as chlordiazepoxide, clonazepam, diazepam, or flurazepam [25].

10.4.3 Opioid Withdrawal

Opioid withdrawal following the cessation of use or following administration of an opioid antagonist develops within minutes (after an opioid antagonist) to hours (after the last opioid dose) and is characterized by dysphoria, nausea/vomiting, muscle aches, lacrimation/rhinorrhea, pupillary dilation/piloerection/ sweating, diarrhea, yawning, fever, and insomnia [16]. Although signs and symptoms of alcohol/ benzodiazepine intoxication and withdrawal are different from opioids, they also share many similarities with opioid intoxication and withdrawal. The frequent use of multiple substances by geriatric patients can contribute to the emergence of multiple signs and symptoms whose etiology may be uncertain within the inpatient setting. In acute opioid withdrawal, buprenorphine/naloxone is thought to be the safest and most effective treatment [26]. The clinical opioid withdrawal scale (COWS) can be used to monitor the severity of withdrawal [27].

10.5 Outpatient Follow-Up Treatment

The appropriate treatment of substance use disorders in the aging adult population includes consideration of pharmacotherapy, psychosocial treatments, and interventions after discharge, to address psychiatric and systemic medical comorbidities. Monitoring and control of medications for the geriatric *outpatient* are crucial for successful treatment of the long-term substance use disorder. This is especially important for patients with neurocognitive disorders.

The literature examining the effectiveness of psychosocial treatment for substance use disorders in geriatric patients is sparse. Few studies of geriatric patients have examined the use of pharmacologic agents such as disulfiram, naltrexone, and acamprosate for the treatment of alcohol use disorders, as well as the use of methadone and buprenorphine/naloxone. Because of its potential for serious interactions and outcomes, disulfiram is not generally recommended in this population [28–30]. There is some evidence to suggest that naltrexone (at dosing equivalent to 50 mg daily) may be a relatively safe and effective agent for relapse prevention in aging adults with an alcohol use disorder [28]. In opioid use disorders, buprenorphine is considered a safer maintenance treatment than methadone [26].

10.6 Summary

Substance use disorders are increasingly prevalent [19] and carry the potential for serious complications and outcomes in the geriatric age group. Syndromes related to substance intoxication and withdrawal are difficult to treat unless the etiology of the symptoms are identified. More definitive research will help clinicians better understand substance use disorders in geriatric patients and provide evidence-based treatment guidelines. The need for expertise and vigilance of these issues within inpatient units is likely to grow.

Take-Away

- Substance use disorders are increasingly prevalent in aging adults [19]. Alcohol, benzodiazepines, and opioids are widely used, easily available, often misused, and are most likely to precipitate acute medical issues within the inpatient setting [31].
- The signs and symptoms of substance intoxication and withdrawal include physical discomfort, suicidality, delirium, respiratory depression, seizures, and death.
- The interaction of substance use disorders and comorbid systemic medical illnesses, polypharmacy, falls, and other psychiatric conditions (including neurocognitive disorders) can complicate the appropriate diagnosis and treatment of substance-related syndromes in geriatric patients.
- Identification and appropriate management of substance use disorders in the inpatient setting, particularly of severe intoxication and acute withdrawal, can prevent serious and life-threatening outcomes.

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Psychiatric Symptoms Comorbid with Neurological Syndromes

11

Elyse Ross, Shaji Khan, and Amer M. Burhan

11.1 Introduction

Complex medical and neurological illnesses are common among patients on inpatient geriatric psychiatry units, due to the high prevalence of Parkinson's disease (PD), stroke (cerebrovascular accidents, CVA), Alzheimer's disease, traumatic brain injuries (TBI), and many other neurodegenerative disorders in the geriatric population [1–3]. Familiarity with their phenomenology and pathophysiology, and the ability to recognize signs of over- or under-treatment, are essential competencies in proper assessment and effective management. Figure 11.1 provides a framework for the assessment of psychiatric/behavioral symptoms in neurological disease.

DSM-5 diagnostic criteria do not necessarily list all neuropsychiatric symptoms or behaviors which are prevalent within the context of neurodegenerative disorders or neurological disorders. Assessment scales, such as the Neuropsychiatric Inventory (NPI) may be used to further describe common phenomenology (Chap. 6: Major Neurocognitive Disorder with Behavioral Disturbance; Chap. 2: Neuropsychological Testing). The NPI is a behavior-rating instrument which is widely used in randomized clinical trials of cognitive enhancers and other medications. It includes ten behavioral and two neuro-vegetative symptom groups [4]. One advantage of the NPI is its established validity in multiple syndromes, including PD, TBI, and vascular neurocognitive disorder [5].

For the purposes of this chapter, common neuropsychiatric symptoms seen in PD, stroke (CVA), and TBI have been divided into symptom clusters. Although not specifically described in the NPI, certain symptom groups such as pseudobulbar affect (i.e. pathological laughing and crying), which are common and potentially disabling, have also been included.

11.2 Vignette 1: Parkinson's Disease and Major Neurocognitive Disorder (MNCD)

A 66-year-old man was brought to the Emergency Department by police for evaluation. His nursing home staff found him forcibly undressing a cognitively impaired female resident. His wife reported the onset of Parkinson's disease (PD) 7 years ago, and he had been followed since then

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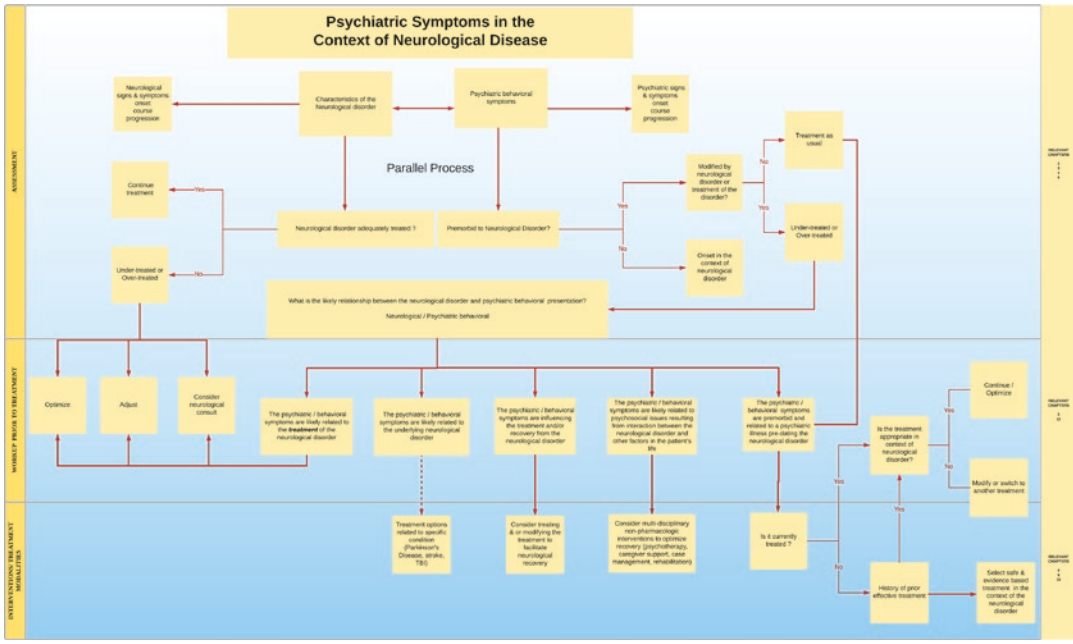

Fig. 11.1 Assessment of neuropsychiatric symptoms in neurological disease

by a neuropsychiatry clinic for motor and behavioral symptoms. PD symptoms were tremor-dominant, with worse tremor and rigidity in the right upper extremity, freezing, and bradykinesia. His PD motor symptoms reached reasonable control with institution and adjustment of dopaminergic medications.

Two years prior to the emergency room visit, the patient had developed symptoms of major neurocognitive disorder (MNCD). A recent neuropsychiatry clinic assessment revealed Mini Mental State Examination (MMSE) score of 23/30, five points lower than an MMSE done 2 years earlier. He had impaired clock drawing, the numbers were not arranged in order, and hands of the clock were incorrectly placed. Major neurocognitive disorder due to PD was diagnosed. He was treated with citalopram 10 mg daily to help evening restlessness and anxiety symptoms, clonazepam 0.25 mg at bedtime to treat REM sleep behavior disorder, and donepezil 10 mg daily for cognitive impairment.

About 18 months prior to the Emergency Department visit, he was referred to a neuropsychiatry clinic for increased restlessness and an increase in inappropriate sexual behavior. He started showing poor impulse control. He demanded sexual intercourse daily from his wife of 20 years. The wife refused her husband's demands. The patient began to leave home without telling his spouse, paying for sex in a crime-ridden part of his town. He showed limited insight into how the behavior impacted his spouse and the risk to himself. Quetiapine 25 mg daily was added in the afternoon, as restlessness and pacing was observed prior to his leaving the home to pursue sex.

His neurologist tapered the dopaminergic medications, in an attempt to reduce sexual behavior and restlessness, but this resulted in an increase in motor symptoms.

His wife could no longer manage his behavior and the patient was admitted to a long-term nursing facility a few months before the incident which led to hospitalization. The nursing facility staff documented the patient's sexually suggestive comments and behaviors. The primary care physician prescribed cyproterone acetate 50 mg daily (an anti-androgen treatment), with no improvement (Chap. 6: MNCD with Behavioral

Disturbance). The patient remained cooperative, did not deny his behaviors, but appeared unconcerned. He had significant tremor and dystonia in right upper extremity greater than left. Some anxiety and restlessness were noted. There was no evidence of psychosis, significant anxiety, or depressed mood. His insight and judgment were impaired.

The patient was admitted to geriatric psychiatry for further management. During a 2-week inpatient geriatric psychiatry hospitalization, medication regimen was revised as follows: citalopram 20 mg daily; quetiapine 12.5 mg at 8:00, 12:00, and 17:00; 25 mg at 15:00; cyproterone acetate 150 mg daily; levodopa/carbidopa/entacapone 150 mg/37.5 mg/200 mg at 6:00; 100 mg/25 mg/200 mg five times daily at 9:00, 12:00, 15:00, 18:00, and 21:00; levodopa/carbidopa 100 mg/25 mg at 12:00 and 15:00; and clonazepam 0.25 mg at bedtime. A behavioral plan included assigning two staff to provide care and monitor his behavior every 2 h. Additionally, his room was reassigned to a location easily observable by staff and away from vulnerable patients. His behavior remained relatively stable with only occasional suggestive sexual comments. He was discharged to the same long-term care facility and then followed by the geriatric psychiatry outreach team.

11.2.1 Discussion

PD affects approximately 1% of the population over 60 years of age [6]. In addition to the motor features, a range of neuropsychiatric behaviors may occur in the context of PD, some of which are the consequences of PD medications rather than the disease itself. The associated signs and symptoms are often under-recognized and undertreated. Non-motor aspects of PD can be separated into "intrinsic" and "iatrogenic" [7]. Intrinsic symptoms include depressive disorders, anxiety disorders, fatigue, apathy, executive dysfunction, and neurocognitive disorders. "Iatrogenic" problems relate to dopaminergic treatment, and include psychotic disorders, affective cycling due to on-off motor fluctuations, and compulsions. Dopamine agonist treatment requires a fine balance between optimal control of motor symptoms and new or worsened psychiatric syndromes, such as impulse control and psychotic disorders [1]. Anticipation of potential harm to others and legal consequences is paramount (Chap. 5: Legal Aspects; Chap. 6: MNCD with Behavioral Disturbance).

"On" refers to periods when the patient experiences a good response to PD medication, while "off" refers to when the medication wears off and motor symptoms reemerge. Strategies for managing the "off" periods include increasing the dose of dopaminergic medication, making individual doses smaller but more frequent, adding an additional PD medication or a catechol-O-methyltransferase inhibitor to prolong the effect [1]. Conversely, if the dopaminergic medication leads to bothersome dyskinesias, medication reduction strategies may improve these symptoms, at the potential expense of worsening parkinsonism [1].

Impulse control disorders, including the hypersexual behavior described in this case, are typically associated with dopamine agonist use in PD [8]. A cross-sectional study of 3090 patients with idiopathic PD (with raters blinded to PD medication status) found impulse control disorders in 13.6% of study patients, including gambling in 5.0%, compulsive sexual behavior in 3.5%, compulsive shopping in 5.7%, and binge-eating disorder in 4.3% [9]. Impulse control disorders were identified in 17.1% of patients on dopamine agonist therapy compared with 6.9% not prescribed a dopamine agonist.

The vignette patient's neuropsychiatric symptoms in PD included anxiety, impulse control disorder, REM sleep behavior disorder, and neurocognitive decline. A study of 360 patients with PD from an academic setting found that 89% exhibited *at least one psychiatric symptom* as identified by NPI. The most common were anxiety disorders (73.1%), depressive disorders (64.7%), apathy (51.7%), and nighttime disturbance (51.3%). Less commonly observed symptoms were disinhibition (3.3%), delusions (1.7%), euphoria/elation (0.3%), and aberrant motor behavior (2.5%) [10].

11.3 Neuropsychiatric Symptoms/Syndromes of Parkinson's Disease (PD)

11.3.1 Depressive, Bipolar, and Anxiety Disorders in Parkinson's Disease (PD)

Depressive disorders affect 35% of patients with PD [11] and are associated with poorer functioning, caregiver stress, and cognitive impairment. A study exploring the profile of neuropsychiatric symptoms in patients with neurocognitive disorder due to PD using the NPI found among the most common symptoms were depressive disorders (58%), apathy (54%), and anxiety disorders (49%) [12].

Differentiating between symptoms of PD and depressive disorders can be challenging, as psychomotor retardation, sleep disturbance, reduced appetite, and altered facial expression are often observed in non-depressed patients with PD [11]. Non-depressed patients with PD may exhibit social withdrawal, as the motor symptoms of PD cause them to feel uncomfortable in social settings [13]. However, anxiety, brooding, irritability, cognitive deficits, pessimism, and suicidal ideation (though lower rates of guilt and self-blame than in typical depressive disorders) are clinical features more common in depressed patients with PD versus depressed patients without PD [14]. Pessimistic thoughts about one's self, the world, and future, early morning awakening, and low mood for >2 weeks with diurnal variation are more suggestive of depressive disorder [15]. Table 11.1 lists features which may help distinguish between major depressive disorder, apathy, and Parkinson's disease motor symptoms.

Major depressive disorder in the setting of established PD should be treated, although the literature is mixed regarding most effective agents. A recent randomized, double-blind, placebo-controlled trial of antidepressants in 115 participants with PD found a mean Hamilton Rating Scale for Depression (HAM-D) score reduction of 6.2 points in the paroxetine treatment group ($p < 0.0007$) and a HAM-D score reduction of 4.2 points ($p < 0.02$) in the venlafaxine XR group, with no worsening of motor function. This study provided class I evidence that paroxetine and venlafaxine XR are effective in treating depressive disorders in PD [16]. A systematic review and meta-analysis of 20 randomized controlled trials including 893 patients with PD and depressive disorder showed a significant aggregate effect of pharmacologic agents on the treatment of depressive disorders. However, stratification of this data showed a statistically significant overall effect only for SSRIs. This review showed a statistically significant effect size (0.87, $p = 0.000$) with the use of behavioral therapy, particularly cognitive behavioral therapy [17].

The decision to use TCAs must be weighed against the risk of cognitive impairment due to their anticholinergic (anti-ACh) effects. A systematic review of evidence for treatment of

Table 11.1 Distinguishing features of major depressive disorder, apathy, and motor symptoms of PD

Condition	Distinguishing clinical features
Depressive disorder	Anxiety, brooding, irritability, pessimistic thoughts, suicidal ideation more common
	Lower rates of guilt and self-blame; early morning awakening
	Diminished interest or pleasure
	Depressed mood with diurnal variation
	Duration of depressive symptoms > 2 weeks
Apathy	Lack of motivation
	Presence of goal-directed speech and activity without hopelessness, depressed mood, or anhedonia
	Neutral mood or indifference
Parkinson's disease: psychomotor and physical features (may overlap with features of DSM-5 major depressive disorder)	Social withdrawal may result from self-consciousness about motor symptoms
	Sleep-wake disorders
	Psychomotor slowing
	Concentration difficulties
	Slow initiation of motor behavior
	Fatigue

depressive disorders in PD with electroconvulsive therapy (ECT) showed improvement in depressive symptoms in 93.1% of patients, and no worsening of cognition in the majority, although delirium and transient confusion led to discontinuation in some patients [18] [Chapter 16: Neuromodulation Interventions (ECT, rTMS)].

11.3.2 Psychotic Symptoms in PD

Psychotic symptoms have been found in 44% of 537 patients with neurocognitive disorder due to PD, assessed with the NPI [12]. If delusions are observed, they are typically paranoid in nature [7]. Generally, hallucinations are seen in later-stage PD (but may occur earlier in older patients) and can be associated with PD medication [1]. Visual hallucinations may require a lower dose of dopaminergic treatment, if feasible. [Chapter 6: MNCD with behavioral disturbance]. If antipsychotic treatment is necessary, clozapine or quetiapine are recommended due to their relatively low D2 blockade, but must be used cautiously, as the geriatric patient with PD is especially vulnerable to adverse events. Quetiapine is the first choice in geriatrics, as clozapine requires frequent blood monitoring and carries the risk of agranulocytosis as well as a significant risk for delirium due to its anti-ACh effects.

Pimavanserin is a novel antipsychotic that works on cortical serotonergic receptors instead of dopamine receptors. A multi-center, open label post-hoc analysis of pimavanserin compared patients who had an established diagnosis of idiopathic PD and moderate-to-severe psychotic symptoms, taking, or not taking, antipsychotics. The adjusted mortality rate was four times higher in participants on atypical antipsychotics [19], in addition to increased risk of serious adverse events.

Evidence suggests ziprasidone may be as effective as clozapine in ameliorating psychotic symptoms in PD, based on a randomized, single blind, open-label comparison [20]. In this study, there were no statistical differences in Abnormal Involuntary Movement Scale (AIMS) scores between the two groups, despite the fact that ziprasidone is known for higher D2 antagonism. Although olanzapine is a less potent D2 antagonist, it has been found ineffective for hallucinations in PD and may cause motor deterioration [21].

11.3.3 Personality Changes, Disinhibition, and Impulse Control Disorders in PD

Pathologic gambling, hypersexuality, and other compulsive behaviors (e.g., excessive shopping and eating) may occur in the context of treatment with dopamine agonists. A history of addictive behaviors, impulsive personality style, and premorbid obsessive-compulsive disorder has been suggested to increase the risk of impulse control disorders [22] in patients with PD. If unclear whether the symptoms are due to dopaminergic treatment, a careful history should help clarify the presence of a premorbid impulse control disorder. As well, in the setting of NCD complicating PD, pathologic sexual behaviors may be related to loss of inhibition [13].

When treating impulse control disorders, consider dopamine agonist dose decrease, or tapering off agonists, particularly if the patient is taking pramipexole. If symptoms spontaneously improve in the inpatient setting without dose changes, consider whether the patient was overusing the agonist at home. Involvement of family members may be required to monitor medication usage. In our clinical experience, if hypersexual behaviors cause significant impairment and cannot be managed by the above, antidepressants, antipsychotics, and/or anti-androgen agents may help given their effect on impulsiveness, goal-directed behavior, and sexual drive, though efficacy is variable. Available literature on this topic is scant.

11.3.4 Aberrant Motor Behavior in PD

Caregivers may also describe the patient's fascination with excessive, repetitive, non-goal-oriented behaviors (e.g., repetitively dismantling electrical equipment). This is known as punding syndrome [23]. Punding may improve with

reduction of dopaminergic medication. One small open-label prospective study showed benefit with amantadine and quetiapine [24].

11.3.5 Agitation/Aggression in PD

Agitation/aggression may occur in the context of neurocognitive disorder due to PD, or psychotic disorders. In a study of 537 patients with neurocognitive disorder due to PD, agitation on the NPI was reported in 5%. Patients with the agitation symptom cluster were among those with the lowest MMSE scores and higher caregiver distress scores [12]. Consider whether agitation is potentially a medication side effect, particularly with dopaminergic treatment.

11.3.6 Sleep-Wake Disorders in PD

Sleep disorders affect 60–98% of PD patients [25]. In the literature, sleep disorders have been divided into two categories: dyssomnia (including insomnia or hypersomnia) and parasomnia [13]. Insomnia includes difficulties initiating sleep, sleep fragmentation, and early morning awakening. Motor symptoms of PD, psychiatric disorders, and neurocognitive disorders may cause insomnia. Hypersomnia refers to excessive daytime sleepiness and affects 20–50% of patients with PD [13]. Parasomnias include phenomena which occur during sleep, including nightmares, night terrors, vivid dreams, sleep walking, hallucinations, and REM sleep behavior disorder.

When complaints about sleep arise, it is useful to review sleep hygiene strategies, and any contributing medical or psychiatric conditions [13]. A medication review should minimize activating medications. Referral for a sleep study may be warranted. Clonazepam may be helpful for REM sleep behavior disorders. Other problems such as pain and stiffness can be a source of insomnia. Appropriate pain management (e.g., adding an analgesic before bedtime), or optimizing dopaminergic treatment, can be tried (Chap. 14: Pain; Chap. 9: Sleep).

11.3.7 Appetite Changes and Eating Disorders in PD

A cross-sectional study found binge-eating disorder (diagnosed using proposed research criteria in DSM-IV-TR) in 4.3% of 3090 patients with treated idiopathic PD [9].

11.3.8 MNCD and PD

Neurocognitive disorder due to PD is not addressed in detail here (Chap. 6: MNCD with Behavioral Disturbance). A systematic review of studies focusing on PD and neurocognitive disorder due to PD in European and North American populations suggests that between 24% and 31% of patients with PD have neurocognitive disorders. Of this subset, 3–4% of these neurocognitive disorders are due to PD [26].

11.4 Stroke/Cerebrovascular Accident (CVA)

11.4.1 Vignette 2

An 84-year-old right-handed gentleman was referred from the CVA unit to geriatric inpatient psychiatry for auditory hallucinations, depressed mood, and apathy. He had a left hemisphere ischemic stroke 2 weeks earlier. It was unclear how to distinguish his apathy from depressed mood. His family reported a prior history of untreated auditory hallucinations for over 40 years that did not affect his functioning. There was a family history of auditory hallucinations in his brother and father, but no clear diagnosis.

Following the CVA, hallucinations worsened. He now described voices of several people, which spoke different languages, conversed with each other, and commented on him. Since the CVA they woke him up nightly, making his sleep nonrefreshing. He became distressed by the voices and depressed. He now was spending most of his time in bed and reduced his participation in rehabilitation and activities.

The brain MRI revealed that along with the CVA affecting frontal white matter, he had an old left temporal pole injury, encephalomalacia, and scattered white matter hyperintensities in subcortical white matter in the parietal and frontal areas bilaterally. The family confirmed he sustained a few falls over the last couple of years and hit his head. As well, his most recent CVA left him with left-sided hemiparesis and left arm spasticity.

On the inpatient unit, geriatric psychiatry consultation suggested a trial of quetiapine for the nighttime hallucinations and to help his sleep, titrated up to 150 mg at bedtime and 25 mg in the morning. The quetiapine was eventually reduced to 100 mg at bedtime due to dizziness. This helped diminish the hallucinations. He coped with the voices through his religious faith. The patient also reported vague gustatory and olfactory hallucinations early on, but these were transient. He denied any anxiety or panic-like symptoms, but had persistently flat affect and lack of interest in activities. He remained largely independent in activities of daily living, but required significant support in attending to his hygiene. He relied on his family for most of his instrumental activities of daily living.

Over the next 6 months, the geriatric neuropsychiatry outpatient clinic treated the hallucinations with a higher dose of quetiapine, limited by tiredness and dizziness. A trial of an activating antidepressant (venlafaxine) was initiated at 37.5 mg once daily. The patient eventually asked to be off all medication. At the last follow-up assessment, he had been off all medications for 3 months and was relatively stable. He received botulinum injections and physiotherapy to the left spastic hand with significant improvement.

11.4.2 Discussion

This patient had a long-standing history of auditory hallucinations, which worsened following brain insults. He had depressed mood and apathy, and it was very difficult to distinguish their respective contributions to his condition. Apathy can easily be mistaken for depressive disorders although the two syndromes *can* be differentiated. A cross-sectional study of neuropsychiatric symptoms using the NPI found no correlation between apathy and depressive disorders in 154 patients with 5 neurodegenerative disorders; furthermore, depressive disorders were associated with irritability, agitation, hallucinations, and anxiety, while apathy was associated with aberrant motor behavior and disinhibition [27].

Although antipsychotic treatment had improved the hallucinations, the side effect of dizziness and sedation from quetiapine limited its use, in an effort not to contribute to depressed mood. Less-sedating antipsychotic medications could have been tried, but the risk/benefit ratio was considered. Non-pharmacological interventions, e.g., psychoeducation and coping strategies, can help in managing these symptoms. Some symptoms may resolve spontaneously. The vignette illustrates how neuropsychiatric symptoms can impede rehabilitation and improvement.

11.4.3 Neuropsychiatry of CVA/Stroke

11.4.4 Depressive, Bipolar, and Anxiety Disorders Post-CVA

A variety of psychiatric symptoms can occur post-stroke (also referred to as cerebrovascular accident–CVA). Depressive disorder is a common neuropsychiatric syndrome following a stroke. In one study, the NPI was administered to 124 post-CVA patients. Depressive disorders were identified in 61%, irritability in 33%, apathy in 27%, and anxiety disorders in 23% [28]. Two recent systematic reviews found that between 29% [29] and 33% [30] of patients experience depressive disorders up to 1 year following a stroke. These studies used various methods of defining post-CVA depressive disorders, including "mood disorder due to a general medical condition" criteria, minor depression (a research diagnosis from DSM-IV), and results of clinical rating scales. The optimal method identifying post-CVA depressive disorders is unclear. In a study examining frequency of depressive

symptoms in patients with post-CVA depression vs. participants without known brain pathology, both groups showed almost identical symptom profiles [31]. Fatigue is a common report in the geriatric population following CVA. Post-CVA fatigue may occur independently of a major depressive episode, preceding the onset of psychiatric symptoms [32].

The etiology of post-CVA depressive disorder is likely a combination of neurobiological effects of CVA and psychosocial factors. A systematic review and meta-analysis of 50 studies revealed a post-CVA depressive disorder prevalence of 29%, with cumulative incidence of 39–52% within 5 years of CVA [29]. The relationship between post-CVA depressive disorder and lesion location is complex and controversial. A meta-analysis of studies comparing frequency of major depressive disorder among different lesion locations found the odds ratio for depressive disorder was 2.29 greater for left anterior vs. left posterior lesions [33].

Best practice recommendations emphasize that all patients with CVA should be considered at high risk of developing a depressive disorder [34]. However, the presence of certain neurologic symptoms (e.g., aphasia) may complicate the assessment process. Alternate tools include the Stroke Aphasic Depression Questionnaire-10 (SADQ-10), with a sensitivity of 70% and specificity of 77% using a cut-off score of greater or equal to 15 [35]. Neurocognitive impairment in post-CVA depressive disorder is an important consideration, although discussion of neurocognitive disorders is beyond the scope of this chapter. One study [36] found that patients with major depressive disorder after left-hemisphere CVA had significantly more impaired MMSE scores than a comparison group of non-depressed participants. As well, both the disorder severity and size of vascular lesion correlated independently with cognitive impairment severity.

Prevention of post-CVA depressive disorders: a Cochrane review of 14 trials with 1515 participants found a small but significant effect of psychotherapy on improving mood and preventing depressive disorders following CVA, but did not find evidence for prophylactic antidepressants preventing depressive disorders in the post-CVA period [37] (Chapter 18: Psychotherapies and Non-pharmacologic Interventions).

A review of 16 trials examining treatment interventions for post-stroke depressive disorder did not demonstrate a benefit of psychotherapy. There was a small but significant improvement with pharmacotherapy (including seven trials comparing SSRIs to placebo, two trials comparing TCAs to placebo, and four trials including deanxit, aniracetam, reboxetine, and trazodone, respectively) on illness remission (pooled odds ratio of 0.47 with 95% CI 0.22–0.98), with a significant increase in adverse events [38]. An *open-label*, controlled clinical trial including 60 patients with recent CVA and post-CVA depressive disorder assigned to receive duloxetine, citalopram, or sertraline [39] showed significant improvement in symptoms of depressive ($p < 0.01$) and anxiety disorders ($p < 0.01$) which were observed in all study groups. However, patients treated with duloxetine improved faster and to a greater degree than patients treated with SSRIs.

A small RCT showed that methylphenidate significantly reduced severity of depressive symptoms in post-CVA cases [40]. However, the American Stroke Association and American Heart Association note that in light of the cardiovascular side effect profile, further studies are needed [41]. In terms of other treatment modalities, electroconvulsive therapy (ECT) has been reported as effective for post-CVA depressive disorder, with few side effects [42].

The prevalence of bipolar disorder post-CVA is relatively low, with prevalence less than 2% [43]; often it is really a misdiagnosed delirium. One study found approximately half of patients with manic symptoms had delirium as a potential cause. As well, about half of patients developed mania within a few days post-CVA, while the other half had onset of symptoms 1–24 months following the stroke [44].

Anxiety disorders post-CVA receive less attention than post-CVA depressive disorders, but are an important consideration. Generally, the use of a structured or semi-structured interview is recommended. A recent study found the prevalence of post-CVA anxiety disorders to be 20.4%

and that lifetime anxiety was a good predictor of these disorders [45]. A 2017 meta-analysis found 13 studies involving 2408 patients demonstrating association between post-CVA anxiety and depressive disorders [46]. An open-label, controlled clinical trial including 60 patients with recent CVA and post-CVA depressive disorder assigned to receive duloxetine, citalopram, or sertraline found that duloxetine was well tolerated and significantly more effective than citalopram and sertraline ($p < 0.01$) for treating anxiety symptoms in post-CVA depressive disorder [39].

Apathy is a relatively common phenomenon post-CVA, with prevalence of 34–36% with a median of 120 days post-CVA, as found in a meta-analysis of 2706 patients from 24 separate studies [47]. As well, comorbid depressive disorders may co-occur in up to 40% of patients with apathy post-CVA. Apathy can be challenging to differentiate from depressive disorders in the clinical setting. The Neuropsychiatric Inventory may be helpful, and Starkstein's Apathy Scale has been found valid and reliable in Japanese patients post-CVA [48]. A study examining the effect of depressive disorder or apathy post-CVA in 237 Japanese CVA patients observed apathy in 19.2% using the NPI. This study also found apathy may be more frequently associated with functional abilities and likely to interact with the recovery process post-CVA as compared to depressive disorder [49].

There is no clear evidence on which treatments are best for apathy. A systematic review of double-blinded randomized controlled trials to determine the effectiveness of dopaminergic agents on various outcomes did not identify any studies focusing directly on apathy [50], although one study did find that combined dopaminergic treatment (methylphenidate and levodopa) significantly improved Geriatric Depression Scale scores compared to placebo at 90 and 180 days [51]. Successful treatment of apathy with the dopamine receptor agonist ropinirole following a prefrontal cortex CVA has been reported [52].

Pseudobulbar affect refers to incongruent emotional expressions disproportionate to the underlying emotional experience. A systematic review and meta-analysis (15 studies with 3391 participants) found the prevalence of pseudobulbar affect was 17% less than 1 month post-CVA, 20% 1–6 months post-CVA, and 12% >6 months post-CVA [53]. This study also found that crying is a more common manifestation of pseudobulbar affect than laughing in the post-CVA population.

A Cochrane review of seven trials involving 239 participants with CVA found that several trials showed antidepressant-related improvements in emotionalism, tearfulness, lability, and improved scores on the Pathologic Laughing and Crying Scale. This effect was not specific to a particular class of medications, and methodological deficiencies in the studies were noted [54].

11.4.5 Psychotic Disorders Post-CVA

Psychotic disorders in the post-CVA setting are relatively uncommon compared to mood disorders. In eight patients without a pre-existing psychiatric disorder, authors of one study discuss onset of psychotic symptoms between 1 month and 11 years following right hemisphere CVA [55]. In terms of treatment, antipsychotic medications have been used with some success in treatment-resistant cases, as well as anticonvulsant medications [56].

11.5 Personality Changes/ Disinhibition/Impulse Control Disorders Post-CVA

A series of 35 caregivers of patients with CVA reported personality changes including reduced patience, increased frustration, reduced confidence, higher dissatisfaction, and a less easy-going nature [57]. Adjustment to new disability and psychosocial consequences related to loss of independence should be considered and treated.

11.6 Aberrant Motor Behaviors Post-CVA

One study examining NPI symptom clusters found < 10% prevalence of aberrant motor behaviors in post-CVA patients, with particular behav-

ior descriptions including repeatedly buttoning/unbuttoning clothes and moving back and forth with a wheelchair [28]. Evidence for management of aberrant motor behavior in the post-CVA setting is lacking.

11.7 Agitation/Aggression Post-CVA

Agitation has been found in 28% of post-CVA patients using the NPI [28]. Elsewhere, an interview of 145 post-CVA patients using the 10-item Spielberger Trait Anger Scale found inability to control anger, or aggression in 32% [58]. A post hoc analysis of results of a treatment study for post-CVA depressive disorder found that when depressive disorder accompanies aggression in the post-CVA setting, successful treatment of the depressive disorder might reduce aggression [59].

11.8 Sleep-Wake Disorders Post-CVA

A relationship between CVA and obstructive sleep apnea has been suggested in epidemiological studies [60]. A prospective observational study of 68 acute CVA inpatients found that sleep-disordered breathing improves 6–9 weeks following CVA, but is still a common finding. The researchers found more severe sleep-disordered breathing in patients with lacunar CVA, in patients of older age, and patients with pre-existing CVA handicap [61]. In the geriatric inpatient, sleep-disordered breathing may be responsible for reversible cognitive impairment [62].

11.9 Appetite Changes and Eating Disorders Post-CVA

Eating disturbances have been described in 33% of post-CVA patients using the NPI [28]. A cohort of 206 patients admitted to a National Health Service trust hospital in the UK used the Modified Barthel Index and Eating Disabilities Assessment Scale to assess for symptoms related to eating [63]. Approximately 12% (11.7%) of patients reported poor or no appetite, and poorer appetite was associated with increased mood disturbance. CVA related dysphagia is an important consideration. 10.7% expressed dissatisfaction with their ability to eat and feed themselves and 32.1% with their ability to prepare meals. Patients should be screened for swallowing deficits in the post-CVA setting and may require swallowing study and assessment by a speech language pathologist, occupational therapist, or dietitian [34].

11.10 Neuropsychiatry of Traumatic Brain Injury (TBI)

Traumatic Brain Injury (TBI) is common in the geriatric population. Assessment of the condition and its residuals is challenging in the context of an aged patient's medical comorbidities and medications. Following mild traumatic brain injury (mTBI), approximately 15% of individuals experience residual symptoms.

11.10.1 Depressive, Bipolar, and Anxiety Disorders in TBI

The Neuropsychiatric Inventory (NPI) is helpful in identifying mood-related symptoms of TBI. In a sample of 120 patients with severe traumatic brain injury, administration of the NPI found apathy in 42%, irritability in 37%, and depressed mood in 29% [64] [Chapter 6: Major Neurocognitive Disorders with Behavioral Disturbance].

Major depressive disorder (MDD) is a frequent neuropsychiatric complication of TBI. In a prospective, case-controlled surveillance study of 91 patients with TBI [65], MDD was observed in 33%, significantly more frequent (p = 0.008, Fisher exact test) in the TBI group compared to control group (7.4% with MDD). The majority of patients with TBI and MDD also had anxiety disorders (76.7%), aggressive behavior (56.7%), and significantly greater impairment in measurements of executive functioning. The Wisconsin Card-Sorting Test number of perseverative errors

showed significant differences ($F_{1,4}$ = 4.76, p = 0.03) as did the Trail Making Test B/A ratio ($F_{1,51}$ = 5.82, p = 0.02). Patients with TBI and MDD were also more likely to have a personal history of depressive and anxiety disorders, suggesting that prior depression and anxiety may constitute risk factors for the development of mood symptoms in TBI. MDD was also associated with reduced left prefrontal gray matter volumes in the ventrolateral and dorsolateral regions [65]. In a retrospective chart review of patients older than 60 years with TBI, divided in depressed and non-depressed groups based on Geriatric Depression Scale scores, the authors found that medical comorbidity, functional limitation, and the presence of bilateral and left hemispheric injury may indicate a higher risk of developing depressive disorders post-TBI [66].

SSRIs are generally recommended as first-line treatment of MDD in the TBI population. The Neurobehavioral Guidelines Working Group [67] recommends SSRIs as first-line treatment for severe, persistent depressive, and anxiety disorders. In terms of prevention of MDD following TBI, a recent study showed efficacy of sertraline 100 mg/day in a sample of hospitalized patients ranging from 18 to 85 years of age [68]. However, as geriatric patients with TBI are especially vulnerable with respect to adverse effects, conservative dosing and avoiding rapid titration is recommended. Paroxetine, which may lead to cognitive side effects due to anticholinergic properties, is not recommended [69], which is of particular concern in the geriatric population.

A review of three randomized controlled trials for psychological treatments in people with TBI and anxiety disorders showed lower post-treatment anxiety symptomatology in participants with mild-moderate TBI in the combined CBT and neurorehabilitation group compared to the control group [70].

In a Cochrane review of randomized controlled trials of interventions targeting apathy in patients with TBI, it was not possible to determine whether cranial electrotherapy led to greater improvements in apathy than in the control group [71]. In the setting of depressive disorder with apathy following TBI, stimulants such as methylphenidate may augment a partial response to SSRIs [72].

11.10.2 Psychotic Disorders in TBI

Psychotic disorders are relatively rare as a neuropsychiatric syndrome following TBI, but symptoms which suggest psychosis warrant prompt attention if suspected, as such symptoms can impact both patient and caregivers significantly. Guidelines recommend referral to a psychiatrist specializing in the treatment of persistent TBI-related symptoms if a psychotic disorder is suspected [67]. In terms of pharmacologic management, it is recommended to use antipsychotic medications using the "start low, go slow" approach beneficial in the general geriatric population. Examples described in the literature include starting daily doses such as risperidone 0.5 mg, olanzapine 2.5 mg, quetiapine 50 mg, and clozapine 6.25–12.5 mg [73].

11.10.3 Personality Changes/ Disinhibition/Impulse Control Disorders in TBI

Following TBI, patients may present with personality changes including disinhibition, affective lability, verbal and/or physical aggression, and apathy. A study using NPI to assess symptom prevalence found disinhibition in 28% [64]. The presence of previous physical limitations, confounding effects of other health conditions are associated with poorer outcomes in TBI. As well, older age may negatively influence the recovery process [67]. In the setting of complicating factors, referral to a multidisciplinary treatment clinic may be warranted.

11.10.4 Aberrant Motor Behavior in TBI

TBI-related aberrant motor behavior has been observed in 9% of patients using NPI [64]. In this particular study, patients with focal lesions (particularly focal unilateral or bilateral) had an increased risk of developing aberrant motor

behaviors. Evidence for management of aberrant motor behavior in the TBI setting is lacking.

11.10.5 Agitation/Aggression in TBI

A study using NPI to identify symptoms in severe TBI found agitation in 24% of patients [64]. Clinical features more common in patients with TBI and aggression include alcohol or substance use, presence of pre-injury mood disorder, frontal lobe lesions, and aggressive behaviors requiring legal intervention [74]. Clear documentation of the precipitants, timing, frequency, intensity, and response to interventions is recommended, followed by a plan for behavioral, non-pharmacological intervention [75] (Chap. 18: Psychotherapies and Non-pharmacological Interventions). The use of medications for non-cognitive symptoms of TBI or MNCD has been reviewed elsewhere [Chap. 6: MNCD with behavioral disturbance]. There are risks associated with use of these medications, and evidence of effectiveness is not robust.

11.10.6 Sleep-Wake Disorders in TBI

The prevalence of sleep disturbances in TBI has been reported at 15% using the NPI [64]. A prospective evaluation of 87 adults at least 3 months post TBI using polysomnography, multiple sleep latency test, Epworth Sleepiness Scale (ESS), and neuropsychological testing found abnormal sleep studies in 46%, and excessive daytime sleepiness in 25%. Twenty-three percent had obstructive sleep apnea, 11% had posttraumatic hypersomnia, 6% with narcolepsy, and 6% with periodic limb movements in sleep [76]. Insomnia disorder is the most common type of sleep disturbance following TBI [77].

Treatment of sleep disorders after TBI (e.g., continuous positive airway pressure for obstructive sleep apnea) may improve polysomnographic findings but not necessarily sleepiness scores or neuropsychological function [78]. Guidelines for treatment of persistent symptoms following mild TBI describe sleep disorders as a "primary symptom" warranting treatment before symptoms related to cognition, balance, vertigo, fatigue, and noise intolerance [67]. In terms of treatment, these guidelines outline the following recommendations: advising patients the goal of treatment is to improve sleep continuity and quality, providing sleep hygiene strategies, relaxation training, compressing the total time in bed to match the total sleep need, and judicious use of short-term pharmacotherapy (i.e., less than 7 days) at the lowest effective dose [67] [Chapter 9: Sleep].

11.10.7 Appetite Changes and Eating Disorders in TBI

One study identified eating disturbances in 27% of patients with severe TBI using the NPI [64]. As well, changes in eating patterns may occur with a comorbid major depressive episode, thus mood should be carefully assessed. A systematic review of 54 case reports of eating disorders in brain injury found certain changes in appetite and eating behavior can occur in hypothalamic lesions (changes in appetite and thirst) and brain stem lesions (difficulty swallowing), while more complex changes are associated with right frontal and temporal lobe damage [79].

11.10.8 Neurocognitive Disorders and TBI

A recent meta-analysis suggests that history of TBI (including mild injuries) is associated with higher odds of later development of Alzheimer disease, PD, mild neurocognitive disorder, depressive disorder, bipolar disorder, and disorders with combined depressive and anxiety symptoms [80]. Cognitive rehabilitation with an occupational therapist, speech therapist, or neuropsychologist is recommended [69].

11.11 Neuropsychiatric Features Among Other Neurologic Conditions

Although this chapter emphasizes PD, stroke/post-CVA, and TBI, the geriatric inpatient may present with neuropsychiatric features of various

other conditions. Examples include multiple sclerosis (MS), neurosyphilis, neuroinflammatory conditions, acquired immune deficiency syndrome (AIDS), seizure disorders, prion disease, paraneoplastic syndrome, and Huntington disease. Regardless of the specific condition, adopting a systematic approach in defining symptoms and understanding the interplay between the neurological, psychiatric, and psychosocial aspects of the illness will guide assessment and management. Table 11.2 summarizes treatment recommendations for common neuropsychiatric symptoms in Parkinson's disease, post-CVA, and TBI.

Table 11.2 Treatment recommendations for neuropsychiatric symptoms in neurological conditions

Psychiatric symptomatology	Parkinson's disease: psychiatric treatments	Post-stroke (cerebrovascular accident–CVA): psychiatric treatments	Traumatic brain injury: psychiatric treatments
Depressive, bipolar, and anxiety disorder symptoms	Depressive disorder: Class I evidence for paroxetine and venlafaxine ER	Prevent depressive disorder: psychotherapy	Depressive disorder: SSRIs first-line sertraline effective both for treatment and prevention
	DA agonists: pramipexole	Treatment: duloxetine improves symptoms faster and to a greater degree than SSRIs (citalopram and sertraline also effective)	Low dosing, avoid rapid titration
	ECT (highly effective)	Methylphenidate may improve depressive disorder cardiovascular SEs	Paroxetine not recommended (cognitive side effects)
	Behavioral therapy, particularly CBT	ECT effective, well-tolerated	Anxiety: SSRIs first line
	Beware of cognitive impairment with TCAs	Anxiety symptoms in MDD: duloxetine well tolerated and more effective than citalopram and sertraline	Combined CBT and neurorehabilitation reduces anxiety symptoms [Chapter on Medical Interventions]
		Apathy: some evidence for dopamine agonists (e.g., ropinirole); structured activities may be helpful	Depressive disorder with apathy: methylphenidate may augment SSRIs
		Pseudobulbar affect: antidepressants improve emotionalism, tearfulness, lability	
Psychotic symptoms	First adjust dopaminergic treatment	Antipsychotics may improve symptoms	Antipsychotics (AP): "Start low, go slow" if APs required: risperidone, olanzapine, quetiapine, clozapine
	Rx: Avoid first-generation antipsychotics (FGAs) and second-generation antipsychotics (SGAs) with high D2 blockade; use clozapine or quetiapine		Referral to psychiatrist/mental health team familiar with TBI symptoms
Personality changes, disinhibition, impulse control disorders	Consider reducing dopamine agonist, particularly pramipexole	Consider adjustment to disability and address psychosocial factors	Consider patient's coping mechanisms and adjustment to disability; address psychosocial factors
	Consider anti-androgens or antidepressants with sexual side-effects for severe sexual disinhibition		Older age may negatively influence recovery process

(continued)

Table 11.2 (continued)

Psychiatric symptomatology	Parkinson's disease: psychiatric treatments	Post-stroke (cerebrovascular accident–CVA): psychiatric treatments	Traumatic brain injury: psychiatric treatments
	Counsel patients, family/ caregivers		
Agitation/ aggression	Consider if agitation is a side effect of dopaminergic treatment	May improve with treatment of comorbid depressive disorder	Document precipitants, timing, frequency, intensity, etc.
	Rx with analgesics for pain		Minimize deliriogenic medications
			Non-pharmacologic strategies first unless imminent risk of DO, DS
Aberrant motor behavior	Punding: first reduce dopaminergic treatment	Evidence is lacking – assess dopaminergic treatment if present; assess for underlying precipitants such as discomfort, boredom, hunger, disorientation, restlessness, etc.	Evidence is lacking – assess dopaminergic treatment if present; assess for underlying precipitants such as discomfort, boredom, hunger, disorientation, restlessness, etc.
	Consider amantadine, quetiapine		
Sleep-wake disorders	Review sleep hygiene strategies, pain, treat medical, and psychiatric conditions	Consider sleep study/referral to sleep specialist	Diagnose and treat sleep disorder – before cognition, balance, vertigo, fatigue, noise intolerance
	Minimize activating medications		Psychoeducation and sleep hygiene
	Clonazepam for REM sleep behavior disorder		Pharmacotherapy: short-term only at lowest effective dose
Appetite changes and eating disorders	Consider reducing dopamine agonist for compulsive eating	Treat comorbid depressive disorder	Treat comorbid depressive disorder
	Counsel patient and family	Swallowing study, assessment by speech language pathologist, OT, dietitian	Swallowing study, assessment by speech language pathologist, OT, dietitian

11.12 Summary

Neuropsychiatric symptoms in the geriatric inpatient have diverse etiologies and their management is complex. Three neurological conditions, CVA (stroke), PD, and TBI exemplify situations in which concomitant psychiatric symptoms need identification and management. A systematic approach starts with assessment of the neurological syndrome and its treatment, followed by formulating the psychiatric diagnosis using DSM-5. The NPI offers a useful framework for describing and organizing diverse symptoms, and has proven validity in characterizing the psychiatric/behavioral symptoms in the context of a neurological condition. Certain symptoms, such as pseudobulbar affect are not specifically outlined in the NPI, but are prevalent.

The two case vignettes illustrate how common neuropsychiatric symptoms can also result in legal and psychosocial problems. Management requires a comprehensive inter-professional team approach. Biological treatment of neuropsychiatric symptom clusters in the geriatric population has not been studied adequately, resulting in little evidence-based guidance. However, recognition of the prevalence and etiologies of neuropsychiatric symptoms can help develop interventions which moderate symptoms and prevent their worsening.

Take-Away

- Recognize and document core features of the neurological disorder.
- Review the current treatment of the neurological disorder, its appropriateness, and effectiveness.
- Diagnose the psychiatric presentation and history using DSM-5 criteria.
- Describe the onset, history, and course of the psychiatric condition.
- With the aforementioned information, develop a formulation about the relationship between psychiatric symptoms and the neurologic syndrome:
- Idenitfy acute psychiatric symptoms which arose as a consequence of new or exacerbated neurological symptoms.
- Idenitfy chronic psychiatric symptoms which were exacerbated/worsened as a result of the neurological disorder or its treatment.
- Suspect psychiatric/behavioral symptoms which arose as psychological reactions to a neurological disorder or its sequelae, e.g., adjustment disorder in reaction to loss of independence, loss of ADLs, loss of IADLs.
- Identify and assess psychiatric symptoms which impede the recovery process and/or rehabilitation.
- Consider any adjustments to the treatment regimen for the neurological disorder (e.g., consider risk/benefit ratio of optimizing motor symptom control in PD vs. exacerbating psychotic disorder).
- Search for *prior* effective treatments of this patient's psychiatric symptoms; determine if these treatments are supported by research evidence and/or clinical guidelines.
- Review the psychotropic medication regimen to avoid worsening of the neurological disorder (e.g., risk of exacerbating motor symptoms in PD with antipsychotic treatment).
- Identify psychosocial interventions which may improve psychiatric and/or neurological symptoms.

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12 Delirium: Risk Factors, Contributors, Identification, Work-Up, and Treatment

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12.1 Introduction

Delirium, depression, and dementia (major neurocognitive disorder—MNCD) constitute the "3 Ds" of geriatric psychiatry [1]. These common syndromes occur both as discrete entities and as co-existing conditions in geriatric patients [2, 3].

Delirium with *hypoactive* features, the most common delirium subtype in the geriatric population [4], has clinical symptoms which overlap with depression and major neurocognitive disorder (MNCD). These include apathy, executive dysfunction, problems with concentration, and anergia. Fluctuation between confusion and transient lucidity, the hallmarks of delirium, complicate the picture. Clinicians often think in terms of a single etiology, whereas several co-morbid causes, as will be discussed later in this chapter, may be involved.

Delirium is prevalent but under-recognized. It has been estimated to be present in 1 out of 10 older patients in the emergency department, and missed 2/3 of the time in the same patient population [5]. It is also estimated that 30–40% of delirium is preventable [6, 7]. The flowchart in Fig. 12.1 summarizes the work-up, identification, risk factors, contributors, and treatment for delirium.

Delirium is particularly common in inpatients. Up to one-half of geriatric patients admitted to the hospital, experience delirium; the rate is much higher for those in intensive care or undergoing surgery, upwards of 90% [6, 7]. Until recent decades, hospital-acquired delirium, which typically lasts a few days to several weeks, but may also last months, was considered a result of old age and not a condition to be prevented or treated [8].

In recent years, delirium has been thought of as acute brain failure similar to acute congestive heart failure, with multiple contributions [9]. Delirium can be conceptualized as a result of varying degrees of noxious insults (e.g., major surgery, psychoactive medications, sepsis, dehydration), overwhelming the brain, especially in someone with variable cognitive reserve. The greater the noxious insult, the greater the cognitive reserve required not to result in delirium. An imbalance of the "stress

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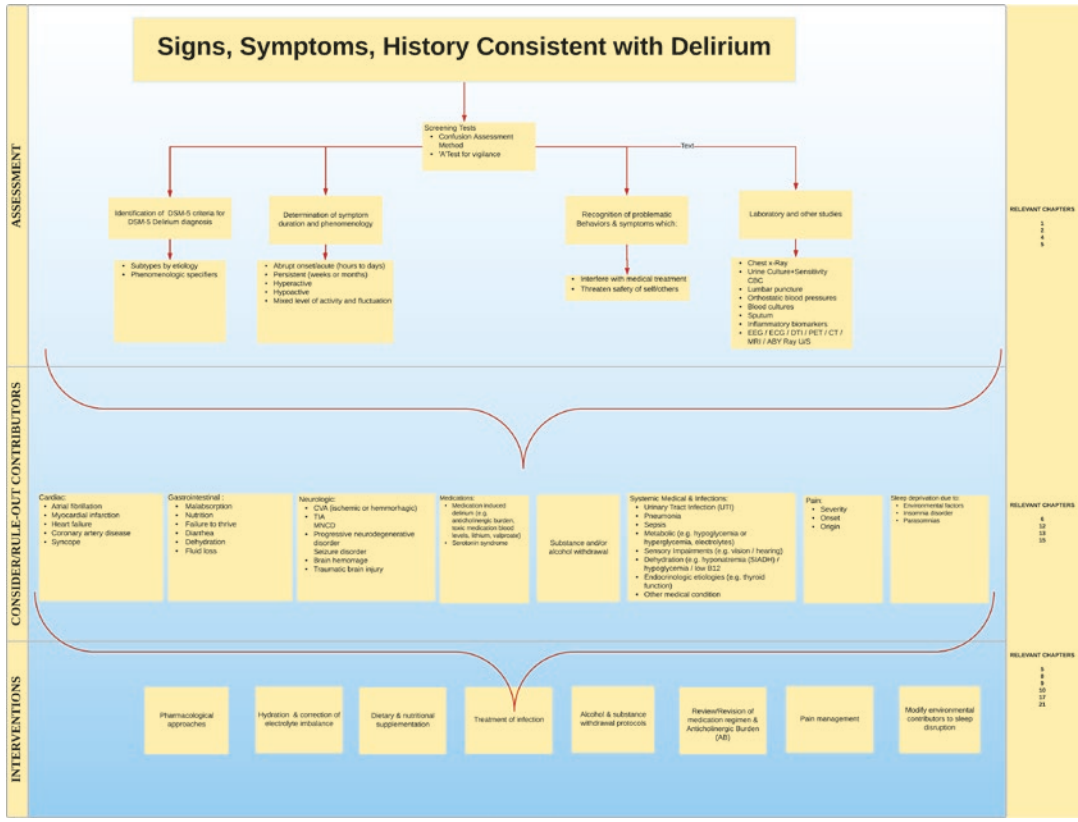

Fig. 12.1 Delirium work-up, identification, risk factors, contributors, and treatment

to reserve" ratio results in delirium. Future research may lead to a better understanding of normal brain functioning, its resilience to external factors, and the factors that contribute to delirium. For example, a transtheoretical model of *delirium disorder* has been proposed to help conceptualize the interacting and discrete neurophysiologic processes which underlie delirium, thereby encouraging novel clinical interventions [10].

While common, delirium is never "normal." Its pathological status is substantiated by its association with increased morbidity and mortality, functional and cognitive decline, increased rates of MNCD, institutionalization, increased length of stay (LOS) and health-care costs, post-traumatic stress disorder, and caregiver burden. According to a 2010 meta-analysis, the hazard ratio of increased risk of death at 22 months was approximately 2 [11]. The same meta-analysis suggested a 2.4 times higher rate of institutionalization and a 12 times higher rate of developing MNCD [11]. One-year mortality following delirium has been estimated to be between 35% and 40% [6, 7].

The onset of delirium is a medical emergency, not only because of the worsened functional outcomes, increased mortality, and increased morbidity, but also because a longer duration of delirium is associated with poorer outcomes. In a cohort of 225 older patients with coronary bypass or heart valve replacement surgery, those whose delirium lasted more than 3 days never returned to their

original baseline [12]. Similar findings among aged surgical patients were described in the short-term and in long-term relationship between delirium and a negative cognitive trajectory (SAGES cohort). In this cohort, 560 patients were followed for 36 months after surgery. Those who had delirium never recovered their baseline function [13].

A prospective cohort study of approximately 10,000 admissions of aging patients from the Fife region of Scotland suggested that cognitive spectrum disorders (CSD—delirium and MNCD), have a 1-year mortality of 40%, compared to a 1-year mortality of 26% for non-CSDs. This finding highlights the impact of brain functioning on survival, regardless of the cause of impairment. Health-care protocols and treatment guidelines should identify and develop discrete care pathways for geriatric patients with CSD as potential medical emergencies [14]. Clinicians who treat geriatric patients should be vigilant for delirium, regardless of the setting.

12.2 Evaluation

Two important steps are needed in the identification of delirium: (1) cognitive assessment to determine that the patient does in fact have cognitive impairment (the specific cognitive tests and screens for delirium will be covered in more depth later in this chapter); and (2) history from an informed observer regarding the patient's baseline, especially a determination of an acute deterioration from baseline. These two elements are important because delirium is defined as an acute onset of a confusional state, characterized by a loss of function, fluctuating consciousness, together with disturbances in attention and disorganization. The presentation can be extremely variable from patient to patient but typically is associated with other cognitive deficits; changes in arousal; perceptual disturbances, including hallucinations, delusions, and paranoia; and an altered sleep-wake cycle. Disturbances in emotion show up as labile mood, depressed mood, irritability, and/or fear.

12.3 Diagnosis

Delirium is not a specific unitary clinical entity due to a discrete cause, but a clinical diagnosis based upon a collection of symptoms. The International Statistical Classification of Diseases and Related Health Problems (ICD) is a medical classification system developed by the World Health Organization. The latest iteration, the ICD-10, which can be found in the publicly accessible "Green book," lists the following diagnostic criteria (Table 12.1).

Table 12.1 Delirium, not induced by alcohol or other psychoactive substances

A	Clouding of consciousness, i.e., reduced clarity of awareness of the environment, with reduced ability to focus, sustain, or shift attention
B	Disturbance of cognition, manifested by both:
	Impairment of immediate recall and recent memory, with relatively intact remote memory
	Disorientation in time, place, or person
C	At least one of the following psychomotor disturbances:
	Rapid, unpredictable shifts from hypo-activity to hyper-activity
	Decreased reaction time
	Increased or decreased flow of speech
	Enhanced startle reaction
D	Disturbance of sleep or the sleep-wake cycle, manifest by at least one of the following:
	Insomnia, which in severe cases may involve total sleep loss, with or without daytime drowsiness, or reversal of the sleep-wake cycle
	Nocturnal worsening of symptoms
	Disturbing dreams and nightmares which may continue as hallucinations or illusions after awakening
E	Rapid onset and fluctuations of the symptoms over the course of the day
F	Objective evidence from history, physical, and neurological examination or laboratory tests, of an underlying cerebral or systemic disease (other than psychoactive substance-related), that can be presumed to be responsible for the clinical manifestations in A–D

12.4 Vignette 1

A 68-year-old woman presented with abrupt onset (over the course of 24 hours) of confusion, gait instability, falls, and incontinence. She was sleeping much of the day and was awake at night. Her medication regimen included beta-blockers, diuretics, and meloxicam. She continued to have 1–2 shot glasses of brandy after supper. When she last came to the clinic for a review, she complained of anxiety and difficulty staying asleep. A locum family physician prescribed 2 mg of clonazepam to help her sleep better and relieve anxiety, which she took for 2 weeks.

At the end of 2 weeks, she was admitted to the inpatient psychiatric unit due to an alteration in mental status. The differential diagnosis included delirium, substance abuse (possible intoxication), depressive disorder, and/or anxiety disorder. Management included full medical work-up of delirium, supportive environment, reducing psychoactive medications, managing withdrawal, rehydration, optimizing sensory impairment, involvement of family, and instituting psychotropic medications to manage safety concerns or severe suffering.

12.5 Vignette 2

A 75-year-old man who lived in a retirement residence presented with a 2-day history of increasing confusion and paranoia. Two months earlier he had moved into the retirement community after the death of his wife 1 year before. He had difficulty managing his daily needs at home and fell at least three times. Over the 2 days prior to admission, he was not sleeping at night and he had periods of some lucidity, punctuated by episodes of confusion and difficulty speaking. He was being treated with anticoagulants for atrial fibrillation. On admission to a geriatric psychiatry unit, he was found to have urinary retention.

12.5.1 Differential Diagnosis

Delirium, major neurocognitive disorder, depressive disorder, urinary tract infection, normal pressure hydrocephalus, head injury, subdural hematoma (SDH), transient ischemic attacks (TIA), and cerebrovascular accident (CVA).

12.5.2 Laboratory Studies and Other Tests

CT/MRI of head, CBC, serum electrolytes, BUN, serum creatinine, urine culture and sensitivity, chest X-ray, and EEG.

12.5.3 Management

Supportive environment, reduction of psychoactive medications, rehydration, improvement of sensory impairment, involvement of family, psychotropic medications to manage safety concerns or suffering.

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) has five diagnostic criteria for delirium, listed under the acronym ACDC [15], shown in Table 12.2. The 5th criterion states that: "…the disturbances in Criteria A and C [attention and cognition] are not better explained by another preexisting, established, or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as coma."

DSM-5 includes several specifiers of delirium, including substance intoxication delirium,

Table 12.2 DSM-5 diagnostic criteria: acronym ACDC

A	ATTENTION: Disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness
C	COGNITIVE CHANGE: Change in cognition (e.g., memory deficit, disorientation, language disturbance, perceptual disturbance) that is not better accounted for by a preexisting, established, or evolving dementia
D	DEVELOPS ACUTELY: The disturbance develops over a short period (usually hours to days) and tends to fluctuate during the course of the day
C	CONSEQUENCE OF ANOTHER MEDICAL CONDITION: There is evidence from the history, physical examination, or laboratory findings that the disturbance is caused by a direct physiologic consequence of a general medical condition, an intoxicating substance, medication use, or more than one cause

substance withdrawal delirium, and medication-induced delirium. There is also a specifier regarding duration of diagnosis: acute vs. persistent, and a specifier of psychomotor activity level: hyperactive, hypoactive, and mixed level of activity. *Hypoactive* delirium is more common in geriatric patients, with symptoms of sedation, psychomotor retardation with slowed physical movements, and delayed verbal response. By contrast, *hyperactive* delirium is characterized by increased activity, agitation, and hypervigilance. Mixed picture states also exist with *both* hypoactive and hyperactive symptoms [4].

Features of delirium may change at different times in the same patient. Although one may display symptoms of a *hyperactive* delirium at one time, the underlying disease process can evolve. For example, during an episode of bacteremia, the patient may show agitation, but may later become hypoactive and less responsive when septic shock develops. The same can happen in acute withdrawal states, wherein a patient may be agitated, but eventually sequelae from hyperarousal, for example, a myocardial infarction, may result in a *hypoactive* delirium due to a combination of the effects of withdrawal together with cerebral hypo-perfusion.

12.6 Evaluation Tools

Formal cognitive screening tests, such as the Mini-Mental State Examination, Mini-Cog, or the Montreal Cognitive Assessment (MoCA), can help determine if cognitive impairment is present. Table 12.3 lists several general cognitive assessment tools available to support the finding of cognitive impairment—necessary criteria for delirium.

When time or limited patient participation does not permit full cognitive assessment, the determination of orientation and attention, such as naming of days of the week or months of the year, subtraction of serial sevens, or digit span (normally three or more) backward, can substitute for a more thorough cognitive evaluation [6, 7]. Several brief assessment tools can assist in the rapid screening, diagnosis, and assessment of motor symptoms of delirium. Brief delirium assessment tests are listed in Table 12.4 and are reviewed in detail by Grover and Kate [16].

Table 12.3 General cognitive assessment

	Mini Mental State Examination
	Clock Drawing Task
	Montreal Cognitive Assessment
	Digit Span Test
	Vigilance "A" Test
	Mental State Questionnaire
	Portable Mental Status

The Confusion Assessment Method (CAM) is the most widely used screening tool which has the core diagnostic criteria embedded. Worldwide, the CAM has been used in over 5000 original studies. It is translated into over 20 languages and comes in 2 forms, a short form CAM (4 item) and a long form CAM (10 item) [6, 7].

12.7 Differential Diagnosis

The differential diagnosis of a confusional state includes delirium, MNCD (in DSM-IV: dementia), depression, mania, psychosis, and catatonia, illustrated in Fig. 12.2.

One key to distinguishing between delirium and other causes of confusion is its *onset.* Delirium shows an acuity of onset, usually measured between hours and days. Major neurocognitive disorder (MNCD) has a more progressive course, generally, stepwise or gradual, over many months to several years. MNCD with Lewy

Table 12.4 Cognitive assessment instruments used to identify delirium

Screening	Diagnostic instruments	Severity measurement	Motor symptoms	Arousal scale
NEECHAM Confusion scale	Delirium Symptom Interview	Delirium Rating Scale	Delirium Motor Checklist, Delirium Motor Symptom Scale	Richmond Agitation and Sedation Scale
Nursing Delirium Screening Scale	Saskatoon Delirium Checklist	Delirium Rating Scale-Revised-98	Richmond Agitation and Sedation Scale	
Delirium Observation Screening Scale/ Delirium Observation Scale	Delirium Rating Scale-revised version	Confusion Assessment Method	Motoric items of Delirium Rating Scale, Delirium Rating Scale-Revised-98, Memorial Delirium Assessment Scale	
Intensive Care Delirium Screening Checklist	Memorial Delirium Assessment Scale	Confusion Assessment Method for Intensive Care Unit Assessment Tool		
Global Attentiveness Rating	Confusion Assessment Method	Delirium-O-Meter		
	CAM-ICU	Delirium Index		
	Clinical Assessment of Confusion – A and B	Memorial Delirium Assessment Scale		
		Confusional State Evaluation Scale		
		Delirium Assessment Scale, Delirium Severity Scale		

bodies is one MNCD that shares several features common in delirium, including fluctuations in attention and consciousness as well has visual hallucinations. The fluctuations seen with Lewy body MNCD (in DSM-IV: Lewy body dementia) occur over hours to days, compared with the fluctuations in delirium which tend to occur from minutes to hours (Chap. 6: MNCD with Behavioral Disturbance). Depressive disorder tends to have a more sub-acute onset with development over weeks to months. Primary psychotic disorders usually have a chronic nature with a natural history punctuated by episodic worsening. Catatonia can mimic some of the symptoms of delirium with periods of excitement fluctuating with periods of stupor or relative unresponsiveness. Catatonia tends to improve with lorazepam, whereas delirium is more likely to be *worsened* by lorazepam unless the delirium is caused by alcohol or benzodiazepine withdrawal (Chap. 15: Special Syndromes: NMS, Catatonia, Serotonin Syndrome).

Delirium, depression, and MNCD are often conceptualized as discrete entities. In fact, the presence of one increases the risk of the other two, and they are frequently overlapping clinical syndromes [3]. From validated predictive models, the relative risk of having depression increases the risk of delirium by 3.2 times on general internal medicine units and 1.2 times on cardiac surgery units. Similarly, having MNCD increases the risk of delirium by a relative risk of 2.3–4.7 on general medical units and 2.8 on non-cardiac surgery units [6, 7].

The connection of delirium with Alzheimer disease specifically is less clear. One finding from the SAGES cohort, which was free of MNCD at baseline, was that APOE E4 status was not a risk factor for the development of delirium [17]. The MRI volumetric changes which are

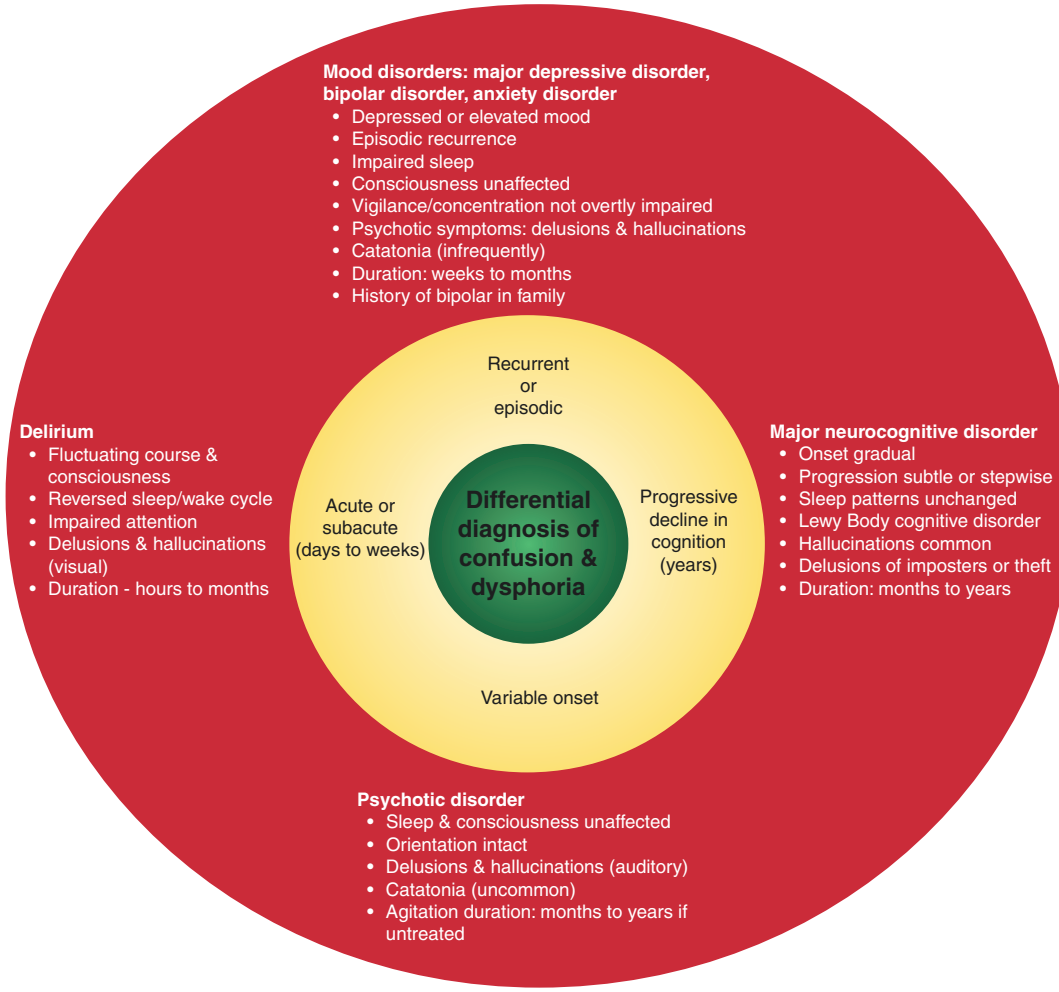

Fig. 12.2 Differential diagnosis of confused/dysphoric states in the context of delirium, major depressive disorder, bipolar disorder, psychotic disorder, and major neurocognitive disorder

typical of Alzheimer disease were not a risk factor for delirium in SAGES either [18]. At least in this cohort of 560 older patients followed prospectively for incident delirium following elective surgery, the risk factors for Alzheimer disease (APOE E4 status and hippocampal atrophy) did not confer increased risk for delirium, suggesting separate pathophysiologic pathways. Figure 12.2 illustrates the differential diagnosis between a confused, dysphoric state in the context of delirium, major depressive disorder, bipolar disorder, psychotic disorder, and MNCD.

12.8 Management

Once the diagnosis of delirium is made, the first step in the management includes identifying immediate safety concerns. Figure 12.3 illustrates the common causes of delirium.

Examples of safety concerns include the interruption of essential medical therapies (e.g., intubation, ventilation, IVs), or danger related to the patient or others from physical agitation or aggression. Unsteady gait and need for best rest immediately following surgery are other exam-

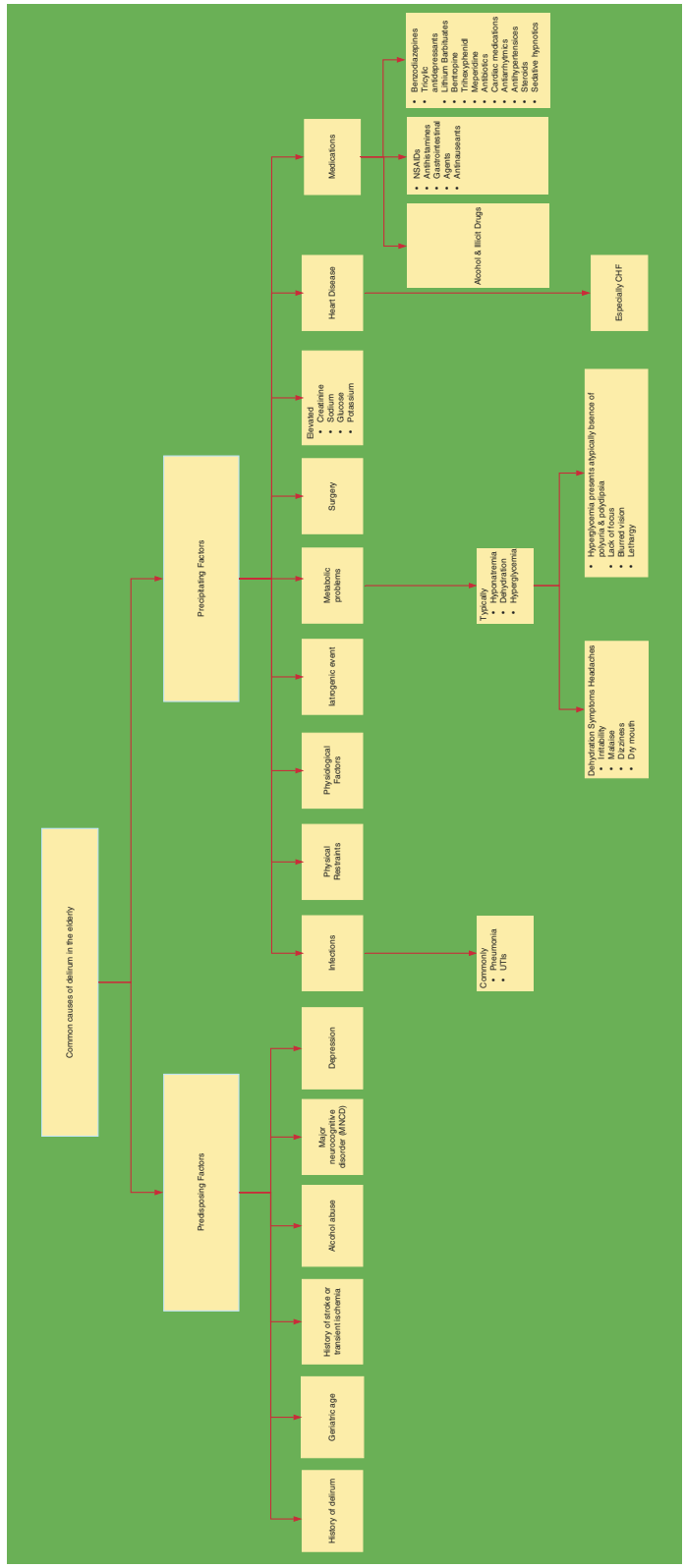

Fig. 12.3 Most common causes of delirium. (Adapted from Inouye et al. [6, 7]; Rockwood [19])

ples of potential safety concerns requiring immediate attention. If safety is imminently at risk, medications and restraints may be necessary, recognizing, of course, that the delirium may be worsened by these interventions (Chap. 13: Involuntary Treatment). Patients who are at high risk of falls may need chemical or physical restraint, as a last resort. Other interventions, including family involvement, are preferable, but may not be possible in urgent or emergency settings. The medications for this circumstance are discussed later, together with non-pharmacologic interventions.

Identification of cause(s) of the delirium is critical in trying to reverse the confusion and its sequelae. Although a single factor can precipitate an episode of delirium, usually delirium is multifactorial, particularly in geriatric patients [20]. The way these factors contribute in vulnerable patients will depend also on the current treatment setting.

A 2018 prospective study following over 2500 community-dwelling aging patients admitted consecutively to 3 acute care medical units in France was designed to identify those diagnosed with delirium ($n = 208$) to determine the contributing causes [21]. Ninety-one percent had cerebral imaging. Fifty-two percent of the patients were found to have an underlying MNCD, supporting the idea of decreased cognitive reserve contributing to the development of delirium.

Regarding specific causes, one study [21] found that infection was the most common cause, present in 49% (mostly pulmonary and urinary tract infections), followed by medications (31%), dehydration (27%), and electrolyte disturbances (19%). Neurological conditions were thought to be contributory in approximately one-eighth of the cases, including epilepsy, subdural hematoma, stroke (both ischemic and hemorrhagic), and head trauma. The main causes of delirium are shown in Fig. 12.3. Other acute conditions accounted for less than 10% of the etiologies of delirium, including heart failure, hypoglycemia, alcohol abuse, pulmonary embolism, fall-related fractures, cancer (disseminated), hyperthyroidism, sleep apnea, and temporal arteritis. Less than 10% of patients had urinary retention, pain, fecal impaction, and arrhythmias, although their role in delirium was thought to be less clear. The percentages of different causes from this study at diagnosis and at discharge are shown in Fig. 12.3, which represent the most prevalent causes of delirium.

Findings from a recent study by Magny et al. [21] support the multi-factorial causal model of delirium in older adults, and the need for identification of other causes of delirium during an admission. This study suggested that *early* diagnostic and management of precipitating factors, especially infection, electrolyte imbalance, and medications, is important, as a significant number are *missed at the initial assessment*; continuous search for *all* potential causes is warranted. Figure 12.4 summarizes precipitating factors by percentage at time of diagnosis, compared to a time of final assessment (Data derived from [21]).

Ken Rockwood, a geriatrician from Dalhousie University, suggests looking first to *medications* as a cause, presumably because this cause is most correctable [19]. Review of all current drugs is imperative (including over-the-counter and herbal medications), with a focus on new drugs and drug-drug interactions. Assessment of drugs is a high-yield procedure in delirium work-ups [6, 7]. Reducing psychoactive drugs should be a first step whenever possible. Various medications are associated with delirium; the most common categories include sedatives, opioids, benzodiazepines, anticholinergics, and calcium channel blockers. Figure 12.5 shows the odds ratios for developing delirium among several classes of medications.

The geriatric inpatient psychiatry setting, wherein patients are treated for depression, anxiety, or major neurocognitive disorders, adds an iatrogenic component to the onset of delirium. Close attention is prudent, especially if medication regimens have recently changed. The use of tricyclic antidepressants (e.g., amitriptyline, doxepin), and first-generation antipsychotics (FGAs) (e.g., chlorpromazine), should be noted, since these categories have greater anticholinergic properties than other psychotropics. Some relatively newer medications, in a dose-dependent

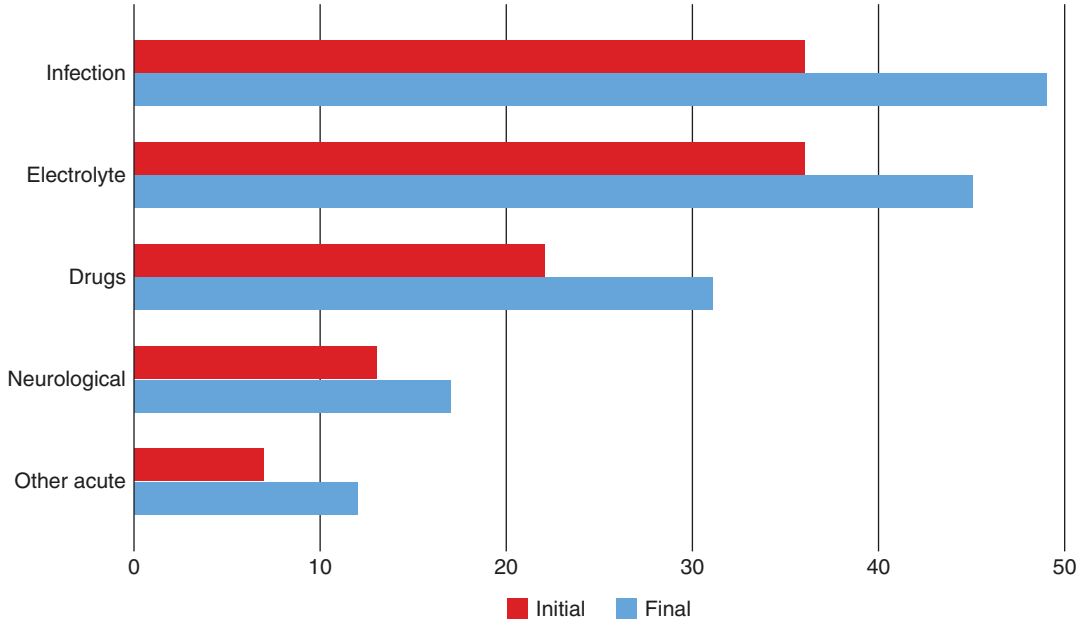

Fig. 12.4 Precipitating factors by percentage at time of diagnosis, compared to a time of final assessment. (Data derived from [21])

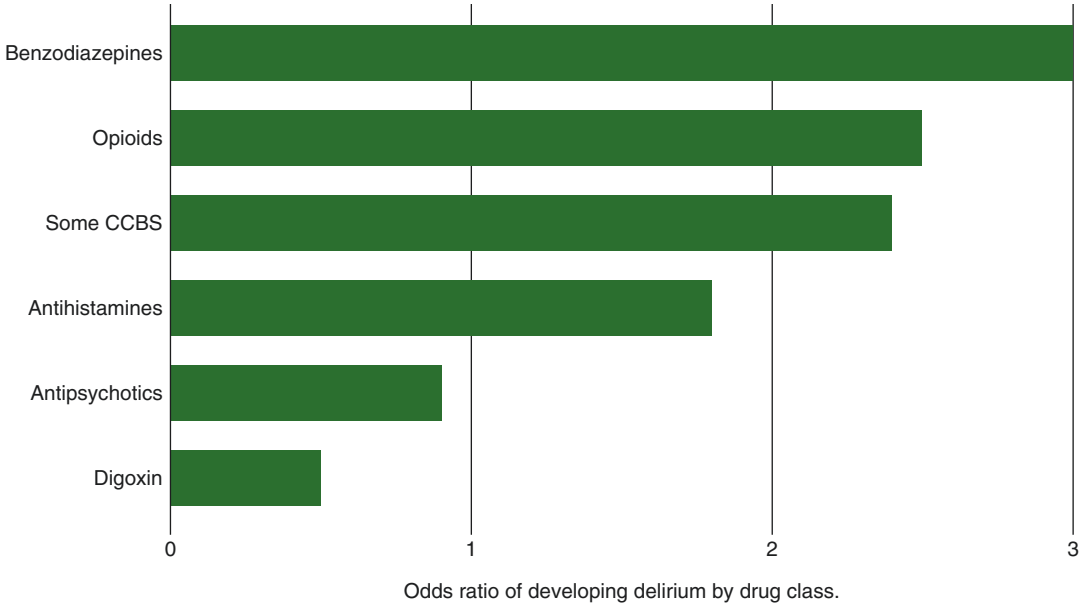

Fig. 12.5 Odds ratio of developing delirium by drug class. (Data from Clegg and Young [22])

fashion, are also associated with delirium, although the responsible mechanisms are less clear. Citalopram in the CitAD trial increased confusion as measured by the Mini-Mental State Examination (MMSE), when the dose was increased from 20 to 30 mg per day [23].

Several antidepressants, as well as some mood stabilizers and antibiotics, also cause hyponatre-

mia through induction of the syndrome of inappropriate anti-diuretic hormone (SIADH). Management may involve medication adjustment/discontinuation, in addition to fluid restriction and hypertonic saline, although the rate of correction needs to be carefully considered [24].

Serotonin syndrome (excessive serotonin accumulation), which is usually the result of medication interactions, has a varying degree of severity and is characterized by confusion, headache, agitation, hypomania, hallucinations, coma, autonomic effects (hyperthermia, sweating, tachycardia, nausea, diarrhea), and somatic effects (myoclonus, hyperreflexia, clonus, tremor). The Hunter Criteria outline this syndrome [25]. Management consists primarily of discontinuation of the offending agents, combined with adequate support, including intravenous fluids, and close observation [26]. (Chap. 15: Special Conditions).

Neuroleptic malignant syndrome (NMS), seen with antipsychotic medications, is also associated with cognitive changes [27]. The classic symptoms include mental status changes, fever, muscle rigidity and autonomic instability. While uncommon, when fever and mental status changes occur it remains an important and life-threatening component of the differential diagnosis. Elevated creatine phosphokinase (CPK) levels are helpful in making this diagnosis and can be done serially to monitor evolution. Treatment involves discontinuing the offending drug and aggressive supportive care and monitoring (Chap. 15: Special Conditions).

Mood stabilizers, including lithium, valproic acid, and carbamazepine, may also contribute to delirium, an effect which is also dose dependent.

Second-generation antipsychotic (SGA) quetiapine has been shown to worsen confusion in patients with MNCD at doses between 25 and 50 mg BID [28]. Clozapine, although used in lower doses in geriatrics for treatment-resistant schizophrenia, Parkinson disease psychosis, and Lewy body MNCD, can also cause confusion and sedation, likely due to its anticholinergic effects resulting from muscarinic receptor antagonism. Consultation with a pharmacist can be invaluable. Figure 12.5 shows the odds ratios for developing delirium among several classes of medication [22].

Knowledge of alcohol and sedative drug history can prompt laboratory evaluation to determine if levels of these drugs are still in the patient's system, and this can clarify if withdrawal states, like delirium tremens, are possible. From validated predictive models, a history of alcohol abuse increases the risk of delirium by a factor of 5.7 [6, 7]. Chronic users of alcohol may develop hallucinations within 12–24 h of abstinence, which may be mistaken for delirium. These symptoms usually resolve within 24–48 h. Hallucinations are usually visual; however, auditory and tactile hallucinations are also seen. In contrast to delirium tremens, alcoholic hallucinosis is not associated with significant global cognitive impairment and they may or may not have withdrawal symptoms as their vitals are often normal.

Approximately 5% of patients who experience withdrawal from alcohol suffer from delirium tremens and virtually all patients with delirium tremens experience symptoms of alcohol withdrawal preceding the confusion [29]. It is recommended that management of alcohol withdrawal delirium be done in an inpatient setting, preferably an ICU. Medical conditions need to be worked up and supportive measures are recommended as the primary symptoms of withdrawal are treated by lorazepam and diazepam, usually intravenously, as per established recommended protocols [29] with careful monitoring (Chap. 10: Alcohol and Substance use Disorders in the Geriatric Psychiatry Inpatient: Acute Treatment, Detoxification, Withdrawal).

Chronic alcohol abuse is associated with thiamine (vitamin B1) deficiencies which can lead to alcohol MNCD, amnestic-confabulatory type (DSM 5), previously called Wernicke-Korsakoff Syndrome (WKS). WKS is a combination historically of two disorders, Wernicke's Encephelopathy and Alcoholic Korsakoff syndrome. Wernicke's Encephalopathy which is characterized by confusion, ataxic gait, nystagmus, and ocular gaze palsies can be a life threatening emergency with lasting neurological and ophthalmological complications. The treatment is acute thiamine replacement. Although the amnesia of Korsakoff syndrome is often preceded by an episode of Wernicke Encephalopathy it can

Table 12.5 Investigations to work-up delirium [30]

Type of study	Test	Investigate/rule-out
Lab	CBC	Infection or anemia
	Electrolytes	Hypo or hypernatremia, dehydration
	Glucose	Hypoglycemia, diabetic ketoacidosis, and hyperosmolar nonketotic states
	Renal function	Renal failure and dehydration
	Liver function	Liver failure or hepatitis, hepatic biliary obstruction, albumin and ammonia, and transaminases
	Vitamin B12, thiamine	Nutritional deficiency states
	Levels of therapeutic drugs	Toxic serum levels of: lithium, valproic acid, carbamazepine, clozapine, tricyclic antidepressants
	Tests for specific bacterial or viral etiologies, VDRL for syphilis, HIV	Specific virus infections: Selected based on clues from history or physical
	Other	CK looking for neuroleptic malignant syndrome) (NMS) and tacrolimus for transplant patients
Microbiology	Urine analysis and culture	Urinary tract infection
	Blood cultures if temperature in excess of 38.5	Bacteremia, sepsis
	Sputum	Pneumonia, in patients with productive cough
	Lumbar puncture	Encephalitis – or assessment of fever with headache and meningeal signs or suspicion of encephalitis
Drug screen	Urine or blood tests	Acute alcohol intoxication: alcohol level, illicit drug use, excess levels of therapeutic drugs (like acetaminophen)
Imaging	Chest X-ray	Pneumonia, heart failure
	CT head	Acute change in arousal or mental status, suggesting hemorrhage, stroke, or space occupying lesion
	MRI head	Stroke, hemorrhage, and structural lesions. Has better soft tissue definition and can identify smaller strokes
Cardiac	ECG	Evidence of ischemia, arrhythmia, QTc prolongation
	Echocardiography	Congestive heart failure, if suspected, to evaluate cardiac output and function
Other		Occult seizures, differentiating psychiatric disorders from delirium
		Slowing of the posterior dominant rhythm and increased generalized slow-wave activity are observed on electroencephalogram (EEG) recordings which is typical of delirium
		Delirium – resulting from alcohol/sedative withdrawal, increased EEG fast-wave activity occurs
		Hepatic encephalopathy, look for diffuse EEG slowing
		Look for triphasic waves in toxicity or metabolic derangement, continuous discharges in nonconvulsive status epilepticus, and localized delta activity in focal lesions
Under investigation	Calcium-binding protein S-100B	Sepsis: levels are associated with mortality and delirium in sepsis
	Neuron-specific enolase	

also develop in individuals who have not had a prior episode of Wernicke's encephalopathy. Confabulation is often associated with the amnesia of Korsakoff syndrome and is a characteristic symptom of WKS. Table 12.5 summarizes the necessary work-up to assess delirium.

12.8.1 Non-Pharmacologic Interventions

Multi-component non-pharmacologic interventions which are delivered by an interdisciplinary team are ideal (Chap. 18:

Table 12.6 Early non-pharmacological recommendations for at-risk delirium patients

Early mobilization and promotion of walking
Orientation to surroundings
Enhancing sleep hygiene
Providing adequate oxygenation, fluids, and nutrition
Removing lines if possible reduces iatrogenic complications of invasive therapies, like infection
Avoid physical restraints
Education programs

Table 12.7 The six items of the HELP program to minimize delirium

Reducing cognitive impairment
Reducing sleep deprivation
Addressing vision impairment
Addressing hearing impairment
Correcting dehydration
Mobilization

Table 12.8 Delirium non-pharmacological interventions

Intervention	Examples of strategies and suggestions
Family involvement	Allow family members to visit frequently and often
Remove lines if possible	Includes IV and Foley catheters
Reorient	Clocks and calendars in the room
Correct day-night reversal	Leave lights on during the day
Nutrition	Ensure adequate oral intake
Decrease environmental stimuli	Try to reduce noise, evaluate need for noisy monitors and alarm bells
Mobility	Mobilize out of bed early if possible
Review medication list	Reduce or stop medications associated with delirium risk unless clinically indicated
Correct sensory impairment	Glasses and hearing aids should be brought in and be available at the bedside

Psychotherapies and Non-pharmacologic Interventions). Table 12.6 summarizes some early non-pharmacological interventions for patients at risk of delirium.

To help with sleep, avoid nursing or medical procedures during sleeping hours; if possible, schedule medication delivery to avoid disturbing sleep and reduce noise to a minimum during sleep periods (Chap. 9: Sleep in Geriatric Psychiatry Inpatients). Avoid physical restraints if possible, as evidence suggests that this worsens agitation and symptoms of delirium. Educational programs for health-care professionals are recommended and need to be ongoing. Assess for pain and discomfort (e.g., urinary retention, constipation, thirst) [31].

The Hospital Elder Life Program (HELP) has demonstrated efficacy and cost-effectiveness across different countries. There is evidence that HELP can assist in the prevention of delirium [32], lead to a reduction in cognitive and functional decline [33], result in decreased hospital length of stay and reduce rates of institutionalization [34]. There is also evidence that falls can be reduced, and as well as the need for 1:1 sitters [34]. Table 12.7 represents the six items that the HELP program focuses on. Examples of more non-pharmacological interventions are provided in Table 12.8.

12.8.2 Pharmacologic Interventions for Delirium

The first prospective randomized, double-blind (but not placebo-controlled) study that demonstrated efficacy in the management of agitation in delirium was described in 1996 [35]: 244 patients were hospitalized for AIDS and 30 patients were enrolled in a study comparing haloperidol, lorazepam, and chlorpromazine. The study did demonstrate a reduction in severity of delirium as measured by the Delirium Rating Scale and an improvement in cognition as measured by the mini-mental status exam. However, the lorazepam arm had to be stopped because of concerns of falls and worsening confusion. Since then several studies have looked at the use of antipsychotics to manage delirium. A systematic review and meta-analysis of 15 randomized controlled trials suggested that antipsychotics are better than placebo and that second-generation antipsychotics (SGAs) may be better than first-generation agents (FGAs).

A recent study compared olanzapine, haloperidol, risperidone, and aripiprazole used to treat delirium in a palliative care setting. Olanzapine had significantly more adverse side effects, perhaps owing to its relatively higher anticholinergic potential [36]. The daily mean doses of the four medications were haloperidol 5.5 mg, risperidone 1.3 mg, olanzapine 7.1 mg, and aripiprazole 18.3 mg. In the ICU setting, a 2018 study of patients with acute respiratory failure or shock, along with hyperactive or hypoactive delirium, found that haloperidol or ziprasidone, compared with placebo, did not significantly alter the duration of delirium [37].

Another meta-analysis of 19 randomized controlled studies found that the evidence was less clear that antipsychotics helped; however, the meta-analysis included seven studies that were prevention studies rather than pure treatment studies [6, 7].

The main limitations of the current studies of delirium are due to the following:

- Mixing treatment with prevention
- Different settings (ICU, palliative care, general medicine, post-surgery)
- Single site, non-blinded for many studies
- Lack of placebo control
- High dropout rates
- Different measures
- Non-uniform dosing

In sum, medication treatment may reduce agitation, but may not diminish the duration of delirium, and may prolong delirium and cognitive decline. The conclusion reached by several systematic reviews, meta-analyses and guideline panels, is that drug treatment for prevention or routine management at this time is not recommended [31, 38, 39]. Medications should be reserved for patients with severe agitation which will cause interruption of essential medical therapies (e.g., intubation), or pose a safety hazard to patient or staff members. Table 12.9 lists recommendations among the antipsychotic medication categories.

If SGAs are used, a similar approach should be taken: low doses with increased frequency, tapering the dose within a few days. One limitation is that SGAs are mostly oral medications with the exception of olanzapine, which is available as a rapidly acting intramuscular injection. For recommended doses, from existing studies, please refer to Table 12.9 [36].

There are many studies suggesting the use of adjunctive pharmacologic agents in intensive care settings; these studies are often retrospective and lack a placebo control. Consultation-liaison (CL) psychiatrists may be involved in their management both there and on medical-surgical units, and many have clinical experience using different agents.

As mentioned above, a proposed transtheoretical model of *delirium disorder* may encourage recognition of discrete neurophysiologic processes which underlie delirium, thereby encouraging novel clinical interventions [10]. For example, CL psychiatrists may be able to recommend using lactulose to treat hyperammonia levels during hepatic failure when patients are admitted to internal medicine. Medications such as valproic acid, ketamine, lorazepam, clonidine, dexmedetomidine, and propofol are currently being studied in ICU settings [40] to manage delirium; however, each of these medications may also cause delirium. Caution and close mon-

Table 12.9 Medications recommended for treatment of delirium [36]

First-generation antipsychotics (FGA)	
Haloperidol	1. Haloperidol 0.25–0.5 mg po or IM (IV short acting, risk of torsades). Range < 5 mg per day
	2. Repeat dose q30 min until patient manageable (max haloperidol dose 3–5 mg over 24 h)
	3. Maintenance: 50% loading dose in divided doses over next 24 h
	4. Taper dose over next few days
Second-generation antipsychotics (SGA)	
Risperidone	1. Risperidone total daily dose < 4 mg per day (0.25 mg increments)
Olanzapine	2. Olanzapine < 7.5 mg per day (1.25 mg increments)
Aripiprazole	3. Aripiprazole < 15 mg per day (1 mg increments)
Quetiapine	4. Quetiapine < 50 mg per day (6.25 mg increments)

itoring are warranted, in order to prevent a reduction of agitation at the cost of prolonging a delirium.

One of the challenges in treating delirium is seeking consent for treatments where decisional capacity may be questionable and fluctuates over time. The fluctuations may mean that a patient is capable one day and not the next. Establishing a plan of treatment when a person is well, based on best interests and prior wishes when capable, is an important principle in guiding decision-making. An alternative/surrogate decision-maker for when the patient becomes incapable, is also a priority (Chap. 5: Legal Aspects).

Although delirium is often considered a transient condition, chronic or persistent delirium does occur; estimates of its prevalence in geriatric patients approach 44% on discharge from hospital [41]. It is important to continue the search for all contributing factors, given the wide acceptance that delirium is multifactorial; residual cognitive deficits *without the fluctuations* may represent a new cognitive baseline for a given patient. A significant proportion of patients with delirium will develop a major neurocognitive disorder over time [41].

12.9 Summary

Delirium is serious and life threatening, but often under-recognized. It is preventable to some extent and efforts to manage this condition make a real difference in outcome. Optimal management includes an interdisciplinary approach that is both multi-component and comprehensive. Delirium is now a well-recognized indicator of overall health-care quality across various settings. Owing to the fact delirium is multifactorial, and linked to many other common syndromes associated with aging (such as falls, limited mobility, pressure ulcers, functional decline, and incontinence), focusing on delirium in general provides a practical and effective strategy to improve outcomes, decrease costs, and raise the quality of health care.

Delirium is a prevalent and life-threatening challenge for geriatric psychiatry that, if identified and addressed early, can be responsive to appropriate treatment. In sum, the approach to delirium should focus on identification of its etiologies and provision of interventions that enhance recovery, maximize functional status, and improve clinical outcomes.

Take-Away

- Review current regimen for medications which can contribute to delirium.
- Recognize delirium as a sixth vital sign and correct its precipitating factors.
- Look for delirium thoroughly with interdisciplinary screening.
- Address work-up as a team, systematically, in a proactive fashion (e.g., HELP).
- Search for multi-factorial causes.
- Mobilize immediately, early in its course.
- Search past history for previous episodes of delirium, etiologies, and treatments.
- Use non-pharmacologic interventions to manage sleep, anxiety, and agitation.
- Aim for the best outcome.
- Ensure patients have their glasses, hearing aids, and dentures.
- Involve patients/families: communicate regularly with all, and apprise them of risks.
- Consider medications as a *cause* of delirium, rather than routinely as a *solution.*
  - Review all medication regimens, both current and recent, including agents *not* formally or currently listed.
  - Reserve medication interventions for patients whose delirium causes severe agitation, risks an interruption of essential medical treatment (e.g., intubation), and may result in self-injury or severe psychotic symptoms (e.g., hallucinations, delusions).

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13 Involuntary Interventions: Medications, Forced Feeding, Restraints, and Prevention of Wandering

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13.1 Introduction

Geriatric patients who are deteriorating due to severe psychiatric symptoms may require involuntary treatment measures, including medications and other interventions, to ensure safety. This need becomes pivotal when the risk of significant harm to oneself or others is imminent, and when the patient has limited insight. In geriatric populations, symptoms requiring the use of involuntary treatment can result from new or preexisting psychiatric condition(s), medical condition(s), or a combination of both. The least restrictive and least intrusive interventions are to be utilized first. Throughout the course of intervention, constant and careful evaluation must be given to balance a patient's rights, safety, and the benefits/risks of treatment, versus no treatment. Respect for the preferences of the patient should be given where possible and safe (Chap. 5: Legal Aspects).

Treatment options and interventions are based on the patient's primary presenting symptomatology and harmful behavior, such as refusal of food/drink, severe depression, suicidality, psychosis, catatonia, aggression, wandering, and homicidality. In addition to medications, electroconvulsive treatment (ECT) is a highly effective treatment, and its use, even though without the informed consent of the patient, can be lifesaving. An evaluation of the risks and benefits of this intervention is needed on a case-by-case basis, as ECT is not suitable for all (Chap. 17: Neuromodulation Interventions). Figure 13.1 provides an overview of decisions revolving around involuntary treatment of the geriatric psychiatry inpatient.

13.2 Vignettes

Vignette 1

A 78-year-old widowed man presented with depressed mood, poor sleep, poor concentration, and worsening memory for over 2 months. He lost 30 pounds in that time, remained in bed all day crying, and refused to eat or drink over the previous 2 weeks. He believed that he did not deserve food or water because he was responsible for all world suffering. When his children visited, he refused to leave the house and said, "…just let

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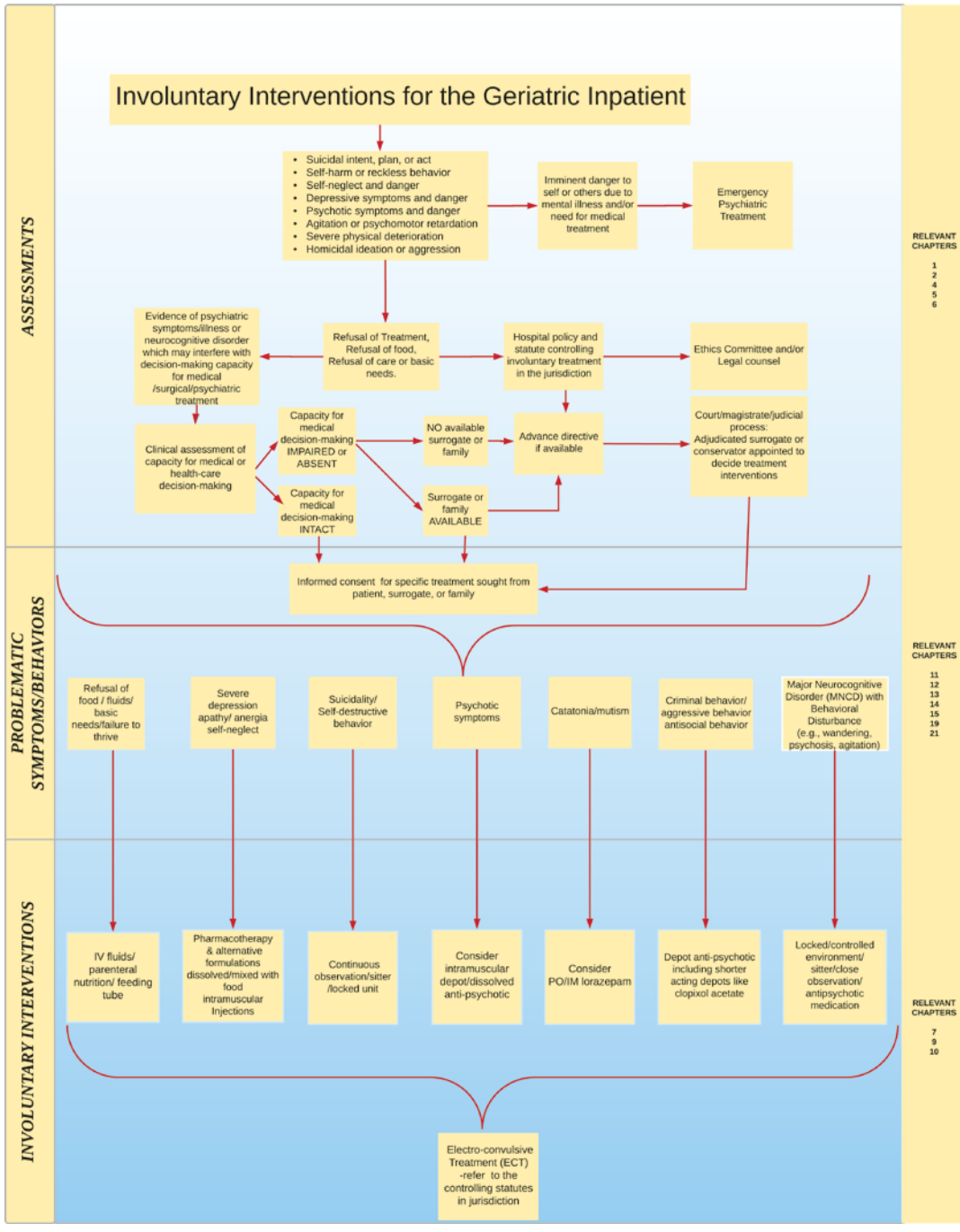

Fig. 13.1 Overview of involuntary treatment interventions

me die." On their last visit, he could no longer get out of bed due to weakness. He had no prior psychiatric history. On admission to the inpatient psychiatry, the differential diagnosis included delirium, major depressive disorder with psychotic features, bereavement, passive death wish, and weight loss due to a general medical condition (e.g., undiagnosed neoplasm). A systemic medical work-up revealed an elevated creatinine, elevated blood urea nitrogen (BUN), and hypernatremia and led to intervention with intravenous fluids and consideration of other nutritional interventions; e.g., nasogastric (NG) tube, PEG, and parenteral nutrition. He refused all treatments, but his family obtained a guardianship which permitted the surrogate to give informed consent for psychiatric treatment, including ECT. Medication treatment with sertraline was titrated to 150 mg orally once a day, but was ineffective. After five sessions of ECT, he began to eat.

Vignette 2

A 67-year-old female with a 35-year history of bipolar I disorder, who was living in a retirement residence, presented with multiple episodes of physical and verbal aggression over the previous 3 weeks. She slapped a co-resident across the face unprovoked and stated: "If I see her again, I am going to kill her." She was irritable and stayed awake all night talking to herself and engaging in projects. The retirement home staff described her as normally pleasant and friendly but had become unmanageable. The staff found a stockpile of antipsychotic medications in her room and the patient admitted to not having taken them for the previous month.

Her daughter was her legal guardian but lived out of town, unreachable either by telephone or email. The daughter was the only one who could provide consent for the treatment, and the patient would not passively assent to treatment. The patient was admitted to hospital involuntarily under the civil psychiatric commitment code due to the expressed homicidality. Her daughter was eventually contacted and gave full informed consent to lithium citrate at a dose of 300 mg twice daily, which was mixed into the patient's morning juice. The patient refused blood draws, but her daughter authorized the treatment of low-dose lithium *without* serum lithium levels. Over the next week, as the serum lithium apparently reached therapeutic range, the patient's irritability decreased and her cooperation with medication and laboratory improved.

13.3 Management Setting and Consent

Involuntary admission to an acute psychiatric unit and psychiatric treatment may be indicated when a patient's psychiatric symptoms threaten his/her safety, and there is no other way to ensure delivery of the needed treatment. Civil commitment, or consent from an adjudicated substitute decision-maker, guardian, or family, should be obtained. Familiarity with local statutes which control civil commitment and hospital policy regarding assessment of decisional capacity are paramount (Chap. 5: Legal Aspects).

An optimal acute inpatient psychiatric setting delivers comprehensive assessment, 24-hour care, close observation, monitoring of compliance, and assessment of response to treatment, without which a medical and psychiatric condition can result in morbidity and mortality. A secure or locked unit may be needed for dangerous behaviors such as wandering, aggression, suicidality, or homicidality. In cases where the primary etiology may be a medical illness, admission to an acute geriatric medicine unit with close psychiatric consultation-liaison may be a suitable model of care.

Involvement of family members and key social supports in the decision-making process may increase patient buy-in and quality of care. Involuntary treatment interventions are most often used when there is imminent risk to the patient and/or others, and timely decision-making is crucial. The risks and benefits of each treatment intervention, as well as consequences of no treatment, should be explained

clearly to the patient, family, and surrogates. It is helpful to document the patient's cognitive status and lack of insight/judgment due to medical and/or psychiatric illness [1]. Complex ethical dilemmas are common, and consultation with a medical center ethics committee prior to initiating treatment, as well as throughout the course of treatment, is valuable, especially when the treatment team encounters an ethical dilemma which causes uncertainty and reluctance to proceed.

13.4 The Role of Involuntary Treatment

Active and voluntary patient participation in treatment decisions is the cornerstone of a strong therapeutic alliance and effective patient-doctor relationship. This involvement should be obtained where possible and safe [2]. In some cases, this is not achievable, and involuntary treatment options are necessary, at least in the short term, to prevent irreversible deterioration or harm to self/others [1]. Clinical judgment, hopefully achieved as a consensus of the treatment team, is often the only guide to determine if and when involuntary treatment is warranted.

Clinicians should consider involuntary interventions when significant safety concerns or severe suffering is present [3]. Issues of safety include suicidal or homicidal ideation, significant self-harm, and marked physical deterioration, such as when an elderly patient stops eating or drinking [4]. Though a degree of subjectivity is involved, distress and suffering can be determined from careful patient interview, clinical observation, and collateral history. As soon as it is safe and possible, involuntary interventions should be replaced with less restrictive and voluntary measures.

13.5 Refusal of Oral Intake

The refusal of food and drink by a geriatric patient can be distressing for caregivers, family members, and the treatment team [5]. The refusal to eat and drink, combined with a decrease in physical reserves and altered regulation of appetite with normal aging, may result in serious consequences: malnutrition, sarcopenia, frailty, functional deterioration, morbidity, and/or mortality [6]. Ethical dilemmas emerge over the professional duty to do what is in the patient's best interest versus the patient's right to decide [7]. Obtaining an ethics consultation may provide some clarity and direction.

From a psychiatric perspective, common reasons for refusal to eat or drink include: major depressive disorder, suicidal ideation, loss of will to live, apathy, presence of active psychosis with hallucinations, and/or delusions (e.g., belief that food is poisoned, preventing oral intake). The history of a prior primary eating disorder should be considered and treated according to past successful treatment. In some cases, food refusal may be an attempt to hasten death, or control the outcome of intergenerational family conflict as a form of protest or hunger strike [8].

Food refusal in the presence of a major neurocognitive disorder (MNCD) presents the challenge of delineating unwillingness, from a lack of/reduced capacity or lack of/impaired ability to comply. With the progression of a MNCD, many patients may lose the ability to coordinate tongue, mastication, and related musculature to ensure effective swallowing and/or the cognitive ability to participate in meals. In advanced MNCD, patients may be unable to recognize food (agnosia), forget how to use utensils (apraxia), and experience confusion with the need to eat [9] (Chap. 19: Medical Nursing Care and Communication Barriers; Chap. 6: MNCD). Mechanical factors such as strictures and obstruction may play a role and require further systemic medical investigation of the dysphagia. The "pocketing" of food in the cheeks and mouth without swallowing is common in moderate to severe MNCD, and often is a result of forgetting the need to swallow [10]. As the MNCD progresses, the staff should pay special attention to a patient's mealtime performance and nutritional status [11]. Patients may ingest more and more food until choking occurs. Encouragement and reminders to chew and swallow may be helpful to

avoid choking or aspiration risk and increase intake (Chap. 7: Acute Medical Events).

Often caregivers and clinicians become frustrated due to a misunderstanding that any behavior which includes refusal is an intentional act of defiance. Recall that deficits in language (aphasia) and agnosia can contribute to difficulty with eating (Chap. 19: Medical Nursing Care and Communication Barriers). Psychoeducation must be provided for caregivers and the front-line staff, to ensure proper awareness of these neurological phenomena. Individuals with severe cognitive dysfunction require more physical assistance to eat, and are more responsive to one-on-one feeding assistance [9]. Alternate means of supplementing and maintaining nutrition may be required when oral intake is no longer safe or sufficient. Behavioral disturbances (noncognitive) symptoms associated with MNCD are often present in the geriatric psychiatry inpatient setting and have been found to negatively affect eating behavior, resulting in malnutrition, and contribute to perceived food refusal [12].

If the refusal of oral intake is persistent and the patient remains at risk of further deterioration, treatment with intravenous fluids and parenteral nutrition should be considered [13]. Subcutaneous fluid replacement may also be considered in cases of mild to moderate dehydration. Careful discussion with the substitute/surrogate decision-maker and/or family members is needed on the risk and benefits of intervention. Regular monitoring for dehydration, electrolyte abnormalities, weight changes, and tracking of oral intake and urinary output can provide clues to the urgency of initiating treatment [14].

Intravenous fluid administration remains the fastest route for fluid replacement, electrolyte correction, and medication administration due to direct entry into venous circulation [15]. Particularly, intravenous fluid replacement should be used for severe dehydration or malnutrition, where electrolyte disturbances are present and when rapid and large-volume fluids administration is required. In less acute circumstances, hypodermoclysis can be considered.

Subcutaneous fluid infusion, also known as hypodermoclysis, is a technique by which fluids are infused into the subcutaneous tissue, and enter into circulation via diffusion and perfusion [16]. Hypodermoclysis can be an effective route for administering fluids for hydration or nutrition, particularly in patients with mild to moderate dehydration or malnutrition, when oral intake is insufficient and when placement of an intravenous catheter is not possible or tolerated. A lower risk of infection and ease of set up and maintenance make this an attractive alternative to intravenous administration for a subset of patients; however, hypodermoclysis is not indicated for severe dehydration and malnutrition [17].

Enteral options such as the insertion of a nasogastric (NG) tube may allow fluid and nutrition needs to be met and medications to be administered while psychiatric treatment is administered and planning for further interventions can be made. The NG tube can be left in for a period of 1–6 weeks and replaced as needed [18]. If a longer duration is required, a percutaneous endoscopic gastrostomy (PEG) placed through the abdominal wall directly into the stomach can be utilized for a period of months to years [19, 20]. In patients with advanced MNCD, there is no current evidence to suggest any improvement in quality of life or long-term survival rates in those who have undergone PEG tube insertion [21]. Ethical consideration is required to achieve a balance among quality of life, provision of nutrition, and the degree of intervention, particularly in advanced MNCDs.

Enteral nutrition should be considered prior to the decision to commence parenteral nutrition. A comprehensive multidisciplinary nutritional assessment should be conducted prior to the initiation of parenteral nutrition, ideally by a nutrition support team, to determine the patient's nutritional requirements [20]. Parenteral nutrition is the intravenous administration of the nutrients, fluids, and electrolytes, and allows for the direct delivery of nutrition into the bloodstream, bypassing the gastrointestinal system [22]. This is achieved via the insertion of a peripherally inserted central catheter (PICC) which can be used for a medium duration of between 4 weeks and 6 months or a central

venous catheter (CVC) which is inserted for long-term vascular access [20].

For patients where artificial nutrition and fluid replacement is ineffective or not tolerated, or in cases where a primary psychiatric illness is the suspected etiology underlying food refusal and serious risk of deterioration, electroconvulsive treatment (ECT) should be considered [23]. ECT is not suitable for all illnesses wherein refusal to eat or drink is present. Potential side effects must be weighed against the potentially life-saving character of ECT, particularly in patients with severe psychiatric illness, such as depressive, bipolar, psychotic disorders, catatonia, and NMS (Chap. 16: Neuromodulation Interventions). Table 13.1 lists primary reasons for the use of involuntary treatments.

Table 13.1 Indications for involuntary interventions

Common rationale for involuntary treatment			
If there is significant deterioration in mental and physical health and/or			
Acute safety concerns (homicidality and/or suicidality), and/or			
Poor or limited insight and judgment			
	Presenting symptoms	**Reasons for treatment**	**Treatment options**
	**Severe depression**	Severe deterioration in social and daily functioning	Antidepressants
		Marked cognitive deterioration due to depression (pseudodementia)	Concurrent psychotherapy
		Psychomotor slowing and agitation	Medication formulations rapidly dissolved in food
		Frailty and physical decline	Medications concealed/crushed in food (consent granted by substitute-decision maker)
		Psychotic symptoms	ECT if clinical indication exists and if no contraindications are present
		Suicidality	
		Moderate to severe unipolar or bipolar	
	**Psychosis**	Presence of an active primary psychotic disorder or primary mood disorder with psychotic features (e.g., schizophrenia, bipolar disorder, depression with psychotic features)	Oral antipsychotic medication
		Acute delirium	Depot antipsychotic medication
		Brain tumor or brain metastasis	Long-acting injectable formulations (not with delirium or dementia)
		Major neurocognitive disorder	Consider ECT if clinical indication exists and if no contraindications are present
		Acute risk of harm to self or others	
		Severe deterioration in daily function	
	**Catatonia**	Severe mood disorder	Oral benzodiazepines
		Symptoms present as a result of medical illness	Intramuscular or intravenous benzodiazepines if oral route not tolerated
		Severity of symptoms of catatonia, including notable rigidity, staring, posturing, verbigeration, excessive motor activity, immobility, negativism	ECT if clinical indication exists and if no relative contraindications are present
		Medical instability as a result of catatonia	
		Neuroleptic malignant syndrome	

Table 13.1 (continued)

	**Refusal of food or drink**	Malnutrition	Intravenous fluids
		Sarcopenia	Hypodermoclysis
		Frailty	Temporary nasogastric tube
		Functional deterioration	PEG tube
		Morbidity	ECT if clinical indication exists and if no relative contraindications
		Risk of mortality	Parenteral nutrition
		Symptoms present are due to underlying psychiatric disorder, such as a psychotic disorder, depression with psychotic features, eating disorder	
	**Self-harm and suicide potential**	Active plan and intent (higher completion rates in the elderly, especially in males)	Close observation
		Unable to identify any reason for living	Certification and commitment to hospital
		Presence of poor impulse control or disinhibition	Locked unit if needed
		Past history of serious suicide attempt	ECT if clinical indication exists and if no relative contraindications
	**Aggression or violence**	Delirium	Admission to secure unit
		Symptom of major neurocognitive disorder	Chemical restraints
		History of traumatic brain injury	Physical restraints
		Deterioration of a psychiatric illness such as the presence of psychosis, mania, or depressive symptoms	Injectable intramuscular formulations for acute agitation and aggression
		Substance intoxication	Consider ECT if clinical indication exists and if no contraindications are present
		Adjustment disorder	
	**Homicidality**	Symptom of major neurocognitive disorder	Admission to secure unit
		History of traumatic brain injury	Certification and commitment to hospital
		Deterioration of a psychiatric illness such as the presence of psychosis, mania, or depressive symptoms	Chemical restraints
		Adjustment disorder	Physical restraints injectable intramuscular formulations
		History of previous violence and criminal behavior	Consider ECT if clinical indication exists and if no contraindications are present
	**Wandering**	Delirium	Constant observation
		Acute deterioration of a psychiatric illness	Locked unit
		Symptom of a major neurocognitive disorder	Civil commitment
		Significant patient distress and suffering as a result of lapping/pacing/random ambulation	Non-pharmacological interventions
		Interferes with needed care	Pharmacological interventions – antipsychotic
		Exit seeking behaviors	Wander bracelet or device
		Anxiety	
		Agitation	
		Rule out akathisias	

13.6 Severe Depression

Major depressive disorder (MDD) is a heterogeneous and sometimes debilitating disorder with poorer outcomes in geriatric patients compared to younger patients [24]. Significant social isolation, marked cognitive impairment, psychomotor slowing or agitation, somatic complaints, psychotic symptoms, and suicidality are often present. Depressive episodes can occur in the context of a recurrent depressive disorder or bipolar illness, or can present for the first time in late life. Major depressive disorder in the aging patient has also been associated with higher levels of medical morbidity, cognitive impairment, and mortality, both from suicide and systemic medical illness [25] (Chap. 8: Suicide).

Pharmacological treatments in geriatric depressive disorders are frequently limited by medical comorbidities and pharmacodynamic factors that decrease response rate and increase the risk of drug interactions. For severe depressive disorder, a combination of antidepressants and psychotherapy should be offered routinely if no contraindication to either treatment exists [26] (Chap. 18: Psychotherapies and Non-pharmacological Interventions). If routine oral medication is refused, consideration should be given to a trial of an antidepressant medication with a rapid dissolving formulation, which can increase compliance. Ethical considerations arise in patients presenting with suicidality, psychosis, and diminished capacity for decision making, as a result of the depressive disorder. After careful consideration, transparent discussion, and informed consent from the surrogate decision-maker, concealment of antidepressant medications in the patient's food or beverages, such as in applesauce or pudding, may be justified and effective.

If depressive symptoms suggest a high risk of rapid and imminent deterioration or the presence of significant health consequences, ECT can be an alternate or adjunctive treatment and should not be delayed. Consultations should be obtained to determine patient suitability for ECT, including an anaesthesia consult.

ECT remains the gold standard for the treatment of severe depression in geriatrics, even in cases where there is a need for involuntary treatment. The risks and benefits of ECT must be assessed for each individual and explained clearly to the patient and/or the substitute decision-maker. Studies suggest that in the aging adult population, ECT is equally or more effective and has a faster response and remission rate than the use of antidepressants in the treatment of severe, unipolar, geriatric depression [27]. Due to the severity of symptoms required to necessitate involuntary treatment, the faster response and remission rate results in earlier relief of suffering for the patient. There is evidence that the adverse cognitive effects of ECT experienced are usually transient and not typically severe [28]. ECT as an alternative to pharmacologic intervention for geriatric depression also has the potential to minimize systemic side effects and medication interactions (Chap. 17: Neuromodulation Interventions).

13.7 Suicidality

Suicide in the elderly is an underestimated and complex issue. Suicide completion rates are higher in aging adults compared to most other age groups [29]. The World Health Organization has estimated suicide rates among those aged 75 and above to be 50/100,000 for men and 16/100,000 for women [30] (Chap. 8: Suicidality in Geriatrics).

Management and/or interventions for suicidal potential require a detailed risk assessment especially if there is a history of psychiatric illness, such as a depressive disorder, bipolar and related disorder, schizophrenia spectrum and other psychotic disorder, history of serious suicidal ideation or actions, recent losses and stressful life events, functional impairment, and/or medical illness. Patients with a detailed suicide plan, active intent, those who are unable to identify any

reason for living, and those with poor impulse control are at the highest risk and require immediate psychiatric assessment and/or civil commitment to hospital. Patients with a substance-related and addictive disorder or schizophrenia spectrum and other psychotic disorder are also at risk and may require urgent psychiatric assessment and/or civil commitment to hospital.

Continuous observation with suicide precautions should be initiated, and modifiable underlying risk factors, such as the treatment of any underlying psychiatric illness, should be addressed. When involuntary treatment is required, a locked unit may reduce risk, especially with a commitment order to prevent the patient from leaving the hospital to act on their suicidal ideation. Careful assessment of the treatment environment helps ensure the environment is safe and risk is mitigated. Collateral history is invaluable, particularly in patients who are not forthcoming. Such information can assist with the decision as to whether the patient requires involuntary treatment.

Should suicidality persist despite biopsychosocial interventions, a trial of ECT may be warranted. Any escalation of risk should prompt increased observation and consideration for ECT. This is especially true if the underlying etiology is a primary psychiatric disorder wherein symptoms are severe and involuntary treatment is required [31]. ECT should also be considered in patients who are deemed high risk and whose suicidality is present predominately as a symptom of active depression [25].

When suicide risk is related primarily to personality disorder or substance use disorder, pharmacological interventions and ECT are minimally effective, and involuntary treatment may be limited to periods of active crisis stabilization, intoxication, and/or withdrawal [31]. Following stabilization and management of acute suicide risk and crisis, treatment of the underlying personality disorder with psychotherapeutic interventions, such as dialectical behavioral therapy, can be beneficial. Treatment of the substance use disorder is also critical.

13.8 Psychotic Symptomatology

Symptoms of psychosis can emerge as a result of a primary psychiatric disorder (e.g., schizophrenia, bipolar I disorder or major depressive disorder with psychotic features), or secondary to other etiologies (e.g., delirium, brain tumor, or major neurocognitive disorder). The content of psychotic symptoms and the patient's level of insight help the mental health provider assess the related risk and the appropriate intervention. Involuntary treatment may be needed to ensure the safety of the patient and/or others, particularly in the presence of persecutory and command hallucinations and/or bizarre delusions. Disorganization and confusion may also pose an imminent risk of harm or deterioration. A history of chronic psychiatric illness, past baseline functioning, and medication compliance can help guide current interventions. If the patient has required involuntary treatment to achieve remission in the past, involuntary treatment may be indicated again.

Routine depot (intramuscular) antipsychotic medication for chronic psychotic symptoms is appropriate for the geriatric patient, if the risk-benefit ratio supports its use. As in younger populations, geriatric patients with chronic mental illness may not be reliable in adhering to antipsychotic medication regimens when not closely supervised. The lack of compliance may lead to recurrent episodes of acute psychosis, detrimental behavior, and involuntary hospitalizations. When dangerous behavior due to psychotic symptoms outweighs the adverse effects, long-acting injectable antipsychotic medication formulations may be approved by surrogates to prevent decompensation, hospitalization, and serious harm. The inpatient psychiatric unit is an excellent setting in which to safely stabilize the geriatric patient on an equivalent dose of an oral antipsychotic agent and transition to depot medications.

Few studies of depot antipsychotics in geriatric patients have been conducted; to date research indicates that first-generation antipsychotic

(FGA) agents as depot injections are associated with positive outcomes in geriatric populations. Long-acting injectable formulations of second-generation antipsychotics (SGA) such as risperidone and aripiprazole have also been shown to have good effect and tolerability [32, 33]. In settings where involuntary treatment is required, long-acting injectable formulations provide an added advantage of decreasing the frequency of medication administration from daily to every 2–4 weeks, which can improve patient quality of life and distress, while ensuring consistency and compliance.

The American Psychiatric Association (APA) practice guidelines suggest that long-acting injectable antipsychotics should not be used in patients with MNCD unless administered for the treatment of a co-occurring chronic psychotic disorder [34].

The choice of antipsychotic medication and its dose should take into consideration previous history of antipsychotic use and effectiveness, active symptoms, current medications and interactions, medical history, and medical comorbidity. In general, in a geriatric patient, the starting dose of the chosen antipsychotic medication should be 25% of the usual adult dose. The total daily maintenance dose should be titrated to the minimal effective dose, typically 25–50% of the adult dose [32]. In aging patients with a history of chronic primary psychotic illness, such as schizophrenia, and who have been treated for multiple decades on antipsychotic medications, higher dosages of antipsychotic medications are often better tolerated than in other geriatric patients, and required for stabilization. Regular monitoring for metabolic side effects is recommended for SGAs, and regular EKG with QTc interval monitoring is recommended for both FGA and SGAs.

Geriatric patients initially treated with antipsychotic medications via involuntary treatment can often be managed on a voluntary basis following the improvement of psychotic symptoms. For those who continue to lack insight into their psychiatric illness and display poor medication compliance, a community treatment order by the court may mandate involuntary treatment in the community. The controlling statute in the jurisdiction should guide the process (Chap. 5: Legal Aspects).

Involuntary treatment with ECT should be considered for geriatric patients with psychotic symptoms if severe health consequences are imminent and rapid response is required. In elderly patients who are unable to tolerate medication side effects or who are at a high risk for drug-induced toxicity or interactions, ECT may be the safer treatment option. Treatment-refractory psychotic disorders have been shown across the lifespan to respond well to ECT. Patients requiring involuntary treatment, with a history of treatment refractory psychotic disorders, should be evaluated for ECT if no contraindications exist. To date, no randomized controlled data specifically evaluating the use of ECT in geriatric patients with primary psychotic disorders exists [35]. In patients with schizophrenia, ECT has shown greatest efficacy in the treatment of positive rather than negative symptoms of schizophrenia [35]. Involuntary treatment is more frequently required when positive symptoms of schizophrenia are present, as a higher degree of clinical distress and risk to the patient and/or others exist compared to the presence of predominately negative symptoms of schizophrenia. In geriatric inpatients with psychotic symptoms as a result of severe depression, ECT should be offered if no contraindications exist, or following a trial of a combination of antidepressant and antipsychotic medication therapy [26].

13.9 Catatonia

Catatonia is a behavioral syndrome that presents with a heterogeneous constellation of specific signs and symptoms [36]. Common presentations of catatonia include rigidity, staring, posturing, verbigeration, excessive motor activity, immobility, and negativism [37]. In aging adults, catatonia occurs most frequently in the context of a severe depressive disorder or systemic medical illness [38]. Catatonia is more prevalent in geriatric psychiatric patients than among younger patient cohorts. Failure to recognize and properly treat catatonia can result in a high risk of morbid-

ity and mortality (Chap. 15: Special Syndromes: Neuroleptic Malignant Syndrome, Catatonia, and Serotonin Syndrome).

A study of 106 patients admitted to an acute geriatric psychiatry unit found that catatonia was present in between 17.9% and 39.6% of geriatric psychiatric inpatients, depending on the diagnostic criteria used [38]. Catatonia was more frequent in patients with depressive disorder, manic episode, and delirium. Other studies suggest that catatonia is a common syndrome among patients with MNCD with a frequency of 42.8% in the inpatient setting [39].

Benzodiazepines are the first-line treatment for catatonia alongside treatment of the underlying cause if known or suspected [36] and lorazepam is the most common benzodiazepine used. Hospital and unit policy may often prohibit the use of IV access on inpatient geriatric psychiatry units, in which case other options can be considered. Intramuscular administration of lorazepam should be started if oral administration is not possible, until an IV line is established. The severity of the catatonic condition and the patient's inability or reluctance for oral intake, justifies the intramuscular formulation. While this treatment can be considered an involuntary intervention, the absence of full informed consent from the patient may simply reflect an inability to provide a meaningful answer or an assent to the treatment.

Lorazepam is usually initiated at a dose of 0.5–2 mg four times per day, and increased every 1–2 days, depending upon patient response, tolerability, and clinical urgency [37, 40]. A total daily dose of 8–24 mg has been found effective for most patients without significant sedation [40]. The use of benzodiazepines for catatonia has an estimated effectiveness of remission rate of 60–70% of cases [37]. For medication-refractory and clinically urgent patients, ECT should be considered [36].

Table 13.2 Lists medication formulations appropriate for the involuntary or otherwise uncooperative patients. Most catatonic patients present in a state of diminished capacity and thus, involuntary treatment is frequently required. Informed consent from the substitute decision-maker or surrogate should be sought, and in its absence, a consultation with the hospital ethics committee and/or legal consultation.

ECT is also a first-line intervention for catatonia in cases of severe illness, such as with malignant catatonia and severe catatonic excitement [35]; and it remains an effective treatment of catatonia, regardless of comorbidity or underlying diagnosis. Limited studies have been conducted specifically in geriatric patients.

13.10 Aggression

Aggression and assaultive behaviors in a geriatric inpatient setting constitute serious safety concerns, with the risk of escalation if not properly managed. Regardless of etiology, the aim is to use the least restrictive means possible, while ensuring the safety of all parties involved. Common causes for aggression and agitation include delirium, the presence of psychotic symptoms, and MNCD. The precipitant of aggressive behavior can differ significantly based on the patient's initial presentation and underlying illness (e.g., geriatric patients who are delirious may display random nontargeted episodes of aggression; in an acute psychotic disorder, paranoid delusions and command hallucinations can result in intentional and targeted displays of verbal or physical aggression) [4].

In patients with MNCD, aggression is common during periods of personal care and is often directed toward caregivers (Chap. 6: MNCD with Behavioral Disturbance). Aggression may also occur randomly when too much or too little environmental stimulation is present. In a study of 146 aggressive incidents involving 66 patients, Paschali et al. found that over half of the incidents involved patients with MNCD, and in 55% of the incidents, a link to the patient's medical condition could be made, whereas in 20% of incidents no precipitating factor could be identified [41]. Aggression was directed at nurses in 82% of the cases in this study, emphasizing the importance of ensuring adequate psychoeducation, training, and support for nursing staff. Interestingly, when looking at predictors of

Table 13.2 Formulations of psychotropic medications for involuntary use

Medication	Class	Geriatric dosing (maintenance dose range)	Mix/crush in food	Injectable	Other formulation
Antidepressants					
Buproprion SR	NDRI	150–300 mg/day	No	No	
Buproprion XL	NDRI	150–300 mg/day	No	No	
Citalopram	SSRI	20 mg/day	Yes	No	Oral liquid
Desvenlafaxine	SNRI	50 mg/day	No	No	
Duloxetine	SNRI	60–120 mg/day	No	No	
Escitalopram	SSRI	10 mg/day	Yes	No	Oral liquid, oral disintegrating tablet
Fluoxetine (daily capsule)	SSRI	20–80 mg/day	Yes	No	Oral liquid
Fluoxetine (weekly capsule)	SSRI	90 mg/week	No	No	
Mirtazapine	NaSSA	15–45 mg/day	Yes	No	Oral disintegrating tablet
Paroxetine	SSRI	10–40 mg/day	Yes	No	Oral liquid
Paroxetine CR	SSRI	10–50 mg/day	No	No	
Sertraline	SSRI	50–200 mg/day	Yes	No	Oral liquid
Trazodone	SARI	75–150 mg/day	Yes, poor taste	No	Oral liquid
Venlafaxine	SNRI	75–225 mg/day	Yes	No	
Venlafaxine XR	SNRI	75–225 mg/day	No	No	
Antipsychotics					
Aripriprazole	SGA	2–15 mg/day	Yes	Yes, long acting (immediate acting discontinued)	(Oral disintegrating tablet and oral liquid)
Clozapine	SGA	12.5 mg–50 mg/day (given in evening)	Yes	No	Oral disintegrating tablet, oral liquid
Haloperidol	FGA	0.25 mg–6 mg/day (divided doses)	Yes	**Yes,** immediate acting and long acting	Oral liquid
Loxapine	FGA	2.5 mg–100 mg/day	Unclear	**Yes,** immediate-acting (In Canada, not in USA)	Oral liquid, Aerosol powder
Lurasidone	SGA	20–60 mg/day	Yes	No	
Olanzapine	SGA	2.5 mg–15 mg/day	Yes	**Yes**, immediate acting and long acting	Oral disintegrating tablet, oral liquid
Paliperidone ER	SGA	3 mg–6 mg/day	No	Yes, long acting	
Quetiapine	SGA	12.5 mg–200 mg/day	Yes	No	
Quetiapine XR	SGA	12.5 mg–200 mg/day	No	No	
Risperidone	SGA	0.25 mg–2 mg/day	Yes	**Yes, long acting**	Oral disintegrating tablet, Oral liquid
Zuclopenthixol	FGA	10 mg–100 mg/day	No	**Yes**, immediate acting and long acting	

Note:
Not all formulations and medications are available in every jurisdiction/State
Off label use of antipsychotic medications listed is for the management of psychosis, agitation – major neurocognitive disorder with behavioral disturbance
An FDA-mandated black box warning exists: elderly patients treated with antipsychotics are at an increased risk of death compared to placebo
NaSSA noradrenaline serotonin-specific antidepressant, *NDRI* norepinephrine dopamine reuptake inhibitor, *SARI* serotonin antagonist and reuptake inhibitor, *SNRI* serotonin norepinephrine reuptake inhibitor, *SSRI* selective serotonin reuptake inhibitor, *FGA* first-generation antipsychotic medication, *SGA* second-generation antipsychotic medication

length of stay at a geriatric psychiatry hospital, geriatric patients who had a greater number of assaults were found to be more likely to have longer lengths of stay than those with fewer assaults [42]. This finding emphasizes the importance of developing strategies and effective interventions for aggression in patients at risk for assaultive behavior.

In a subset of geriatric patients, homicidality may be present. Studies suggest that patients at high risk for future violent behavior include individuals with a prior history of violent behavior, substance abuse, and noncompliance with psychiatric medications [43]. Similar to aggression, homicidal ideation may be present as a result of a psychiatric disorder, including psychosis, substance intoxication and withdrawal, manic episode, adjustment disorder, delirium, MNCD, or traumatic brain injury.

Treating any underlying psychiatric illness or medical condition is paramount to reducing aggression and risk. Admission or transfer to a secure unit with close observation should be considered, especially if a history of violence or aggression has been previously noted. Chemical and physical restraints may be required if behavioral approaches and redirection are ineffective. If rapid onset of action is required for stabilization, and patient cooperation and compliance is absent, injectable intramuscular formulations are recommended. Common agents include haloperidol, loxapine, and lorazepam (short-term), and in severe aggression, shorter acting agents such as zuclopenthixol acetate may be considered. Dosages should be adjusted downwards from adult dosing for geriatric populations.

Aggression is a common behavioral symptom of MNCD (Chap. 6: MNCD with Behavioral Disturbance) and is associated with greater severity and cognitive decline [44]. Patients displaying severe aggression as a result of a MNCD often require involuntary treatment to manage episodes of aggression and ensure the safety of caregivers. Ideally, a multidisciplinary inpatient team performs a comprehensive assessment of medical, psychological, and environmental factors. The inpatient unit provides an opportunity to monitor and observe the patient in a controlled environment where the patient is safe and triggering stimuli can be reduced. Assessment of patient capacity and thorough neuropsychiatric assessment may be needed to delineate cognitive deficits.

Patients with MNCD may display impaired moral judgment, and a decline in social interpersonal conduct [45]. Patients with frontal lobe deficits and damage often have personality changes and an increased impulsivity without concern for the consequences, which can result in aggressive, violent, and potentially criminal behaviors. Physical assaults may be a result of the patient's misperception of stimuli and the external environment. With progression of MNCD, increased combativeness, biting, and hitting behaviors are commonly witnessed during periods when personal care is provided by staff and caregivers [46]. Involuntary admission and treatment may be the only available setting option available, when family and caregivers are the targets for aggression from the patient, and where the patient can continue to receive care while aggressive behaviors are not yet controlled. Aggression is most often seen as a result and expression of frustration in patients with major neurocognitive disorders.

Interventions which can minimize external stimuli and reduce the complexity of day-to-day tasks for the geriatric patient may be effective in reducing risk and incidents of aggression (Chap. 19: Psychotherapies and Non-pharmacological Interventions). Aggression and increased risk of violence are also more frequent when psychosis and a history of alcohol use disorder are present [4, 47]. In cases of minimal cognitive reserve and behavioral and environmental modifications have had minimal success, the use of pharmacological agents such as an SGA and physical restraints may be necessary to ensure the safety of the patient and caregivers.

Emerging evidence suggests that ECT may play a valuable role in the treatment of aggression and agitation in patients with major neurocognitive disorders, particularly those with severe aggression. In addition, ECT provides an alternative treatment option where regular and frequent encounters to administer involuntary oral medications for aggression can be avoided. Clinically,

ECT remains minimally used to date for the treatment of aggression.

A study ($n = 23$) examined the safety and efficacy of ECT in the treatment for behavioral disturbances in MNCD, including agitation and aggression, and suggests that ECT may be a safe treatment option to reduce symptoms of agitation and aggression in patients with MNCD, whose symptoms have been refractory to medication management [48]. A systematic review conducted by Glass et al. identified a total of 216 patients who received ECT for aggression in the context of a major neurocognitive disorder and suggests promising results for decreasing symptoms of agitation associated with aggression [49]. Most recently, van den Berg et al. (2018) [50] reviewed 17 articles which studied ECT for agitation and aggression in dementia and found that 88% of the 122 patients showed clinically significant improvement. One prospective cohort study, 1 case-control study, and 15 retrospective chart reviews, case series, or case reports were identified [50]. Both authors conclude that preliminary results are promising, but higher-quality studies are required.

To date, ECT has generally been considered when all behavioral and pharmacological interventions have been exhausted, and a return to the community remains unsafe for the patient and/or others in the patient's environment. Case studies suggest that maintenance ECT, alternating with acute treatment course may be effective in managing severe aggression in patients with MNCD, particularly in individuals where pharmacologic interventions are ineffective or not tolerated [51].

The criminal justice system may need to be involved with individuals who display severe aggression, violence, homicidality, and other criminal behavior (Chap. 5: Legal Aspects). The study of criminality in aging patients is limited to date. Existing studies indicate that up to 80% of aging offenders had a history of prior psychiatric hospitalization [52]. Homicides committed by the geriatric population remain rare. Most criminal offences in aging adults are minor in severity (e.g., driving offenses, shoplifting) [45]. In the United States, with an aging population, the number of older adults who have been arrested or incarcerated has increased in the last 20 years. In a study of older adult offenders, Reutens et al. found that older offenders were more likely to have more physical illness, cognitive impairment, or psychotic illness; and victims were more likely to be female and in a domestic relationship with the offender [47].

There is also an increased risk of aggressive behavior by *caregivers* toward geriatric patients with MNCD. This presents a unique challenge in involuntary treatment. Though limited studies exist and such events are rare, homicidal ideation has been found to be a real and significant phenomenon among family caregivers of people with MNCD [53]. Untreated depressive disorder in older male caregivers, especially those with feelings of hopelessness or helplessness, raises the possibility of resulting in homicide–suicide or mercy killing [54]. Risk of homicidality is increased when there is the belief that nothing more can be done to make a patient, frequently a spouse, better, and in caregivers who are unable or unwilling to accept help.

When significant risk is present, commitment to hospital and involuntary treatment of the patient may be necessary in order to ensure the mental well-being and safety of both the caregiver and individual with MNCD. The perpetrators in the majority of cases are male, and homicide-suicides frequently involve couples married for long periods of time [54]. Half of perpetrators have been found to have an undiagnosed depressive disorder, emphasizing the importance of ongoing monitoring and support for caregivers to reduce burnout and ensure stable mental health in the caregiver. Should involuntary treatment and hospitalization be required for the caregiver, respite care must be arranged for the individual with MNCD in their care.

13.11 Use of Restraints

The principles and frequency of chemical and physical restraint use in inpatient settings is a contentious topic, and practitioners and institutional approaches differ. Restraints can be primarily classified as chemical – administration of

medications to control behavior – or physical, by means of seclusion and devices aimed to physically and temporarily limit movement. Examples of chemical restraints include the use of sedatives or antipsychotic medications administered either orally or intramuscularly. Examples of physical restraints include seatbelts, harnesses, and straps. Both chemical and physical restraints should be used sparingly, and with the goal of the least restrictive intervention possible for stabilization. At times, the use of both physical and chemical restraints in conjunction is required. A behavioral management plan is best that factors in restraint use, a graduated stepwise, interdisciplinary approach, with a hierarchy of interventions corresponding to the severity of observed behavior and mental status deterioration.

Despite attempts to reduce the use of restraints, the prevalence of restraint use in the general adult psychiatric inpatient setting is between 3.8% and 20% [55]. In a systematic review of 49 studies on restraint use in adult inpatient psychiatry units, variables most frequently associated with the use of restraints included male gender, younger age, foreign ethnicity, schizophrenia, involuntary admission, aggression or trying to abscond from hospital, and the presence of male staff [55]. Though no specific data is available on prevalence, and associated variables specific to a geriatric psychiatry inpatient setting, the prevalence of restraint use is believed to be lower than in the general adult inpatient psychiatry population. A qualitative study of nurses in an inpatient geriatric psychiatry environment suggests that adverse interpersonal environments, defined as behaviors of, and relationships between, patients and staff, and physical environments (e.g., noisy, crowded environments) contribute to restraint and seclusion use, which are also influenced by the practice environment [56]. From a frontline perspective, a lack of accessible and effective alternatives to restraint and seclusion often exists [56]. The use of restraints is also related to the availability of staffing, comfort, attitudes of frontline staff, and culture of the inpatient unit.

Noncoercive and minimally restrictive approaches such as decreasing environmental stimuli and redirection should be utilized first before chemical and physical restraints [57]. Whenever restraints are used, the infringement of individual liberty is present, risking further distress and injury [58]. Thus, there is a shift toward the reduction of restraint use in the inpatient psychiatric setting. Restraint use and other control interventions should be methods of last resort to prevent self-harm or harm to others [59]. Despite differences in perspective, there are times where it is necessary for restraints to be considered to ensure the safety of all involved on the inpatient psychiatry setting [60]. Some patients remain persistently restless, intrusive, or aggressive as a result of MNCD or psychosis to the extent that co-patients, staff, and the patients themselves all suffer and where restraints are necessary [58]. The consequences of restraint and seclusion should not be considered lightly, as studies have shown detrimental physical trauma, and psychological effects [55].

Frequent monitoring of response and patient distress, and regular reassessments should guide whether use is constructive and if use of restraints is still required. Restraint use ordered for an indefinite period is illegal in many jurisdictions. In jurisdictions where restraint use can be ordered with an indefinite timeline, practitioners should avoid such practice to minimize psychological damage, harm, and distress. Chemical restraints orders should be on a one-time or an as-needed basis and accompanied by specification of the frequency by which the medication can be administered and the behavioral signs and symptoms needed to trigger administration. Physical restraint orders should be on a one-time or on an as-needed basis if legally permitted within the jurisdiction of practice, accompanied by an indication of reassessment period, which should not exceed a 24-hour period.

In the inpatient geriatric psychiatry setting, common physical restraint interventions range in their degree of restrictiveness to movement. Minimally restrictive physical restraints include seclusion where an individual is confined to a room and is not able to exit freely for a period of time. The use of locked seatbelts where individual may be confined to a wheelchair or a static chair such as a Broda chair, where primarily

standing and walking movements are limited but hands and feet are free is also an option.

In some cases it is necessary to restrict a patient's movement to ensure safety. The use of mechanical restraints may thus involve immobilizing one or more limbs using straps attached to the bed to restrict movement and promote calmness. In patients with neurocognitive disorders, such practices can be quite distressing and may increase agitation in the short term. Physical restraints may be better than chemical restraints for patients with significant medical comorbidities such as cardiac abnormalities and with history of multiple medications with potential interactions. In cases of a limited response to medications in the past, in circumstances where the period of restraint is anticipated to be for a short period of time, and when containment and response is required immediately, physical restraints may be useful. Effective physical restraint use can be as short as several minutes, especially in cases where pharmacological interventions are concomitantly administered or where a clear trigger is identifiable and can be removed. A low-dose pharmacological agent may also be helpful as an adjunct to reduce psychological suffering in individuals who are very distressed with physical restraints.

Common chemical restraints include antipsychotics: First-generation antipsychotic medications (FGA) such as haloperidol or loxapine or second-generation antipsychotics (SGA) such as quetiapine, risperidone, or olanzapine. Sedative medications such as benzodiazepines are to be used only in the short term, if at all, due to their increased risk in the geriatric population of falls and adverse events in the geriatric population. Oral medications are offered first, if the patient is able to participate and if it is safe. If the patient refuses, is unable to participate, or if the safety concern is imminent and severe, intramuscular injection may be indicated.

In a study of the relationship between the frequency and duration of restraint use in a geriatric psychiatric inpatient population, self-aggression led to fewer but longer restraint episodes, whereas aggression directed toward others led to fewer and shorter restraint episodes. Restless patients were restrained more often but for briefer periods [61]. While the use of pharmacological agents had no influence on the frequency of restraints, pharmacological agent use did influence the duration of restraints. Patients treated with low-potent neuroleptics had particularly short intervals of restraints, suggesting a role for a comprehensive approach involving pharmacological and physical interventions in effectively managing of distressing behaviors such as agitation and aggression [61].

Patients with a diagnosis of a MNCD, common in inpatient geriatric psychiatry, were 10.6 times more likely to be restrained than patients with other illnesses [62]. Patients incapable of consenting to treatment were 39.3% more likely to be restrained than patients able to provide consent [59]. Patients with MNCDs often have difficulty communicating their needs as the disease progresses (Chap. 19: Medical Nursing Care and Barriers to Communication). Individuals with difficulties making themselves understood were more than twice as likely to be restrained than those who had no such difficulties [59]. Impaired mobility and unsteady gait frequently resulted in restraint use by frontline staff in order to ensure safety [59].

Restraints should be used with caution in geriatric patients who may have a history of PTSD and for whom restraint use may be a source of re-traumatization. Refugees from combat zones, survivors of genocide, combat veterans, sexual trauma victims, victims of assault, and prisoners of war are at risk. Chemical restraints are preferred to physical restraints.

13.12 Prevention of Wandering

Wandering is a challenge for both patients and caregivers, and is commonly observed in the acute clinical setting. Different definitions of this complex behavior exist; typically wandering refers to seemingly aimless or disoriented ambulation throughout a facility, often with observable patterns such as lapping, pacing, or random ambulation [63]. The underlying etiology is poorly understood [64]. Persistent wandering behaviors have been found to be a major reason

for nursing home admission [63]. In the inpatient setting, wandering may impede or interfere with the provision of necessary daily care (e.g., assistance with feeding, bathing, or toileting). Conflict can also occur if the patient enters into another patient's room. Wanderers who present with exit-seeking behaviors pose a challenge in the inpatient setting and frequently require involuntary treatment – civil commitment, constant observation, and transfer to a locked unit may be required.

Robinson et al. found that there is currently no robust evidence to recommend any one intervention. There was some weak evidence for exercise in decreasing wandering behaviors [65]. A Cochrane review by Hermans et al. in 2007 looking at the use of non-pharmacological interventions for wandering in MNCD in the domestic setting found no high-quality or controlled studies and was not able to provide support for any specific interventions [66]. Higher-quality research studies are required to determine the effectiveness of non-pharmacological interventions in the management of wandering behaviors.

In the absence of any one intervention to recommend, reversible contributors to wandering behaviors should be addressed, and involuntary treatment required should a lack of insight and/or an acute safety concern be present. Clinical judgment is required to distinguish wandering from behavioral symptoms that may present a similar clinical picture (e.g., anxiety, agitation, hypomanic episode, and akathisias) [67]. Environmental modifications such as a locked unit where an individual can wander safely and freely may be less restrictive on individual liberties than in an open unit setting, where constant monitoring by staff is required. Frequent redirection in an empathetic manner may be required. Those with exit-seeking tendencies should have their room located close to the nursing station and away from exits and elevators. Psychological factors should also be considered and careful clinical interview with the individual, if able to participate, is helpful in developing appropriate strategies. For example, individuals who are disoriented and are trying to return home or look for a family member, may benefit from reminders and reorientation that they are in hospital and that their family members are safe. Wandering may also be in response to an unmet care or personal need, which can be reduced if addressed by staff. While trying to determine the underlying cause of wandering behaviors, clinicians must constantly assess whether the patient poses an acute danger to themselves and/or others in the environment and if present, involuntary treatment, including restraints may be necessary.

Wandering may be present in the context of an acute condition such as delirium, deterioration of a primary psychiatric illness, or more as a symptom of a major neurocognitive disorder. Regardless of acuity and underlying etiology, safety of the individual and others is paramount. The acute management of wandering in the inpatient setting is across settings, but prognosis and persistence of wandering differs with diagnosis. In patients where wandering is a symptom of acute decompensation, resolution of the underlying etiology should result in the termination of wandering behavior. In the context of a major neurocognitive disorder, or other chronic degenerative process, mitigation of harm and reduction of wandering behavior is the goal rather than complete cessation of wandering.

The wandering behavior occurs in 15–60% of people with MNCD [65], and begin to cease when the MNCD progresses in severity or a decline in mobility occurs. When wandering is a result of temporary, acute, or fluctuating presence of confusion, such as with a delirium or deterioration of a physical or mental condition, the treatment and management of the underlying condition often results in resolution. The presence of pain, however, may be associated with less wandering (Chapter: Pain). In addition to ensuring safety acutely until wandering behaviors resolve, the focus is treatment of the underlying condition (Chap. 12: Delirium).

The management of wandering requires a holistic and comprehensive multidisciplinary approach. Evidence on the effectiveness of pharmacological and non-pharmacological interventions is limited though growing. A thorough risk and safety assessment must be conducted, and commitment to hospital and transfer to a locked unit should be considered. In instances where

wandering presents an imminent risk or aggression is present when confronted about the behavior, the use of temporary chemical and physical restraints may be required. These should be evaluated constantly and as soon as it is safe to do so discontinued. The least restrictive intervention that achieves control and safety should be utilized (Chap. 6: MNCD with Behavioral Disturbance).

Limited evidence exists to support the use of pharmacological interventions for wandering behavior. Pharmacological agents may be beneficial if wandering is accompanied by other symptoms such as aggression, agitation, or depression. Low-dose risperidone has shown some efficacy in reducing aggression and wandering in patients with Alzheimer's disease, though is associated with an increase in fall risk [68]. Despite limited empiric evidence, a low-dose atypical antipsychotic medication such as quetiapine or risperidone is frequently used in clinical practice to reduce psychological distress in individuals with wandering behaviors with some effect, including short courses in acute delirious patients.

Wandering has been correlated with increased severity of cognitive impairment along with presence of disruptive behaviors (e.g., resistance to care and socially inappropriate behaviors) [67]. Schonfeld et al. also found that though wanderers are typically more independent in ambulation than non-wanderers, they are more dependent in their hygienic ADLs [67]. Wheelchair bound patients are also capable of wandering and remain understudied.

Subjective barriers by way of exit modifications using mirrors, camouflage, and grids/stripes of tape are currently used in many centers as a means to reduce wandering and exit-seeking behaviors [69]. Though some studies support their use, unfortunately no high-quality or controlled studies have been conducted to date on subjective barriers. A Cochrane review by Price et al. in 2004 [69] found that there is currently no evidence that subjective barriers prevent wandering in cognitively impaired individuals.

Several non-pharmacological approaches to managing wandering have also been proposed and anecdotal evidence given for their use including music therapy, exercise programs, massage, electronic devices, and multisensory stimulation (Chap. 6: MNCD with Behavioral Disturbance). Robinson et al. conducted a systematic review to evaluate effectiveness of non-pharmacological interventions in reducing wandering in MNCD and to assess acceptability and ethical issues associated with their use [65]. Exercise and music therapy were the most acceptable interventions, with no ethical concerns. Tracking and tagging devices were acceptable to caregivers but raised significant ethical considerations. In the involuntary treatment setting, tracking and tagging devices may be a means to monitor and ensure patient safety remotely.

13.13 Summary

Involuntary interventions may be needed when a geriatric inpatient refuses or obstructs treatment which is essential to prevent irreversible and harmful behavior, and/or is necessary to ameliorate the serious consequences of medical or psychiatric conditions. Such interventions are considered in the context of a patient with impaired decision-making capacity because of psychiatric/behavioral symptoms. Appropriate therapeutic intervention is a priority especially on an inpatient unit, wherein the safety of *all* patients and staff, in addition to the patient, must be considered. The responsibility falls to the psychiatric health-care provider to assess and document the patient's lack of capacity to give full informed consent to the necessary treatment Chap. 5: Legal Aspects provides guidance on the assessment of capacity. When the capacity to give informed consent is lacking, surrogate decision-makers such as family, those who have Durable Power of Attorney for Health Care (DPOA), or adjudicated conservators are enlisted to give consent for the proposed treatment.

An individual's right to refuse treatment may conflict with the need to prevent self-harm, or avert serious physical deterioration or harm to others. This may require administration of treatment without the patient's expressed informed consent, or even without assent, and require substitute informed consent from a surrogate

decision-maker or family member. An early and enduring alliance with key participants, therefore, is important to institute safe, respectful, and effective involuntary interventions.

Understanding the legal environment, hospital policies, and the statutes controlling involuntary interventions is necessary, and access to legal consultation is needed where uncertainty exists. Clinical situations which may require some degree of involuntary intervention include refusal of food, self-harm, aggression, homicidal behavior, wandering, severe depression, and catatonia.

In sum, involuntary treatment may play a critical role in ensuring safe recovery from acute and dangerous psychiatric/behavioral symptomatology. Attention and re-evaluation of the individual's mental state is required in order to ensure that, as an individual's risk decreases and condition improves, his/her decision-making should be reassessed and her/his preferences (documented in an Advance Directive if available) are respected.

Take-Away

1. Geriatric psychiatry inpatients who lack decision-making capacity may require involuntary medical/psychiatric interventions when the risk-benefit ratio weighs in favor of intervention.
2. Comply with local statutes which control involuntary medical interventions.
3. Document the rationale for any involuntary intervention, including risks and benefits.
4. Document informed consent from surrogates and family for any involuntary intervention.
5. Document risk-benefit information provided to decision-makers about a proposed treatment, as well as the risk-benefit for *no* intervention or alternative interventions.
6. Guiding principles of involuntary treatment include safety, least restrictive approach, best interests of patient, avoidance of undue suffering, prior wishes of the patient when he/she *had* decisional capacity, and the risks and benefits of the treatment (Chap. 5: Legal Aspects).
7. Involuntary interventions and treatments should be appropriate to the diagnosis and behavioral need.
8. Patients with major neurocognitive disorders (MNCD) may require involuntary and restricted treatment settings tailored to individual needs (e.g., a locked unit for wanderers, wheelchair with a safety belt for an imminent fall risk).
9. Patients with major neurocognitive disorders with behavioral disturbance may need medications concealed in food or drink in order to minimize aggression, maximize quality of life, and minimize suffering.
10. ECT may be considered, with appropriate informed consent, from surrogates, if other options are not feasible.
11. Consultation with a hospital ethics committee, and/or legal counsel, may be needed if the treatment team is uncertain about ethics or legality of the involuntary intervention.
12. Alliance between family, caregivers, and treatment team should be established as early as possible in the process of delivering involuntary treatment.

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Pain Management

14

Calvin H. Hirsch

14.1 Introduction

Older adults admitted to psychiatric inpatient units likely will have a prevalence of chronic pain that is similar to, if not higher than, the prevalence found in community-dwelling adults. Epidemiological studies have estimated the prevalence from 25% to 76% among adults aged 65 and older, reflecting differences in the population surveyed and the methodology used. Between 83% and 93% of patients admitted from long-term-care settings can be expected to suffer from chronic pain [1]. Most studies have found that chronic pain disproportionately affects older women, compared to older men [1, 2]; the hip, knee, back, and other joints represent the most common anatomical sites of pain symptoms [1]. A study of geriatric patients admitted for depressive disorders to an inpatient psychiatric unit at a large Canadian teaching hospital reported that patients acknowledging pain had a 39% shorter length of stay than those without pain (18.6 days v 30.4 days) [3]. The authors speculate that pain may have played a role in the severity of their depressive disorders, and that treatment of the pain may have accelerated their improvement. In the general adult population, however, the comorbidity of chronic pain appears to reduce the likelihood of a full remission of depressive symptomatology [4]. Figure 14.1 summarizes the overall approach to inpatient pain management. This chapter will highlight:

- The relationship between psychiatric illness and chronic pain
- The challenges of recognizing chronic pain in older adults, especially those with neurocognitive or thought disorders compromising communication
- The potential for atypical presentation of acute pain in older adults
- Strategies for the assessment and management of pain in the older psychiatric inpatient

14.2 Clinical Vignette

Mr. S is a 76-year-old man admitted to the inpatient psychiatric facility for major depressive disorder and an apparent suicide attempt by consuming an unknown quantity of combination tablets of acetaminophen (APAP, paracetamol) and hydrocodone.

For months, Mr. S complained to his wife that he was "sick and tired" of the sharp, lightning bolt pains in his feet and chronic low back pain with left-sided sciatica that were not controlled with the APAP and gabapentin that his

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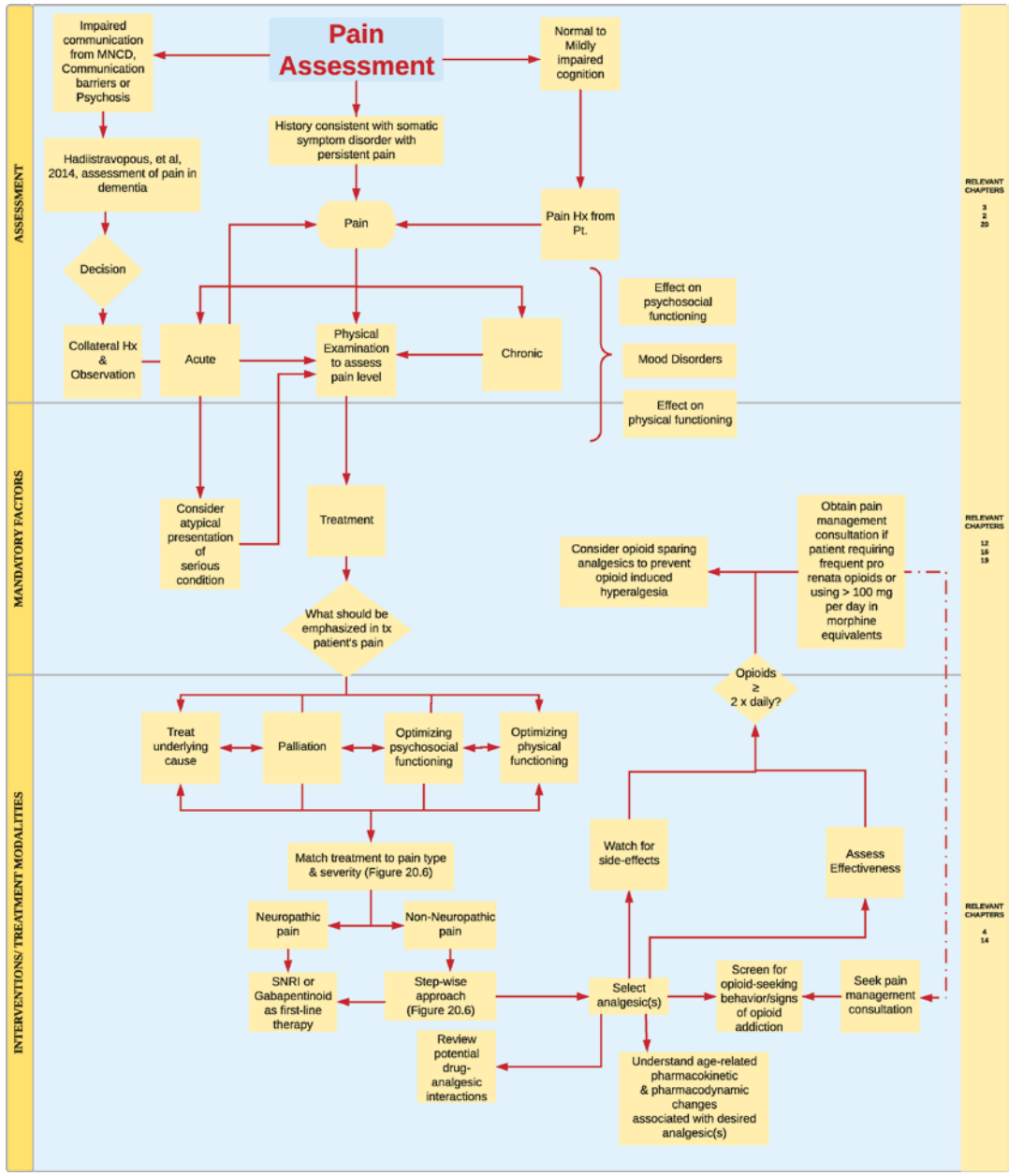

Fig. 14.1 Approach to inpatient pain management

physician prescribed; of feeling tired all the time; of walking like he was drunk and falling; and of feeling confused and forgetful. In the past 2 months, he had tripped and fallen twice, the second time resulting in two fractured ribs and a broken nose. He began to talk about wanting to die. His family physician prescribed paroxetine 20 mg daily for depressive disorder, but Mr. S did not feel that the medication was working. Because of difficulty falling and staying asleep at night, his physician also prescribed zolpidem extended-release 6.25 mg at bedtime, which had recently been increased to 12.5 mg (maximum dose).

His physician also prescribed 30 tablets of APAP 325 mg/hydrocodone 5 mg to take up to every 6 hours as needed for severe pain. The prescription was renewable every 30 days. After 6 weeks on this regimen, he complained to his physician that he still had moderate pain all the time, especially when he walked, and needed a stronger analgesic. His physician changed the opioid prescription to 1–2 tablets every 6 hours as needed, with a 120-tablet supply expected to last at least 30 days.

Over the next 3 months, Mr. S's wife grew concerned about his poor appetite, evident weight loss, irritability, and tendency to sit in his recliner and just stare at the wall much of the day. She became concerned that he sometimes asked her the same question repeatedly, despite being told the answer. He complained that his daughter never came for a visit, although she might have stopped by earlier in the day. He stopped bathing regularly and refused to change his clothes despite episodes of urinary incontinence. He repeatedly told his wife that he was tired of living with his pain and felt he would be "better off dead." He demanded that she drive him to the sporting goods store so he could buy a pistol, and got angry when she refused. One morning she found her husband in his recliner unarousable with vomitus on his chin and chest. She called the paramedics, who transported him to the nearest emergency department (ED). She noticed that his APAP/hydrocodone bottle on the table by his recliner was empty and on its side, although it had contained at least a week's supply of tablets the day before.

On presentation to the ED, Mr. S was arousable to sternal rub but incoherent. Out of concern that he had ingested a large quantity of APAP/hydrocodone, a nasogastric tube was inserted and activated charcoal was administered. Simultaneously, he was given a 9750 mg parenteral loading dose of N-acetylcysteine based on his estimated weight of 65 kg. An acetaminophen level obtained at admission was 55 μ/mL (364 μmol/L) and 4 hours later dropped to 17.5 μg/mL (102.6 μmol/L), both considered within the "safe" range. His hepatic aspartate aminotransaminase (AST) peaked at 55 units/L (mild elevation). After 12 hours in the ED, he became alert and conversant. He was medically cleared and transferred to the inpatient psychiatric unit. His admission serum creatinine was 2.1 mg/dL (185.6 μ mol/L; baseline = 1.8 mg/dL [159 μmol/L]).

Mr. S's medical history was obtained by chart review. His problems included diabetes mellitus type 2 complicated by retinopathy and peripheral neuropathy, hypertension, hyperlipidemia, peripheral vascular disease, stable angina pectoris, prostatic hypertrophy with secondary urge incontinence, lumbar spinal stenosis, knee osteoarthritis, weight loss, hypovitaminosis D, stage 3 chronic kidney disease, insomnia, and depressive disorder. His medications included metoclopramide 1000 mg twice daily for diabetic gastroparesis, gabapentin 600 mg three times daily for peripheral neuropathy, APAP 325 mg/hydrocodone 5 mg: 1–2 tablets up to four times daily as needed for severe pain, simvastatin 40 mg daily for hyperlipidemia, benazepril 20 mg twice daily and metoprolol succinate 50 mg daily for hypertension, aspirin 81 mg daily and tamsulosin 0.8 mg daily for benign prostatic hypertrophy, tolterodine 4 mg daily for urinary incontinence, paroxetine 20 mg daily for depressive disorder, vitamin D3 1000 IU daily, and zolpidem extended-release 12.5 mg at bedtime. Physical examination was notable for a cachectic appearing, unkempt man with a strong odor of urine and generalized muscle wasting. His blood pressure (after rehydration in the ED) was 108/76 mm Hg, heart rate 62 BPM and regular. He reported that his pain was a "6 out of 10" in his feet at rest, and rose to a "10 out of 10" when he tried to ambulate, which he described as "walking on sharp, blisteringly hot rocks." His low back pain was a constant, dull ache of "5–7/10." On neurological exam, he had markedly decreased sensation to light touch and vibration in his feet up to the mid shins. He required moderate effort in pushing off with his hands to stand from a seated position. He was unable to stand with his feet together even with eyes open, and his gait was broad-based and unsteady. On neurocognitive testing, he scored 21 out of 30 on the Montreal Cognitive Assessment (MoCA), failing the Mini-Trails test, clock draw (set time incorrectly), and recall

(missed all 5 items at 5 minutes), and he was disoriented to day and date.

Questions:

1. What is the relationship between Mr. S's pain and his major depressive disorder (MDD)?
2. What neuropathological factors contributed to the severity of his pain?
3. How should Mr. S's pain be treated in light of his MDD and comorbid conditions?

For an in-depth discussion of the answers, see section "Case Vignette: Answers to Questions and Discussion".

14.3 The Interaction of Psychiatric Disorders and Pain

14.3.1 Mood Disorders

Between 10% and 20% of adults age 65 and older suffer from substantial depressive symptoms, which commonly co-occur with anxiety [5–7]; it has been estimated that approximately 13% of older adults concurrently suffer from both depressive disorder and chronic pain [8]. There is a graded association of both depressive and anxiety disorders with the reported severity of pain. In the Netherlands Study of Depression and Anxiety, adults with moderate depressive disorder were three times more likely than nondepressed patients to experience severely disabling pain that limited their activities. If their depressive disorder was severe, the odds ratio (OR) of reporting severely disabling and limiting pain approached 8, compared to nondepressed adults. The study observed a similar, graded relationship between anxiety disorders and pain, with moderately and severely anxious adults being 7 and 13 times more likely, respectively, of reporting severely disabling and limiting pain, compared to non-anxious adults. The combination of depressive and anxiety disorders increased the risk of severely disabling pain 30-fold relative to nondepressed, non-anxious adults. All analyses were adjusted for age, sex, antidepressant use, chronic disease burden, and other demographic factors [9]. The relationship reciprocally works in the opposite direction. In a multinational study of pain in adult primary care conducted at 15 different sites across 15 separate countries and comprising interviews with 5438 patients, depressive and/or anxiety disorder was present in one-third of patients with persistent pain, compared to 10% of patients without pain (OR 4.14, 95% confidence interval 3.52–4.86). That these findings span so many distinct cultures supports the hypothesis that a biological explanation underpins the relationship between pain and depressive and anxiety disorders.

14.3.1.1 Neuroinflammation as a Common Denominator in Depression, Insomnia, Pain, and Aging

Although it has been proposed that chronic pain is a variant expression of depressive disorder [10], the evidence now points to neuropathology that also overlaps with the neuropathology of insomnia. Aging, by itself, produces many of these changes and may amplify the neuropathology of chronic pain and depressive disorders.

Aging is associated with alterations in the structure and function of peripheral sensory nerves, particularly the A∂ fibers, resulting in unchanged to a slightly reduced perception of pain. However, when exposed to the identical noxious stimulus, older adults perceive a greater intensity of pain than young adults [11]. Chronic pain leads to central pain hypersensitivity that amplifies this phenomenon, and pain hypersensitivity can be found in up to 80% of patients attending a pain referral center [12].

Mast cells play a central role in the process of age-related pain hypersensitivity. By being located near nerve endings and the vasculature, mast cells release mediators (e.g., bradykinin, prostaglandins, histamine), which elicit a physiological response to the nociceptive stimulus, which for acute pain can be adaptive and protective. Mast cells also are concentrated in the thalamus, and, in older rodents, more readily release nociceptive mediators that stimulate microglia. These microglia exist in an activated state that results in more robust and sustained production of pro-inflammatory cytokines [11].

Associated with increased central cytokine production is dysfunctional pain modulation caused by reduced inhibition of nociceptive receptors in the spinal cord by descending seroto-

nergic (5-hydroxytryptamine (5-HT)) pathways. Chronic pain causes central hypersensitivity through mechanisms similar to normal aging, potentially increasing the susceptibility of older adults to moderate to severe chronic pain states. Altered serotonergic pathways also have been implicated in mood regulation and sleep disturbances [13]. Depressive disorders and sleep disturbances also share with chronic pain and aging increased levels of pro-inflammatory cytokines within the brain and reduced brain-derived neurotrophic factor [13, 14] (Fig. 14.2).

14.3.2 Other Conditions and Pain Perception

14.3.2.1 Substance Misuse and Schizophrenia

There appears to be a bidirectional relationship between substance use disorder and chronic pain. Patients with fibromyalgia, chronic neck or back pain, and migraine have been found to have an increased risk of misuse of opioids and alcohol. In contrast to what is seen in depressive and anxiety disorders, adults with schizophrenia have a relatively *lower* prevalence of chronic pain, along with a *higher pain threshold*, compared to adults without schizophrenia [15]. It is unknown whether this relationship holds true in old age.

14.3.2.2 Multimorbidity's Influence on Chronic Pain

Most older adults admitted to a psychiatric facility will have one or more chronic systemic medical conditions that can impact chronic pain, which, in turn, may negatively impact depressive disorders. The German Multi-Care Cohort study assessed chronic pain in 3189 primary care patients (mean age 74 years), who had an average of 7 chronic medical conditions. Moderate to severe pain limiting their daily activities affected 22% of women and 15% of men. The notable finding of the study was that, for a given condition commonly associated with chronic pain (e.g., low back problems), the prevalence of chronic pain varied according to the types of morbidities coexisting with that condition [16]. The implication for the treating psychiatrist is that successful management of a depressive disorder may depend not only on managing the chronic pain itself, but also on adequately managing other existing comorbidities.

14.4 Recognizing Chronic Pain in the Older Psychiatric Inpatient

14.4.1 Under-recognition, Underreporting, and Undertreatment

Because chronic pain rarely is the principal reason for admission, failure to recognize and properly address it is common in the inpatient setting, including the psychiatric setting, despite its potential contribution to the patient's psychiatric illness. Among 387 consecutive admissions to the geriatric units of 8 Italian hospitals, roughly two-thirds reported moderate to severe pain, but fewer than half of these patients received adequate pain relief [17]. The reasons are multifactorial. Clinicians' concern over complications from opioids, such as delirium, constipation, urinary retention, falls, and

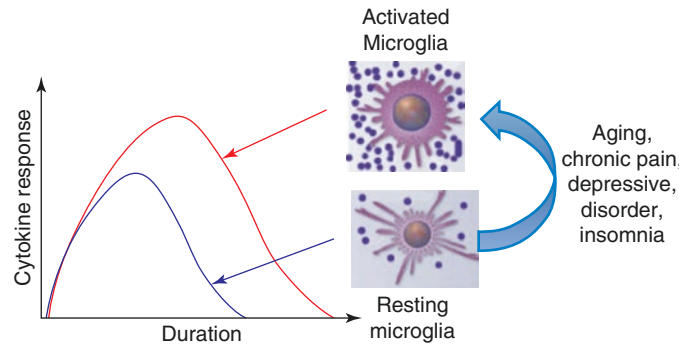

Fig. 14.2 Neuroinflammation associated with aging, chronic pain, depression, and insomnia

opioid dependence, as well as the risk of acute kidney injury or gastritis from non-steroidal anti-inflammatory drugs (NSAIDs), may result in the undertreatment of acute and chronic pain [1, 17]. Increased regulatory oversight in countries like the United States, resulting in part from rising mortality rates from opioid overdoses, have placed pressure on clinicians to limit opioid prescribing. Patients, on their part, may underreport their pain for a variety of reasons, including cultural or socially mediated stoicism, fear of addiction, fear of losing their independence if they admit to pain, language barriers, impaired hearing limiting comprehension of the examiner's questions, major neurocognitive disorder, and an acute psychiatric condition preventing communication of pain [18]. Busy nurses and therapists may overlook important clues about pain, such as grimacing, quiet moaning, or refusing to change position in bed or chair. Agitation from pain may be misinterpreted as a manifestation of the comorbid psychiatric illness.

14.4.2 Pain Assessment

14.4.2.1 Pain History

The assessment of pain should be integrated into the psychiatric history. Figure 14.3 provides a schema for a patient who can provide a reliable history. For those who are unable to give an accurate history, questions can be posed to a knowledgeable informant. The 12-item Geriatric Pain Measure (Fig. 14.4) [19] below may be substituted for a pain review of systems; this tool is not copyrighted and can be duplicated freely. In the hospital setting, validated observational pain scales used by nurses can facilitate pain assessment. The commonly used numeric rating scale (0 = no pain, 10 = the severest pain) is appropriate for verbally responsive patients with normal or mildly impaired cognition. For those with moderate to severe cognitive impairment due to psychiatric illness including neurocognitive disorder, the Wong-Baker FACES Pain Rating Scale [20] allows the examiner to choose the cartoon facial expression most appropriate for the patient's level of distress. The longer PAINAD instrument provides a more informative assessment, and is accessible without copyright restriction through www.geriatricpain.org [21].

As neurocognitive disorders worsen, self-report of pain becomes less accurate and the clinician must turn to a caregiver or family member to identify clues to the existence of acute and chronic pain; these generally consist of changes

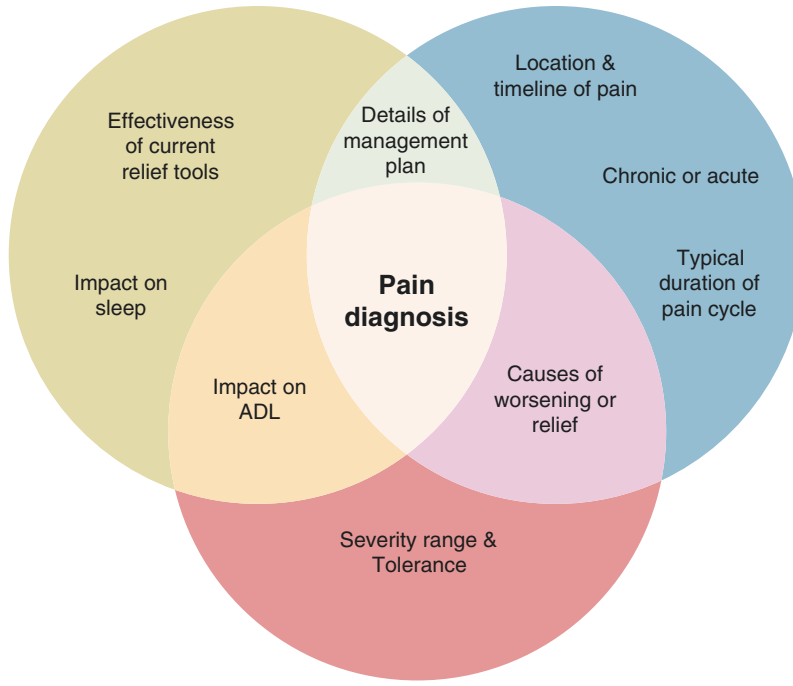

Fig. 14.3 Pain diagnosis

- Do you currently have pain with, or have you stopped, moderate activities such as moving a heavy table, pushing a vacuum cleaner, bowling, or playing golf because of pain?
- Do you currently have pain with, or have you stopped, climbing more than one flight of stairs because of pain?
- Do you currently have pain with, or have your stopped, walking more than 200 yards because of pain?
- Do you currently have pain with, or have you stopped, walking 200 yards or less because of pain?
- Because of pain, have you cut down the amount of time you spend on work or other activities?
- Because of pain, have you been accomplishing less than you would like to?
- Because of pain, have you limited the kind of work or other activities you do?
- Because of pain, does the work or activities you do require extra effort?
- Because of pain, do you have trouble sleeping?
- Does pain prevent you from enjoying any other social or recreational activities (other than religious services)?
- On a scale of 0 to 10, with 0 meaning no pain, and 10 meaning the worst pain you can imagine, how severe is your pain today?
- In the last 7 days, on a scale of 0 to 10, with 0 meaning no pain and 10 meaning the worse pain you can imagine, how severe has your pain been on average?

Fig. 14.4 Geriatric pain measure – short form

in behavior or activity. In advanced major neurocognitive disorder, severe distress can present paradoxically as withdrawal or involution. The patients may stop communicating, resist opening their eyes, tightly clench their fists, stop eating and drinking, and/or resist opening their mouth.

14.4.2.2 Physical Examination and Signs of Distress

The physical examination should include assessment of body sites where the patient complains of pain. In patients incapable of providing a history or with advanced neurocognitive disorder, look for the following signs or history elicited from others:

- Does the patient, when touched or moved, withdraw or attempt to push away the examiner's hand?
- Does patient moan, frown, or grimace during a specific examination maneuver like sitting up?
- Are there signs of recent trauma like ecchymoses or musculoskeletal injury, especially if the patient has a history of falls?
- On examination of the thorax, is there splinting of a hemi-diaphragm during deep breaths, raising concern about a fractured rib?
- Does the patient rub or hold a body part, or brace it during movement?
- The mouth is a frequently overlooked source of pain. Check the dentition for hypersensitive or broken teeth and obvious caries. If the patient

resists opening the mouth, a dental panoramic X-ray or a facial computerized tomographic scan can be obtained if there is reasonable suspicion of a dental source for the pain.

14.5 Atypical Presentation of Acute Pain

Acute pain occurring in a psychiatric inpatient can herald a medical emergency or represent a minor condition that can be handled on the floor. Most pain in the older adult presents *typically* for the underlying pathology, but with increasing age and frailty, *atypical* presentations occur more frequently, and failure to correctly diagnose the patient in a timely fashion can adversely affect outcomes. Vague or nonspecific pain in the older, frail patient may have a serious underlying cause and should not be dismissed without an evaluation.

A complete absence of chest pain may characterize acute myocardial infarction in patients over the age of 70 (and especially over age 80). "Silent" myocardial infarctions also are more common in women and in diabetics [22]. Instead of a burning sensation during urination with or without urgency, urinary tract infections in older patients are more likely to present with nonspecific symptoms that do not immediately implicate the urinary tract (e.g., malaise, loss of appetite, poor balance or falls, delirium).

Although abdominal pain is the fourth most common complaint of older adults presenting to the emergency department [23], the initial symptoms may be vague and nonspecific. In a series of patients 80 years and older requiring emergent surgery for abdominal pain, less than one-third had a fever or a white blood cell count >15,500/mm^3 [24]. Typically, small bowel obstruction presents with diffuse abdominal pain and distention, nausea, vomiting, and obstipation or constipation. However, the full spectrum of symptoms may be absent in older patients at initial presentation, and diarrhea paradoxically may occur. Acute pancreatitis may begin with a vague, poorly localized abdominal pain without radiation, nausea, or vomiting. Peritonitis may initially present without classic rebound tenderness. Like acute pancreatitis, cholecystitis and appendicitis may present with vague, poorly localized abdominal pain [23]. Over half of older patients presenting with acute cholecystitis lack one or more of the classic signs of fever, nausea, and vomiting; 40% fail to show a leukocytosis. An acute, nonspecific, and nonlocalizing pain or symptom in an older patient therefore should be taken seriously and prompt a careful history and physical examination.

14.6 Strategies for Chronic Pain Management

14.6.1 Establishing a Framework for Treatment

The goals of treatment can be categorized into four overlapping domains (Fig. 14.5).

The emphasis given to each domain will vary from patient to patient, but improving *psychosocial functioning* is integral to the treatment of

- Palliation (reducing suffering)
- Treat the underlying cause
- Optimize physical functioning (the ability to perform basic, instrumental, and advanced activities of daily living)
- Optimize psychosocial functioning (social and emotional skills that will enable the patient to resume or find fulfilling relationships and roles within the community, workplace, and home).

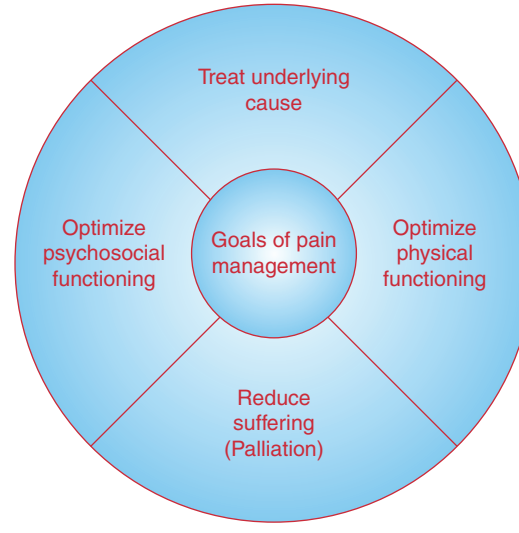

Fig. 14.5 The goals of pain management

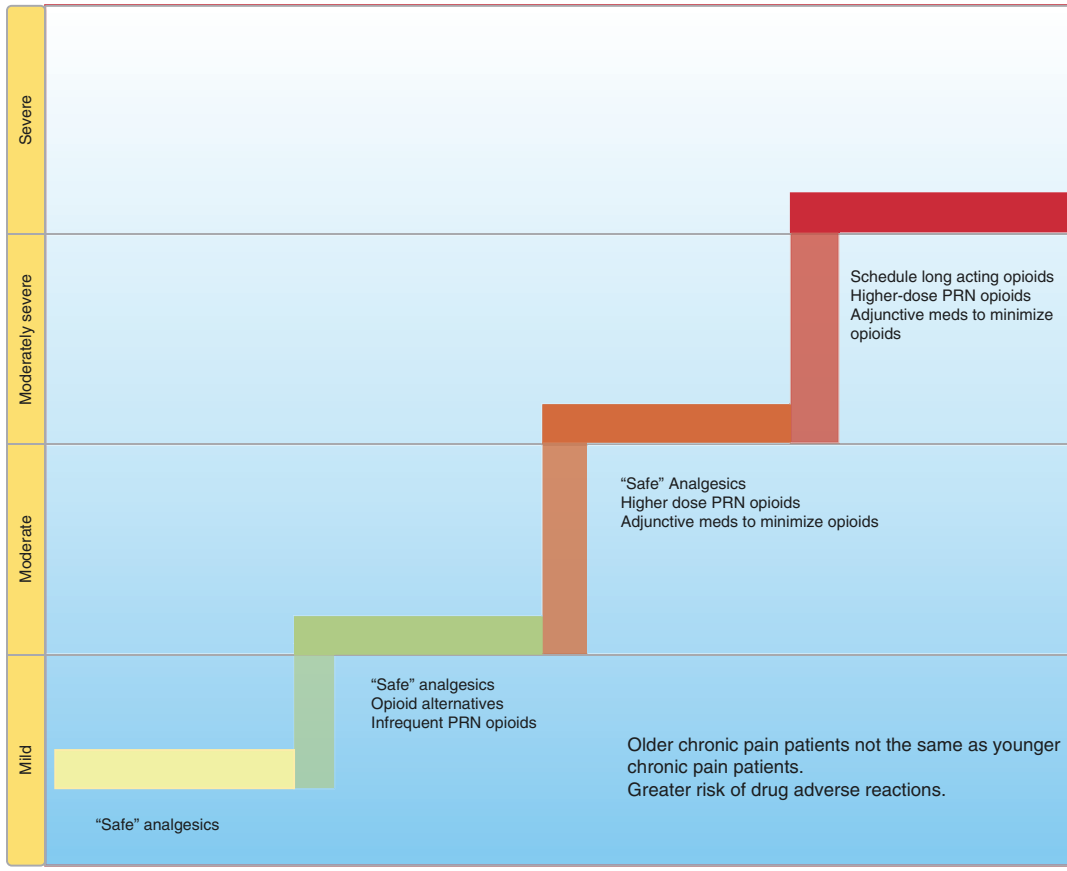

Fig. 14.6 Analgesic regimen

chronic pain in patients with psychiatric illness. In the outpatient sector, chronic pain management usually entails an interdisciplinary collaboration of health professionals coordinated by the primary care physician or pain specialist, but for inpatient psychiatry, this responsibility devolves to the admitting psychiatrist.

14.6.2 Pharmacologic Management of Pain

14.6.2.1 Basic Principles

Compared to younger adults, older patients with chronic pain have a greater risk of adverse reactions to opioid and other analgesics as a result of age-related changes in drug metabolism and sensitivity, as well as drug-drug and drug-nutrient interactions, requiring a careful balancing of benefits versus harm to achieve the goals of chronic pain management. The pharmacologic management of chronic pain entails a stepwise approach based on the severity of pain and whether it is neuropathic, nociceptive, or multifactorial. The selection of an analgesic regimen must be informed by age-related changes in drug metabolism and the hazards of adverse reactions caused by polypharmacy (Fig. 14.6).

14.6.2.2 Age-Related Alterations in Drug Metabolism and Important Drug Interactions

The multimorbidity prevalent among older adults tends to result in the prescription of multiple medications. Polypharmacy, conventionally defined as five or more routinely taken medications, directly correlates with the risk of adverse drug events (ADE) as well as potentially harmful drug interactions. Patients taking five or more medications accounted for >90% of all ADE admissions in a study of US Veterans Administration hospitals

[25]. Age-related changes in body composition and organ-system function cause alterations in the pharmacokinetics (metabolism) and pharmacodynamics (action) of drugs. Lean body mass, consisting primarily of muscle, declines, and along with it total body water, leading to a higher concentration (per dose) of hydrophilic drugs. At the same time, the proportion of body mass made up of fat increases, leading to a higher volume of distribution of lipophilic drugs. The reduction of liver mass with age leads to a slight reduction of phase I metabolism of drugs that utilize the cytochrome P450 (CYP) family of enzymes to oxidize, reduce, or hydrolyze the drug. Phase II metabolism, involving glucuronidation, acetylation, and sulfation, does not significantly change with aging, leaving the metabolism of drugs that utilize these pathways largely unaffected.

For CYP-metabolized drugs, the possibility of adverse drug interactions increases with the number of administered medications, including opioid-drug interactions. Drugs can serve as a substrate for a given CYP, competitively *inhibiting* the metabolism of another drug that uses that CYP, or they can directly inhibit the CYP, in either case prolonging the other drug's half-life. Conversely, a drug can *induce* the metabolism of another drug, reducing the second's duration of action. Drugs metabolized by CYP2D6, for example, can directly or indirectly inhibit the metabolism of codeine, hydrocodone, oxycodone, and tramadol. Commonly prescribed CYP2D6-metabolized drugs include the antidepressants duloxetine, venlafaxine, citalopram, escitalopram, fluoxetine, paroxetine, sertraline, and bupropion, as well as metoprolol and other beta-blockers. Numerous medications are metabolized by CYP3A4, including calcium-channel blockers, cardiovascular agents like amiodarone and digoxin, the anticoagulant warfarin, at least six different benzodiazepines, and zolpidem. Coadministration of these medications with oxycodone, methadone, tramadol, or fentanyl can prolong the serum half-life of these opioids. Only morphine, hydromorphone, and oxymorphone do not interact with CYP enzymes, being metabolized by glucuronidation [26]. Morphine is relatively contraindicated in older adults because of the age-related reduction in renal clearance. These potential drug interactions do not preclude co-prescription of the opioids, but underscore the need to start at a dose lower than the conventional starting dose for younger adults and to titrate up slowly ("Start low, go slow").

14.6.2.3 Other Factors Affecting Drug Metabolism

Pharmacodynamic Changes

In addition to age-associated changes in pharmacokinetics, many drugs show age-related differences in the extent and type of their pharmacologic actions. Compared to younger adults, older adults, on average, experience greater overall analgesia from a given dose of morphine that is unrelated to the longer half-life due to reduced clearance by the kidneys. Another example illustrating pharmacodynamic changes with aging can be found with benzodiazepines and benzodiazepine-GABA receptor agonists (e.g., zolpidem, eszopiclone). Compared to younger adults, these medications cause relatively greater postural sway in older patients that increases their fall risk. These medications can precipitate acute delirium and/or lead to chronic delirium, which merges with major neurocognitive disorder.

Sex Differences and Ethnicity

Regardless of age, a given mg/kg dose of hydromorphone or oxycodone will reach up to a 25% higher peak dose in women than men. Patients from different ethnic backgrounds may show different metabolism of an opioid, likely reflecting genetic polymorphisms [26].

14.6.2.4 Stepwise Approach to the Pharmacotherapy of Pain

The selection and dosing of analgesic medications must take into account the purpose and expected duration of analgesic therapy, the patient's comorbidities, and the use of other medications. A guiding principle is to choose the safest analgesics at the lowest effective dose, and to utilize opioids only when necessary to achieve adequate analgesia. For mild chronic pain, acetaminophen (APAP; paracetamol) has had a favorable safety profile for chronic use, although recent data have shown it to be associated with a slightly increased risk of gastrointestinal (GI) bleeding and cardiovascular (CV) events, but much less than from non-steroi-

dal anti-inflammatory drugs (NSAIDs). Its potential hepatotoxicity is well known, especially in doses >4.0 g/day [27], and it should be used cautiously at doses ≤2 g/day in patients with mild to moderate cirrhosis. Patients with advanced cirrhosis or hepatic dysfunction should not be prescribed APAP. Despite a meta-analysis demonstrating the inferiority of APAP compared to NSAIDs for pain control in osteoarthritis [28], APAP continues to have a safety profile superior to NSAIDs, especially in older adults, and should be the drug of choice for the initial treatment of mild to moderate acute or chronic pain.

All NSAIDs, whether nonselective or COX-2 inhibitors, increase the risk of GI bleeding, not merely in the stomach and duodenum but throughout the intestine [27]. In contrast to the nonselective NSAIDs, COX-2 inhibitors do not inhibit the platelet-aggregating effects of thromboxane A2, which therefore increases the risk of cardiovascular events. The early belief that COX-2 inhibitors would not impair renal function only holds true for healthy elderly with preserved renal function and neither on a very low-salt diet nor a salt-depleting diuretic. If they are, COX-2 inhibitors, like the nonselective NSAIDs, can reduce renal blood flow and increase the risk of acute kidney injury [29]. Increasing age elevates the risk of NSAID-associated GI bleeding and dysfunction, acute kidney injury, and CV events. As a consequence, NSAIDs play a limited role in chronic pain in older adults, and their use should be restricted to short-term use (i.e., 1 week or less) for acute mild-moderate pain unresponsive to APAP in order to avoid or minimize the use of opioids. Unless contraindicated, daily use of NSAIDs should be accompanied by a proton pump inhibitor (PPI) [27, 29]. It should be noted that selective serotonin reuptake inhibitors (SSRIs) and selective norepinephrine reuptake inhibitors (SNRIs) can inhibit platelet aggregation, have independently been associated with serious bleeding, and augment the platelet inhibition of nonselective NSAIDs, aspirin, and clopidogrel.

Patients with moderate to moderately severe chronic pain usually require an oral opioid to supplement the "safe" analgesic (usually APAP), which is kept to minimize the amount of opioid required. At this level of pain, the opioid should be short-acting and dosed pro re nata (PRN). Clinicians can prescribe a combination pill with APAP 325 mg plus hydrocodone or oxycodone, or separately prescribe the opioid and APAP, which allows more flexibility in dosing. Tramadol binds to mu opioid receptors and has an abuse potential that is less than more traditional opioids, but still significant [30]. As a weak inhibitor of serotonin and norepinephrine reuptake, tramadol can increase brain serotonin levels, and alone or in combination with other serotonergic agonists can precipitate serotonin syndrome. If patients require, or have been requesting, regular daily dosing of an opioid, additional "opioid-sparing" medication should be tried while bearing in mind potential side effects and drug interactions. Adjunctive pain medications historically have consisted of the gabapentinoids (gabapentin and pregabalin), which are classified as anticonvulsants; the tricyclic antidepressants (particularly amitriptyline); and the SNRIs (duloxetine and venlafaxine). All anticonvulsants have been associated with falls, and an increased, but relatively smaller, fall risk has been associated with the SNRIs. The gabapentinoids can cause sedation and delirium, and their use should be initiated with a small, nighttime dose and titrated up gradually as tolerated. In neuropathic pain, the gabapentinoids and SNRIs move to first-line therapy. In older adults, tricyclic antidepressants are relatively contraindicated because of potential cardiotoxicity and central anticholinergic activity that can affect cognition. Comorbid conditions can help guide the selection of therapy. If, for example, the patient has concomitant depression, an SNRI may be a more appropriate choice for neuropathic pain than a gabapentinoid. Due to hepatotoxicity risk, avoid duloxetine in hepatitis or cirrhosis.

Patients with severe chronic pain often require or request the maximum frequency of an oral short-acting opioid. Allowing the analgesic effect of a short-acting opioid to wear off substantially often requires a higher repeat dose to reach the threshold of analgesia than would be needed if an adequate serum level of opioid were maintained. This, in turn, results in the patient potentially receiving a higher daily amount of opioid (usually measured in morphine equivalents), compared to being on a long-acting preparation. Therefore, the patient with severe chronic pain usually is a candidate for a long-acting opioid supplemented by a PRN short-acting opioid for breakthrough pain. Although

there is no role for plain APAP (paracetamol) in severe pain, an adjunctive analgesic (e.g., SNRI, gabapentinoid) may mitigate opioid-induced hyperalgesia (see section "Opioid-induced hyperalgesia (OIH)"), and the dose should gradually be titrated up to the maximum or as tolerated, closely monitoring for side effects. It is recommended that patients who may need conversion from PRN to long-acting opioids or whose pain is so severe as to require parenteral opioids receive a consultation from a pain specialist or pharmacist. A consultation by a pharmacist can help identify the safest analgesic regimen based on the patient's renal, hepatic, and medication profiles.

Multiple conversion tables exist to facilitate conversion from one opioid to an equianalgesic dose of another, either oral to oral or parenteral to oral. Most are derived from single-dose studies, have limited validation across pain types and age groups, and do not account for interpatient variations in organ-system function and drug-drug interactions [31]. Due to this imprecision, the conversion doses should be viewed as approximate, and a good rule of thumb is to start the alternate opioid at a dose 15–25% less than the calculated equianalgesic dose.

14.6.2.5 Opioid-Induced Hyperalgesia (OIH)

Patients receiving chronic opioids may develop opioid-induced hyperalgesia, characterized by paradoxically increased pain sensitivity as a result of alterations in the closely interconnected GABA-ergic (pro-nociceptive) and dopaminergic (anti-nociceptive) pathways found in the thalamus, midbrain, and mesolimbic system. These pathways have been shown to modulate pain perception, pleasure sensations, and mood, and may underlie the close association of depressive disorders and pain. As in the phenomenon of pain hypersensitivity associated with aging and chronic pain, the brains of patients with OIH demonstrate microglial activation and increased inflammatory cytokines [32]. The consequence is that a state of opioid "tolerance" supervenes, with the increased sensitivity to pain leading to higher doses in order to achieve the same analgesic effect. Unfortunately, untoward side effects like constipation, sedation, and cognitive impairment are unaffected by this opioid tolerance and tend to increase. For patients requiring >100 mg in morphine equivalents over a 24-hour period, consultation with a pain pharmacist or physician should be considered.

14.6.2.6 Analgesic Medications and Cognitive Impairment

All opioids, including tramadol, can precipitate delirium and impair cognitive function in older patients, and should be used cautiously in patients receiving other psychoactive medications that may affect cognition. In addition to obvious culprits, like benzodiazepines, many commonly prescribed medications can independently and in combination affect cognition. These include all medications with centrally acting anticholinergic properties, such as bladder antispasmodics (e.g., oxybutynin, tolterodine), first-generation (H_1) antihistamines (e.g., meclizine, diphenhydramine), first-generation H_2 blocking antacids (e.g., ranitidine), tricyclic antidepressants (e.g., amitriptyline, doxepin), and phenothiazine derivatives (e.g., prochlorperazine, chlorpromazine). To complicate matters, many common medications have weak to moderate anticholinergic activity at usual therapeutic doses, and, when taken with other anticholinergic drugs, can produce a substantial cumulative anticholinergic burden. Examples of these weaker anticholinergic medications include digoxin, warfarin, metoprolol, atenolol, isosorbide, chlorthalidone, furosemide, paroxetine, and olanzapine. It should be noted that among existing expert-based anticholinergic scales, disagreement exists regarding the magnitude of anticholinergic activity of individual medications. For example, the Anticholinergic Cognitive Burden Scale lists bupropion as a weakly anticholinergic medication, whereas neither the Anticholinergic Drug Scale nor the Anticholinergic Risk Scale mentions any anticholinergic activity associated with bupropion [33].

14.6.2.7 Analgesics and Fall Risk

There is a dose-response relationship between the morphine equivalents administered and the risk of falls. Opioids increase the risk of falls – common in older adults – as much as fivefold

compared to NSAIDs [25]. As mentioned earlier, the gabapentinoids and virtually all classes of antidepressants have been linked to a statistically significant but small increase in fall risk.

14.6.3 Non-pharmacologic Approaches to Pain Management for the Inpatient Psychiatric Patient

A number of behavioral therapies have been used to modify the patient's beliefs about and attitude toward their pain and to increase the patient's perception of control over the pain. A number of small studies, mostly taking place in the long-term care setting, have examined the effects of behavioral therapy on a variety of outcomes, including self-reported pain, mood, sleep, and quality of life. Mindfulness, cognitive behavioral therapy, guided imagery, biofeedback, and self-efficacy training have been tried as supplemental or alternative interventions for mild to moderate pain, although the studies have been small with heterogeneous study designs and patient populations. Their results, although promising, remain inconclusive. An important limitation for the inpatient setting is the duration of the studies, with many requiring multiple sessions over many weeks [1].

14.6.4 Pain Management and the Risk of Addiction and Dependence

Older adults with a history of substance misuse may also have chronic pain for which they request opioids after admission to the hospital. It is often unclear whether the pain is real or part of opioid-seeking behavior due to addiction. It has been estimated that between 18% and 41% of older adults who receive prescription opioids misuse their prescription (take more than prescribed, request early refills, or divert the medication) [34]. However, most of the misuse results from trying to manage their pain rather than to get "high" [35]. In many patients who display addictive behaviors toward opioids (e.g., requesting higher doses), these behaviors disappear when their pain is adequately treated (a phenomenon known as *pseudo-addiction*). In such patients, a careful pain history should seek disparities between reported severity of pain and level of function or behavior (e.g., complaining of "10 out of 10" pain but smiling and joking). A legitimate basis for the pain should be sought through a search of medical records and a careful physical examination that includes discrete observation of the patient performing activities that would be expected to exacerbate the pain. If available, a government-sponsored Internet database for monitoring scheduled drugs, such as those in Canada and California [36, 37], should be searched for multiple opioid prescriptions from different providers over a short period of time. For newly admitted patients who claim regular opioid consumption, a urine toxicology screen can confirm recent use of opioids; a "clean" screen should alert the clinician to possible opioid-seeking behavior or diversion of their prescription opioid.

14.7 Case Vignette: Answers to Questions and Discussion

1. What is the relationship between Mr. S's pain and his major depressive disorder (MDD)?

   As discussed in section "Mood Disorders", there is a strong graded relationship between depressive disorders and chronic pain. Patients with depressive disorder are more likely to have moderate to severe chronic pain, compared to nondepressed patients; the obverse also holds true.

2. What neuropathological factors contributed to the severity of his pain?

   Chronic pain, advanced age, and major depressive disorder are associated with central pain hypersensitivity. This is believed to result from impaired serotonergic modulation of pain by the central nervous system as a consequence of neuroinflammation induced by pro-inflammatory cytokines released by activated microglia that have been stimulated by mast

cells in the thalamus. (section "Neuroinflammation as a Common Denominator in Depression, Insomnia, Pain, and Aging"). Mr. S also regularly ingested hydrocodone for his moderate to severe chronic pain. This likely resulted in opioid-induced hyperalgesia (section "Opioid-Induced Hyperalgesia (OIH)") that compounded his existing pain hypersensitivity.

3. How should Mr. S's pain be treated in light of his MDD and comorbid conditions?

Mr. S has moderate to severe neuropathic pain, despite the maximum recommended dose of gabapentin, and has been regularly using opioids, resulting in probable opioid-induced hyperalgesia. His comorbid conditions include peripheral neuropathy, unstable gait and falls, peripheral vascular disease, insomnia, major depressive disorder, prostatic hypertrophy with urge incontinence, weight loss, spinal stenosis, knee osteoarthritis, and mild cognitive impairment. He has significant polypharmacy. Figure 14.7 provides a schematic representation of the interrelationships between Mr. S's active comorbidities at the time of admission. The patient's chronic pain is integrally associated with his major depressive disorder and therefore requires aggressive treatment. At the same time, the side effects of his analgesic

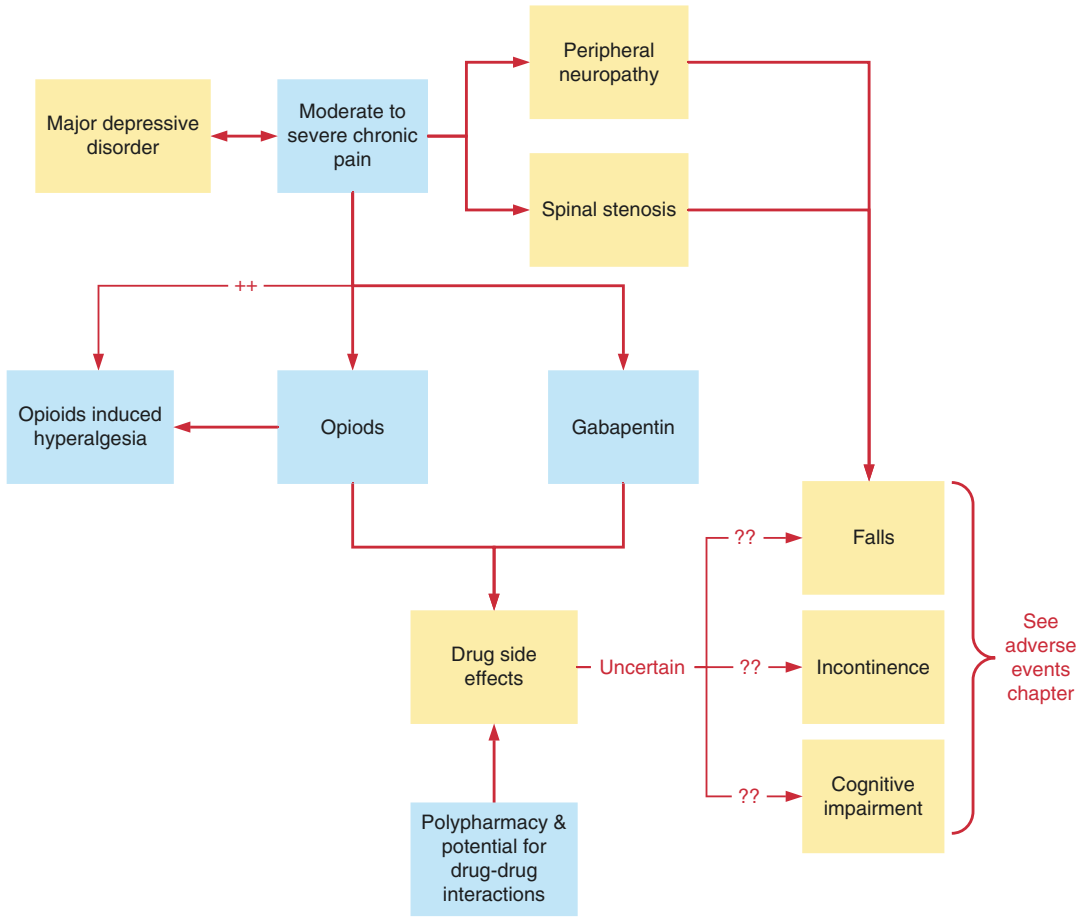

Fig. 14.7 Schematic representation of the interrelationships between the comorbities and chronic pain in the case vignette

regimen must be weighed against their potential benefits. He already has a high fall risk due to his peripheral neuropathy and spinal stenosis. All opioids increase fall risk, as does the gabapentin. Opioids also can adversely affect his cognition, which is impaired based on history and neurocognitive testing. The cause of his neurocognitive disorder likely is multifactorial, with an unknown reversible component contributed by drug side effects and cognitive symptoms of depressive disorder. He takes 12 prescription medications, with significant potential for adverse side effects and drug interactions. Notably, the zolpidem could contribute to his confusion and fall risk. Both tolterodine and paroxetine have significant anticholinergic properties, which could worsen his confusion. His total anticholinergic burden (including from the opioids) could lead to urinary retention; a post-void bladder residual volume therefore should be checked, as overflow may have replaced urge incontinence. His blood pressure is low-normal and he appears beta-blocked; orthostatic hypotension should be ruled out as a contributor to falls, and the antihypertensive drugs may require a dose reduction. SSRIs (and SNRIs) affect platelet function and increase the risk of bleeding, especially in light of daily aspirin for his coronary heart disease. Paroxetine, metoprolol, and hydrocodone are metabolized by CYP2D6, and an SSRI or SNRI could prolong the half-life of the hydrocodone and increase the risk of adverse effects (section "Age-Related Alterations in Drug Metabolism and Important Drug Interactions").

The goal of pain management is to find the best possible balance between the risk of adverse effects, including drug-drug interactions, and sufficient control of the pain to enable resumption of usual physical and psychosocial activity. Neuropathic pain dominates his pain syndrome. Consideration should be given to substituting duloxetine, an SNRI, for the paroxetine. This potentially could help both the neuropathy and the major depressive disorder, allowing a taper of the gabapentin. Although SNRIs have been associated with fall risk, it appears less than with high-dose gabapentinoids. Mirtazapine could be substituted for the zolpidem, although a taper of the latter might be required because of likely physical dependence from long-term daily use. The mirtazapine could help with sleep, potentially improve his appetite (his glucose intolerance notwithstanding), and augment the antidepressant effects of duloxetine. The duloxetine may have the added benefit of helping to stabilize, if not reduce, his presumed opioid-induced hyperalgesia.

At this stage, it is premature to start tapering his opioids. Based on his report of chronic pain and its impact on his physical and psychosocial functioning, he likely would benefit from conversion to a long-acting opioid. Because of his chronic kidney disease and age-related reduction in renal mass, morphine is relatively contraindicated. Despite potential drug interactions with duloxetine, a long-acting scheduled opioid, such as oxycodone or a fentanyl transdermal patch, should be considered, and the APAP/hydrocodone dose commensurately reduced and reserved for breakthrough pain. In light of Mr. S's suicide attempt, the advantage of a fentanyl patch is the inability to ingest a large, potentially fatal dose. Given the complexity of Mr. S's pain management and its integral relationship to his depressive disorder, consultation with a pain specialist is recommended.

14.8 Summary

Chronic pain affects between 25% and 76% of older adults; between 10% and 20% of older adults experience significant depressive disorders. The graded, bidirectional relationship between depressive symptoms and pain severity mandates that pain assessment and management be integrated into the treatment of severe depressive disorders. In all adults, neuroinflammation, involving the increased

production of inflammatory cytokines within the brain by activated microglia, is caused by chronic pain, major depressive disorder, and insomnia, leading to pain hypersensitivity. In older adults, age-related changes to the central nervous system also contribute to pain hypersensitivity.

Pain in older adults often goes underappreciated or undertreated as a result of patient underreporting, failure to screen for pain by the inpatient medical team, focus on the acute medical or psychiatric illness, or fear on the part of clinicians of utilizing opioids because of potential side effects and an anti-opioid regulatory climate. In the older psychiatric inpatient, a standardized pain assessment should be integrated into the admission history and physical exam. In patients with impaired communication or major neurocognitive disorder, pain recognition requires a collateral history or structured observation looking for signs of distress at rest or upon movement. Severely cognitively impaired patients may withdraw, refuse food or fluids, or resist personal care. Although most older adults with acute pain present the typical symptoms for the cause of the pain, clinicians should be aware that, especially among frail older patients, symptoms may initially present atypically (e.g., poor localization of the pain, lack of associated symptoms like fever, leukocytosis, delirium, or other change in behavior).

There are four overlapping goals for chronic pain management: palliation, treatment of the underlying cause, optimizing physical functioning, and optimizing psychosocial functioning. How much each domain is emphasized will depend on the source, intensity, and effects of the pain on the patient's ability to carry out basic activities and fulfill social roles. The pharmacologic management of chronic pain entails a stepwise approach based on pain severity and type. For nociceptive pain, treatment begins with safer analgesics like acetaminophen (paracetamol) for mild-moderate chronic pain, escalating to the addition of as-needed short-acting opioids when the pain is moderate-severe, and culminating in scheduled long-acting opioids when the pain is severe or disabling, reserving shorter-acting opioids for severe breakthrough pain. Long-term use of non-steroidal anti-inflammatory drugs is relatively contraindicated in older adults because of the drugs' effects on kidney function and risk of gastrointestinal bleeding, as well as other side effects like salt and water retention and (for COX-2 inhibitors) increased risk of myocardial infarction and stroke. Adjunctive analgesics, particularly the gabapentinoids and selective norepinephrine reuptake inhibitors (SNRIs), usually are added when opioids are taken regularly in order to minimize opioid requirements (and side effects) and to mitigate opioid-induced hyperalgesia. A gabapentinoid or an SNRI is considered first-line therapy for neuropathic pain. For long-term psychiatric inpatients, cognitive behavioral therapy, mindfulness-based cognitive therapy, guided imagery, biofeedback, or self-efficacy training may be suitable as an alternative or adjunctive nonpharmacologic intervention for mild to moderate chronic pain, despite limited evidence for their efficacy.

Polypharmacy coexists with the multimorbidity prevalent in older adults and poses a challenge to clinicians because of drug-drug interactions, including analgesic-drug interactions, and the risk of adverse drug events. Clinicians also should be aware of age-associated changes in the metabolism (pharmacokinetics) and action of analgesics. For patients with substantial opioid requirements, evidence of opioid-seeking behavior, or the need to convert from one type of opioid to the equianalgesic dose of another, a consultation from a pain specialist or pharmacist is recommended.

Take-Away

- Chronic pain among older adults is very common.
- Chronic pain is closely associated with depressive and anxiety disorders, as well as other medical comorbidities.
- Chronic pain is frequently underreported by the patient, underrecognized by the clinician, and undertreated.
- Acute systemic medical conditions that typically involve pain may have *atypical* presentations in the frail, older adult.
- In managing chronic pain, there are four overlapping goals of treatment: palliation, treating the underlying cause, optimizing physical functioning, and optimizing psychosocial functioning.
- Nociceptive pain management in the hospital setting involves a stepwise approach utilizing safer analgesics first and escalating to opioids for moderate to severe pain.
- Pregabalin, gabapentin, or an SNRI should be considered for first-line therapy for neuropathic pain and as adjunctive therapy in moderate-severe pain.
- Clinicians must be aware of pharmacokinetic and pharmacodynamic changes that occur with normal aging and which potentially can affect the metabolism of analgesics and other drugs.
- Patients taking multiple prescription medications (polypharmacy) are at risk of drug-analgesic interactions; it is useful to check for drug interactions before prescribing an analgesic, using a drug interaction tool available as an app on smartphones, an online tool, or the advice of a pain pharmacist.

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15 Special Syndromes: Serotonin Syndrome, Neuroleptic Malignant Syndrome, and Catatonia

Julia Kulikowski and Usha Parthasarathi

15.1 Introduction

Serotonin syndrome (SS), neuroleptic malignant syndrome (NMS), and catatonia are often seen in the context of the acute psychiatric unit and have serious consequences if not recognized early in the geriatric population. The following vignette shows how the syndromes overlap in presentation, and are challenging to diagnose and to differentiate, especially in the geriatric patient. Figure 15.1 illustrates in overview, the assessment and management of the special conditions SS, NMS, and catatonia. A more specific introduction to each syndrome is offered below.

15.2 Vignette

An 81-year-old man presented to hospital due to the onset of disorientation within the prior 24 hours. He was irritable, agitated, and repeated, "Sam, Sam, where are you?" He lived with his wife and needed minimal assistance with activities of daily living (ADLs). Three years ago, he was diagnosed with Parkinson disease and began treatment with carbidopa/levodopa 25/100 mg po t.i.d. He was also prescribed amlodipine 25 mg po daily for hypertension. He had no known past psychiatric history nor any illicit substance use or alcohol abuse.

Six weeks prior to admission, he was diagnosed with complicated bereavement, after the sudden death of his young grandson, Sam. Family members reported that the patient began hallucinating the voice of his deceased grandson. He was started on fluoxetine 20 mg po daily but unable to tolerate it, and a week prior to admission he was switched to escitalopram 10 mg po daily. Within that same week, he was diagnosed with a urinary tract infection (UTI), and a course of ciprofloxacin was started. The day before admission, the patient developed confusion, anxiety, headache, dizziness, restlessness, shivering, tremor, dysarthria, diarrhea, and diaphoresis.

On admission, his blood pressure was 130/75 mm Hg, heart rate 116 beats/min, and axillary temperature 98.6 F. The complete blood count, electrolytes, glucose, calcium, liver, kidney, creatine phosphokinase (CPK) levels, and thyroid function tests were within normal limits. Electrocardiogram (ECG) was normal. Computed tomography (CT) of the head showed no acute abnormalities. A provisional diagnosis of major depressive disorder secondary to complicated bereavement was made. Physical symptoms were considered manifestations of anxiety, and escita-

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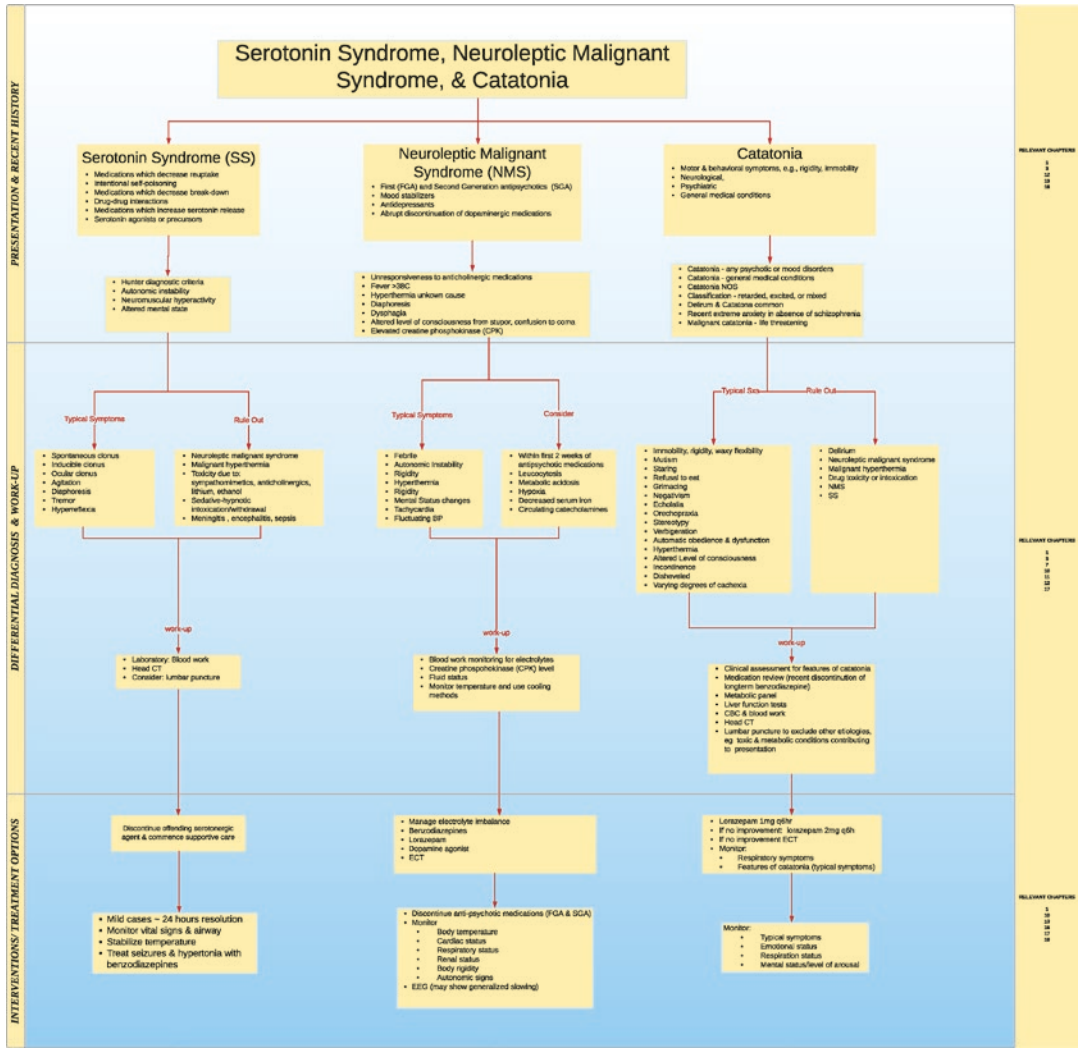


Fig. 15.1 Special syndromes: serotonin syndrome, neuroleptic malignant syndrome, and catatonia

lopram was increased to 20 mg po daily; olanzapine 5 mg daily was added to treat hallucinations.

On the second hospitalization day, his physical status worsened, with increased confusion, mydriasis, tachycardia, hyperreflexia, myoclonus, muscular rigidity, and inability to walk, but there were no focal neurological findings. A diagnosis of SS was offered using Hunter Criteria [1], and all serotonergic agents were discontinued, and he was prescribed lorazepam 1 mg t.i.d. On the third hospitalization day, carbidopa/levodopa (C/L) was discontinued due to the possibility that this medication contributed to his auditory hallucinations. Over hospitalization days number 3–7, the patient became more alert to his environment.

On hospitalization day #8, he developed hyper-rigidity, temperature of 100.58 °F, and CPK rose to 1622 IU/L; and his blood pressure was labile. These characteristic symptoms and signs confirmed the diagnosis of NMS. Olanzapine was discontinued and CPK levels were drawn every 4 hours.

Comment NMS is a potentially lethal condition, described in patients with idiopathic Parkinson

disease (PD), after long-term dopaminergic medications are stopped abruptly or moderately decreased. If patients with PD develop severe rigidity, stupor, and hyperthermia, levodopa withdrawal should be suspected and the dopaminergic drug restarted as soon as possible to prevent rhabdomyolysis and renal failure. The vignette patient was also at a high risk for NMS due to the introduction of olanzapine due to dopamine receptor antagonism. The patient was transferred to ICU, where levodopa was reintroduced, while olanzapine was withdrawn. The increase in temperature resolved the next day; rigidity gradually resolved over the next 10 days, and serum CPK levels began to fall. Escitalopram was reintroduced gradually to treat his depressive symptoms.

The differential diagnoses at admission included:

1. *Delirium associated with PD.* Parkinson disease is a progressive neurodegenerative disorder, with three cardinal features: resting tremor, rigidity, and bradykinesia. It mostly affects the geriatric age group. Of note, delirium has been reported in 5–25% of Parkinson disease patients treated with levodopa. High doses of levodopa can also lead to confusion and psychosis [2].
2. *SS*: SS typically presents with autonomic changes, delirium, and neurological findings of hyperreflexia/clonus, particularly in association with recent medication changes; e.g., introduction or increased doses of Selective Serotonin Reuptake Inhibitors (SSRIs). In the vignette, the diagnosis of SS was worth considering. Clinical presentation in the vignette was dynamic, and therefore, catatonia, SS, nor NMS could not be ruled out initially. Several concurrent factors in the vignette patient's presentation clouded the diagnosis:
   - An acute change from baseline
   - Autonomic changes (i.e., shivering, diaphoresis)
   - Psychiatric symptoms (i.e., anxiety, delirium)
   - Neurological symptoms (i.e., headache, confusion, dizziness, dysarthria, gait disturbances)
   - Gastrointestinal symptom (i.e., diarrhea)
   - History of sub-acute symptoms consistent with a depressive disorder (i.e., complicated grief disorder)
   - Comorbid neurological condition (i.e., Parkinson disease)

The primary psychiatric diagnoses were considered only *after* exclusion of other systemic etiologies. The patient was initially evaluated for delirium, with the delirium protocol (Chap. 12: Delirium). Cerebrovascular events were important diagnoses to rule out, due to the risk factors of neurological symptoms, autonomic symptoms, advanced age, and hypertension. Isolated dizziness and headache symptoms are common clinical contexts for missed cerebrovascular disease. A risk of misdiagnosis is much greater when presenting neurologic complaints are mild, nonspecific, or transient [3]. It was important to consider the possibility of medication adverse effects, such as adverse effect of antibiotics, as an etiological factor for delirium. The polypharmacy and recent medication changes were also risk factors for delirium. Treatment with ciprofloxacin complicated the differential diagnosis of the vignette patient's acute symptomatology. Tandon et al. [4] reviewed 39 studies of fluoroquinolones and found the most commonly reported adverse effects were nausea, vomiting, diarrhea, headache, dizziness, and rash.

In a study by Halkin, 1988, overall rates of adverse reactions associated with fluoroquinolones (ciprofloxacin in the vignette) have been shown to be 4–8%, and adverse reactions necessitated discontinuation of the therapy in 1–2.6% of patients. Patterns of organ system involvement and of signs and symptoms were quite similar, with gastrointestinal effects predominating (nausea, vomiting, diarrhea, or abdominal pain in 1–5% of the patients), followed by effects on the central nervous system (dizziness, headache, and/or insomnia in 0.1–0.3% of the patients), and skin (0.5–2.2% of the patients). Elevation in levels of hepatic enzymes occurred in 1.8–2.5% of the patients, azotemia in 0.2–1.3%, and eosinophilia in 0.2–2.0%. These adverse effects were reversible after drug withdrawal and were

generally not dose-dependent [5]. More recently, Tandan et al. [4] have confirmed many of the central nervous system (CNS) and gastrointestinal adverse events associated with fluoroquinolones, including nausea, vomiting, diarrhea, headache, dizziness, and [4].

15.3 Serotonin Syndrome (SS)

15.3.1 Introduction

SS is an adverse medication effect, characterized by excess serotonergic agonism in the central and peripheral nervous systems. It can result from therapeutic drug use, intentional self-poisoning, or drug-drug interactions. It is a well-known and predictable drug reaction; hence, it is also often preventable and usually treatable. Its early recognition can be challenging because the syndrome includes a spectrum of clinical symptoms ranging from mild to severe and life-threatening.

SS is not an idiosyncratic drug reaction or allergy; it is rather a predictable and direct response to increased concentration levels of serotonergic activity in the body. SS has been described classically as a triad of distinct clinical symptoms – *autonomic instability, neuromuscular hyperactivity, and altered mental status/delirium*. In its early stages, a patient seldom presents with all three of the triad of symptoms simultaneously, and the condition can be missed or misdiagnosed. Frequently, the symptoms are mild and self-limiting; however, in severe cases, and if the syndrome is not identified early in its course, progression can lead to organ failure and death [6]. Figure 15.2 provides a summary of the clinical triad of symptoms associated with SS.

15.3.2 Epidemiology

Approximately 15% of patients who overdose on SSRIs develop SS [7]. But SS often goes unrecognized and unreported, so the incidence of SS is difficult to establish. Almost 85% of family physicians reported that they were not aware of SS as a clinical entity [8]. Some of the clinical symptoms associated with SS, such as diarrhea, fever,

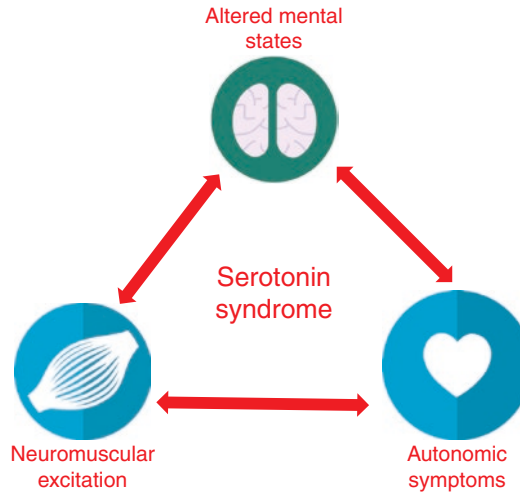


Fig. 15.2 Serotonin syndrome symptoms

and tremors, may be dismissed by patients and clinicians as unrelated to medication use, or misdiagnosed as another condition, such as an infection or another psychiatric disorder [6].

SS has been recognized in all age groups, including children and newborns. However, the aged have increased vulnerability to SS, due to changes in drug metabolism which occur with aging (e.g., a decline in liver volume), susceptibility for increased polymorphism in P450 cytochromes, and loss of inducibility of metabolizing enzymes. A decline in drug clearance in heart and kidney disease may lead to a hyper-serotonergic state with common medications such as SSRI and serotonin and norepinephrine reuptake inhibitors (SNRIs) [9] (Chap. 3: Pharmacological Overview). Geriatric patients are also at a higher risk for polypharmacy that can lead to complex drug interactions that result in SS. Diet influences drug metabolism, and a high prevalence of protein-calorie malnutrition in sick hospitalized geriatric patients may add a risk for the development of SS. Trauma and ill health can also have substantial effects on enzymes of drug metabolism in geriatric patients [9].

15.3.3 Etiology

Serotonin (5-hydroxytryptamine, 5-HT) is a neurotransmitter concentrated within neurons located in the raphe nuclei. Serotonin neurons

play a part in sleep-wakefulness cycles, mood, emotional and food-seeking behaviors, and thermoregulation. All drugs that directly or indirectly increase central serotonin neurotransmission at postsynaptic receptors 5-hydroxytryptamine 1A (5-HT1A) and 5-hydroxytryptamine 2A (5-HT2A) can produce SS.

Many psychotropic and nonpsychotropic drugs modulate serotonin receptors. SS is associated with excessive levels of serotonin, which can be altered by dosage timing, dosage frequency, drug interactions, drug overdoses – accidental and intentional – and inadequate washout periods between medication changes [10]. This phenomenon has implications for medication revision strategies such as drug discontinuation, tapering, and switching (Chap. 17: Medication Strategies).

Wang et al. [10] described five mechanisms by which commonly prescribed medications can alter serotonin levels and cause SS:

- *Decreased serotonin breakdown*: Medication classes include monoamine oxidase inhibitors, antibiotics including linezolid and tedizolid and methylene blue, procarbazine, and Syrian rue may decrease serotonin breakdown, and thereby, increased serotonin availability.
- *Decreased serotonin reuptake:*
  - SSRIs: fluoxetine, fluvoxamine, paroxetine, citalopram, escitalopram, sertraline
  - SNRIs: venlafaxine, duloxetine, milnacipran
  - Tricyclic antidepressants (TCAs): clomipramine, imipramine, amitriptyline
  - The herbal remedy, St. John's wort
  - Opioids: meperidine, buprenorphine, tramadol [11], tapentadol, dextromethorphan
  - Antiepileptics: valproate, carbamazepine
  - Antiemetics: ondansetron, granisetron, metoclopramide
- *Increased serotonin precursors or agonists*: Agents include tryptophan, lithium, fentanyl, and lysergic acid diethylamide (LSD).
- *Increased serotonin release within the central nervous system*: amphetamines, anorectics (e.g., fenfluramine), dexfenfluramine and phentermine, cocaine and methylenedioxymethamphetamine (ecstasy), as drugs of abuse.
- *Drug-drug interactions*: Medications that inhibit drug metabolism can cause a higher serum concentration of serotonin. Specifically, inhibitors of CYP2D6 and CYP3A4, including the antibiotics erythromycin and ciprofloxacin, the antifungal fluconazole, and the antiretroviral ritonavir.

Table 15.1 lists pharmacological mechanisms which can lead to increased central serotonin transmission [10].

15.3.4 Clinical Description

As noted above, the SS is classically composed of a triad of symptoms: *neuromuscular excitation, altered mental status, and autonomic dysfunction.* Many patients will not exhibit the full clinical triad, and symptoms may not occur simultaneously; altered mental status and autonomic dysfunction are present in roughly 40% of the cases and neuromuscular excitation in 50% [12]. SS may also present in mild, moderate, and severe forms, with most cases mild and self-limiting [13]. The mild form can present with sub-acute symptoms, whereas the most severe SS is life-threatening and, within hours of onset, can rapidly progress to organ failure. Patients typically come

Table 15.1 Pharmacological mechanisms leading to increased central serotonin transmission

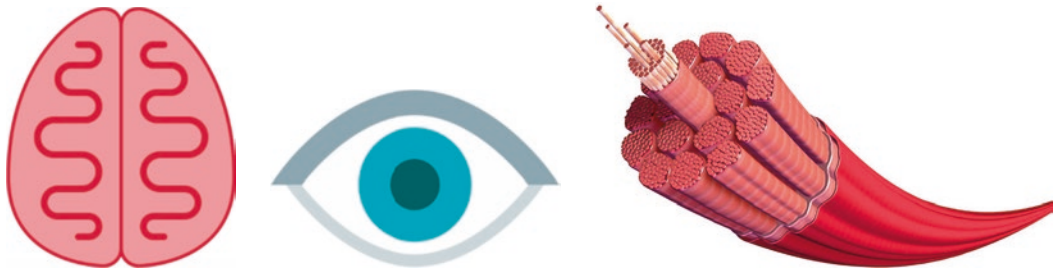
- Augmentation of serotonin synthesis
- Increased serotonin release
- Inhibition of serotonin uptake
- Inhibition of serotonin metabolism leads to direct stimulation of postsynaptic serotonin receptors

to attention within 24 hours of overdose, dose adjustment, and/or drug initiation [14]. The spectrum of presentation includes aspects within the triad of symptoms (neuromuscular excitation, altered mental status, and autonomic dysfunction) but differs in severity. Figure 15.3 summarizes the differential diagnosis of SS.

Mild SS starts with neuromuscular excitation, seen as hyperreflexia, tremor, and myoclonus, altered mental status in the form of anxiety, restlessness, and insomnia, and is associated with autonomic dysfunction as diaphoresis, mydriasis, and tachycardia. The symptoms of toxicity with serotonergic agents arise within 1 hour of a precipitating event in approximately 30% of patients and within 6 hours in 60% [14].

Moderate symptoms of SS include *opsoclonus* (uncontrolled eye movements which are rapid, involuntary, unpredictable, conjugate, and fast), spontaneous or inducible clonus, neuromuscular excitation, agitation, hypertension, hyperthermia (< 40 °C, < 104 °F), hyperactive bowel sounds, and diarrhea, nausea, and vomiting.

Severe cases of SS include neuromuscular excitation in the form of rigidity, respiratory failure, tonic-clonic seizures, altered mentation (manifested as coma or delirium), autonomic dysfunction with severe hyperthermia (> 40 °C, > 104 °F), and fluctuations in blood pressure (Chapter: Delirium). If rigidity is not treated properly, the resulting hyperthermia can lead to cell damage and rhabdomyolysis, myoglobinuria, renal failure, metabolic acidosis, acute respiratory distress syndrome, disseminated intravascular coagulation, and death [15]. A distinct neurological finding with SS is that spontaneous/induced clonus and hyperreflexia are both more pronounced in the lower extremities than in the upper extremities [1]. Table 15.2 summarizes the SS spectrum of severity.



Mental state changes	Autonomic instability	Neuromuscular abnormalitis
• Agitation • Pressured speech • Confusion	• Tachycardia • Shivering • Mydriasis • Diaphoresis • Diarrhea	• Hyper-reflexia (lower extremities > upper) • Clonus • Tremor • Mydriasis • Seizure

Risk factors:
- history of exposure to a serotonergic drug

Presence of one or more of the following:
- spontaneous clonus
- inducible clonus with agitation and diaphoresis
- ocular clonus with agitation and diaphoresis
- tremor and hyperreflexia
- hypertonia
- temperature over 38 °C / 100.4 F with ocular or inducible clonus

Fig. 15.3 Hunter serotonin toxicity criteria

Table 15.2 SS severity spectrum

Serotonin Syndrome	*Mild*	*Moderate*	*Severe*
Neuromuscular excitation	Hyperreflexia Tremor Myoclonus	Opsoclonus Spontaneous/inducible clonus (pronounced in lower limbs)	Rigidity Respiratory Failure Tonic-clonic Seizures
Altered mental status	Anxiety Restlessness Insomnia	Agitation	Delirium Confusion Coma
Autonomic dysfunction	Diaphoresis Mydriasis Tachycardia	Hypertension Hyperthermia (< 40C or < 104F) Hyperactive bowel sounds Diarrhea Nausea Vomiting	Severe Hyperthermia (> 40C or > 104F) Fluctuating BP

15.3.5 Diagnostic Evaluation

Serotonin syndrome more commonly presents on a continuum rather than in clear-cut clinical stages, and the diagnosis can be difficult to recognize, easily missed, or misdiagnosed. The Hunter diagnostic criteria, based predominantly on physical signs and observable symptoms, were developed to use clear markers to identify SS. Using a decision tree algorithm, the Hunter criteria include spontaneous clonus, inducible clonus, ocular clonus, agitation, diaphoresis, tremor, and hyperreflexia in the context of serotonin exposure [16], which determine the likelihood of SS. Figure 15.3 summarizes the key features of the Hunter serotonin toxicity criteria. In the case vignette, the patient's medications, escitalopram, olanzapine, and levodopa were discontinued, and he was given lorazepam 1 mg t.i.d.

The clinical symptoms in the vignette likely occurred following the introduction of the antidepressant escitalopram, which began without an adequate washout period from fluoxetine. Hence, the initial presentation may have in fact been SS – mild and most likely missed and misdiagnosed as major depressive disorder (MDD) and anxiety. Increase in escitalopram, in combination with olanzapine, which also has serotoninergic activity, likely worsened SS (Chap. 17: Medication Interventions).

15.3.6 Differential Diagnosis

The differential diagnosis of SS includes, among other conditions, NMS; malignant hyperthermia; toxic levels of sympathomimetics, anticholinergics, lithium, and ethanol; sedative-hypnotic withdrawal; meningitis; intracranial bleeding; or encephalitis [16] (Chap. 10: Alcohol and Substance intoxication and withdrawal).

In an aging patient, the anticholinergic burden of the medication regimen and possible toxicity must always be considered due to the pervasive use of medications with anticholinergic properties. The anticholinergic syndrome typically presents with hyperthermia, agitation, confusion, mydriasis, dry mucous membranes, urinary retention, constipation, and decreased bowel sounds. As opposed to those in the SS, muscular tone and reflexes are normal in anticholinergic toxicity.

Workup should include a routine metabolic panel, liver function tests, complete blood count (CBC), a head CT, and, in some cases, a lumbar puncture to exclude other etiologies like toxic and metabolic conditions attributing to the presentation [16]. Figure 15.4 summarizes the differential diagnosis for SS.

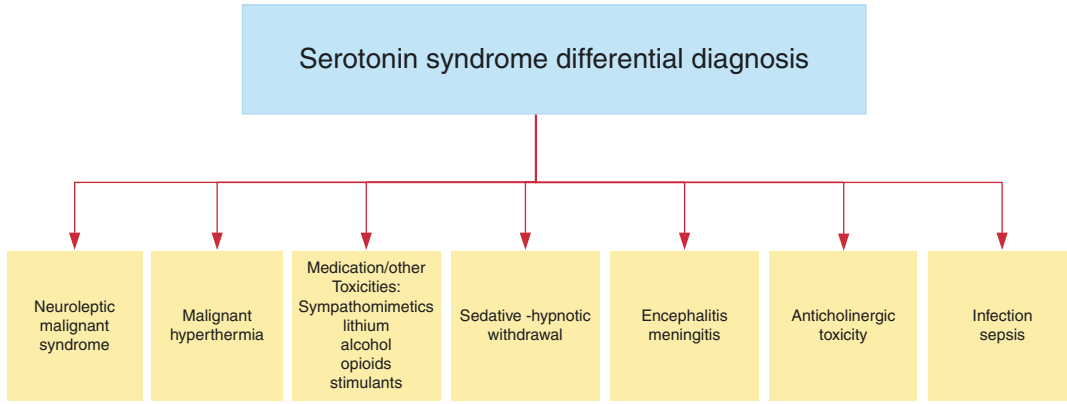


Fig. 15.4 SS differential diagnosis

15.3.7 Treatment

The first steps for management of SS should include discontinuation of all serotonergic agents and provision of supportive care; mild forms of SS can resolve within the first 24 hours. Special attention should be paid to vital signs and airway to dictate further management. Hyperthermia should be managed with cooling measures and the physician should avoid the use of antipyretics, as SS temperature instability is not centrally mediated, but is rather secondary to increased muscle tone. Increased muscle tone leading to myoclonus, seizures, and hypertonia should be managed by benzodiazepines. Consider intubation and muscle paralysis with severe degrees of muscle rigidity.

Mason et al. [14] reviewed the role of antiserotonergic agents like cyproheptadine for the reversal of SS in human case reports. Cases demonstrated resolution occurring within the first 1 hour. Treatment is, however, limited to those who are able to swallow, but this agent may also be administered by nasogastric tube. The initial cyproheptadine dose of 4–8 mg can be titrated up to a maximum of 20 mg daily dictated by clinical improvement. Further human trials to determine the efficacy of this medication are needed to establish guidelines.

15.4 Neuroleptic Malignant Syndrome (NMS)

15.4.1 Introduction

NMS is an idiosyncratic drug reaction, of uncertain etiology, wherein patients become hyperthermic, display signs of autonomic instability, rigidity, and delirium. It can be fatal if not recognized and treated promptly [15].

Delay and Deniker [17] recognized NMS for the first time, and described it as a syndrome of extrapyramidal symptoms with four principal symptoms: hyperthermia, rigidity, mental status changes, and autonomic dysfunction. Another description of the distinct clinical features of NMS includes rigidity unresponsive to anticholinergic medications, hyperthermia of *unknown* cause, diaphoresis, dysphagia, changes in level of consciousness ranging from confusion to coma, and elevated CPK levels [18].

15.4.2 Epidemiology

The overall incidence of NMS ranges between 0.02% and 3.23% [18]. NMS is less common in older adults for unknown reasons. Men are approximately 50% more likely than women to be

Table 15.3 First-generation antipsychotics (FGAs) and second-generation antipsychotics (SGAs) associated with NMS

Antipsychotics	
First Generation	Second Generation
• Flupentixol	• Clozapine
• Haloperidol	• Olanzapine
• Fluphenazine	• Risperidone
• Thioridazine	• Quetiapine
• Chlorpromazine	• Aripiprazole
• Trifluoperazine	• Paliperidone
• Loxapine	• Asenapine
• Periciazine	• Ziprasidone
• Methotrimeprazine	
• Prochlorperazine	
• Zuclopenthixol	

diagnosed at any age [19]. Antipsychotic use has been the most common etiological factor; other implicated drug classes include mood stabilizers (e.g., lithium, carbamazepine), antidepressants (e.g., paroxetine, sertraline, amitriptyline), and antiemetic agents (e.g., metoclopramide) [20].

The proposed mechanism for SSRI/TCA leading to NMS is based on the pharmacodynamics of SSRIs, in which an excess of serotonin inhibits dopamine release, worsening the hypodopaminergic state [21]. The TCA mechanism with clomipramine primarily works on serotonin, while amitriptyline and imipramine are norepinephrine−/serotonin-acting agents, which would cause a hypodopaminergic state similar to SSRIs.

NMS has also been associated with use of the SGAs as well as FGAs. The rate of NMS resulting from SGA administration is similar to the rate resulting from administration of FGAs [20]. The risk is not well known; however, it has been cited as 0.02–2.44% [22]. Table 15.3 lists First Generation and Second Generation antipsychotics commonly associated with NMS.

15.4.3 NMS Precipitating Factors

Careful monitoring is necessary to recognize NMS symptoms promptly and initiate early intervention (Chap. 3: Pharmacological Overview). Situations which have the potential to precipitate NMS include:

1. The use of more than one antipsychotic at a time. FGA have been associated with NMS in approximately 68% of NMS cases, and almost 72% of these cases were co-medicated with an SGA. Median length of exposure to an SGA prior to the onset of NMS was 23 days, while the median length of exposure to a FGA before onset was 6 days. Mortality rate was 11% for SGA-induced NMS and 12% for FGA-induced NMS [20].
2. Age: Independent predictor of mortality; each 10-year increase in age is associated with a 40% increased odds of mortality [23]. Antipsychotic treatment is hazardous in the aged population even with SGAs.
3. Major neurocognitive disorders, especially Lewy body type, are at risk of antipsychotic hypersensitivity when exposed to antipsychotics. Extrapyramdal symptoms and NMS have been attributed to a depletion of nigrostriatal dopaminergic neurons and acetylcholinergic receptors [24], although other explanations have been offered. NMS has developed in patients with major neurocognitive disorder with Lewy bodies, even when treated with SGAs [25–28].
4. Abrupt cessation or reduction in the dose of dopaminergic medications, such as levodopa in Parkinson disease, may precipitate NMS. A rapid switch from one type of dopamine receptor agonist to another has also been associated with NMS. An abrupt withdrawal of medications to treat Parkinson disease, which do not possess *direct* dopaminergic activity, such as amantadine (a tricyclic amine) and tolcapone (a catechol-O-methyltransferase – COMT) inhibitor [29], can result in NMS. A common complication of NMS has been rhabdomyolysis (30.1%), and of those patients, 30% developed acute kidney injury, out of which 5.9% underwent hemodialysis. Other complications included respiratory failure requiring ventilator support, pneumonia, and sepsis.

The current overall reported mortality rate for NMS is between 4% and 30%, with a 50% mortality rate for cases in which renal failure develops [30]. There is a slight increase of NMS reported cases in summer seasons, due to higher likelihood of increased heat and dehydration [30]. Depot antipsychotic-induced NMS lasts twice as long as NMS induced by oral medications [30]. Table 15.4 shows a summary of common inciting and predisposing factors associated with NMS.

15.4.4 Etiology of NMS

The etiology of NMS has been explained as an adverse effect of antipsychotic drugs which function as dopamine (D2) receptor blockers. Approximately 0.5–1.0% of those treated with FGA and SGA will develop NMS within the first 2 weeks, during which antipsychotics are titrated to their therapeutic dose [15]. Patients taking carbamazepine, valproic acid, or those who have undergone rapid discontinuation of levodopa, amantadine, and benzodiazepines are also at risk [31–33].

Early in its recognition, NMS was viewed as an example of malignant catatonia, secondary to antipsychotic use. Similarly, dantrolene and dopamine agonists were proposed as treatment because the symptoms of NMS were so similar to those of malignant hyperthermia [34]. Consensus recommendations advised treatment with benzodiazepines and electroconvulsive therapy (ECT) if treatment had failed with benzodiazepines [33, 35–38].

The original hypothesis that malignant hyperthermia and NMS each had a different etiology has become less prominent. The finding that NMS can be provoked by medications *other than* antipsychotics supports the notion that this syndrome was not due solely to antipsychotics and/or dopamine blockade. Other instigating medications include disulfram, corticosteroids, phencyclidine, abrupt withdrawal of anticholinergic and antihistamine drugs, and phenelzine with lithium or dothiepin [15].

15.4.5 Clinical Description

NMS remains an unpredictable and potentially life-threatening neurologic condition that requires early identification and proper medical management to ensure improved patient outcomes. As mentioned, it most often presents within the first 2 weeks of antipsychotic initiation during drug titration to therapeutic doses. Patients typically present with hyperthermia >100.4 °F, autonomic instability, rigidity, mood alterations, and delirium. The DSM-5 identifies defining characteristics of NMS to include "lead pipe rigidity," most often resistant to antiparkinsonian agents, and other symptoms summarized in Table 15.5.

Table 15.4 Inciting or predisposing factors in neuro malignant syndrome

N	• Dehydration
	• Malnutrition
	• Exhaustion
M	• Intramuscular injection of antipsychotics
	• Advanced age
	• Neuropsychiatric disorders
	• Traumatic brain injuriy
	• Antipsychotic dose increases
	• Neurodegenerative brain disease (e.g., MNCD)
S	• Infections
	• Ethanol (EtOH) intoxication
	• HIV infection
	• Concomitant use of lithium, anticholinergic agents, and some antidepressant agents

Table 15.5 DSM-5: NMS and signs

• Rigidity • Tremor • Sialorrhea • Akinesia • Dystonia • Trismus • Myoclonus • Dysarthria • Dysphagia • Rhabdomyolysis • Extremely high temperatures • Creatine kinase levels 4x higher than upper limit of normal • Significant impairment in mentation • Altered levels of consciousness from delirium to coma.		• Dazed apperance - may be mistaken for a catatonic like picture • Autonomic dysregulation - tachycardia > 25% baseline • Diaphoresis • Fluctuations in blood pressure - > 20mm Hg diastolic or > 25mm Hg systolic • Urinary incontinence • Pallor • Respiratory distress - tachypnea > 50% above baseline • Metabolic acidosis • Hyper-metabolism • Restricted chest wall expansion, • Aspiration pneumonia • Pulmonary emboli - may lead to respiratory arrest

15.4.6 Differential Diagnosis of NMS

A workup for NMS needs to exclude infectious, metabolic, substance-related, or other neuropsychiatric conditions, but no specific laboratory test is yet specific to NMS. Laboratory results will show leukocytosis, metabolic acidosis, hypoxia, decreased serum iron, and an elevation in creatine phosphokinase (CPK) and circulating catecholamines. EEG demonstrates a generalized slowing, while cerebrospinal fluid (CSF) and neuroimaging are nonspecific.

15.4.7 Treatment

Mild cases of NMS should be treated conservatively with fluid replacement, correction of electrolyte imbalance, and normalization of body temperature with active cooling methods. Cardiac, respiratory, and renal status should be carefully monitored [18, 39]. A trial with lorazepam 0.5–1.0 mg IM q4–6 hours has been demonstrated to be effective as a primary approach by Woodbury and Woodbury [40]. Severe cases of NMS may require the use of a dopamine agonist to correct the depleted dopaminergic state of the body. Velamoor [41] considers dopaminergic agents when temperatures fall between 38.3° and 40 °C (100.9 °F and 104 °F). Bromocriptine 2.5 mg or amandine 100 mg every 8 hours continued for 10 days with a gradual taper [18]. If temperatures exceed 40 °C, IV dantrolone 2–3 mg/kg body weight may be warranted to decrease the muscle spasticity and rigidity [41]. If ineffective, treatment with ECT is recommended [33, 35–38], especially in cases where delirium, rigidity, and catatonia have not resolved [39].

15.5 Catatonia

15.5.1 Introduction

Catatonia is a complex neuropsychiatric condition characterized by particular motor and behavioral signs and symptoms that can manifest as a consequence of many neurologic, psychiatric, and/or general medical conditions, which was first described in 1874 [42]. For decades, catatonia was thought to be a subtype of schizophrenia and appeared this way in the early versions of the DSM [43]. Because of its complexity and variation in presentation, the DSM-5 has since modified the phenomenological approach to define catatonia with specifiers. These include: catatonia as a result of all psychotic, depressive, and bipolar disorders, as a result of non-CNS general medical conditions, or as a syndrome *not otherwise specified*. It is characterized by psychomotor, autonomic, and behavioral abnormalities [44], occurring in general medical, neurological, and psychiatric conditions as well as due to medications and illicit substances.

15.5.2 Epidemiology of Catatonia

Psychiatrists and other physicians tend to underrecognize catatonia [45] despite the reported high prevalence rate in some studies. Prevalence varies between 5% and 50.8% in acute psychiatric admissions, based on the diagnostic method used [33]. The prevalence of catatonia is estimated to be 5–18% in inpatient psychiatric units, 12% in drug-naïve patients with first episode psychosis, 3.3% in a neurology/neuropsychiatric tertiary care inpatient

unit, 3.8% in the intensive care unit, 1.6–1.8% in psychiatry consultation-liaison services, and 8.9% in geriatric patients referred for psychiatric consultation [46].

Catatonia has been commonly associated with depressive and bipolar disorders, particularly manic episodes [42, 47, 48]. The prevalence of catatonia due to a general medical condition may also vary from 20% to 39% [49]. Catatonia in older patients frequently appears in association with a systemic medical condition rather than secondary to a primary psychiatric presentation [50]. Delirium and catatonia can often coexist at the same time in a patient, this of course makes the diagnosis of catatonia difficult to recognize as a syndrome separate from delirium. Catatonia was present in at least 12% of patients with delirium [46] (Chap. 12: Delirium). For example, delirium and catatonic features are indistinct and often difficult to delineate [51]. In the geriatric population, superimposed cognitive and somatic conditions (i.e., emotional dysregulation caused by a physical symptom) may complicate the symptom presentation [52]. Immobility associated with major neurocognitive disorder has been postulated as a catatonic state, and some authors suggest that this may respond to lorazepam [53].

15.5.3 Etiology

No consensus exists about the specific pathophysiological mechanism underlying catatonia. Often patients have retrospectively reported feeling extreme levels of anxiety immediately prior to a catatonic episode. Because of these consistent reports, one theory of the neuropathology is that catatonia is the result of intense anxiety that reflects a functional GABA deficit [54]. This supports its response to benzodiazepines by acting on the GABA-A receptor ion channels, the mechanism thought to be responsible for the treatment of catatonia. However, patients with schizophrenia who have had a catatonic episode do not endorse the same heightened levels of anxiety as those with depressive or bipolar disorders report [43].

Another pathophysiological theory is that catatonia is a movement disorder. Rasmussen et al. [43] report the overlapping nature of catatonia as parkinsonism, a dysfunction within the basal ganglia. In support, functional imaging has demonstrated altered activity in the orbitofrontal, prefrontal, parietal, and motor cortical regions in catatonia [55]. This author reinforces that GABA-A binding is also reduced in catatonic patients, motor and mood symptoms coincide with GABA-A binding abnormalities, and cortical abnormalities in catatonic patients resolve with lorazepam.

15.5.4 Clinical Description of Catatonia

Catatonia is subtyped into three clinical forms: retarded, excited, or mixed. Table 15.6 summarizes the presentations of catatonia, with malignant catatonia requiring urgent assistance [56].

Immobility and mutism are the most commonly identified signs of catatonia, observed in 90.6% and 84.4% of catatonic patients, respectively [57]. Rasmussen et al. [43] report incontinence, disheveled appearance, and varying degrees of cachexia as other common symptoms. In another report, the most prevalent catatonic signs were excitement (64.3%), verbigeration (61.9%), negativism (59.5%), immobility/stupor (57.1%), and staring (52.4%) [42].

15.5.5 Treatment

Benzodiazepines remain the gold standard treatment for catatonia. The intravenous route is preferred because of quick onset of action, length of action, and ease of administration. Initial dose should be 1 mg of lorazepam IV q6h. If no improvement, lorazepam should be increased to 2 mg q6h. Maintenance doses should total 6–8 mg daily with divided doses occurring every 6–8 hours for 2–3 days, which should be followed by oral treatment and tapered before discontinuing to prevent relapse into catatonia. During this

Table 15.6 Various presentations of catatonia

CATATONIA

Type	Presenting symptoms
Retarded	Presents with: • immobility • mutism • staring • rigidity • refusal to eat • grimacing • negativism • waxy flexibility • echolalia or echopraxia • stereotypy • verbigeration • automatic obedience
Excited	• less frequent • periods of psychomotor agitation, life threatning with hyperthermia • altered level of consciousness • autonomic dysfunction
Mixed	• heterogenous presentation • patients may demonstrate symptoms of retarded and excited form
Malignant	• highly lethal form - demands early recognition (Mann 1986)

time, workup for ECT should be completed (Chap. 16: Neuromodulation Interventions).

If catatonic symptoms have not improved within 48–72 hours, or if symptoms of malignant catatonia emerge, treatment with ECT is recommended (Chap. 13: Involuntary Interventions; Chap. 16: Neuromodulation interventions). Resolution may occur within 1–2 treatments but may require 10–20 sessions.

If ECT is not available at the treatment location, a glutamate antagonist, amantadine, should be administered in a dose of 100 mg daily or memantine 10 mg daily titrated to 600 mg and 20 mg daily, respectively. If failure occurs, antiepileptic medications like carbamazepine and valproic acid should be administered 300–600 mg po daily or valproic acid 500–1500 mg po or IV daily. If an antiepileptic drug fails, treatment with an SGA in conjunction with lorazepam should be trialed. Aripiprazole 10–30 mg, olanzapine 2.5–10 mg, and clozapine 200–300 mg daily are the preferred among available SGAs [58].

15.6 Summary

SS, NMS, and catatonia are three syndromes that have several overlapping features. If untreated or unrecognized, each of these presentations can be life-threatening, especially in the geriatric population. Each syndrome has its own unique guidelines for treatment. The psychiatric inpatient setting affords the necessary resources to monitor and evaluate, in order to recognize distinguishing features of each syndrome, make an early diagnosis, and choose the specific treatment. A careful and timely approach will help to manage these patients and limit the progression of these life-threatening conditions. Table 15.7 summarizes the distinctions between SS, NMS, and catatonia, including key features of the Hunter serotonin toxicity criteria.

Table 15.7 Summary of SS, NMS, and catatonia

Syndromes	Serotonin Syndrome (SS)	Neuroleptic Malignant Syndrome (NMS)	Catatonia
Etiology	• Hyper-serotonergic state • Clinical symptoms related to increase in serotonin levels • Predictable adverse effect to medications • SSRIs the most common medications as a common causative agent	• Idiosyncratic reaction to medications	• Many possible etiologies • Neurological conditions, such as encephalitis, stroke • Psychiatric conditions such as schizophrenia, bipolar, major depressive disorders
Clinical Symptoms	• Triad of clinical symptoms - autonomic symptoms, altered mental symptoms, and neuromuscular excitation	• Hyperthermia- Temp >38 c • Extrapyramidal symptoms - tremors, parkinsonism • Altered mental state • Hypermetabolic state • Autonomic disturbance - Labile Heart Rate • Labile blood pressure • Tachycardia,Tachypnea	• Three out of 12 clinical symptoms, catalepsy, mutism stupor, agitation, stereotypy, negativism, waxy
Prodromal Symptoms	• Commonly have gastrointestinal symptoms, such as, nausea, vomiting and diarrhea	• Some patients have insidious alteration in mental status and other neurological signs in days prior to the clinical presentation	• EARLY abnormal motor signs: retardation, excitement, or mixed
Distinguishing Features	• Spontaneous/induced clonus and hyperreflexia - generally more pronounced in the lower limbs	• Hyperthermia • Lead pipe rigidity • Increased CK • Hyper-metabolic state	• Unusual motor symptoms like posturing are more typical of catatonia • Intense and uncontrollable emotions, behavior abnormalities such as stereotypy, negativism and automatic disobedience
Onset	• Typically within 24 hours of initiation of new medications or medication change typically SSRI	• Typically within 2 weeks of initiating medications or change in medications typically antipsychotics • Rarely, fulminant course, onset and peak of the illness course within 24 hours of initiating medications	• Typically acute and sudden
Screening scales/ criteria	• Hunters/ Strenbach criteria	• Francis-Yacoub NMS Rating Scale • Woodbury Staging Method for NMS	• Bräunig-Catatonia Rating Scale • Bush-Francis Catatonia Screening Instrument • Bush-Francis Catatonia Rating Scale • Rogers Scale • Northroff Scale • Catatonia Rating Scale
Treatment	• Withdraw all serotonergic agents • Supportive therapy • Benzodiazepines for myoclonus, seizures, hypertonia	• Withdraw all antipsychotic medications • Supportive therapy • Benzodiazepines • Dopaminergic agonists	• ECT and benzodiazepines • Treat underlying disorders - psychiatric, medical, neurological

Take-Away

- First: rule out acute syndromes, common in the geriatric population, such as delirium, substance intoxication, and anticholinergic toxicity.
- Review medication history and risk factors for SS, NMS, or catatonia.
- Attend carefully to clinical symptoms and their progression.
- Delineate history, symptoms, signs, which can differentiate SS, NMS, and catatonia.
- Institute treatment *specific* to the working diagnosis of SS, NMS, or catatonia.
- In the geriatric population, intervene early to minimize progression of SS, NMS, or catatonia.
- Continue to consider alternative diagnoses and adjust treatment accordingly.

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16 Neuromodulation Interventions: ECT and rTMS – Work-Up, Preparation, and Posttreatment Care + Ketamine in Inpatient Psychiatry

Lisa A. McMurray and Barbara Deren

16.1 Introduction

Geriatric patients with mood disorders, suicidality, aggression, or resulting physical deterioration often need urgent intervention. They may be unable to tolerate adequate pharmacotherapy due to age-related pharmacokinetic and pharmacodynamic changes, as well as the presence of medical comorbidities (Chap. 3: Pharmacological Overview). Furthermore, their fragile physiological homeostasis presents a risk of physical deterioration as a result of prolonged episodes of mental illness.

Many patients admitted to a geriatric psychiatry inpatient unit will have major depressive disorder [1]. Other common diagnoses in this setting include bipolar disorder, psychotic disorder, and major neurocognitive disorder with behavioral disturbance. A substantial proportion of patients with late-life depression (30–50%) will be resistant to pharmacotherapy [2, 3]. ECT can be helpful for all of these diagnostic indications.

In the context of clinical acuity, frailty, intolerance of pharmacotherapy, and treatment resistance, electroconvulsive therapy (ECT) is an essential treatment modality, often administered on a geriatric psychiatry inpatient unit. It can be lifesaving, with its potential to dramatically and rapidly reduce symptoms and improve quality of life. ECT is a well-established treatment, with superior efficacy for major depressive disorder. Furthermore, aging adults respond preferentially to this treatment [4]. ECT acts faster than pharmacotherapy to relieve depressive symptoms, sometimes resulting in substantial clinical improvement even after the first treatment. In addition to its efficacy for major depression, ECT is also useful for treatment-resistant mania and as an adjunctive treatment for psychotic disorders. There is also emerging evidence for its efficacy in treating behavioral disturbances in major neurocognitive disorders. Figure 16.1 offers a recommended process of assessment for ECT, rTMS, and other treatments.

While ECT is the mainstay of somatic therapies in geriatric psychiatry, alternatives are emerging. Repetitive transcranial magnetic stimulation (rTMS), in which the brain of a conscious patient is stimulated by a magnetic coil applied to the scalp, is a brain stimulation treatment with some evidence for efficacy in older adults [5]. It may initially seem more acceptable to patients than ECT, but the efficacy of rTMS is markedly lower [6], with treatment courses taking 4–6 weeks. Moreover, it is not available in many treatment sites and therefore has limited utility in

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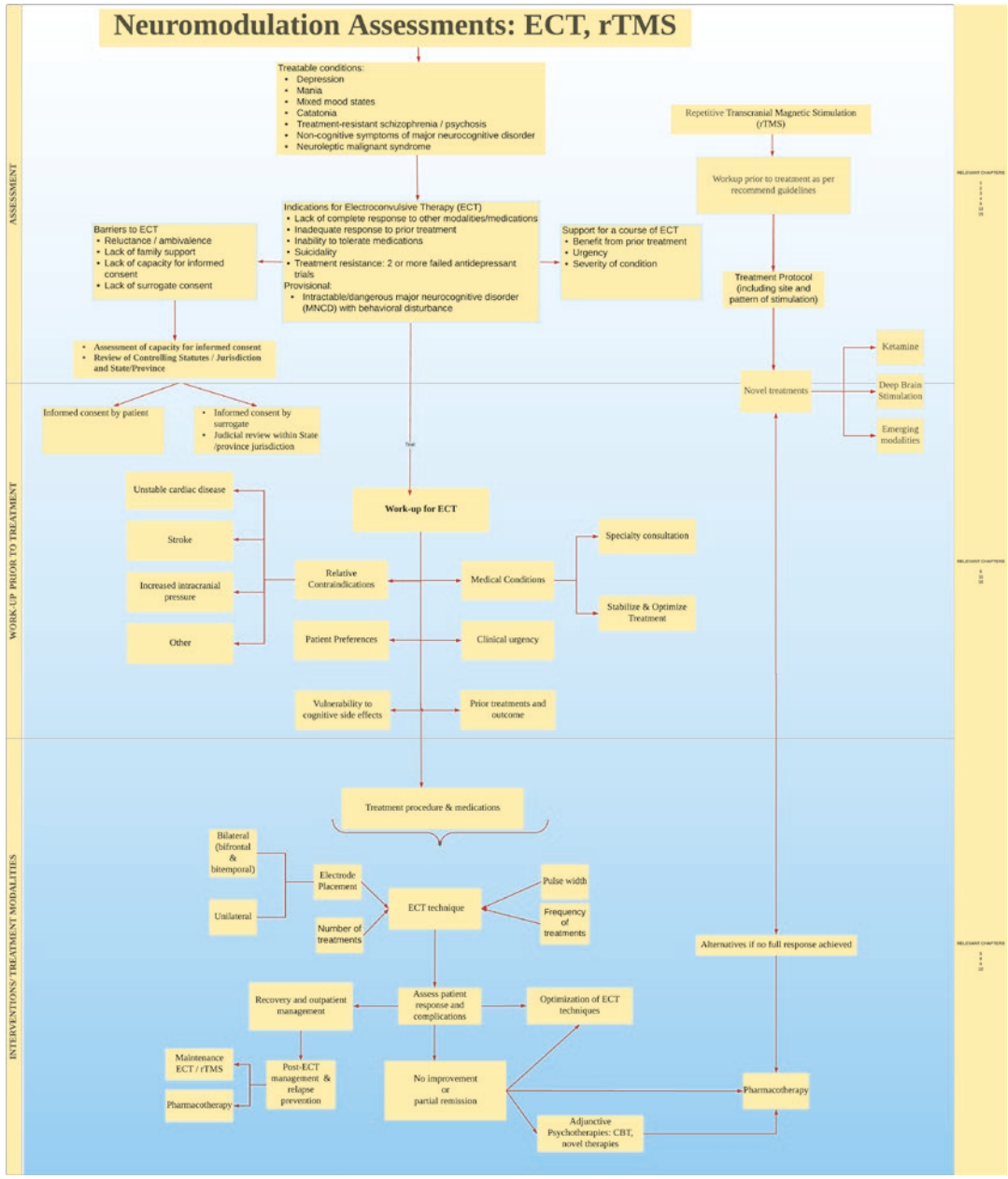


Fig. 16.1 Flowchart of patient assessment for ECT, rTMS, and other treatments

the geriatric psychiatry inpatient setting. Future treatment protocols may improve response and remission rates and may ultimately make this intervention more useful. Similarly, IV infusion of ketamine, another emerging treatment for reduction of suicidal ideation and treatment of depression [7], has very limited evidence for efficacy and safety in geriatric patients. Ketamine is currently an investigational treatment in the general adult population, and its role in the geriatric psychiatry inpatient unit warrants further research.

16.2 Vignette 1: Depression with Melancholic Features

An 82-year-old married woman presented to geriatric inpatient psychiatry requesting ECT. She demonstrated psychomotor slowing and debilitating depressive symptoms, including depressed mood with minimal reactivity, anxiety, anhedonia, and significant diurnal variation of mood which was worse in the morning. She reported that she was no longer socializing with friends, not attending church, and not participating in her bird-watching hobby for several months. Her score on the Montreal Cognitive Assessment (MoCA) [8] was 25/30.

The patient had her first episode of depression in her 30s and was treated with multiple medications with limited benefit. She subsequently received four ECT treatments, after which she achieved remission of symptoms and was discharged on maintenance pharmacotherapy with a tricyclic antidepressant. She remained well for 50 years. Three years prior to the current admission, the patient was diagnosed with mild cognitive impairment based on brief cognitive screening, and her driver's license was revoked.

Fluoxetine 20 mg, on which she had been stable for decades, was changed to the norepinephrine-serotonin reuptake inhibitor (SNRI) duloxetine at a dose of 30 mg, in hopes of alleviating her chronic back pain. Six months later, she had relapsed and was referred to psychiatry for assessment and treatment of recurrent major depressive episode. Optimization of duloxetine and subsequent switching to other antidepressants had been ineffective.

Given the history of mild cognitive impairment, right unilateral ultra-brief pulse ECT was recommended to minimize any potential cognitive side effects. Seizure threshold was determined by stimulus dose titration, and subsequent treatments were given at 6 times seizure threshold. A custom bite block was fashioned out of rolled gauze to protect her single remaining natural tooth. She tolerated the treatments well, with no reported cognitive or physical side effects. Posttreatment evaluation indicated remission of depressive symptoms, with scores on the Montgomery-Asberg Depression Rating Scale (MADRS) [9] of 32 pretreatment to 6 posttreatment (Fig. 16.2).

She was discharged on maintenance pharmacotherapy, consisting of nortriptyline 45 mg and lithium carbonate 300 mg, both given at bedtime. Serum levels of nortriptyline were maintained in the therapeutic range, and lithium levels were kept in the range of 0.4–0.6 mmol/L. At discharge, she reported feeling that ECT had given

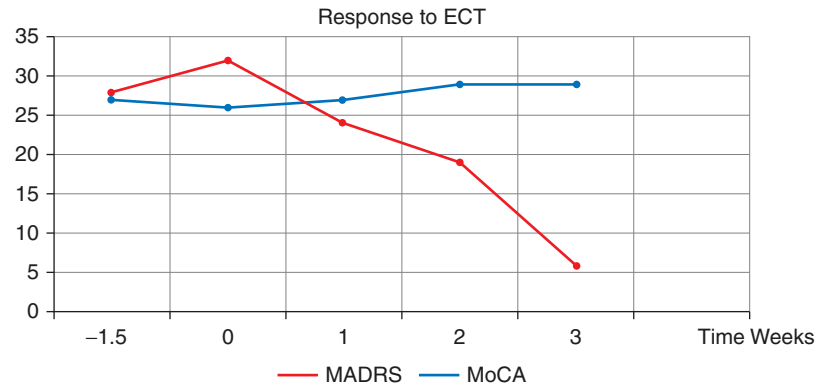


Fig. 16.2 Vignette 1 patient: depressive symptoms and cognitive impairment, pre-ECT and post-ECT treatment. (X-axis: The number of weeks of ECT. The vignette patient's ECT treatment began at 0 on the X-axis. Y-axis: Montreal Cognitive Assessment (MoCA) (green line) tracks severity of cognitive impairment, with 26 or higher indicating no cognitive impairment. Montgomery-Asberg Depression Rating Scale (MADRS) (red line) tracks the severity of depressive symptoms, with 60 as the most severe depressive symptoms)

her back her life and expressed interest in providing peer support to other patients receiving ECT. MoCA performed 1 month after ECT was 28/30. Six months after ECT, she regained her driver's license.

16.3 Vignette 2: Neuropsychiatric Symptoms and Major Neurocognitive Disorder (MNCD)

A 77-year-old married man living in a nursing home for the past year was admitted to inpatient psychiatry service for agitation. His wife had been unable to care for him at home. He was currently healthy, although he had been admitted to hospital one year prior for delirium in the context of pneumonia. Aside from a diagnosis of major neurocognitive disorder (MoCA 17/30), there was no previous history of mental health problems or hospitalizations.

He had little insight and resented living in long-term care. After a few months of living there, he lost interest in previously enjoyed activities such as listening to music, reading, and word-search puzzles. He became irritable, negative, and dissatisfied. Visits from family did not cheer him up, his appetite declined, and he lost approximately 5 lbs. in a 6-month period. He said that he wanted to die and was verbally and physically aggressive toward caregivers.

In the geriatric psychiatry inpatient unit, he pushed, hit, kicked, and spit at staff. He paced, was intrusive and hostile, and yelled negative, critical, persecutory comments. There were at least two physical fights with other patients. His wife explained that he had previously been sociable, kind, and gentle. A probate court hearing ruled that he lacked capacity to make medical or psychiatric treatment decisions, and wife was appointed surrogate decision-maker (Chap. 5: Legal Aspects).

Several trials of antidepressants and antipsychotic medications were administered, and although he tolerated the medications well, there was no improvement in symptoms or aggressive behavior. His wife and two daughters were apprehensive about ECT. The staff offered education and encouraged collaborative decision-making. Ultimately, his wife approved ECT treatments, given his symptoms, suffering, and danger to himself/others.

Right unilateral ultra-brief pulse (RUL UBP) ECT was chosen to minimize cognitive side effects and given at 6 times seizure threshold. After six treatments, he showed a slight reduction in symptoms. He was no longer physically aggressive, but stayed irritable, hostile, and resistive to care. His affect remained dysphoric with limited reactivity. ECT was changed to brief pulse (RUL BP) ECT, at which point he began to improve. He became calmer and less distressed, spent more time sitting in the common areas, and seemed to enjoy watching old movies. All agreed he was less irritable, had a more reactive affect, and no longer made critical and persecutory comments. He was more cooperative with care. He soon enjoyed walking around hospital grounds with his wife.

MoCA was 14 immediately after his acute course of ECT, and his wife reported that he was more confused, with worsened memory and more pronounced word-finding difficulties. He gradually regained the weight he had lost prior to admission. His wife did not consent to antidepressants and preferred to continue maintenance ECT. ECT treatments were gradually spaced out to once every 3 weeks. By 6 months, his MoCA score had improved to 18. A medical transportation service took him to maintenance ECT treatments. He was seen regularly in long-term care by the same geriatric psychiatry outreach team.

16.4 Vignette 3: Frailty

An 86-year-old widowed man was admitted to inpatient geriatric psychiatry service for social withdrawal and failure to thrive. He had no previous history of mental health concerns, although a diagnosis of major neurocognitive disorder (MNCD) due to Alzheimer disease had been considered 2 years prior to admission. He did not seek medical assistance for his hypertension.

His wife died 3 years prior to admission. Shortly after that, he fell and broke his pelvis. He

was not engaged in rehabilitation and only regained the ability to walk short distances. He became reclusive, spending most of his time in bed. He complained of low mood, poor appetite, and no longer worked on small projects around his home. He developed delirium due to a urinary tract infection and was admitted to a general hospital. The medical staff noted depression when he expressed a wish to die and refused to eat. He was transferred to inpatient geriatric psychiatry.

Trials of SSRIs, SNRIs and stimulant medications were ineffective for his severe major depressive episode. He was felt to be at risk of physical deterioration due to malnutrition and dehydration. His daughter consented to ECT. Given the need for a rapid response, he was treated with bitemporal brief pulse (BT BP) ECT. His baseline heart rate was 58, and he was taking bisoprolol for hypertension. The anesthesiologist felt the risk of bradycardia was significant, and so his ECT dose was chosen using the half-age method rather than by stimulus dose titration. After his first two treatments, he began to accept some oral intake and became less resistive to care. His initial treatments were complicated by bradycardia. He was given glycopyrrolate, resulting in the development of intermittent urinary retention, and he required occasional urinary catheterization. His bladder was scanned prior to each ECT session to ensure that he was not in retention and to avoid the risk of bladder rupture during ECT.

The ECT frequency was reduced to twice per week to minimize his exposure to glycopyrrolate. This patient continued to improve gradually, but by 15 treatments, had reached a plateau in improvement. He remained reluctant to get out of bed, only acquiescing when encouraged by nursing staff. He began to go to the dining room and ate 75% of each meal. He gained weight, was less irritable, and no longer expressed a wish to die. He enjoyed visits with his daughter, especially when she brought his favorite foods or took him outdoors in his wheelchair.

Venlafaxine was introduced for post-ECT relapse prevention, but he developed hypertension of 180/95. He was switched to sertraline and tolerated it well. The dose was gradually increased to 200 mg daily. The treating team elected not to prescribe lithium, despite its evidence for relapse prevention in the context of major depression. Lithium was thought to be associated with unacceptable risk given the patient's intermittent refusal of food and drink, as well as his relative bradycardia.

ECT was gradually reduced in frequency, but if the interval between treatments increased to greater than every 2 weeks, he became irritable, hostile, and resistive to leave the bed or eat. In the days leading up to each ECT session, he would begin to take to his bed, express dissatisfaction with his life, and express passive suicidal ideation. For 2–3 days after each ECT session, he experienced transient increases in agitation and confusion and had at least one fall as he attempted to get out of bed during the night. Environmental modifications to mitigate the risk of falls included lowering the bed height, partial railings, and optimizing sleep.

Aripiprazole was added as an augmenting agent, allowing the frequency of ECT to be reduced to every 3 weeks. He was discharged to a long-term care facility and returned for ECT using medical transportation. His daughter indicated that maintenance ECT was contributing significantly to her father's quality of life, despite the substantial cost and inconvenience associated with travel. After 6 months, the frequency of ECT was gradually extended to once per month, and he did not relapse. Ultimately, ECT was discontinued and the patient remained stable at his relatively low level of functioning in long-term care.

16.5 Vignette 4: rTMS

This 66-year-old woman had a history of recurrent major depressive disorder, with no medical comorbidity. She had two depressive episodes in adulthood, both of which responded to pharmacotherapeutic interventions and psychotherapy. Her third episode began approximately 1 year prior to hospitalization. At that time, she was unable to tolerate fluoxetine, due to stomach upset. Venlafaxine produced restlessness. Mirtazapine 15 mg po qhs helped with sleep, but she was unable to tolerate higher doses. She was

admitted to the geriatric psychiatry inpatient unit for suicidal ideation. Score on the MADRS was 28. She declined ECT.

The patient was started on vortioxetine 10 mg daily. Her suicidal ideation improved, and she was discharged from the inpatient unit. However, she soon found the antidepressant intolerable and discontinued it. She decided to pursue rTMS treatment at a private center. She received stimulation of the left dorsolateral prefrontal cortex (left DLPFC) for 20 min per day, 5 days per week, with a theta burst pattern of pulses. The patient complained of some mild scalp discomfort at the site of simulation during the procedure but was able to tolerate this without difficulty. At week 2, her MADRS score had decreased to 23, and by week 4 it was 9. When rTMS was stopped after 6 weeks of treatment, she was in remission and maintained her improvement thereafter. She was advised that many patients relapse within 4–6 months and to return to the rTMS clinic for consideration of maintenance rTMS if symptoms recurred.

16.6 Overview of Clinical Use of ECT and rTMS

Electroconvulsive therapy (ECT) is a somatic therapy, first developed by Cerletti and Bini in Italy in the 1930s, which treats mental illness by inducing a generalized seizure with the application of an electrical stimulus. It was widely used in psychiatry before the development of pharmacotherapy, alongside other somatic therapies such as insulin coma therapy. In the 1960s and 70s, the general public developed the perception that ECT was overused and punitive. As a result, ECT began to be used less often. As it became clear that medications had limitations, and that some patients required the substantial benefits provided by ECT, its use gradually re-emerged.

Standard ECT procedure has been refined by the introduction of general anesthesia with muscle relaxation, and by the development of alternate electrode placements, as well as stimulus waveforms, to reduce cognitive impairment. The treatment is safe, effective, and tolerable. Its use remains limited due to the need for general anesthesia and concerns about side effects such as autobiographical memory loss.

Research is underway to develop alternate forms of brain stimulation which might be more focal and result in less cognitive impairment. Examples include magnetic seizure therapy and focal electrically administered seizure therapy (FEAST) [10]. However, these methods remain investigational.

Repetitive transcranial magnetic stimulation (rTMS) has been approved for the treatment of depression in many locales, including the United States and Canada, and has a favorable side effect profile [11]. It is not as effective as ECT, not available in all centers, and not covered by all insurers. Given its variable availability, as well as its lesser efficacy, rTMS does not yet represent a true alternative to ECT.

A significant proportion of all patients receiving ECT in North America are in the geriatric age range. ECT is used more frequently in the geriatric population than in younger patients [12]. Greater medical comorbidity and frailty, preventing tolerance of pharmacotherapy at adequate dosage and duration, may favor the use of ECT in the older population. With its need for general anesthesia and electrical stimulation, ECT may appear to patients as riskier than pharmacotherapy. However, in older adults, the use of ECT has been associated with longer survival and greater clinical improvement than pharmacotherapy [13].

16.7 Overview of Indications for ECT

Severe and treatment-resistant mood disorders – both unipolar and bipolar – respond well to ECT. Inpatient admission is frequently necessary, due to safety issues requiring urgent intervention (e.g., inanition, severe functional decline, or acute suicidality). ECT is most indicated when rapid decline and loss of functioning are imminent. Geriatric patients with major depressive disorder (MDD) are more likely to have psychotic features, which are associated with greater response to ECT, and greater remission rates

after ECT, than seen in nonpsychotic depression [4]. As they age, patients with MDD may develop episodes of increasing severity, frequency, treatment resistance, as well as increased likelihood of relapse. Aging patients are less physically robust, with lower physiologic reserves than younger patients. They are therefore more likely to develop dangerous complications when they stop eating and drinking, and so are more likely to require the rapid response which can be achieved with ECT. Suicide rates in older adults are high, especially in men, who frequently attempt suicide with lethal means (Chap. 8: Suicide). When suicide risk is detected, or an attempt is thwarted, patients are admitted to inpatient units for safety, as well as definitive and rapid treatment.

Patients with complex comorbidity, both psychiatric and medical, may also be admitted for diagnostic clarification and treatment. Patients with prominent somatic symptoms and/or severe anxiety may be unable or unwilling to take medications at effective doses, making ECT a useful treatment option. Uncooperative and potentially violent patients with appropriate diagnostic indications may also be good candidates for ECT, provided appropriate local legal process has been followed with respect to informed consent (Chap. 5: Legal Aspects).

Agitated behavior in the context of major neurocognitive disorder (per DSM-IV: dementia) may prompt an admission to a geriatric psychiatry inpatient unit (Chap. 6: Major Neurocognitive Disorder with Behavioral Disturbance). To date, evidence-based pharmacotherapy for this indication has significant limitations, due to limited efficacy and substantial side effects. When agitation and aggression are severe, nonpharmacological options exhausted, and pharmacotherapy has failed, ECT can be considered. Although primarily retrospective, evidence is emerging for the use of ECT in this context [14, 15].

Once stabilized on a medical unit, medically ill patients can be transferred to a psychiatry unit for ECT. If the patient remains critically ill, she/he may need ECT in a medical unit. For example, a patient with a severe mood disorder and malignant catatonia, autonomic instability, and rhabdomyolysis may have to remain in an intensive care unit (ICU), while an unstable cardiac patient with severe depression may remain in the cardiac care unit (CCU). Close collaboration with medical teams and ECT practitioners is essential.

16.8 Pre-ECT Evaluation

Implementation of a course of ECT requires a thorough patient assessment and an evidence-based indication for ECT. Assessment of each patient's physical health and concomitant treatments helps to determine the risk/benefit ratio of ECT. It is essential that informed consent be obtained from the patient or substitute decision-maker. Pre-ECT assessment will identify necessary treatment modifications, ensuring safety, and may include medical investigations, consultations, stabilization of medical conditions, and modifications to concurrent medication treatments. Baseline psychiatric symptoms, and level of cognitive functioning, should be established using standardized rating scales. Figure 16.3 shows the essential components of the pre-ECT evaluation.

16.9 Specific Indications for ECT

16.9.1 Major Depressive Episode of Bipolar I Disorder and Bipolar II Disorder and Major Depressive Disorder with Psychotic Features

Late-life depression is a common problem, and as many as 1/3 of aging patients have treatment resistance [2]. It is associated with substantial impairments in quality of life, caregiver stress, as well as increased morbidity and mortality, making it a major public health issue as well as a challenge for clinicians. ECT is a fast-acting and effective treatment for unipolar and bipolar depression [16] and is more effective than any pharmacotherapeutic intervention. The CORE (Consortium for Research in Electroconvulsive

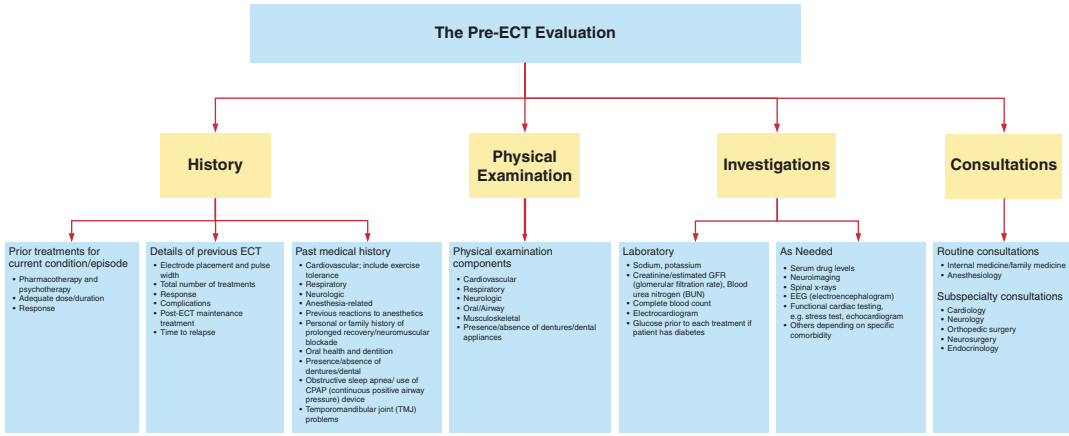


Fig. 16.3 The essential components of the pre-ECT evaluation

Therapy) study, a large multicenter trial of bitemporal vs right unilateral vs bifrontal ECT in a depressed mixed-age population (age 18–85), showed overall remission rates of 87% with ECT treatment. When a depressive episode is resistant to medication treatment, response rates to ECT are somewhat lower, closer to 50–60% [17]. In the CORE study, greater severity of depression and older age were associated with better response to ECT [18]. The median number of ECT treatments to achieve remission was 7 (mean 7.8), illustrating the rapid improvement which can be obtained with this treatment modality.

As noted above, major depressive disorder with onset in late life is more likely to be associated with psychotic features [17]. This condition is a severe form of depressive disorder which often requires inpatient admission and is associated with significant suicide risk. Major depressive disorder with psychotic features appears to respond particularly well to ECT. The CORE study, mentioned above, showed remission rates with ECT for psychotic depression of 95%, vs 83% for nonpsychotic depression [18]. In contrast, aggressive pharmacotherapy for psychotic depression in older adults produces lower remission rates than ECT (e.g., 41.9% after 12 weeks in the STOP-PD/Study of Pharmacotherapy of Psychotic Depression trial) [19]. Psychosis was a predictor of response to ECT in a prospective study of late-life depression [20]. However, a recent meta-analysis failed to confirm the presence of psychosis as a predictor of response [21]. Despite this mixed evidence in the literature, most geriatric psychiatrists consider psychotic depression to be a condition particularly responsive to ECT.

In aging adults, ECT may be somewhat less effective for early-onset depression vs late-onset depression. In a prospective study [20], early-onset patients had only a 67.3% response to right unilateral (RUL) ECT vs 86.9% for late-onset depression. Vascular changes on MRI did not affect response rates.

Most depressed patients require between 6 and 12 treatments to achieve remission. However, substantial improvement in depressive symptoms may be observed even after the first treatment [22]. Furthermore, aging patients may require a longer course of ECT to obtain remission [4, 17].

It is common for objective improvements to precede subjective improvements. Thus, the use of a rating scale can provide early evidence of response, plateau, or lack of response, which can guide clinical decision-making more accurately than routine clinical observation. The Montgomery-Asberg Depression Rating Scale (MADRS) [9] and the Hamilton Depression Rating Scale (HAM-D) [23] are well-validated measures of depressive symptoms that are useful on the geriatric psychiatry inpatient unit. If no improvement is observed after six treatments, the clinician should consider a modification in ECT technique to optimize the chances of response.

Depressive episodes in the post-stroke context respond well to ECT [4, 17], though a 1-month delay is suggested because of a theoretical risk of ischemia or hemorrhage [24]. Depression due to medical conditions and medications (e.g., depressive disorder due to steroids) can also respond well to ECT [25].

For conditions other than major depressive disorder with psychotic features, ECT is rarely used as a first-line treatment. Patient preference and past response to ECT are appropriate indications for ECT. Patients who have failed rTMS may respond to ECT [6].

16.9.2 Bipolar Disorder with Manic Episode

Patients with bipolar disorder account for 8–10% of geriatric psychiatry inpatient admissions [26]. ECT can be a helpful part of the inpatient geriatric psychiatrist's repertoire in treating this complex and challenging disorder. Bipolar depression responds as well to ECT as unipolar depression. Response can be relatively rapid, with a lower number of treatments required. Some patients may respond in as few as two to three treatments. The presence of mixed features is not a contraindication to ECT, but there is some evidence that such patients may require more treatments [4].

The use of ECT in geriatric patients with mania has not been studied extensively, and one must often rely upon research from adult psychiatry for this indication. There are many effective medications for mania, and most patients with mania can be treated with pharmacotherapy. However, mania does have a robust response to ECT. Specific situations in inpatient geriatric psychiatry in which ECT can be helpful for the manic patient include intolerance to effective pharmacotherapy, severe disorganization with threat to life (e.g., delirious mania), and treatment resistance [4]. In geriatric patients, bipolar disorder can become more unstable and brittle, with more frequent episodes and more treatment resistance. When pharmacotherapy is ineffective or poorly tolerated, maintenance ECT can be effective, with or without pharmacotherapy, to achieve or improve stability [4].

16.9.3 Major Depressive Disorder, Major Depressive Episode, and Manic Episode with Comorbid Mild or Major Neurocognitive Disorder

The presence of a comorbid mild or major neurocognitive disorder does not affect the response rates of depression or mania to ECT. However, such patients are more likely to become transiently confused during the acute period of ECT [27]. By 6 months following completion of ECT, these patients are likely to have returned to baseline levels of cognitive functioning [28, 29]. The increased incidence of cognitive impairment, and post-ECT delirium, must be balanced against the possibility of cognitive improvement if a depression is effectively treated. If the patient has significant cognitive impairment, a structured rating scale, such as the Cornell Scale for Depression in Dementia [30], can be helpful as an adjunct to diagnosis and evaluation of response to ECT.

16.9.4 Psychotic Episodes in Schizophrenia

Based on the adult literature, ECT has efficacy in schizophrenia, though it is less robustly effective than it is for mood disorders [31]. In North America and Europe, the use of ECT in schizophrenia is most common in cases of treatment resistance, in combination with antipsychotic medications [32]. In the adult population, the combination of clozapine with ECT has been shown to have substantial efficacy in treatment-resistant schizophrenia [32]. Patients with severe, treatment-resistant schizophrenia have a shorter life expectancy than the general population, and positive symptoms may become more attenuated over time [33]. As such, the use of ECT in geriatric patients with schizophrenia is less common than for patients with mood disorders. However, as the population ages, the need to consider using ECT in this population may increase.

Table 16.1 Factors suggesting presence of depressive disorder in patients with cognitive impairment

Personal history of depressive disorder
Family history of depressive disorder
Onset following a stressor
Onset and progression over months rather than years
Poor effort on cognitive testing
Self-critical or hopeless statements during cognitive testing
Prominent psychomotor slowing or ambivalence
Dysphoric affect

16.9.5 Dementia Syndrome of Depression

Depressive disorders may present in aging patients with psychomotor slowing and cognitive impairment, which can mimic a Major Neurocognitive Disorder (MNCD). This presentation, once known as "pseudo-dementia" or "dementia syndrome of depression," can result in a false diagnosis of MNCD, and an opportunity to reverse a treatable condition can be missed. Clues to the presence of a primary mood disorder in the patient with a neurocognitive disorder are listed in Table 16.1 [34]. In such patients, ECT can dramatically improve cognition despite it being associated with the post-ECT side effect of cognitive disturbance.

16.9.6 Major Neurocognitive Disorder with Behavioral Disturbance

Patients with major neurocognitive disorder (MNCD) suffer often from non-cognitive (NCS) or neuropsychiatric symptoms (NPS), also known as behavioral and psychological symptoms of dementia (BPSD). Symptoms can include aggression, anxiety, depressed mood, irritability, restlessness, pacing, delusions, hallucinations, inappropriate sexual behavior, and vocalizations (Chap. 6: MNCD with Behavioral Disturbance). When behaviors and/or symptoms are unmanageable in the community, or in long-term care settings, patients are often admitted to the geriatric psychiatry inpatient unit. Yet patients may still be at risk of harming others, or being harmed, on an inpatient unit. Interventions on an inpatient unit can include a thorough multidisciplinary evaluation, observation, person-centered treatment based on recognition of unmet needs, utilization of residual capacities, non-pharmacological approaches, and, finally, judicious, evidence-based pharmacotherapy. Evidence-based pharmacotherapy (e.g., antipsychotics or antidepressants) shows significant limitations, and in many cases efficacy is accompanied by unacceptable side effects or risks. For many patients, these approaches may be sufficient, but for those with intolerance of pharmacotherapy, treatment resistance, or dangerousness, ECT can be effective.

In cases where there is a depressive episode, comorbid with MNCD, ECT is indicated and effective [27].

Behavioral disturbance, such as agitation, may also present in a patient with a MNCD who *does not* meet criteria for a major depressive episode. An emerging literature suggests the possible benefit of ECT in the *non-depressed* patient diagnosed with MNCD with behavioral disturbance [14]. Disruptive behavior might include yelling, insulting, threatening, throwing things, hitting, kicking, biting, scratching, pacing, and fidgeting. One open-label study of 23 patients [35] showed that ECT can result in significant improvement in such behavior. In the study, 3 out of 23 patients had an adverse event from ECT, resulting in discontinuation (e.g., atrial fibrillation, delirium), and 20% of the patients developed delirium. But the remaining medication-treatment-resistant patients demonstrated significant benefit from ECT, a finding that warrants further research. At present, the use of ECT in the BPSD population is not a traditional indication, and evidence-based treatments should be employed first [36]. Caution must be exercised because ECT may also be associated with risks in patients with severe BPSD. A recent population-based study showed a significant incidence of falls, hip fractures, and pneumonia during ECT, as well as a 6% mortality rate in patients with this indication [37].

Treating MNCD geriatric patients with ECT requires some practical adjustments. Patients may not cooperate with preparation for the

treatment and may require additional supervision. For example, they may forget to adhere to the "nil by mouth" recommendation prior to anesthesia or not understand, or wish to cooperate with, the need for an IV. Staff may need to hold the patient's arms in place during preparation for the procedure, resulting in some infringement of their personal autonomy, with potential risk of injury for staff from patient's resistive behavior. Cognitively impaired patients can be poor health historians, and corroboratory information may be needed. Transportation of patients with cognitive and physical disabilities can be challenging, with potential for significant costs to patients and families. ECT may not have a durable effect, and maintenance ECT may be necessary [14, 15]. Continuation of outpatient ECT for patients with cognitive impairment in long-term care settings must take logistical issues into account.

16.9.7 Parkinson's Disease

Parkinson's disease is a complex neurological disorder with prominent neuropsychiatric symptoms, including depression and psychosis. Persistent dyskinetic movements may also develop in response to dopaminergic agents. ECT can be effective for the motor symptoms of Parkinson's disease, though the effect is transient. In practice, ECT is *rarely* used solely for the motor manifestations of the disease but can have a beneficial effect on these when used for neuropsychiatric symptoms. In the patient with Parkinson's disease and comorbid depression or psychotic depression, ECT is effective and generally well-tolerated. It may have a secondary positive impact on motor symptoms, allowing reduction in antiparkinsonian medication which, in turn, may improve psychiatric symptoms. Borisovskaya et al. reported that, in a series of 116 patients with Parkinson's disease and comorbid depression, depressive symptoms improved in 93.1% and motor symptoms improved in 83% with ECT treatment [38]. Most patients did not experience worsening of cognition, though there was a significant incidence of post-ictal confusion and delirium. Patients with Parkinson's disease, and a comorbid neuropsychiatric disorder which responds to ECT, often require maintenance ECT. Collaboration with the patient's neurologist, or primary care provider, is essential to optimize treatment. Given the challenge of treating depression with pharmacotherapy in patients with Parkinson's disease and comorbid depression, and the benefits of ECT in multiple domains, ECT should not be a treatment of last resort, but rather considered earlier in the course of illness. Case reports illustrate the efficacy of combined ECT and clozapine in the treatment of psychosis in Parkinson's disease, a relatively common problem in inpatient geriatric psychiatry [39] (Chap. 12: Delirium).

16.9.8 Catatonia

Aging patients with major depressive disorder are more likely than younger patients to present with catatonic signs and symptoms and are especially at risk of developing malignant catatonia, which requires urgent treatment to prevent morbidity and mortality [4]. Benzodiazepines are commonly used to relieve such symptoms, but aging adults may not tolerate these medications well, being more at risk for sedation, confusion, and falls than their younger counterparts. Thus, ECT may be of particular utility for aging patients with catatonia. ECT is effective for catatonia regardless of etiology, so the presence of a mood disorder is not necessary for ECT to be indicated (Chap.15: Serotonin Syndrome, Neuroleptic Malignant Syndrome and Catatonia).

16.9.9 Neuroleptic Malignant Syndrome (NMS) and Malignant Catatonia

Case reports suggest that ECT is effective for the treatment of NMS and malignant catatonia. These conditions are relatively rare and are more likely to present in the inpatient setting. Such patients will often require intensive medical care, and ECT may be invoked by the consultation-liaison team [40]. Milder forms of NMS may present in the geriatric inpatient unit, and ECT can sometimes be helpful,

either by treating the condition directly (as in the case of malignant catatonia) or by allowing removal of an antipsychotic agent, while still providing effective treatment for the underlying disorder (Chap. 15: Special Syndromes).

16.9.10 Medicolegal Considerations

Many older patients, even those who are depressed, retain their capacity to provide informed consent for ECT. In such cases, the usual procedures for obtaining informed consent are appropriate, with modifications for the aging patient as necessary [41]. These may include optimizing, and accommodating, hearing and visual deficits, for example, by using a larger typeface on consent forms and discussing the information at an appropriate pace and level to help the patient integrate the information. If desired by the patient, involving family members or trusted friends in the consent process can be helpful to assist in processing and retaining the relevant information. Issues specific to particular patients should be addressed when obtaining informed consent. Psychiatric and medical comorbidities should be considered when personalizing the discussion of risks and benefits (e.g., patients with comorbid major neurocognitive disorder are more vulnerable to transient cognitive impairment during ECT) [27]. Many legal jurisdictions, such as California, require extra judicial review of patient decision-making capacity specifically for ECT (Chap. 5: Legal Aspects).

Some otherwise suitable candidates for ECT may have an illness of sufficient severity to compromise their capacity to consent (e.g., comorbid neurocognitive disorders that diminish their capacity to understand the relevant information). Mood disorders, for which ECT is commonly used, may be accompanied by impaired cognition (e.g., "pseudo-dementia," dementia syndrome of depression, delirious mania). When ECT is used to treat agitated behavior in MNCD, patients are typically too impaired to consent to the procedure. Consultation with family, friends, or the hospital ethics committee, is recommended. These significant others may be strong advocates for ECT or may have preconceptions or misgivings regarding ECT treatment.

When family members have conflicting views, this can create stress for patients and undermine the successful implementation of a course of ECT. Meeting with members of patients' support networks to address questions regarding ECT can help to resolve these conflicts, moving the focus to the clinical aspects of ECT instead. When maintenance or outpatient ECT is proposed, the engagement of a support network for transportation and supervision is necessary. Involvement of these stakeholders can greatly facilitate ECT. Memory deficits that may emerge following ECT can also be mitigated with support of family members, who can remind patients of their illnesses and the need for ECT treatment.

The risk of falls after ECT treatment, less common with younger patients, must be considered when administering ECT to frail older adults [37]. Aging adults with severe osteoporosis may be more vulnerable to musculoskeletal complication during ECT. Loose teeth may be dislodged during ECT, and dental work is at risk of being damaged with the strong bite inherent in ECT treatments [42]. When only some teeth are present, a custom bite block may have to be fashioned out of gauze [42]. On occasion, it may be preferable to keep a partial denture in place during the ECT treatment. In such cases, dental consultation should be considered [43] (Chap. 7: Acute Medical Events).

16.10 Work-Up for ECT

No specific set of investigations is considered mandatory prior to a course of ECT. Possible components of the pre-ECT work-up are listed in Fig. 16.3. Conditions associated with increased risk need to be evaluated and stabilized by the treating psychiatrist, with the aid of consultants if necessary. Aging patients typically have multiple medical comorbidities, which may require optimization prior to ECT. A full medical history, physical examination, with attention to the cardiovascular, respiratory, and neurological systems, should be completed. Consultation with a

family physician and/or internist may be necessary to ensure stability of any pertinent medical conditions. The mouth, oral hygiene, and dentition should be examined. Considerations pertinent to the airway should be explored. A personal and family history of anesthesia, including any previous complications, should be obtained. Anesthesiology should be consulted if there are conditions which may require attention or modification to intra-suite anesthesia procedure. Prior to ECT, electrolyte levels should be restored to the normal range at an appropriate rate, and hydration should be optimized. Hyponatremia increases the risk of prolonged seizures, and hyperkalemia increases the risk of cardiac complications associated with succinylcholine.

16.11 Mortality

ECT mortality is typically quoted as 1/10,000 per 12 treatments, or 1/80,000 treatments (APA text, 2001). More modern estimates suggest that the mortality rate is even lower, estimated at 2–3/100,000 treatments [44]. Aging patients with multiple medical comorbidities, especially cardiac comorbidities, are at higher risk of dying during ECT. The risk is comparable to the risk associated with any minor surgical procedure done under anesthesia. It is also important to consider the mortality associated with *no* treatment or medication treatment. Frail older patients may tolerate repeated brief anesthesia better than medications. Aging patients who receive ECT have lower morbidity and mortality, over the subsequent 2–3 years, than patients who receive more conventional treatment [4].

16.12 Specific Medical Conditions Associated with Increased Risk

There is no absolute contraindication to ECT. It can always be considered and pursued if the risk/benefit ratio favors it. However, some medical conditions are associated with possible morbidity and mortality. Such relative contraindications are listed in Table 16.2. In the presence of these conditions, ECT should only be considered when the clinical condition is life-threatening and alternatives are not viable.

Table 16.2 Conditions associated with increased risk during ECT

Conditions	Specifics
Unstable cardiovascular disease	Uncontrolled hypertension
	Unstable angina
	Significant cardiac arrhythmia
	Severe cardiac valvular disease
	Recent myocardial infarction
	Decompensated congestive heart failure
Fragile aneurysms	Cerebral
	Aortic
Hyperthyroidism	Pheochromocytoma
Increased intracranial pressure	Headache
	Vomiting without nausea
	Ocular palsies
	Altered level of consciousness
	Back pain
	Papilledema
Recent stroke	Face drooping
	Arm weakness
	Speech difficulty

16.12.1 Physiologic Changes Induced by ECT

Risks posed by ECT need to be considered in the context of the physiologic changes occurring during treatment. The electrical stimulus produces parasympathetic activity marked by initial bradycardia and, occasionally, asystole. If a seizure is initiated, a surge of sympathetic activity ensues, accompanied by tachycardia and hypertension. The increased sympathetic activity occurs only if a seizure is induced. During the seizure, intracranial and intraocular pressure also increases. During the initial determination of seizure threshold by titration, when multiple sub-convulsive stimuli are given, there is a particular risk of bradycardia. Premedication with an anticholinergic drug, such as atropine or glycopyrrolate, may be given, although this is not necessarily routine. Instead, some anesthesiologists prefer to treat with an anticholinergic

agent only when clinically significant bradycardia or asystole occurs during the procedure. The routine use of anticholinergic agents during ECT may contribute to hypertension and tachycardia during the procedure; these are clinical problems which may require management in and of themselves. For example, glycopyrrolate is associated with less tachycardia/hypertension during ECT and so may be preferable to atropine [24]. If asystole occurs during ECT, the use of glycopyrrolate or atropine premedication should be considered for subsequent ECT treatments. Short-acting beta-blockers (e.g., esmolol and labetalol) can be used to manage hypertension and/or tachycardia of sufficient severity. Labetalol has a somewhat longer duration of action than esmolol, a difference which can be applied to manage specific clinical situations (e.g., persistent hypertension in the recovery area) [45]. Advanced age, bitemporal electrode placement, and subconvulsive stimuli are risk factors for asystole [24]. Most changes in heart rate and blood pressure resolve within 20 min [24]. The anesthesiologist may attempt to attenuate the cardiovascular tachycardia and hypertension during the procedure to increase its tolerability.

16.12.2 Cardiac Disease

Of all patients receiving ECT, geriatric patients are the most vulnerable to cardiac complications. Patients over the age of 80 have a 36% incidence of cardiovascular events during ECT, compared to 12% in their younger counterparts. Most of these are minor and do not interfere with the completion of course of treatment [24]. Hypertension is a chronic medical condition prevalent in the geriatric psychiatry inpatient population. Blood pressure should be stabilized prior to ECT according to current guidelines. If possible, beta-blockers should be avoided, as they can shorten seizure duration and interfere with the efficacy of ECT, although this empirical finding is somewhat controversial [24].

Atrial fibrillation is common in geriatric patients. ECT can still be given safely, with therapeutic anticoagulation continuing during treatment. Patients on warfarin should have their international normalized ratio (INR) monitored weekly, and ECT can be given with INRs up to 3.5 [24]. ECT is relatively contraindicated in patients with INRs greater than 3.5. Patients may have spontaneous return to sinus rhythm during their course of ECT. The effects of this conversion on rates of embolic stroke are unknown, but thromboembolic events remain a theoretical risk.

Patients with cardiac pacemakers or implantable cardioverter-defibrillators (ICDs) can receive ECT safely, if a careful evaluation is performed. A cardiologist should be consulted prior to ECT, and the type of pacemaker ascertained. The settings of the device may need to be adjusted. The functioning of the device must be checked regularly [46]. A magnet should be available in the procedure room to turn off ICDs whose functioning might be perturbed by the electrical stimulus of ECT or which might discharge inadvertently. This is done while cardiac monitoring is in place. After the treatment, the ICD is turned back on by removing the magnet.

16.12.3 Respiratory Disease

Patients with chronic obstructive pulmonary disease (COPD) or asthma may be taking theophylline. This medication has been associated with status epilepticus during ECT treatment and is therefore relatively contraindicated. If possible, theophylline should be tapered and discontinued in collaboration with the medical practitioner managing the patient's respiratory disease. If any exacerbation of COPD or asthma is present, it should be stabilized with standard treatments before ECT is started. The patient's outpatient regimen of bronchodilators and/or inhaled corticosteroids should be continued during ECT [24].

16.12.4 Osteoporosis, Fractures, and Other Musculoskeletal Issues

Fractures were more common when ECT was performed without general anesthesia. In

contemporary practice, with the use of anesthesia and muscle relaxants during the procedure, fractures are rare. Many geriatric inpatients have osteoporosis and are more vulnerable to fractures if muscle relaxation is inadequate. Patients with rheumatoid arthritis may have an unstable cervical spine. Spinal radiographs are not recommended for all patients, but are indicated in a variety of such situations. Spinal radiographs can help to evaluate the stability of the cervical spine in rheumatoid arthritis and identify pre-existing spinal compression fractures prior to ECT. Collaboration with the anesthesiologist is necessary to ensure adequate paralysis during ECT, particularly for patients at increased risk of fracture.

Some patients with recent fractures may require ECT. Consultation with the patient's orthopedic surgeon to explore the risks of destabilizing the repaired injury, a risk/benefit analysis, and collaboration with the anesthesiologist to ensure adequate muscle relaxation are all essential. A higher dose of muscle relaxant may be considered. Wrist fractures have been reported in patients with osteoporosis when the cuff method is used to isolate an upper extremity during ECT [47]. The lower limb is the preferred site for the cuff method and prevents this complication.

16.12.5 Gastroesophageal Reflux Disease (GERD)

Aging patients frequently present with asymptomatic, mild gastroesophageal reflux disease (GERD) and are not thought to be at increased risk for aspiration during ECT [48]. Patients should continue their medications during ECT. Patients thought to be at increased risk of aspiration after assessment by anesthesiology can be pretreated with citric acid/sodium citrate immediately prior to the ECT treatment to neutralize gastric acids. Intravenous medications may be considered by the anesthesiologist in severe cases [48].

16.13 Management of the Acute or Index Course of ECT

16.13.1 Electrical Aspects of ECT in Geriatric Patients

In ECT, electricity is applied to the skull to induce a generalized seizure. Most of the electrical energy is shunted into the scalp and skull; only a small proportion of it reaches the brain. The amount of energy required to induce a seizure is known as the seizure threshold.

Older patients typically have higher seizure thresholds than younger patients. The seizure threshold varies substantially between patients, and age accounts for much of this variability. It is for this reason that age-based dosing, in which the patient's age is used to determine the dose of electrical energy likely to induce a seizure, is possible. Gender is also known to play a significant role, with men having higher seizure thresholds than women. Skull thickness does not change substantially with age, but scalp thickness does in fact increase with age in women (correlated with estrogen levels over the lifetime). Brain atrophy occurs with aging and results in reductions in the amount of stimulated brain volume, as well as in a decrease in the maximum induced electric field in the brain [49]. This accounts, in part, for the higher seizure thresholds observed in aging patients, particularly in men.

16.14 Administration Technique

16.14.1 Choice of Electrode Placement and Pulse Width

Standard ECT placements include bitemporal (BT), right unilateral (RUL), and bifrontal (BF). These placements are illustrated in Table 16.3. Pulse width is also an important variable, with brief pulse ECT typically being given with a pulse width of 1 ms and ultra-brief pulse less than 0.5 ms. Some practitioners choose an intermediate pulse width of 0.5 ms. The choice of electrode placement, illustrated in Table 16.3, is an

Table 16.3 Choice of electrode placement

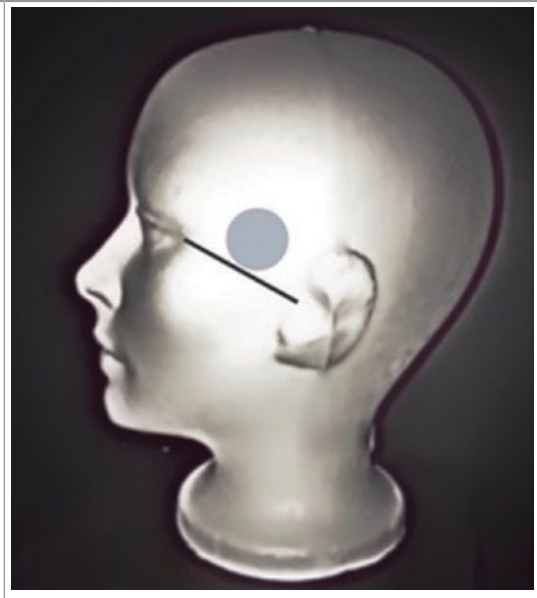
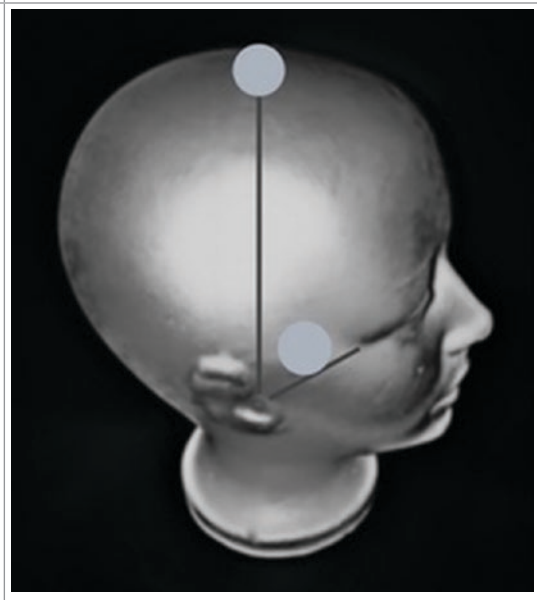
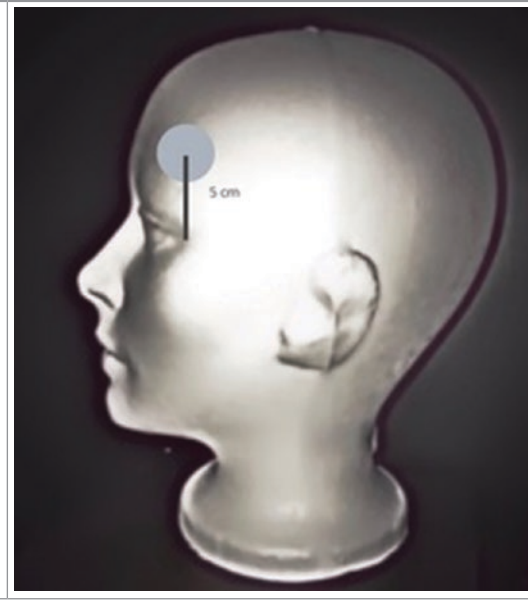
Electrode placement	Landmarks	Diagram
Bitemporal	Center of each ECT electrode is placed 1 inch (2.5 cm) above a line connecting the outer canthus of the eye to the tragus of the ear on either side of the head	
Right unilateral	Right temporal electrode uses the same landmarks as for bitemporal electrode placement The second electrode is placed 1 inch (2.5 cm) to the right of the vertex, on a line connecting the tragi of the ears	

Table 16.3 (continued)

Electrode placement	Landmarks	Diagram
Bifrontal	Center of each ECT electrode is placed 2 inches (5 cm) above the outer canthus of the eye	

important consideration when attempting to minimize the cognitive adverse effects of ECT. BT, high-dose RUL, and BF electrode placements have all been shown to have equivalent efficacy in meta-analysis [50]. Some data suggests that BT ECT may be faster-acting and require fewer treatments [16]. BT placement is associated with more cognitive side effects than high-dose RUL placement. BT ECT may be indicated when urgency for clinical response is of importance (e.g., severe suicide risk or inanition).

Limited data is available on the cognitive side effects associated with BF ECT. BT and high-dose right RUL ECT appear to be as effective as BF. The data on BF ECT's cognitive profile is equivocal. In the CORE (Consortium for Research in Electroconvulsive Therapy) study, bifrontal ECT was no better than bitemporal ECT in terms of cognitive side effects [16]. However, a meta-analysis found that both BF and high-dose RUL caused smaller declines in the mini mental state examination (MMSE) scores than did BT ECT. BF had more of an impact on word recall than RUL, with RUL having more of an impact on recall of a complex figure [51]. Bifrontal ECT may, based on this study, have an advantage for some specific domains of cognition.

RUL ultra-brief pulse ECT has been shown to be effective in treating geriatric depression [52] but may be associated with lower response and remission rates than RUL brief pulse ECT [53]. The cognitive side effect profile of RUL ultra-brief pulse ECT is favorable, although the

treatment is less robustly effective. The potential need to treat, to observe the lack of complete response, and to adjust the pulse width and electrode placement may prolong the treatment course. This may be a factor which influences the choice of ECT modality in the inpatient geriatric psychiatry setting. However, RUL ultra-brief pulse may be particularly useful when minimizing cognitive side effects is a clinical priority.

Bitemporal ECT is effective at moderate multiples of seizure threshold (ST), typically 1.5xST, whereas right unilateral ECT (RUL) must be given at higher doses (e.g., 6xST to be effective). Aging patients have higher seizure thresholds compared to their younger counterparts. These higher thresholds may make it impossible to administer RUL ECT at adequate doses. Ultra-brief pulse (UBP) ECT is more efficient in inducing seizures than brief pulse ECT. RUL ultra-brief pulse may be helpful in the context of high seizure thresholds [4].

For most geriatric inpatients, it is appropriate to start with right unilateral brief pulse or bifrontal brief pulse ECT. Patients who fail to respond to these modalities may still respond to bitemporal brief pulse ECT. A common strategy is to switch to bitemporal ECT if there is no sign of a clinical response after six unilateral ECT treatments [54].

Note that most left-handed patients are left-dominant, and so right unilateral ECT is used even in left-handed patients. If the left-handed patient experiences excessive memory impairment early in the treatment course, the clinician can consider a switch to left unilateral placement.

16.14.2 Frequency of Treatments

In North American inpatient units, ECT is typically given 3 times per week. This differs from practice in the UK and Europe, where ECT is typically given 2 times per week [55]. Thrice-weekly ECT produces a somewhat more rapid response but also produces more cognitive side effects [54]. Both treatment frequencies are effective in achieving response and remission. Twice-weekly treatment may be associated with a small increase in total duration of treatment course and fewer treatments overall [56]. If a patient develops significant cognitive side effects while receiving ECT 3 times per week, a reduction in frequency to 2 times per week may be helpful. Rarely, in severe clinical situations such as malignant catatonia, ECT can be given daily if necessary.

16.14.3 Dosing Methods

Stimulus dose titration is a method that determines the seizure threshold or the minimum charge necessary to produce an adequate generalized tonic-clonic seizure. The ECT practitioner starts at a low dose and stimulates. If no seizure is produced, the dose is doubled and the stimulus re-administered after 10–20s. The stimulus dose titration method allows subsequent dosing at specific multiples of seizure threshold and was developed to minimize the total energy given to the patient, thus reducing cognitive side effects. This method is the most accurate [4] but does involve the administration of multiple subconvulsive stimuli at the time of the first treatment. In patients, already at risk of asystole, this can be associated with increased risk. After consultation with the anesthesiologist, these patients may require premedication with an anticholinergic agent. Following stimulus dose titration, the clinicians can confidently administer ECT at multiples of seizure threshold consistent with the evidence for efficacy, while avoiding giving excess energy, and potentially greater cognitive side effects to patients with low seizure thresholds. Knowledge of seizure threshold is especially important when using RUL ECT, as the dose of electrical energy needs to be 6–8 times threshold for this modality to be effective. Low-dose RUL ECT may produce generalized tonic-clonic seizures which are ineffective in relieving symptoms of depression [4].

Age-based dosing is another method of ECT dose determination. The total output of the ECT device (576 millicoulombs [mC] for MECTA, 504 mC for Thymatron, in North America) is set

as 100%, then the dose of ECT is determined based on the patient's age. Age accounts for much of the variance in seizure threshold, with younger patients having lower thresholds and older patients' higher thresholds. With age-based dosing, the patient's age is used to determine the energy administered, with the patient's age in % energy given for unilateral ECT and ½ age in % energy given for bilateral ECT. Age-based dosing may be advantageous for bilateral ECT, which is less sensitive to dose effects than unilateral treatments [57]. It offers a less accurate approach to determination of seizure threshold but has the advantage of simplicity, reducing the risk of errors in dosing. It also reduces the risks of asystole and bradycardia associated with subconvulsive stimuli during dose titration.

In the fixed high-dose method for administering ECT, all patients receive the same dose of electrical energy. It is not recommended for routine practice, due to the risk of stimulating some patients with excessive doses and increasing cognitive side effects.

16.15 Integrating ECT and Pharmacotherapy

It is generally preferable to discontinue ineffective antidepressants prior to ECT treatment, unless the use of those medications is planned as maintenance pharmacotherapy after ECT. For aging patients, this principle is of special importance given their vulnerability to cognitive and other side effects [25]. Some evidence supports the principle of using ECT in conjunction with antidepressants such as venlafaxine or nortriptyline, with this approach yielding a 15% improvement in remission rates [58].

16.15.1 Psychiatric Medications

Prior to ECT, the medical provider should consider the management of concomitant medications during ECT. There are no absolute rules. The risk/benefit ratio of combination treatment should be carefully considered for each patient. The following section describes general principles of combining ECT with psychiatric medications.

16.15.1.1 Antidepressants

There is some controversy about the management of antidepressant treatment during ECT. ECT is effective as a stand-alone treatment. Many clinicians combine ECT with pharmacotherapy, either to enhance efficacy or to ensure that post-ECT pharmacotherapy is in place prior to discontinuation of ECT. Concomitant use of antidepressants and ECT may be associated with complications [43]. When combining treatments in aging patients, a risk/benefit analysis should be conducted prior to treatment selection. Tricyclic antidepressants (TCAs) may be associated with an increased rate of cardiovascular complications, and venlafaxine has been associated with asystole at doses above 300 mg per day. Monoamine oxidase inhibitors (MAOIs) can affect the choice of pressor agent in case of hypotension, as some agents may provoke a hypertensive crisis. Therefore, the anesthesiologist needs to be aware of concomitant use of MAOIs. SSRIs and other antidepressants are commonly combined with ECT, though less is known about the safety of the combination. Drug-drug interactions should also be considered. Sertraline has been associated with prolonged action of succinylcholine, and bupropion may lower seizure threshold [48]. However, in clinical practice both sertraline and bupropion are commonly combined with ECT.

16.15.1.2 Cholinesterase Inhibitors

Cholinesterase inhibitors may prolong the action of succinylcholine given their mechanism of action and may theoretically increase the risk of cardiovascular complications such as bradycardia. However, despite this theoretical concern, cholinesterase inhibitors are routinely continued during ECT without complication. Some studies and case reports suggest that they protect against post-ECT confusion [43]. The data is not strong enough to recommend the use of these agents routinely, but their presence does not usually affect the administration of ECT.

16.15.1.3 Anticonvulsants

Older adults may take anticonvulsants for mood stabilization and/or for the treatment of seizure disorders. Patients with seizure disorders may have to continue their anticonvulsants during ECT. In these patients, the adequacy of seizures, and clinical response, should be evaluated, with treatment adjusted accordingly. In patients who are taking anticonvulsant agents for mood stabilization, it is ideal to discontinue anticonvulsants, or reduce the dose, during ECT, with the possible exception of lamotrigine. If seizures are inadequate, the evening dose of the anticonvulsant can be held. Lamotrigine appears to have relatively little effect on seizure threshold and ECT [43, 59].

The use of lamotrigine in combination with ECT has been described in several case series [60]. For most patients, concomitant use of lamotrigine during ECT was not associated with any reduction in the ability to produce a seizure of adequate duration or in the ability to achieve remission with ECT. In a case series of nine patients who had ECT both with and without lamotrigine, seizure duration was reduced in two of those patients from an average of 47.1 s without lamotrigine to 40.6 s with lamotrigine [60]. For most patients on lamotrigine, ECT may be given without changes in the lamotrigine regimen, especially if the use of lamotrigine is planned as a post-ECT maintenance treatment. However, if it is observed that it is difficult to obtain a seizure of adequate duration, consideration should be given to reducing the dose of lamotrigine and/or discontinuing lamotrigine during ECT. Care should be taken to observe the manufacturer's recommendation for restarting lamotrigine and achieving a therapeutic dose.

16.15.1.4 Lithium

Concerns that lithium worsens confusion after ECT have primarily stemmed from case reports [61]. Lithium has been safely combined with ECT in another case series [62]. In a more recent report, lithium has also been safely combined with maintenance ECT [63]. Aging patients may be especially sensitive to post-ECT confusion when ECT and lithium are combined. In general, lithium should be discontinued prior to ECT in an index or acute series if there is no compelling reason to continue it. If the clinician chooses to continue lithium, it should be kept at the minimum therapeutic dose, and held the night before ECT. The risk of psychiatric decompensation should be considered, especially in patients with brittle bipolar disorder. Lithium can safely be combined with maintenance ECT, but it should be held the night before ECT in this context.

16.15.1.5 Benzodiazepines

Ideally, benzodiazepines are minimized or discontinued prior to ECT. Abrupt discontinuation can result in confusion and withdrawal seizures. If seizures are inadequate during ECT treatment in the presence of benzodiazepines, flumazenil can be used to reverse the action of benzodiazepines in the treatment room [48]. Some clinicians give a dose of midazolam after ECT to prevent benzodiazepine withdrawal symptoms post-procedure after the use of flumazenil.

16.15.1.6 Antipsychotics

Antipsychotics are commonly administered with ECT, and this combination is generally thought to be safe [64]. ECT and clozapine are both commonly used in treatment-resistant conditions and have been combined with evidence of added efficacy over each agent alone. The safety of this combination is based on use in a small number of patients [65].

16.15.1.7 Medications for Medical Conditions

Most medications can be held the morning of ECT and given after the patient returns from treatment. Cardio-protective medications (except diuretics) and anti-reflux agents should generally be given with sips of water in the morning prior to ECT treatment. Decisions about administration of medications in the peri-ECT period should be made in collaboration with the anesthesiologist. Premedication with anticholinergic agents (e.g., atropine or glycopyrrolate) should be discussed with the anesthesiologist. The use of these agents is not necessarily routine, but can be considered if there is a risk of excess secretions, bradycardia, or asystole [48].

16.15.1.8 Other Considerations in Patient Preparation

Patients must be kept nil per os (NPO) for solids after midnight the night before ECT. Local policies should determine the timing of NPO status for liquids. Some anesthesiologists prefer adequate hydration and suggest that patients can have clear fluids up to 3 h prior to the treatment [66]. It can be a challenge to keep patients with cognitive impairment NPO, as they sometimes cannot understand or remember the instructions. It is ideal to treat such patients earlier in the treatment day to prevent accidental oral intake. Staff may have to remove personal food items from patients' rooms and observe patients closely to prevent them taking food from others. Patients with diabetes should be treated early in the day if possible to minimize the risk of hypoglycemia.

Anxiety prior to ECT is common [67] and can usually be managed with education, accompaniment, and reassurance. If medication is required, it is best to avoid benzodiazepines prior to the procedure due to their anticonvulsant properties. In some extreme situations (e.g., catatonia or severe anxiety), concomitant benzodiazepine administration can be reversed with flumazenil to permit the elicitation of an adequate seizure [48].

16.16 Response Assessment

16.16.1 Monitoring During the Acute or Index Course of ECT

The acute or index course of ECT refers to the period in which patients are receiving two to three treatments per week, with the goal of relieving symptoms of the current episode. Possible outcomes are remission, response, and no response.

Patients should be assessed after each treatment to determine if they have experienced any adverse events from the procedure (e.g., headaches, muscle pain, nausea). They should be assessed prior to each treatment to ensure that they remain medically stable and fit for the procedure. Standardized symptom rating scales can be helpful to determine if a clinical response is beginning to occur, as it is common to note some response even after the first treatment [22]. Typically, rating scales are done weekly to permit adjustment of the treatment. Interviews with informants can also be helpful.

16.16.2 Cognition

ECT is associated with cognitive impairment in older patients without neurocognitive disorders, with 40% experiencing anterograde memory impairment and 11% experiencing retrograde memory for current events. However, ECT is also associated with overall improvement in performance on most neuropsychological tests [68]. Patients without pre-existing cognitive impairment showed overall improvement in cognitive functioning 6 weeks after ECT, while patients with pre-existing cognitive impairment took longer to recover but were improved by 6 months after ECT [68]. Overall, the cognitive profile of ECT in older adults is similar to that in younger adults, with transient and reversible cognitive deficits. Patients with neurocognitive disorders may be at higher risk of transient delirium with ECT [68].

Cognition should be evaluated prior to treatment, after treatment, and periodically during a treatment course. There is no consensus on the specific instruments to be used, though some authors have attempted to make recommendations [69]. The MMSE [70] and MoCA [8] are familiar to clinicians and are commonly used measures. The frequency of assessment should consider acceptability to patients, practice effects, and the need to identify emergent cognitive problems in time to adjust the electrical parameters of treatment. A practical approach might be to apply a standard global measure of cognition before treatment, to monitor cognition periodically during the index course with that measure or another shorter one, and to repeat the global measure both at the end of the index course of ECT and again 2–3 months after ECT. Asking patients and caregivers specifically about autobiographical memory loss can also be as helpful as bedside cognitive testing. Formal neuropsychological

testing may also be helpful in specific clinical situations [17].

16.17 Assessment of Completion of ECT

An index course of ECT should be continued until remission is obtained or until a plateau of clinical improvement is reached. Typically, plateau refers to a lack of improvement after two to three consecutive treatments.

For perspective, the multi-site PRIDE (Prolonging Remission in Depressed Elderly) study of older patients with RUL UBP ECT had a mean number of treatments of 7.3. In that study, 19.6% of remitters required fewer than four treatments and only 25% of remitters required more than ten treatments [52]. A meta-analysis found that the mean number of treatments to achieve remission with RUL UBP ECT was 9.6, one more treatment than for RUL BP ECT [53]. There is considerable variability between patients, with some patients requiring more than 12 treatments to achieve remission or maximum response [4].

16.18 Posttreatment Maintenance

Although ECT is a very effective treatment acutely, many patients will relapse following ECT despite adequate pharmacotherapy. Continuation/maintenance ECT on a fixed schedule is equivalent to pharmacotherapy in preserving remission post-ECT, so many clinicians will choose pharmacotherapy first [71]. This approach avoids the need for travel, accompaniment, and repeated procedures, which can be burdensome for some older patients [71]. Evidence-based pharmacotherapy post-ECT consists of an antidepressant in combination with lithium [4].

When patients who have responded to ECT relapse despite adequate post-ECT pharmacotherapy, consideration should be given to continuation ECT (for the first 6 months following remission) and/or maintenance ECT (ongoing treatment beyond 6 months) to preserve remission and quality of life [72, 73].

The PRIDE study focused on a symptom-titrated approach to maintenance ECT, providing a "rescue" treatment as needed when depressive symptoms emerge and delaying treatment if patients developed cognitive impairment with ECT [73]. These rescue treatments allowed patients to minimize the number of maintenance ECT treatments, while preserving remission. In this study, pharmacotherapy (lithium plus venlafaxine) was compared to pharmacotherapy plus continuation ECT. Patients who received pharmacotherapy along with symptom-titrated ECT over 6 months (the average number of ECT treatments was 4.5) had better quality of life over 6 months in both physical and mental domains than patients who received pharmacotherapy alone.

Ideally, patients who receive continuation/maintenance ECT as outpatients meet the following criteria:

(a) Are able to comply with restrictions such as nil per os (NPO) status.
(b) Are not actively using substances.
(c) Have a responsible adult to accompany them home from the procedure.
(d) Can be accompanied/monitored 24 h after the procedure.

For older patients, these requirements can sometimes pose challenges. They may have cognitive deficits, which interfere with their ability to comply with the preparations for the procedure. They may take multiple medications which need to be held the night before (e.g., lithium) or given in the morning prior to the procedure (e.g., antihypertensives), creating opportunities for error. They may live in institutional settings, such as retirement residences or long-term care facilities, in which such behavioral restrictions must be coordinated with the relevant care team. They may also be unable to tell ECT staff about any recent changes in their medications or physical health. In such cases a close relationship between the psychiatric treating team, the local medical team, and the ECT team is essential, and substantial coordination may be required. Many younger ECT outpatients are physically healthy, but older patients are more likely to have multiple medical comorbidities and to have between-treatment

medical changes or accidents which require vigilance to ensure the safety of ECT treatments on an ongoing basis.

16.19 Adjunctive Treatments to ECT

Older adults have fewer misconceptions regarding the nature of ECT [74] than do their younger counterparts. However, older patients and their families do require ongoing education and support throughout a course of ECT. Initially, it is helpful to address issues of stigma, treatment-specific anxieties, and misconceptions and to assist with perseverance with the treatment until clinical benefit is seen.

ECT is associated with high relapse rates. Although this is discussed before beginning a course of ECT, patients and families are often discouraged when relapse occurs and may see it as a failure of ECT. Considerable effort needs to be made to ensure that patients have the best chance of remaining well after an index course of ECT. This usually involves pharmacotherapy along with supportive therapy as part of routine clinical care. It may also involve continuation or maintenance ECT after initial treatment series.

In a case series, cognitive-behavioral psychotherapy has been used as an adjunctive treatment to prevent relapse after ECT with reported benefit [75]. Although existing evidence is weak, evidence-based psychotherapies can be considered as an adjunct for relapse prevention after an index course of ECT, particularly if a patient wishes to pursue such treatment or if the clinician can identify a relevant therapeutic focus.

Anxiety related to ECT is common and multifactorial but may persist throughout the index course of ECT and after its completion. It can be a significant barrier to patient acceptance of ECT treatment [67]. Common themes include anxieties related to:

(a) The use of electricity.
(b) The possibility of brain damage or memory impairment.
(c) Fear of being made unconscious and losing control.
(d) Fear of the intravenous injection.
(e) Uncertainty during the waiting period prior to ECT.

Very limited data suggests that patient accompaniment prior to the procedure, talking through of the intervention, as well as interaction with a therapy animal during the ECT waiting period, can reduce these anxieties [67].

16.20 Repetitive Transcranial Magnetic Stimulation (rTMS)

Repetitive transcranial magnetic stimulation (rTMS) is an emerging neuromodulation treatment. It is primarily used for depression, but its use in other disorders is being explored. Its current utility in inpatient geriatric psychiatry is limited. It is not available in many centers, or it is prohibitively expensive.

In rTMS, the patient remains awake. An electrical wire coil, encased in plastic, is placed on the head. The coil generates a pulsed magnetic field which can stimulate selected areas of the cerebral cortex repeatedly, without inducing a seizure. Treatment sessions are typically done 5 days per week and last between 20 min and 1 h, depending on the stimulation protocol, although some newer protocols such as theta burst can be much shorter [76]. In rTMS, patients are required to sit quietly for the duration of the stimulation period. Patients' capacity to adhere to this requirement should be assessed.

16.20.1 Patient Assessment for rTMS

Prior to initiating rTMS, a psychiatric assessment should confirm the presence of a disorder for which rTMS is indicated. The one absolute contraindication to rTMS is the presence of any metallic hardware in close contact with the stimulus coil [77]. Such hardware might include cochlear implants, deep brain stimulation electrodes, and medication pumps. The magnetic pulses administered in rTMS can cause implanted devices to malfunction. The presence of a cardiac pacemaker or implanted defibrillator is of concern

for similar reasons [78]. However, there is one case report of successful rTMS administration with a cardiac pacemaker in a 72-year-old patient [79]. A pretreatment screening questionnaire can minimize risks associated with rTMS [77].

Induction of a seizure is one of the major risks associated with rTMS (incidence 1/30,000 treatment sessions) [80]. Patients should be assessed for the presence of disorders associated with lower seizure thresholds, such as seizure disorders and irritative brain lesions. Consideration should be given to state-dependent factors, such as the use of medications which lower seizure threshold, heavy alcohol use with potential for withdrawal, and sleep deprivation, and attempts should be made to mitigate these. Consideration can also be given to the use of anticonvulsant medications if necessary [77].

16.20.2 Diagnostic Indications for rTMS

rTMS is primarily indicated for the treatment of unipolar major depressive episodes. It is considered a first-line treatment for patients who have failed at least one antidepressant [59]. Treatment resistance is associated with a lack of response to rTMS, and so it may be preferable to turn to rTMS early in the course of illness. The utility of rTMS in bipolar depression is unclear at present [76].

Psychotic depression appears to respond less well to rTMS than does nonpsychotic depression [5]. Patients with psychotic depression are more likely to respond to ECT than to rTMS [81]. White matter changes do not seem to affect the response to rTMS [78]. Little is known about how depression in the context of neurocognitive disorders might respond to rTMS.

16.20.3 Site of Stimulation and Pulse Pattern

rTMS is administered with a treatment coil, which is an electrical wire coil (encased in plastic) placed on the scalp surface. Coils of various shapes are available, but the practitioner will typically be limited to what is available in the local setting. The figure-8 coil, with two round coils placed side-by-side, is the most commonly used.

Stimulation at low frequencies (1 Hz or less) produces cortical inhibition, while frequencies above 1 Hz produce excitation. High-frequency rTMS (HF-rTMS) over the left dorsolateral prefrontal cortex (DLPFC) is the most commonly used method of treatment for major depression. However, low-frequency rTMS (LF-rTMS) over the right DLPFC, or a combination of the two, is also used [80].

Stimulus intensity is typically determined in relationship to the motor threshold, which is the lowest intensity of stimulation causing muscle contraction. The stimulus intensity, the total pulses delivered per session, and the pattern of pulses (e.g., theta burst) are important variables in rTMS [59]. Older patients may require longer courses of treatment and higher stimulus intensities for rTMS to be effective [5].

16.20.4 Management of the Acute or Index Course of rTMS

16.20.4.1 Medication Management During rTMS

Psychiatric medications can be continued during rTMS. It should be noted that many psychiatric medications, in particular antipsychotics and antidepressants, alter the seizure threshold. If medications change during rTMS, the patient's motor threshold may change, and settings may need to be reassessed [80]. Caution should be exercised with concomitant medications, as most seizures induced by rTMS have occurred in patients taking medications which lower the seizure threshold. Withdrawal from benzodiazepines can produce the same effect. Starting a new antidepressant medication along with rTMS may improve the rate of response [59].

16.20.5 Response Assessment

During a course of rTMS, the practitioner should inquire about side effects (e.g., headache and discomfort at the site of stimulation) [82]. As with

ECT, the use of structured rating scales for depression can help with decision-making during the management of a course of rTMS. rTMS does not affect cognition [5], so the cognitive monitoring required during a course of ECT is not required. Treatment is usually administered for a total duration of 4–6 weeks. If a patient has only a partial improvement, and no clear plateau in improvement, it can be appropriate to extend treatment beyond 6 weeks. If patients show no response at 6 weeks, they are unlikely to respond to further rTMS.

16.20.6 Post-rTMS Management

There is limited evidence available to guide post-rTMS management. Most patients are continued on pharmacotherapy after a course of rTMS. Many patients will remain in remission, but some will relapse, with an average time to relapse of around 4–6 months [83]. Limited data in aging adults suggests a somewhat higher relapse rate, although numbers are too small to draw definitive conclusions [84].

Consensus recommendations are to discontinue rTMS gradually, reducing the frequency over 3 weeks prior to discontinuation. If patients relapse, a return to rTMS is likely to result in a return to previous levels of response [80]. When patients relapse often, maintenance rTMS at a frequency of once per week to once per month can be helpful. In a naturalistic Australian study, 12.6% of adult rTMS patients went on to require maintenance rTMS [85].

16.21 Novel Treatments

There are several novel somatic treatments under investigation for the treatment of severe depression, including:

(a) Deep brain stimulation (DBS).
(b) Magnetic seizure therapy (MST).
(c) Focal electrically administered seizure therapy (FEAST).
(d) Transcranial direct current stimulation (tDCS).

These treatments are investigational and not routinely available.

Ketamine is an investigational treatment for depression and suicidal ideation, in which there is significant interest at present. Intravenous ketamine is a fast-acting, novel, and promising treatment for depression. Clinicians have an interest in developing its clinical applications, and it may be available in some centers.

16.22 Ketamine in Inpatient Geriatric Psychiatry

There is emerging evidence for the rapid efficacy of ketamine in treatment-resistant major depression in the general adult population [7, 86]. Ketamine is an N-methyl-D-aspartate receptor (NMDA) antagonist which is used as a general anesthetic but which appears to have rapidly acting antidepressant and anti-suicidal ideation effects after intravenous (iv) infusion at sub-anesthetic doses. For depression, ketamine is typically given as a 0.5 mg/kg total dose, in an intravenous infusion over 40 min. Ketamine infusion is of particular interest in the inpatient setting due to its rapid onset of response. Between 43% and 90% of patients experience symptom relief (50% reduction in baseline symptoms) immediately after a single infusion, and suicidal ideation is independently reduced as well.

The evidence for the efficacy of ketamine in late-life depression (LLD) is limited at present. Some older patients are included in the randomized controlled trials demonstrating the efficacy of ketamine, but it is difficult to determine any differential efficacy with the data reported. Szymkowicz et al. [87] reported on four heterogeneous older patients, with different ages of onset and comorbidities, who failed to respond to repeated ketamine infusions. A subsequent case report by Srivastava et al. was more favorable [88]. More recently, a report describing two more aging patients who did respond to ketamine infusions appeared in the literature [89].

Ketamine infusions require monitoring of vital signs, as hypo/hypertension, tachycardia/

Table 16.4 Research studies of ketamine for treatment of depression in aging adults

Ketamine study	Subjects	Response	Remission	Tolerability	Duration of effect
Szymkowicz et al. [87]	4	0/4	0/4	Dissociation, paresthesias in 3/4	N/A
Srivastava et al. [88]	1	1/1	1/1	Transient mild dissociation, memory	1 year
Medeiros da Frota Ribairo and Riva-Posse [89]	2	2/2	2/2	1/2 Mild dissociation	1 subject relapsed after 5 weeks

bradycardia, and reduced respiratory rate have been reported. Aging patients may be more sensitive to these physiological effects. Ketamine infusions can also produce psychotomimetic effects (e.g., feelings of alteration of the passage of time, feelings of floating, and feelings of dissociation), which can be anxiogenic and distressing for patients. In the general adult population, these appear to be transient and of mild-moderate intensity. However in an aging population, these effects may be somewhat more common [87].

Intranasal ketamine has also been examined as an alternative. At present, relatively few patients have been studied using this formulation.

Prior to widespread adoption of ketamine, more studies are needed regarding its safety and tolerability in the geriatric population and its place in current treatment algorithms. Table 16.4 lists some research studies of the application of ketamine to treat depression in older adults.

16.23 Summary

ECT is a valuable treatment option in the geriatric psychiatry inpatient unit. Emerging treatments such as rTMS and intravenous ketamine are of considerable interest, but at present are not realistic or evidence-based alternatives to ECT. This chapter has concentrated primarily on the practical aspects of using ECT in the inpatient setting, where it can be lifesaving, due to its rapid efficacy in treatment-resistant conditions common in geriatric psychiatry.

There are many reasons why geriatric patients receive proportionally more ECT than their younger counterparts. Aging patients with depression respond preferentially to ECT compared to younger patients. They are also more likely to have depression with psychotic features, for which ECT is a first-line treatment. Geriatric patients are also likely to have multiple comorbid medical conditions and decreased ability to maintain their physical health and stability in the context of severe mental illness, and may therefore be less likely to tolerate adequate pharmacotherapy, or to be stable enough to wait for clinical response to medications. Many of the conditions commonly seen on the geriatric psychiatry inpatient unit, such as major neurocognitive disorder with neuropsychiatric symptoms, have the potential to respond to ECT, although the use of ECT for MNCD with behavioral disturbance is an emergent indication, with a limited evidence base. ECT is generally safe for the frail elderly and may be safer than psychotropic medications, provided that medical conditions are adequately stabilized in collaboration with the primary care provider and medical specialists prior to ECT. Consultation with anesthesiology and careful management of the peri-ECT period is essential. In most cases, ECT is integrated with pharmacotherapy to achieve the best results and to prevent relapse, although ECT is effective a stand-alone treatment.

Right unilateral brief pulse ECT may have the most favorable balance of efficacy and cognitive side effects, although right unilateral ultra-brief pulse ECT may be almost as effective and be cognitively neutral. Bitemporal brief pulse ECT retains a role as the gold standard for robust efficacy and rapidity of action, as well as for use in conditions such as schizophrenia, in which alternate electrode placements and pulse widths have not been studied.

The availability of multiple treatment approaches makes careful monitoring of treatment response and side effects essential. Structured rating scales and bedside cognitive assessment tools can be helpful to detect emergent clinical response and side effects to guide decision-making, as can the observations of caregivers, in addition to standard clinical reassessment. If there is no response or a plateau of response after four to six treatments, the clinician should consider modifying the electrode placement or pulse width.

Relapse rates following an acute or index course of ECT are high, and so careful attention should be paid to the immediate post-ECT period to maximize the patient's chances of staying well. Evidence-based pharmacotherapy following ECT consists of an antidepressant combined with lithium, provided the patient's clinical condition and concomitant medications permit the use of this combination. Many clinicians choose to taper ECT slowly rather than stop it abruptly. For the prevention of relapse, maintenance ECT is equivalent to lithium combined with an antidepressant. Combined maintenance ECT and pharmacotherapy may be even more effective than either alone, and a symptom-titrated approach to maintenance ECT has evidence of efficacy.

rTMS is not available in many clinical settings and is most effective in cases of mild treatment resistance. Modern treatment protocols and longer treatment courses may be necessary for it to be as effective in older patients as it is for the general adult population. However, there is rapid evolution in rTMS treatment protocols, and the place of rTMS in the inpatient setting is likely to evolve over time. The same is true for intravenous ketamine, an investigational treatment which appears to have rapid efficacy in reducing suicidal ideation and depressive symptoms. Its place in the inpatient geriatric psychiatry setting will emerge as the evidence base develops.

At present, ECT remains the most useful somatic treatment, with the greatest body of evidence to support its benefit compared to rTMS and other non-pharmacological treatments.

Take-Away

- ECT is a faster and more effective treatment than pharmacotherapy for major depressive disorder.
- Older patients respond better to ECT than do their younger counterparts.
- ECT can be considered a first-line treatment for depression with psychotic features.
- ECT should be considered when there is a need for urgent response.
- ECT has effects on episodic memory and cognition in geriatric adults, but overall, cognition may improve with ECT.
- Right unilateral ultra-brief pulse ECT has a favorable cognitive side effect profile but is slightly less robust in efficacy than right unilateral brief pulse and bitemporal brief pulse ECT.
- Bitemporal brief pulse ECT is the most effective form of ECT and is indicated when there is clinical urgency, when patients fail to respond to unilateral placement, or when medical comorbidities are such that minimizing the number of ECT treatments is desirable.
- ECT is effective for patients with depression comorbid with major neurocognitive disorder, though such patients are more likely to have more transient worsening in cognition than their non-cognitively impaired counterparts.
- ECT is a safe and effective treatment even for frail older adults with medical comorbidities.
- Collaboration with medical and anesthesiology consultants is necessary to optimize patients' medical conditions prior to ECT.
- There is no absolute contraindication to ECT.
- Relative contraindications to ECT include recent myocardial infarction and recent stroke.

- ECT can be useful for conditions other than depression, such as treatment-resistant mania, schizophrenia, and catatonia.
- Evidence is emerging to support the use of ECT in major neurocognitive disorder (MNCD) with behavioral disturbance.
- The average number of treatments to achieve remission is between 6 and 10.
- Relapse rates following ECT are high.
- Nortriptyline plus lithium and venlafaxine plus lithium have the best evidence for preventing relapse after an index course of ECT.
- Maintenance ECT can maintain remission when pharmacotherapy post-ECT fails to prevent relapse.
- A combination of pharmacotherapy and symptom-titrated ECT improves remission rates and is associated with increased quality of life.
- rTMS may be indicated when patients have failed one antidepressant trial. It may be less effective when there is a history of greater treatment resistance.
- rTMS has few side effects and a high degree of acceptability to patients.
- Although rTMS was initially thought to be less effective in older adults than in younger ones, it may be as effective when used with treatment protocols of higher intensity and longer duration.
- Intravenous ketamine remains an investigational treatment for depression and suicidal ideation.

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17 Medication Strategies: Switching, Tapering, Cross-Over, Overmedication, Drug-Drug Interactions, and Discontinuation Syndromes

Elizabeth Kozyra and Timothy Lau

17.1 Introduction

For the geriatric patient, an inpatient hospitalization can be an optimal setting in which to review a medication regimen, implement safe medication prescribing, minimize polypharmacy, assess for adverse effects, and institute processes to prevent overmedication after discharge. For example, inpatient care can address a common result of geriatric pharmacokinetics and pharmacodynamics: the risk of overmedication. Factors which contribute to excessive medication in the geriatric patient include higher doses than tolerated, use of medications for which there is no longer a valid indication, age effects on pharmacokinetics and pharmacodynamics, or drug-drug interactions. Studies estimate that about 50% of individuals aged 65 years or older are prescribed an average of five or more medications [1]. There is a linear relationship between the number of medications prescribed and the likelihood of being admitted to hospital as a result of adverse effects and inappropriate medication use [2]. This pervasive problem, and one amenable to correction, is termed polypharmacy: a complex medication regimen, with usually five or more medications. Some of the medications may no longer be current or indicated, and some of the medications may have been prescribed to treat adverse effects of other medications [3]. Figure 17.1 provides a summary of the medication interventions for geriatric inpatients.

17.2 Vignettes

17.2.1 Vignette #1

A 74-year-old female was admitted to the inpatient geriatric psychiatry unit, after she presented to the emergency department (ED) earlier that day with complaints of lethargy, paresthesia, tremor, anxiety, insomnia, and profuse sweating. The patient had a 25-year history of depression, hypertension, atrial fibrillation, osteoporosis, chronic renal failure, and type 2 diabetes. A review of medications identified the recent discontinuation of paroxetine and a plan to switch to venlafaxine. No other recent medication changes appear to have been made. Prior to her ED presentation, her paroxetine 40 mg was tapered by 10 mg daily, up to her last dose, taken 48 hours prior to admission. The patient was advised to begin taking venlafaxine one week after discontinuing paroxetine.

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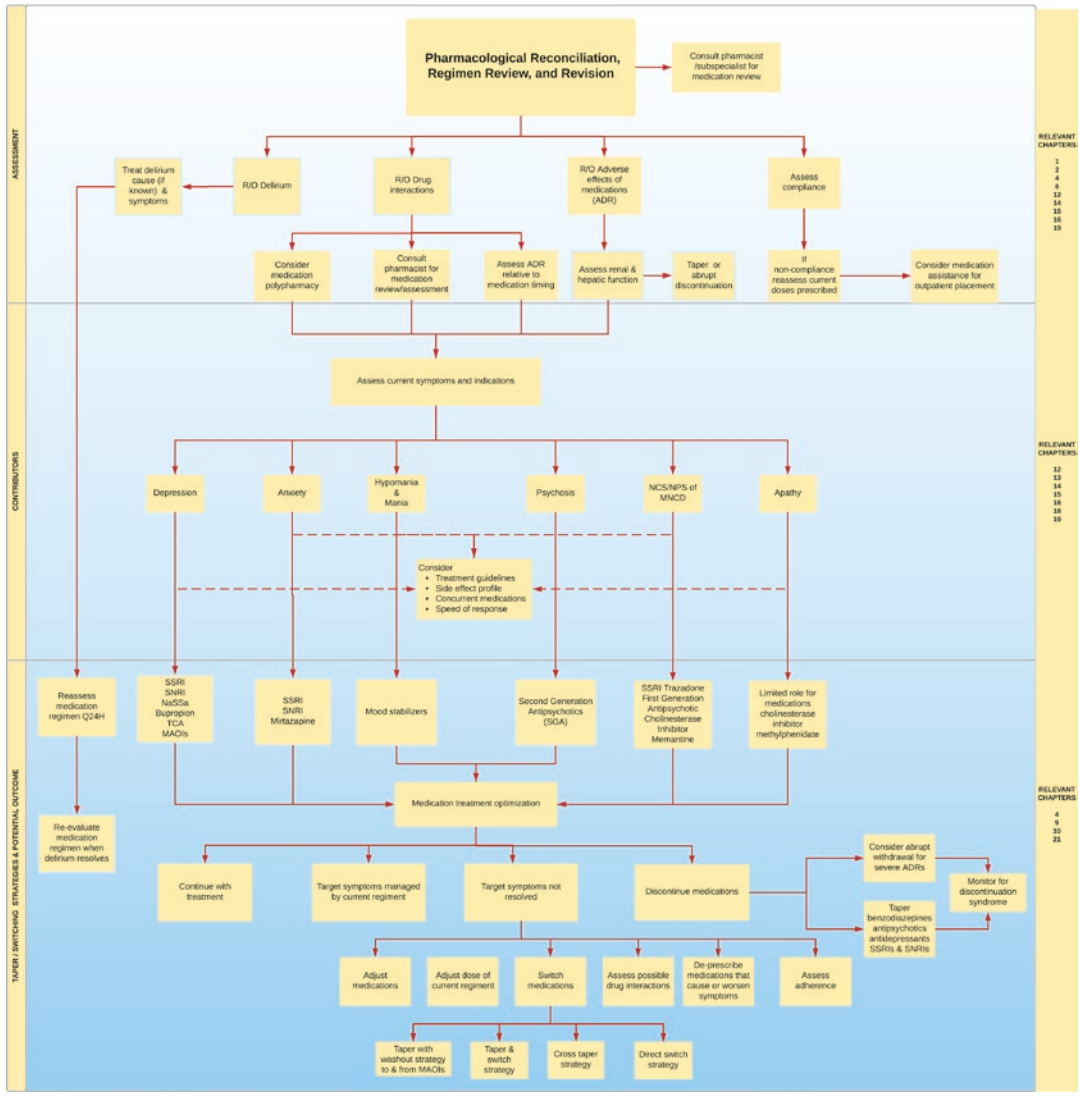

Fig. 17.1 Flowchart of medication interventions

17.2.2 Vignette #2

A 77-year-old man with neuropsychiatric symptoms (NPS)—behavioral disturbance—of major neurocognitive disorder (MNCD) was admitted to the geriatric inpatient unit for the management of agitation and aggression. Prior to admission, the patient was prescribed daily risperidone 2.0 mg, but after an increase to 3.0 mg, he developed axial dystonia. Lower doses were not effective in managing his psychotic symptoms. Previous trials of antipsychotics included quetiapine, which resulted in constipation, excessive daytime drowsiness, and falls. During his hospitalization, the patient continued to require doses of as needed medications (PRNs) throughout the day. Nursing staff reported that he was sleeping well through the night. His constipation was well managed with regular bowel medications. His rheumatoid arthritis was managed with acetaminophen 650 mg four times daily. Delirium screening was negative.

Behavioral approaches to manage his NPS were implemented as part of his nursing care plan. This behavioral plan was not successful, and it was decided to switch from his regimen of risperidone to aripiprazole to manage agitation and aggression (Chap. 6: Major Neurocognitive disorder with Behavioral Disturbance).

Rationale: There was a need to discontinue the first antipsychotic medication, risperidone, due to an adverse reaction and lack of efficacy. The disruptive behavior demanded a prompt intervention, and the new antipsychotic was started immediately. The choice of aripiprazole was made because aripiprazole has evidence of benefit for behavioral symptoms in MNCD, yet has less D2 receptor blockade compared to risperidone. Differences in the pharmacodynamic profile and the pharmacokinetic profile of both agents, as well as past responses to the new agent, were considered.

17.3 Pharmacodynamic and Pharmacokinetic Changes

Pharmacokinetic changes in the geriatric patients involve processes of absorption, distribution, metabolism, and excretion, which have a significant impact on efficacy, tolerance, adverse effects, and drug-drug interactions. These physiological processes can be summarized as follows:

- *Absorption*: the proportion of medication absorbed orally is usually not significantly changed in the elderly. However, aging does reduce gastric acidity and splanchnic blood flow and lowers intestinal motility. As a result, some medications may have slower absorption and a delayed onset of effect (Chap. 3: Pharmacologic Overview in Geriatrics).
- *Distribution***:** aging is usually accompanied by a loss of lean body mass and an increase in the percentage of the total body fat, resulting in an increase in the volume of distribution and longer duration of action for some fat-soluble drugs (e.g., diazepam). Typical decreases in the total body water with age result in a decreased volume of distribution of water-soluble medications (e.g., lithium), leading to an increase in concentration.
- *Metabolism*: reduced liver mass, with age, leads to decreased hydroxylation and demethylation, resulting in the accumulation of medications (e.g., diazepam).
- *Excretion*: many factors contribute to a reduced renal function in geriatric patients; there is a 35% loss of function by 65 years of age and a 50% loss of function by 80 years of age. Reduced renal clearance causes the accumulation of many medications, necessitating a reduction in dosing and/or dosing frequency.

17.3.1 Receptor Sensitivities Also Change with Aging

- Increased sensitivity of the GABA-aminergic system, resulting in heightened response to benzodiazepines
- Decreased cholinergic activity resulting in increased sensitivity to anticholinergic agents
- Decreased serotonin reuptake with an increase in 5-hydroxy-tryptamine (serotonin) 2A (5-HT2A) receptor concentrations
- Decreased dopamine turnover resulting in increased risk of developing drug-induced parkinsonism

The presence of multiple medications can compete and/or antagonize each other at the receptor site, to a lesser effect or an enhanced effect (e.g., anticholinergic medication in combination with acetylcholinesterase inhibitors can result in competition for the cholinergic receptor. This can result in worsening of cognitive effects due to the anticholinergic effects and the blunting of the acetylcholinesterase inhibitor effect).

The benefit of treating patients with multiple medications must be balanced with the known risk of prescribing multiple medications, especially those medications listed on the Beers criteria list [4]. The Beers criteria provide guidance on the avoidance of medications that may place the geriatric patient at risk.

17.4 Medication Interventions Which Are Best Accomplished in an Inpatient Setting

17.4.1 Switching Antidepressants

There are four common strategies for switching antidepressants. The choice of strategy depends upon many factors, including the reason for switching, the adverse effect profile, efficacy, interactions between the medications, elimination half-life of both current and new antidepressant, the current medical condition(s), and medication history.

- *Direct switch strategy*: the current antidepressant is abruptly discontinued, and the new antidepressant is initiated. The main advantage of this approach is that it is quick and simple. This strategy is more suitable if the current antidepressant has been taken for 4 weeks or less and as a result is less likely to cause discontinuation syndrome. Direct switching can be considered where a severe adverse effect has occurred with the current antidepressant. The strategy carries the least risk if the current and new antidepressants have a similar mode of action (e.g., switching from one selective serotonin reuptake inhibitor (SSRI) to another; the exception is if the current antidepressant is fluoxetine). The main disadvantage of this approach would be the risk of discontinuation syndrome with the current antidepressant.
- *Taper and switch strategy*: the current antidepressant is gradually tapered and stopped; and the new antidepressant is initiated after tapering the first antidepressant is completed. The main advantage of this approach is that it can reduce the risk of discontinuation syndrome; however, this strategy requires more time than a direct switch. The strategy is recommended when switching from one SSRI to another or to an serotoninnorepinephrine reuptake inhibitor (SNRI), when there is concern over excess serotonin, seen in the serotonin syndrome (Chap. 15: Special Syndromes: Serotonin Syndrome, Neuroleptic Malignant Syndrome, and Catatonia).
- *Taper and washout strategy*: the current antidepressant dose is gradually lowered and discontinued, followed by a *washout (drug-free interval)* before the new antidepressant is started at a low dose. The advantage is safety. However, the risk is that the patient is *without treatment for a period*. The rationale is that the patient has not been responding fully to the first antidepressant and there is little to lose during a washout of that medication. The washout period is dependent on the half-life of the first drug. Most of the drug is eliminated from the body within five half-lives. This strategy is most commonly recommended when switching from non-selective, irreversible monoamine oxidase inhibitors (MAOIs) due to the risk of serotonin syndrome. If the current antidepressant has a relatively short half-life, a washout period of 2 weeks is required before starting an MAOI. If the current antidepressant has a long half-life (e.g., fluoxetine), a washout period of 5–6 weeks is recommended before starting an MAOI.
- *Cross-taper strategy*: this is the most widely used strategy. The current and first antidepressant dose is gradually tapered while simultaneously initiating the new antidepressant at a low dose. The tapering process continues while the new antidepressant is increased to reach a patient response and/or therapeutic range. In this approach, the patient will briefly use both medications at the same time. The advantage of this strategy is that the patient does not have an interruption in therapy. The risk is that there may be period of time with increased adverse effects (e.g., serotonin syndrome, hypotension, drowsiness) *due to the combination of two drugs* and possible drug-drug interactions.

Whichever strategy is implemented, the patient should be closely monitored for adverse effects. Table 17.1 provides a summary of recommended strategies for switching antidepressants.

Table 17.1 Recommended strategy when switching antidepressants

Switching from→Switching to	Non-selective irreversible MAOI	SSRI (except fluoxetine)	Fluoxetine	SNRI	Tricyclic	Mirtazapine	Moclebemide	Viorxetine	Trazodone	Vilazodone	Bupropion
Non-selective irreversible MAOI	Taper/ washout (2 weeks)	Taper/ washout (1 week)	Taper/ washout (5 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)
SSRIs (except fluoxetine)	Taper/ washout (1 weeks)	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1	Cross-taper	Cross-taper	Cross-taper	Cross-taper
Fluoxetine	Taper/ washout (2 weeks)	Cross-taper		Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1	Cross-taper	Cross-taper	Cross-taper	Cross-taper
SNRI	Taper/ washout (2 weeks)	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1 day)	Cross-taper	Cross-taper	Cross-taper	Cross-taper
Tricyclic	Taper/ washout (2 weeks)	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1 day)	Cross-taper	Cross-taper	Cross-taper	Cross-taper
Mirtazapine	Taper/ washout (2 weeks)	Cross-taper	Cross-taper	Cross-taper	Cross-taper		Taper/ washout (at least 1 day)	Cross-taper	Cross-taper	Cross-taper	Cross-taper
Moclebemide	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (5 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)		Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Taper/ washout (2 weeks)	Cross-taper
Vortioxetine	Taper/ washout (2 weeks)	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1 day)		Cross-taper	Cross-taper	Cross-taper
Trazodone	Taper/ washout (2 weeks)	Cross-taper	Cross-Taper	Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1 day)	Cross-taper		Cross-taper	Cross-taper
Vilazodone	Taper/ washout (2 weeks)	*Taper and switch immediately*	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1 day)	Cross-taper	Cross-taper		Cross-taper
Bupropion	Taper/ washout (2 weeks)	*Taper and switch immediately*	Cross-taper	Cross-taper	Cross-taper	Cross-taper	Taper/ washout (at least 1 day)	Cross-taper	Cross-taper		

17.5 Switching Antipsychotics

In geriatric patients, a switch to an alternative antipsychotic may be necessary in the case of nonresponse, partial response, or adverse effects. A switch may be especially indicated when a patient's behavior or psychiatric symptoms are dangerous to self or others. In considering how to switch between antipsychotics, it is important to consider the clinical situation, as well as the pharmacology and pharmacokinetics of the original antipsychotic, the new antipsychotic, and past responses to other antipsychotics.

Differences in the pharmacodynamic profiles between the two agents are crucial. The pharmacodynamic profile refers to the receptor affinity—how tightly the medication binds to specific receptors. The greater the difference in the receptor affinity between the two agents, the more likely the patient may experience effects related to the switch, requiring more caution. For example, if the current antipsychotic binds tightly to the muscarinic receptor (e.g., olanzapine, quetiapine, clozapine) and the new agent being considered has little to no binding to the muscarinic receptor (e.g., aripiprazole, risperidone, ziprasidone), the patient may experience a transient anticholinergic rebound, with anxiety, insomnia, or agitation.

In a similar manner, when switching from a tighter D_2 binding agent to a looser binding agent (e.g., from risperidone to quetiapine) or to a partial D_2 agonist (e.g., aripiprazole), the patient may experience a transient worsening of psychosis, mania, or aggression/agitation [5]. An abrupt switch has the greatest potential for rebound and withdrawal phenomena [5]. Even a conventional cross-taper can lead to problems when the current antipsychotic has a shorter half-life and/or blocks more tightly the cholinergic, histaminergic, or dopaminergic receptors than the new antipsychotic. Rebound phenomena can be minimized by avoiding abrupt or fast switching when the current, newer antipsychotics vary greatly in their receptor affinities and/or half-life [5]. Table 17.2 can provide guidance when considering potential effects of switching between second-generation antipsychotics (SGAs).

Table 17.2 Switching antipsychotics

	Examples	Rebound receptor effects
D_2 Blockade Switching from an antipsychotics with strong binding affinity to D_2receptor to an agent with a looser binding affinity or to a D_2 partial agonist	Risperidone to quetiapine Risperidone to aripiprazole	Psychosis Mania Agitation Akathisia Dyskinesia
M1 Muscarinic blockade Switching from an antipsychotic with strong binding affinity to the M1 receptor to an agent with weaker or no binding to the M1 receptor	Clozapine to aripiprazole Clozapine to ziprasidone Olanzapine to aripiprazole Olanzapine to ziprasidone	Flu-like symptoms Nausea Vomiting Diarrhea Diaphosesis Insomnia Agitation Anxiety
H1 Histaminic blockade Switching from an antipsychotic with strong binding affinity to the H1 receptor to an agent with weaker or no binding to the H1 receptor	Clozapine to aripiprazole Clozapine to risperidone Olanzapine to aripiprazole Olanzapine to risperidone Quetiapine to aripiprazole Quetiapine to risperidone	Rebound insomnia Restlessness Agitation
Alpha-1 blockade Switching from an antipsychotic with strong binding affinity to the Apha-1 receptor to an agent with weaker or no binding to the Alpha-1 receptor	Risperidone to aripiprazole Quetiapine to aripiprazole	Tachycardia Hypertension
5-HT2 blockade Switching from an antipsychotic with strong binding affinity to the 5-HT2A receptor (5-HT2A antagonist to an agent with weaker binding to the 5-HT2A receptor	Risperidone to quetiapine Risperidone to clozapine	Serotonin syndrome (agitation, diaphoresis, fever, tremor, confusion) NMS symptoms

References to consult when switching between antipsychotics include www.switchrx.ca, which provides recommendations for switching as well as monitoring parameters. Note that most references are tailored to the outpatient setting, but a more rapid switch can be considered in the inpatient setting, depending on patient factors and monitoring parameters that can be implemented [www.switchrx.ca].

In order to mitigate or avoid discontinuation syndromes for various medications, as described below in section "Discontinuation Syndromes", it is useful to consider some recommended steps in the withdrawal and tapering of antipsychotics, antidepressants, and benzodiazepines, as provided in Table 17.3.

17.6 Discontinuation Syndromes

Psychotropic medications may need to be discontinued for several reasons, including adverse drug reactions, drug interactions, absence of a current indication, risks outweighing the benefit, and change in medical status. Each patient should have an individualized assessment of their current medication regimen, which includes a review of the indication and benefit/harm for each drug. Medication discontinuation can often be an appropriate clinical decision and may result in significant clinical benefit. In deciding whether to discontinue a medication, consider the likelihood that the medication is benefiting or stabilizing the current clinical situation, the wishes of the patient or their

Table 17.3 Withdrawal and tapering

Abrupt withdrawal	May be required in situations such as an acute or severe adverse reaction
	May be appropriate in an inpatient setting where patient can be closely monitored
	May be appropriate for medications that have been prescribed at low doses for a short period of time
	Monitor for exacerbation or re-emergence of symptoms
	If medication has anticholinergic properties monitor for cholinergic rebound symptoms
Tapering medications	Start by halving the dose
	Continue to taper by quartering the dose
	Where possible, stop only one medication at a time
	Length of taper will depend on duration of treatment, urgency of discontinuing, patient tolerability
	Adjust/modify the rate of taper depending on patient factors
Antidepressants	If duration of antidepressant treatment has been 4 weeks or more, taper the medication, if possible
	Withdrawal symptoms for antidepressants can include anxiety, gastrointestinal disturbance, insomnia, irritability, headache, and myalgia
	SSRIs/SNRIs
	Tapering is recommended where possible
	Fluoxetine, at low doses, may not need to be tapered due to its long half-life
	Substituting one dose of fluoxetine near the end of the SSRI taper can be used to help reduce discontinuation symptoms
	Due to shorter half-life, a discontinuation syndrome is more likely to occur with paroxetine and venlafaxine
	TCAs
	Taper slowly where possible
	Rapid tapering or abrupt withdrawal may produce symptoms associated with cholinergic rebound (agitation, sweating, gastrointestinal symptoms, and headache)
Antipsychotics	Low dose antipsychotics prescribed for insomnia can generally be discontinued abruptly, especially if it has been used for 6 weeks or less
	The pharmacodynamic and pharmacokinetic profiles of the medication being discontinued should be considered when discontinuing an antipsychotic
Benzodiazepines	A slow taper of a benzodiazepine is recommended, when possible
	The greatest risk of discontinuation syndrome is during the final quarter of the benzodiazepine taper

family, the clinical indications, and harm or potential harm. Full informed consent should be obtained for the discontinuation of medications as well as the addition of medications and even for combinations of medications (Chap. 5: Legal Aspects).

Several resources have been developed to help identify potentially inappropriate prescribing and/or high-risk medications in geriatric patients. These resources include the Beers criteria and the STOPP/START tools. These tools, along with careful clinical judgment, can be useful when reviewing medications. Medications that may be inappropriate in the geriatric patients include tertiary tricyclic antidepressants (e.g., amitriptyline or diphenhydramine due to their strong anticholinergic effects and benzodiazepines due to its risk of falls and sedation). Figure 17.2 provides a summary of tools available for prescribing medications in geriatric inpatient settings including STOPP/START and the Beers Criteria.

- STOPP

Screening Tool of Older Persons' Potentially Inappropriate Prescriptions

- START

Screening Tool to Alert Right Treatment

- BEERS CRITERIA

Medications to avoid in geriatric patients, underprescribing, interactions,and duplications

Fig. 17.2 STOPP/START tools and Beers criteria

17.6.1 Antidepressant Discontinuation Syndrome (ADDS)

Antidepressant discontinuation syndrome (ADDS) has been included as a new category in the DSM-5, as a medication-induced movement disorder. ADDS is defined as a set of symptoms that can occur after an abrupt cessation (or marked reduction in dose) of an antidepressant medication that was taken continuously for at least 1 month [6].

There remains some debate as to the biological mechanism underlying ADDS; there is agreement that it can occur following the withdrawal of any antidepressant. It is commonly seen with tricyclic antidepressants (TCAs), MAOIs, SSRIs, and SNRIs, particularly those with short half-lives. Possible exceptions may be agomelatine or vortioxetine [7].

ADDS symptoms can be vague when reported, and it is sometimes a challenge to determine if a patient is experiencing ADDS, a relapse of the underlying condition, a physical disorder, or side effects of a new or concurrent medication. Consideration should be given to all possibilities when assessing a patient. ADDS is reported in up to 40% of patients [8]. The inpatient setting is particularly well-suited for accelerated tapering and/or abrupt discontinuation, especially if there is an urgent need to discontinue the medication or reduce the dose. The patient can be monitored closely, medication intake controlled, and observations are around-the-clock.

17.6.2 Presentation of Antidepressant Discontinuation Syndrome (ADDS)

Some patients experience little to no discomfort when tapering or discontinuing antidepressants, while others are more severely affected [9]. In general, symptoms of ADDS begin within 2–4 days and typically include reported sensory and somatic symptoms, including flashes of lights, "electric shock" sensations, nausea, and hyperresponsivity to noises or lights. Some may report nonspecific anxiety and feelings of dread [6]. For the diagnosis of ADDS, symptoms should not be present prior to the dose reduction or explained by another psychiatric disorder [6].

There are differences among the classes of antidepressants with regard to specific ADDS symptoms reported. SNRIs and SSRIs are more likely to be reported as "flu-like symptoms," nausea, leth-

argy, dizziness, ataxia, electric shock sensations, anxiety, irritability, insomnia, and vivid dreams [9]. TCAs are associated with nausea, headache, abdominal pain, diarrhea, lethargy, anxiety, insomnia, and vivid dreams [9]. Abrupt discontinuation of TCAs can lead to an anticholinergic discontinuation syndrome characterized by cholinergic rebound. These symptoms include nausea, vomiting, diarrhea, sweating, headache, and muscle cramps. MAOI discontinuation is associated with agitation, irritability, mood disorders, dreams, cognitive impairment, and, occasionally, psychosis and delirium [9]. Table 17.4 summarizes the range of symptoms reported in ADDS by drug class.

Finally, some geriatric patients may be unable to express or report specific symptoms (Chap. 19). Changes in behavior, sleep patterns, and physical function are important monitoring parameters when tapering or discontinuing antidepressants.

17.6.3 Prevalence of ADDS

The prevalence of ADDS is unknown, but is thought to vary according to the dosage prior to discontinuation, the half-life and receptor-binding affinity of the medication, and possibly the individual's genetically influenced rate of metabolism for this medication [6]. Patients who experienced adverse symptoms during the early phase of treatment were more likely to suffer from a discontinuation syndrome upon withdrawal and should be monitored closely during discontinuation [10].

17.6.4 Management of ADDS

When possible, taper antidepressants to lessen the risk of a patient experiencing ADDS. There is no consensus on the optimal rate of tapering an antidepressant to avoid ADDS. A gradual dose reduction does not always eliminate ADDS; some patients and/or practitioners may prefer to abruptly discontinue treatment in order to curtail the period in which symptoms are experienced [10]. The general consensus is that tapering is unnecessary for patients who have been taking an

Table 17.4 Antidepressant discontinuation syndrome (ADDS)—symptoms reported by drug class

Drug class	Symptoms
SSRI & SNRI	Flu-like symptoms
	Nausea
	Lethargy
	Dizziness
	Ataxia
	Electric shock sensations
	Anxiety
	Irritability
	Insomnia
	Vivid dreams
TCA	Nausea
	Headache
	Abdominal pain
	Diarrhea
	Lethargy
	Anxiety
	Insomnia
	Vivid dreams
	Anticholinergic discontinuation syndrome – characterized by cholinergic activity
	Nausea
	Diarrhea
	Rebound
	Vomiting
	Sweating
	Headache
MAOI	Agitation
	Irritability
	Mood disorders
	Dreams
	Cognitive impairment
	Psychosis and delirium
Antipsychotics	Psychosis
	Aggression
	Agitation
	Delusions
	Hallucinations
Benzodiazepines	Anxiety symptoms
	Autonomic instability
	Increased heart rate and blood pressure level
	Tremulousness
	Diaphoresis
	Insomnia
	Sensory hypersensitivity
	Acute withdrawal
	Seizures
	Delirium tremens

antidepressant for 4 weeks or less as this is insufficient time to develop a withdrawal reaction [10]. However, as some short-acting agents have been reported to cause ADDS with missed doses, it is often advisable to taper, where possible.

If symptoms are mild, reassure the patient that symptoms are common, will be short-lived and self-limiting, and do not indicate dependence on the drug. If symptoms are severe or uncomfortable for the patient, reintroduce the antidepressant or increase the dose or substitute with another agent from the same class that has a longer half-life, and gradually taper while monitoring for symptoms [10]. Consideration can be given to adding an agent to help manage ADDS symptoms. For example, an anticholinergic agent may be useful in ameliorating the gastrointestinal (GI) symptoms associated with TCA withdrawal [7, 10]. Benzodiazepines may be useful for those experiencing insomnia [10], although benzodiazepines carry their own risks for geriatric patients.

A concern in treating ADDS with additional medications (e.g., adding a benzodiazepine for the management of insomnia) is the risk that the medication may not be reassessed or removed after that patient no longer had an indication for its use. Clinical experience has demonstrated that for benzodiazepines, once started, subsequent providers may not discontinue it and the patient becomes habituated to its use**.** Adding the indication for use on prescriptions may help reduce the risk of continuing the medication without a current indication.

Fluoxetine, an SSRI with a long half-life, can be a useful tool to manage ADDS. The use of one dose of fluoxetine near the end of the tapering schedule for SRRIs, venlafaxine, or clomipramine can help alleviate discontinuation symptoms. One may also prefer to substitute fluoxetine at the beginning of the tapering process.

17.6.5 Antipsychotic Discontinuation

When discontinuing antipsychotics, a general approach is to decrease the dose by 25–75% weekly. Higher doses should be tapered over a period of 2–4 weeks. If a patient is prescribed a low dose for primary insomnia or secondary insomnia, and underlying comorbidities are managed, abrupt discontinuation can be considered. Mild withdrawal symptoms can include nausea, vomiting, sweating, and insomnia.

Avoidance of the re-emergence/exacerbation of psychotic symptoms, aggression, agitation, delusions, and hallucinations is crucial during antipsychotic medication changes. Guidance should be sought by conducting a past history of prior discontinuations: how the patient's psychotic symptoms have responded to previous antipsychotic medication doses and formulations. It is important to determine doses which, in the past, have effectively controlled psychotic symptoms and doses which have precipitated specific adverse effects and to calculate equivalent doses of the new medication accordingly. The medication adherence history is also important, since it is possible that a patient's lack of full response to a medication may be the result of missing doses, or inadequate dosing, rather than "treatment resistance."

When discontinuing antipsychotics, an abrupt or gradual discontinuation can be considered. Abrupt antipsychotic discontinuation has been linked with dopamine super-sensitivity syndromes, rebound syndromes, and emergence/exacerbation of symptoms. Abrupt withdrawal of a medication that strongly antagonizes one or more receptors results in the exposure of sensitized receptors, leaving them potentially vulnerable to excessive stimulation. Adverse effects including dopaminergic rebound, cholinergic rebound, histaminic rebound, or serotonergic rebound may occur

A gradual tapering approach is generally considered to have less risk of withdrawal effects despite a meta-analysis that demonstrated no significant difference in clinical outcomes between immediate and gradual discontinuation of antipsychotics in schizophrenia [11]. In the absence of any strong scientific evidence or compelling clinical factors to the contrary, it is recommended to follow a slow taper in order to minimize any discontinuation effects.

The pharmacokinetic and pharmacodynamic profiles of the antipsychotics being considered help direct recommendations for switching

between different antipsychotics. Elimination half-life and the metabolism of these drugs are of paramount importance. Antipsychotics that have a long elimination half-life (e.g., risperidone, olanzapine, or aripiprazole) may present with fewer problems when discontinuing or when an abrupt or immediate change is required. Long half-life antipsychotics may present with a greater risk of pharmacodynamic interactions with other antipsychotics that are similar in terms of their affinity for certain receptors.

17.6.6 Benzodiazepines

17.6.6.1 Prevalence

The most common uses of benzodiazepines are to treat anxiety and sleep disturbances. While effective for both conditions, the medications have risks, especially when used over long periods. These risks are highest in the geriatric population. Long-term use can lead to dependence and withdrawal symptoms when discontinued. In geriatric subjects, research has shown that benzodiazepines can impair cognition, mobility, and driving skills and they increase the risk of falls. An association with suicidality has also been reported (Chap. 8: Suicide). Despite the known risks of benzodiazepine use in geriatrics, a patient in the 65-80-year-old range is more likely to be prescribed a benzodiazepine than any other age group [12]. Prescribers, who feel a particular sympathy to the aging patient, may offer a benzodiazepine, in the hopes of an immediate relief of distress and anxiety.

Individuals are more likely to use benzodiazepines if they are older, female, or widowed, separated, or divorced, have lower income, and are experiencing poor physical or mental health [13]. A 2014 National Institute for Health (NIH) study reported that 8.7% of geriatric patients were prescribed a benzodiazepine and 31% of those patients were prescribed a benzodiazepine for long-term use [14]. Inpatient prescribing of benzodiazepines is predicted to be even higher. Geriatric patients are commonly prescribed benzodiazepines during a hospital admission to assist with sleep induction or maintenance or to manage medication side effects. Patients who are prescribed benzodiazepines during a hospital admission are more likely to continue the medication post-discharge, putting the patient at risk of benzodiazepine dependence and risk of experiencing adverse effects.

17.6.7 Presentation of Benzodiazepine Discontinuation Syndrome

Discontinuation syndrome with benzodiazepines typically starts within 2 days after discontinuing a short-acting benzodiazepine and between 2 and 10 days after stopping a long-acting benzodiazepine. In geriatric patients, particularly with poor metabolism, the onset of discontinuation symptoms may not appear until up to 3 weeks later (Chap. 10: Alcohol and Substance Intoxication/Withdrawal).

Withdrawal effects from therapeutic dosages of benzodiazepines are mainly anxiety symptoms. In addition, autonomic instability (i.e., increased heart rate and blood pressure level, tremulousness, diaphoresis), insomnia, and sensory hypersensitivity are common. The most serious acute withdrawal symptoms are seizures and delirium tremens, which most commonly occur with abrupt discontinuation. Most patients who have been taking therapeutic dosages of benzodiazepines for more than a few months are likely to experience withdrawal symptoms, and the severity of withdrawal symptoms generally depends on the amount of the original dosage, the rate at which the dosage is tapered, the selection of patients, and the definition of withdrawal symptoms.

17.6.8 Benzodiazepine Tapering Regimens

There are no standard tapering regimens; the rate of tapering depends on the prescribed dose, duration of therapy, risk of relapse, and how well tapering is tolerated by the patient. To reduce the risk of discontinuation symptoms, abrupt discontinuation after long-term use is generally not indicated; a gradual taper is recommended.

But a more rapid tapering schedule can be implemented in the inpatient setting, where the patient can be medically supervised. In general, at higher doses (e.g., greater than 10 mg diazepam equivalents per day), the dose may be tapered more rapidly. Once the patient achieves 10 mg, the dose should be tapered more slowly [15]. A patient is at the greatest risk of discontinuation syndrome during the final quarter of the benzodiazepine dose reduction. There is no published evidence to suggest that switching to a long-acting benzodiazepine reduces the incidence of withdrawal symptoms or is more effective than tapering a short-acting benzodiazepine. During the latter part of the benzodiazepine taper, consider dosing every 2–3 days or switching to lorazepam or oxazepam for the final taper.

17.6.9 Interventions to Manage Benzodiazepine Discontinuation Symptoms

Help the patient and/or caregivers to understand the rationale for discontinuing the medication, including the risks associated with long-term use of a benzodiazepine and reduced efficacy of the medication with long-term use. Educate the patient and/or caregivers that discontinuation symptoms can occur but are usually mild, transient, or short term. Obtain and document full informed consent for the withdrawal of a medication and the risks and benefits (Chap. 5: Legal Aspects).

Cognitive behavioral therapy (CBT) can help manage symptoms, prevent relapse, and treat the underlying condition that led to the initial use of the benzodiazepine prescription. CBT has been shown to be an effective addition to the gradual discontinuation of benzodiazepines in geriatric patients experiencing symptoms of withdrawal, including anxiety and/or insomnia [16]. Evidence-based psychological interventions to manage symptoms when discontinuing benzodiazepines are available at the National Institute for Health and Care Excellence (NICE) website; these interventions are not specific to geriatrics.

17.7 Drug Interactions and Combinations

Geriatric patients prescribed combination psychotropic medications or multiple medications as a result of comorbidities are at risk of drug interactions. Drug interactions may present in a patient as treatment failure or as side effects. There are two main mechanisms by which drugs interact with each other: pharmacokinetic and pharmacodynamic interactions. Pharmacokinetic interactions are more common and involve alterations in absorption, distribution, metabolism, or excretion as a result of the addition of a second medication. In psychiatry, these interactions commonly involve cytochrome P450 family of enzymes. Many involve medications that can inhibit or induce the metabolism of other drugs through their effect on the CYP450 system (Chap. 3: Pharmacological Overview).

Pharmacodynamic interactions refer to interactions in which drugs influence each other's effects directly (e.g., sedative effects of CNS agents can potentiate each other). These effects can often be seen to a greater effect in geriatrics. For example, the synergistic anticholinergic activity of amitriptyline combined with benztropine can produce constipation, heat stroke, urinary retention, and other related difficulties. Serotonin syndrome, which results from the combination of serotonergic agents, occurs frequently (Chap. 15: Special Syndromes: Serotonin Syndrome, Neuroleptic Malignant Syndrome, and Catatonia). The assessment of drug regimens for possible drug-drug interactions should include reliable drug-drug interaction resources, such as those often built into electronic medical record (EMR) software, and include over-the-counter, natural, and herbal products when assessing interactions. Consider the characteristics of the drug, the width of therapeutic index, and the patient's renal and hepatic function. Table 17.5 provides examples of potentially dangerous drug-drug interactions and mechanisms of action.

Table 17.5 Examples of drug-drug interactions

Interaction	Concern	Mechanism	Comments
Two or more serotonergic agents (e.g., SSRIs, TCAs, SNRIs, tramadol, MAOIs, meperidine, dextromethorphan, St. John's wort)	Serotonin syndrome	Pharmacodynamic interaction: mechanisms that cause serotonin syndrome include increased serotonin production, inhibition of serotonin reuptake, inhibition of serotonin metabolism, increased serotonin release, and stimulation of serotonin receptors	A potentially fatal interaction. Serotonin syndrome usually develops within 2–24 h of the addition of a serotonergic agent and/or an increase in dose of a serotonergic agent. Signs and symptoms include cognitive or mental-status changes (e.g., agitation, confusion, delirium, hallucinations), neuromuscular abnormalities (clonus, hyperreflexia, increased muscle tone and spasms, rhabdomyolysis, rigidity, shivering, tremor); and autonomic hyperactivity symptoms (diaphoresis, diarrhea, fever, flushing, hypotension or hypertension, increased bowel sounds, mydriasis, increased respiratory rate, tachycardia, tearing)
Multiple medications that increase QT interval prolongation (e.g., haloperidol, ziprasidone, TCAs, marcrolide antibiotics)	QT prolongation, leading to risk of torsades de pointes	Pharmacodynamic interaction: additive QT prolongation	Consult references which identify risk (www.crediblemeds.org). Increased QT interval can lead to torsades de pointes, a potentially fatal arrhythmia. Assess patient's baseline risk prior to prescribing medications with known QT prolongation risk. Monitor ECG and electrolytes before and during treatment
Lithium when used in combination with NSAIDs, thiazide diuretics, ACE inhibitors, or ARBs	Lithium toxicity	Pharmacokinetic interaction: increased serum lithium concentration through decreased renal excretion or increased renal reabsorption	Use caution when interpreting lithium levels in the geriatric population. Normal adult therapeutic lithium levels can be toxic in the elderly population If interacting medications with lithium cannot be avoided, it is important to monitor closely.
Multiple medications that inhibit acetylcholine or block	Anticholinergic toxicity	Pharmacodynamic interaction: additive effect of decreased acetylcholine	Can lead to delirium confusion, falls
Smoking and antipsychotics	Decreased antipsychotic plasma concentrations	Pharmacokinetic interaction: polycyclic aromatic hydrocarbons in cigarette smoke induce the activity of CYP1A2, leading to decreased concentration of medications metabolized	Changes in cigarette smoking status (common when admitted to or discharged from hospital) may require an alteration in dosage

17.8 Summary

A geriatric inpatient psychiatry setting has many advantages for safe, efficient, and well-controlled review/adjustment of medication regimens. Indications for an inpatient hospitalization include the need for safe withdrawal, abrupt discontinuation, switching, dose adjustment, or trials of different agents. It is often difficult to determine if withdrawal symptoms or discontinuation syndrome symptoms are present, but close observation and attention to past medication history can help this determination. Consultation with a pharmacist is often helpful.

Take-Away

- Review all medications as potential causes or contributing factors to a patient's clinical presentation.
- Consider pharmacokinetic and pharmacodynamic changes when prescribing psychiatric medications in the geriatric population.
- Switching between antidepressants or between antipsychotics requires consideration of the pharmacokinetic and pharmacodynamic profile of the individual medications to determine the safest approach to switching.
- Discontinuation syndromes can be severe for geriatric patients. Document full informed consent of risks and benefits from patient and surrogate for any medication changes, including discontinuation syndromes as well as starting new medications.
- History of prior medications, dosing, tolerance, adverse effects, outcome of past switches, tapers, and combinations can offer guidance.
- Psychotropic medications can interact with other psychotropic and non-psychotropic medications.
- Benzodiazepine tapering: the greatest risk of discontinuation syndrome is during the final quarter of the benzodiazepine dose reduction.

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18 Psychotherapies and Non-pharmacological Interventions

Laura Kenkel and Caroline Giroux

18.1 Introduction

Many factors impact the mental health and quality of life of aging adults, particularly those residing in institutional settings. Geriatric patients may have experienced many losses, e.g., social connections upon retirement, the death of family members and/or friends, the reduction of independence, and the need to move into assisted living facilities [1]. Geriatric patients may also suffer from cognitive impairment, whereby the reduction of memory and reasoning skills adversely impacts independence and well-being [2]. The flowchart above in Fig. 18.1 displays many psychotherapeutic and non-pharmacological interventions available to improve coping with stressors and adaptation to losses.

Pharmacologic treatment is a standard approach for most psychiatric disorders, but there are many challenges to pharmacological treatment in geriatrics, e.g., pharmacokinetic (e.g., absorption, distribution, metabolism, excretion) changes, greater sensitivity to certain classes of medications (e.g., benzodiazepines, anticoagulants) [3], polypharmacy, and increased risk of adverse drug reactions [3] (Chap. 3: Overview of Pharmacology in Geriatrics). Adjunctive use or substitution of psychotherapy and other modalities for pharmacotherapy (when appropriate) is desirable given the aforementioned factors.

Aging adults have been found to accept psychotherapy as a treatment modality. One study (Hanson et al. 2008) examined aging adults' acceptance of cognitive therapy, antidepressant medication, or a combination of the two, for the treatment of depression. Geriatric patients were shown to find combination treatment more acceptable than either treatment alone [4]. A systematic review by Apóstolo et al. (2016) of non-pharmacological interventions for depression in aging adults indicated that cognitive behavior therapy, competitive memory training, reminiscence group therapy, problem adaptation therapy, and problem-solving therapy decreased depressive symptomatology, but did not lead to changes in secondary outcomes (cognitive function, quality of life) [5].

Research on the outcome of psychotherapy, specifically in geriatrics, has been studied, although to a lesser extent than in other populations. A 2015 systematic review by Jonsson et al. found very few studies which examined the cost-effectiveness, efficacy, and safety of psychological treatments for depressive disorder in adults

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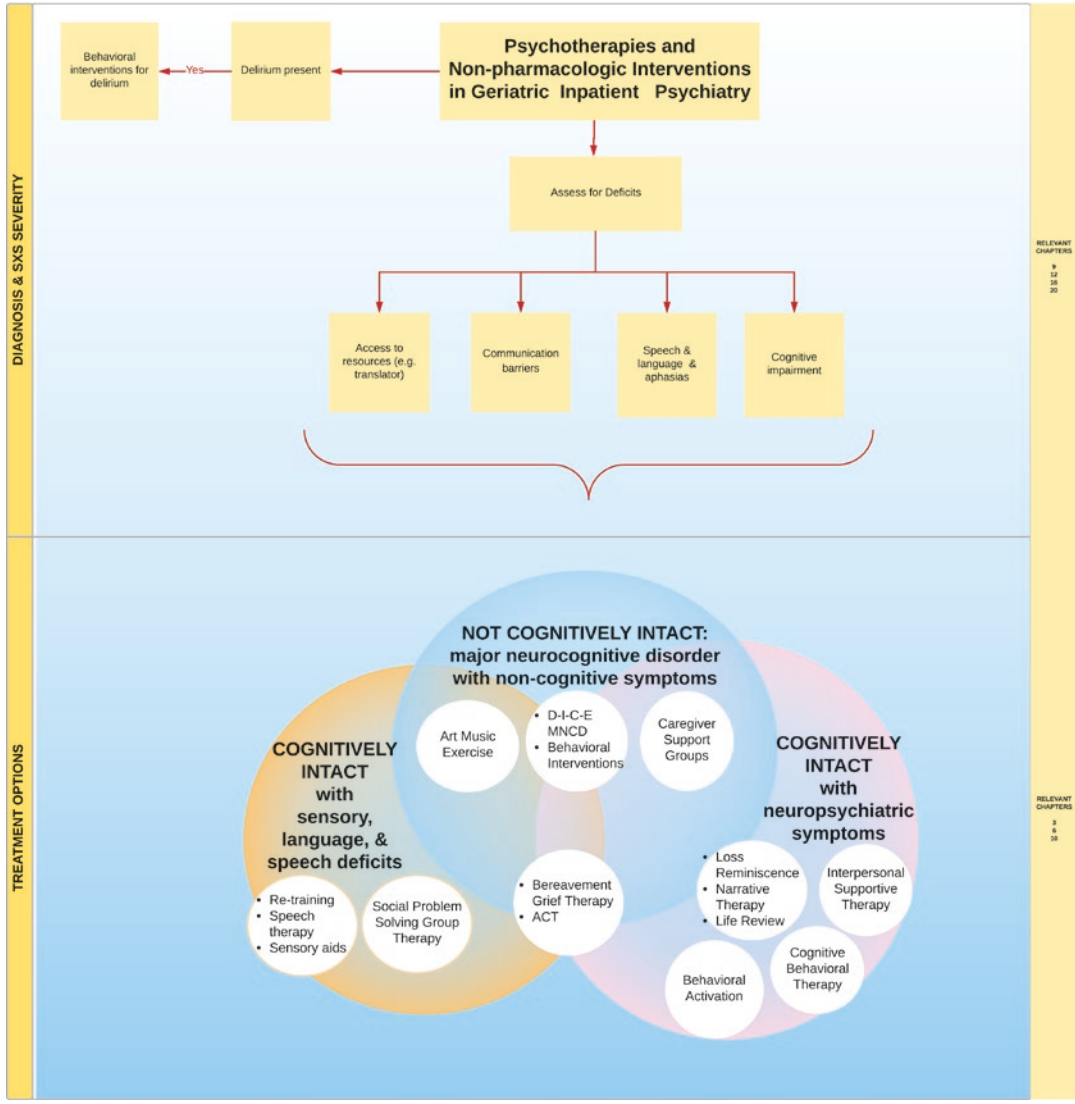

Fig. 18.1 Psychotherapeutic and non-pharmacological interventions

aged 65 and over [6]. The review noted that the generalizability of findings must take into account the deficits related to old age, such as frailty and cognitive impairment, as well as differences in depressive disorder types [6]. Although patients with cognitive impairments may not be suitable for cognitive behavioral therapy (CBT), they may be more able to access problem-solving therapy (PST) [6].

A 2008 Cochrane review of psychotherapeutic treatments for aging adults with depression discovered relatively few trials appropriate for inclusion [7]. Based on a meta-analysis of five trials (153 participants), CBT was more effective than waiting list controls. But in three small trials, no significant difference in treatment effect was found between psychodynamic therapy (PT) and CBT. CBT, however, was found *superior* to active control interventions when the Hamilton Depression Rating Scale was used, and CBT was found *equivalent* to active control when the Geriatric Depression Scale was used to measure outcome [7].

Another review of psychological treatments of late-life depression examined 17 studies of

different therapeutic interventions (Francis et al. 2013). The therapeutic modalities included cognitive behavioral therapy, problem-solving therapy, interpersonal therapy, reminiscence therapy, and brief psychodynamic therapy [8]. This review found that all therapies resulted in reduction of depressive symptoms. But conclusions could not be drawn regarding the superiority of one modality over another nor how to determine which treatment should be used for which patient [8]. Only one study was conducted in an acute inpatient (medical inpatient) setting [8].

When psychotherapeutic approaches alone are not successful, a combination of medications and psychotherapeutic modalities has been found helpful, including deep interpersonal psychotherapy, induction of altered states of consciousness, and art therapy [12–14] (Chap. 16: Neuromodulation Interventions; Chap. 17: Medication Strategies).

There is a paucity of research addressing the use of psychotherapeutic techniques for aging adults with psychotic disorders. Nikolitch et al. concluded that it was feasible to include brief (10 minutes) mindfulness-based approaches for patients with psychotic disorder, given their vulnerability to experience distress from voices or paranoid ruminations [9]. The study found that brief group mindfulness interventions are well-tolerated and suitable for acutely hospitalized psychiatric patients, including those with acute psychosis [9]. Mindfulness-oriented interventions with an active component (e.g., tai chi, mindful walking) may be best suited for this population.

18.2 Clinical Vignettes

Vignette 1

A 77-year-old, right-handed, widowed woman of German descent was admitted to neurology for a left ischemic stroke. There was a history of hypertension, hypercholesterolemia, and borderline diabetes. She was living alone when found on the bathroom floor by her son. In the ER, she presented with aphasia, right hemiparesis, and increased spasticity in on the right. She was distressed, confused, and agitated. She insisted on keeping her belongings because "neighbors are stealing."

There had been no other psychiatric symptoms or documented cognitive symptoms prior to the stroke, although her son had noticed forgetfulness for names, tasks, and deadlines. Some food items were found rotting in her refrigerator when her son found her. She had soiled herself after the stroke but there was no prior report of incontinence. The differential diagnosis included post-stroke hemiparesis, delirium, and neurocognitive disorder.

On the medical unit, a urinary tract infection was diagnosed, blood pressure was elevated, and blood sugar was high; these findings gradually improved. After a few days, the patient's mood seemed calmer. She was happy to see her son but still unable to speak. Physical and speech therapists began working with her. The patient often became frustrated because of her unsteady gait. After a few weeks, she became withdrawn and refused to eat. She was seen crying silently.

She simply stared in response when staff asked if she wanted to die. A psychiatric consultation was obtained due to concerns for a depressive disorder, which affects 30% of post-stroke patients.

At the bedside psychiatric consultation, the patient had hemiagnosia. She was able to answer close-ended questions reliably, but had difficulty forming sentences. Her son facilitated an interview which revealed that she was born during World War II. Her father, a successful merchant, immigrated with family to America when she was 2 years old. Most of her extended family died in concentration camps. The patient's mother died when she was 9 years old in a car accident. The patient denied morbid thinking or former suicidal attempts. Her son worried that she had little motivation to socialize or participate in group activities. She did not smile, was irritable, and looked angry when people didn't understand her. However, she appeared to respond positively and attempted to reposition herself closer to the psychiatrist when she was told that the psychiatric team would continue to see her.

The occupational therapist helped the patient join the unit's art therapy group. It emerged that the patient used to work as a seamstress. She didn't have fine motor control of her dominant right hand, but she was able to hold a piece of fabric while learning to use left-handed scissors. She seemed to enjoy hearing the rhythmic sound of the fabric being cut. An old sewing machine was found, and she practiced pressing the pedal with her right foot, without sewing anything, looking at the needle going up and down. With the assistance of staff, she started to sew straight lines to combine pieces of fabric. She insisted on doing everything her left hand could do, even ironing the fabric under supervision or cutting the thread.

When the patient tried to speak in the group, German words came more easily to her. Her children decided to help her reminisce; her older daughter brought in an old photo album of black-and-white pictures from their life in Europe. The patient joined the ongoing reminiscence therapy and life review group. Even though she was unable to speak English fluently again, she showed meaningful photos which others asked about. One was a photo of a serene woman holding a cello in an orchestra, which the patient examined intently. A male patient commented on how beautiful the woman in the photo was and remarked that the patient looked like her. The patient whispered "Mutti" (German for "mommy") and started crying. She kept repeating the word.

A German-speaking visiting medical student agreed to provide translation for a course of short-term interpersonal therapy. After 2 weeks of interpersonal therapy at three sessions per week, the patient was less anhedonic and more engaged in her autobiographical (reminiscence) therapy group. She started regaining her appetite, and she requested foods from childhood, such as sauerkraut. Her sleep improved. She participated more actively in the art therapy group and created a mixed media tribute to her deceased mother, with a collage of pictures and fabrics. Later, the patient did another collage for the unit which included a tribute to victims of many other catastrophes: a tree with pieces of fabric and glued copies of black-and-white pictures, including those of her relatives. She wrote an epitaph to them on a board with her right hand in German, using a big marker.

The patient's physical therapy progressed to the use of a walker and then a cane. She kept busy with art activities and groups while she was transitioning to assisted living for semi-autonomous people, located near her three children.

Discussion

Effective antidepressant treatment can involve "a combination of antidepressant medication and active behavior that includes search activity and different types of psychotherapy oriented toward the restoration of the right hemisphere functions: interpersonal deep psychotherapy (Schore 2003; Brody et al. 2001), induction of altered states of consciousness, and art therapy. The most modern methods in the treatment of depression help patients use their left hemispheric skills in order to partly compensate for the core and fundamental distortion – the deficiency of right hemispheric skills and an inability to feel themselves integrated in the poly-dimensional world through the creation of a poly-semantic context" (Hecht 2010). In Vignette 1, the patient's left hemispheric stroke had further impacted left brain functions and exacerbated underlying depressive tendencies. Both psychotherapeutic modalities utilized in the vignette (interpersonal therapy (IPT) and art therapy) have demonstrated efficacy for the improvement of brain functional asymmetry in depression. Since the patient in Vignette 1 had verbal abilities affected by the stroke, with possibly an underlying mild cognitive impairment prior to the stroke, cognitive therapy was not an appropriate modality. However, behavioral activation, using reminders from staff, and an aid at home after discharge were provided as modalities which can enhance brain health.

Vignette 2

A 71-year-old man with a history of bipolar disorder was admitted to the acute psychiatric unit with depression and a plan to overdose on Tylenol. His medical history included

uncontrolled hypertension, resulting in chronic kidney disease stage 3. His bipolar disorder had been well-controlled for years on lithium, but in discussion with his psychiatrist and nephrologist, he slowly tapered off lithium 4 months prior to admission. The patient presented to the clinic after his son found him sitting in a messy apartment, not having showered for days, and without food. On admission, he was disheveled and unshaven, wearing clothing that was too loose and showing psychomotor retardation and blunted affect. He had linear, but impoverished, thought process and voiced themes of worthlessness and being better off dead. There were no psychotic symptoms. He scored 30/30 on the Montreal Cognitive Assessment (MoCA). His past failed trials of divalproex, lamotrigine, carbamazepine, and quetiapine, and the declining renal function, led to an inpatient treatment regimen of aripiprazole 5 mg in the morning.

The patient gradually began to show improvement. A few days into the hospitalization, the patient began participation in the unit's Grief and Loss group and talked about lingering symptoms of grief related to the loss of his wife 10 years prior, accompanied by social isolation after retirement from his career as a civil engineer. But cognitive distortions, including "black-and-white thinking" and catastrophizing about the future, were noted. The psychiatrist provided psychoeducation about how cognitive patterns can become maladaptive in depression and briefly guided the patient through the "evidence for and against" his beliefs. CBT self-help materials were provided, including thought records and activity planning worksheets. Over their next few meetings, the psychiatrist reviewed the patient's progress in the readings and helped him sketch out an activity plan for the week following discharge. The group therapist reinforced these efforts with the patient during Discharge Planning and Coping Skills groups. By the time of discharge, the patient's depressive symptoms had significantly improved and he was no longer suicidal. He expressed optimism and a plan to reduce his isolation at home through volunteer activities and spending more time with his son.

Discussion

In cognitively intact older adults with depression, brief CBT interventions can be effective. The insights gained by other treatment team members (social workers, nurses, group therapists) can help to guide the therapeutic approach when there is not enough time for lengthy interviews. In this case vignette, "homework assignments" during his inpatient stay involved the patient actively and gave a sense of agency in his own recovery. Other team members provided assistance with these brief assignments; a discussion of these approaches was fruitful in group sessions, which helped diminish the patient's sense of isolation.

18.2.1 Cognitive Behavioral Therapy, Behavioral Activation, and Cognitive Stimulation (See Vignette 2)

In a 2014 meta-analysis, Cuijupers et al. reviewed 44 studies comparing psychotherapies to control groups, including other therapies and pharmacotherapies. They found that CBT is an effective psychotherapeutic modality in geriatrics. CBT, along with PST and life review therapy, were found to be more effective than the other psychotherapies studied [15]. A 2017 Hummel et al. study examined an early intervention CBT program for depressed geriatric patients who were medically hospitalized with acute systemic illnesses. Participants were limited to those with normal cognitive function or mild cognitive impairment; and all patients eligible for the study could partake in three group psychotherapy sessions during their inpatient stays [10]. Once discharged, patients were randomized to a psychotherapy treatment group or a control group (waiting list with usual care) [10]. Those participants in the therapy group continued to participate in weekly group psychotherapy sessions at the hospital's day clinic [10]. A manualized CBT program was used, with 15 90-minute sessions that were standardized for older adults living at home; sessions were facilitated by experienced psychotherapists who were qualified in geriatric therapy [10]. The psychotherapy group showed

significantly decreased depression scores compared to the control group and also had improvements in secondary outcome measures that examined physical, psychological, cognitive, and functional parameters [10]. Additionally, caregiver burden was decreased for the psychotherapy group [10]. This study demonstrated that CBT can be modified for use in group settings in acute general medical hospitalizations and could serve as a bridge for continued care/treatment upon discharge.

Levin et al. suggest that cognitive therapy (CT) can be integrated into acute general medical settings via psychosomatic medicine consultation [16]. The traditional approach to CT is necessarily modified by the acute inpatient environment, which is often short on time. While an assessment/data-gathering phase in outpatient settings results in a case formulation which precedes psychotherapy, in acute general medical settings, data gathering must be combined with CT interventions [16]. In the initial psychosomatic medicine consultant interview, once a physician has enough information for a preliminary formulation, they should employ appropriate cognitive or behavioral techniques [16].

Behavioral activation (BA) is a therapeutic approach that focuses on maintaining patient involvement in life activities (as opposed to withdrawing and becoming more less active) and helping patients connect the impact of meaningful activities on their mood [17]. Snarski et al. studied the use of BA in an inpatient geriatric psychiatric facility with participants who had a mean age of 72. The participants' diagnoses included schizophrenia, bipolar disorder, major neurocognitive disorder (MNCD) (dementia), schizoaffective disorder, psychotic disorder, and major depressive disorder [17]. The average MMSE (Mini-Mental State Examination) score of the group was 24.80 (cut-off score for inclusion was 18 or above) [17]. Eight 30-minute sessions were conducted over 4 weeks. The study found that participants in the BA group had improved GDS (Geriatric Depression Scale) scores, compared to participants in a treatment-as-usual group [17]. The authors suggested that BA may more useful for geriatric patients with cognitive impairment, than therapies such as CBT [17].

A 2016 study compared CBT to behavioral activation (BA) in terms of clinical effectiveness and cost-effectiveness [18]. Richards et al. recruited 440 participants and randomly assigned them to either BA or CBT groups [18]. Mental health workers were trained in BA and provided 60-minute sessions to the BA group participants; psychotherapists of various disciplines accredited in CBT conducted the CBT sessions [18]. The authors found that BA was more cost-effective than CBT and was not inferior to CBT in terms of improvement of depression [18]. Though this study was conducted in participants with a mean age of 43.5 years and conducted in an outpatient setting, it could still inform approaches to treating older adults in inpatient settings. Training staff of inpatient psychiatric units to conduct a simpler type of therapy is perhaps a more feasible way of improving access to psychotherapy in the acute psychiatric or medical hospital.

Cognitive stimulation has been shown to be effective in reducing symptoms of major NCD, in both individuals living in the community and in long-term care homes [2]. Van Zon et al. examined the use of cognitive stimulation administered by volunteers to residents of long-term care homes [2]. Volunteers were solicited from people already volunteering at the care homes and provided with 2 hours of training in how to structure their 20-minute sessions with each resident and how to administer exercises in reasoning, attention, and memory [2]. The volunteers met with the residents three times a week for 8 weeks [2]. The study found significant improvements in immediate memory (verbal, nonverbal, and learning) and verbal fluency, including in those older adults who carried a diagnosis of major NCD [2].

18.2.2 Acceptance and Commitment Therapy

Acceptance and commitment therapy (ACT) is a contextual behavioral science approach [19]. In this theory, psychopathology is caused by

"cognitive fusion" or the domination of verbal and cognitive processes over emotions, thoughts, memories, and bodily sensations [19]. According to ACT theory, people interpret their thoughts literally and stay in a problem-solving mind-frame, which leads to "experiential avoidance" [19]. As a result, their behavior is inflexible and ineffectual, leading to unhelpful behavioral patterns (impulsivity, inaction, avoidance, excessive social compliance) [19]. The sum of these patterns is termed "psychological inflexibility," which ACT theory posits as the core of most human suffering [19]. The goal of ACT is to increase psychological flexibility and change behavior so that it ultimately serves an individuals' personal/chosen values [19]. Figure 18.2 summarizes the main principles of ACT.

ACT has six core processes [20]. "Acceptance" is an active and conscious acceptance of inner experiences (thoughts, feelings, sensations) without accompanying effort to change or counter them [20]. "Cognitive defusion" refers to techniques that decrease the believability of thoughts/feelings/sensations experienced by a person [20]. "Being present" is the non-judgmental and direct experience of physiological (e.g., physical pain), psychological (e.g., sadness), social (e.g., having a conversation), and environmental (e.g., rain) events [20]. "Self as context" describes a person's awareness of their own internal and external experiences, which is often facilitated by mindfulness exercises [20]. "Values" refers to the qualities of chosen, purposeful action and "Committed action" refers to concrete goals achieved by effective action linked to a person's chosen values [20].

Gaudiano et al. utilized a modified ACT protocol for the individual treatment of psychiatric inpatients with psychotic symptoms in acute inpatient settings [21]. Each patient received an average of three ACT sessions, which were designed to be "stand-alone" sessions; the sessions began with an educational component focused on psychotic symptoms, followed by presentation of the ACT model [21]. Mindfulness and acceptance exercises were taught and practiced, and behavioral goals were discussed and explored within the framework of thoughts/emotions as potential barriers to goal attainment [21]. The sessions ended with a review and suggestions of exercises to practice prior to the next session [21]. This approach could be modified for use in a group format for inpatient psychiatric settings, or modules could be adapted for short-term individual interventions in medical settings. The modules could also be manualized, increasing usability for staff not formally trained in psychotherapeutic techniques.

Fig. 18.2 Main principles of ACT

In their study, Davison et al. found support for a 12-session (individual therapy) ACT-based intervention in reducing depressive symptoms in long-term care residents, which remained at lower levels at a 3-month follow-up [22]. While the study did not focus on acute inpatient settings, it does demonstrate the applicability and efficacy of the ACT model for older adults.

In their 2016 study, Villatte et al. divided acceptance and commitment therapy into two modules (ACT OPEN and ACT ENGAGED), which each had eight treatment sessions [23]. Participants were included not based on diagnostic criteria, but instead on clinically significant psychological distress; diagnostic interviews completed after participant selection revealed a mix of depressive and anxiety disorders [23].

ACT OPEN procedures focused on acceptance and cognitive diffusion processes to increase psychological flexibility and decrease detrimental reactions to feelings and thoughts [23]. ACT ENGAGED procedures focused on values and "committed action processes" in order to increase motivation and reinforce meaningful behaviors [23]. Both modules facilitated flexible self-awareness, as well as action based on awareness and intention [23]. The ACT OPEN module resulted in more improvement after each session, and more of a decrease in symptom severity, when

compared to ACT ENGAGED [23]. The ACT ENGAGED module resulted in better quality of life improvements when compared to ACT OPEN [23]. This modularized approach allows clinicians a choice between a process focused more on increasing self-awareness of reactions to thoughts/behaviors and a more motivation/action-oriented approach. Clinicians could choose the module better suited for their patients, both in terms of personality and physical capability to be more active or engage in behavioral change.

18.2.3 Interpersonal Therapy (See Vignette 1)

IPT is a short-term (12–16 sessions), manual-based treatment that was developed for treating depression in the early 1980s [24]. It incorporates elements of psychodynamic-oriented therapies (exploration, clarification of affect) and CBT (behavior change techniques, reality testing of perceptions) that are used to address four areas of conflict: unresolved grief, role transitions, interpersonal role disputes, and interpersonal deficits. These four areas, especially grief and role transitions (e.g., retirement), represent realities with which older populations are inevitably, sooner or later, confronted. IPT is a psychotherapy that is suitable for primary care and inpatient settings, and its basic principles can be taught to a variety of clinicians.

Within this approach, considerable effort is spent educating patients about the biopsychosocial model of depression (or other psychiatric illnesses, as indicated). Interpersonal relationships are seen as the stage upon which depression and/or other forms of distress are expressed. All of the patient's important relationships are systematically explored with regard to the degree of attachment they contain for the patient that may indicate a causal factor in the development of the depressive episode (e.g., role dispute). This short-term treatment makes no attempt to alter personality, but rather focuses on current problems. The therapist is a benevolent facilitator without inviting a deepened transferential relationship. Focusing on the here and now facilitates problem-solving as well (see below). The conversational style inviting the patient to tell his/her story is comfortable and helpful. Depending on the hospitalization length of stay and the patient's degree of engagement, it might be possible to implement such a therapy and complete the course by planning for three sessions per week, for instance.

Family members often misattribute problem behaviors to volitional acts of defiance, when they actually are features of executive dysfunction, a salient aspect of cognitive impairment. Fortunately, the therapy modality can be adjusted for these patients (IPT-ci) and the approach includes (1) remind the patient of abilities that remain intact that could be further developed to help to compensate for the lost abilities and (2) help the patient to foster new attachments, commensurate with his/her current abilities, and, when necessary, help the patient accept increased dependency on others. Furthermore, it is important to adapt the interventions, especially since deficits in executive functions are associated with a poor and unstable response to antidepressant medications [25].

In one study, IPT was found to be more effective in moderate to severe depression [26]. Mackin and Areán, 2005 [25], systematically reviewed the evidence base for psychotherapy as an empirically supported treatment of late-life depression. The review also found good support for the combination of medication and IPT to prevent relapse and recurrence of major depression in older adults, especially those who have recurrent major depression. Aging patients responded as well, albeit more slowly, than middle-aged patients. IPT may be most effective as a maintenance treatment when combined with an antidepressant medication for more severely depressed older adults [27].

18.2.4 Social Problem-Solving Therapy

Interventions in inpatient settings are usually time-limited in an unpredictable fashion, which can make the determination of a specific therapy timeline challenging. However, some approaches

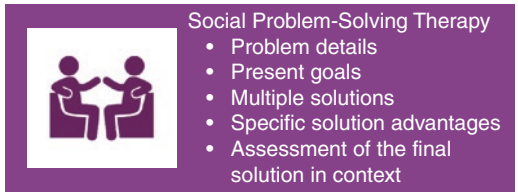

Fig. 18.3 Summary of issues addressed in social problem-solving therapy

can at least be initiated, with the recommendation of following up with a psychotherapist after discharge. Other approaches can be used in the inpatient setting in the form of tools. Social problem-solving therapy (SPST) is a good example of such an approach. Ineffective coping under stress (due to insufficient coping reserve compared to the number or severity of stressors) is hypothesized to lead to distress in the form of anxiety, depression, and even psychotic decompensation. Figure 18.3 provides a summary of issues addressed in SPST.

Such a step-by-step, practical approach presents significant advantages, especially for patients with cognitive decline or predominantly concrete thinking. Social problem-solving therapy has been shown to be associated with significantly greater improvements in depressive symptoms compared with reminiscence therapy or the waiting list [27].

18.2.5 Reminiscence Therapy and Life Review (See Vignette 1)

According to Socrates, the only life worth living is the examined life. Reminiscence and life review therapy, as their names indicate, consist of (1) remembering specific life events and (2) looking back at one's life trajectory. These approaches are derived from Eriksonian developmental theory and were specifically developed for older adults. Both techniques can lead to re-experiencing of personal memories and significant life experiences and to the identification or remembering of personal values and self-identity. Both approaches are totally patient-centered, as each person knows best about her/his own life.

Reminiscence therapy uses the recall of past events, feelings, and thoughts to facilitate pleasure, better quality of life, and better adjustment. It is valuable and very accessible in inpatient settings because it can be conducted during daily activities such as mealtime or walking around a facility. There does not seem to be reported adverse events to reminiscence therapy, and it can alleviate feelings of loneliness, anxiety, and depression [28]. Studies show that reminiscence therapy has a positive effect on older adults' psychological and subjective well-being. Self-esteem, satisfaction, and meaning are all measures that were improved by such interventions, while anxiety and depressive symptoms decreased [29]. They could even prevent depression in late life [29]. The positive effects persisted when measured at 1 and 3 years post-therapy [30]. Areán and Cook [27] say that reminiscence psychotherapy may be useful for treating depression in confused or older adults with early major NCD living in residential facilities.

Life review therapy is an advanced type of reminiscence, exploring problems through narration (verbal, written, or other). The life story is an internalized and evolving myth of the self, which provides unity and purpose in the individual's life [29]. It helps create a sense of internal coherence, which is healing especially when traumatic experiences have created a rupture in the person's life story. It is an especially relevant process for older adults as they face their last opportunity to sum up their life and its meaning, and hospitalization (whether for physical conditions or psychiatric decompensation) can act as a catalyst because of the sense of urgency older patients may feel regarding unresolved issues when they are acutely ill. Erikson emphasized that studying one's life story enhances an individual's sense of integrity, gratitude, and acceptance. In life review, individuals are encouraged to acknowledge past conflicts and to consider their meaning in their life as a whole. It is more structured and focused on both positive and negative life events. The life story reflects what a person leaves behind and how he/she wants to be remembered. Reviewing and writing one's life story appears to be therapeutic [31]. Authors of memoirs also mention the

significant advantage of keeping their linguistic skills alive. Additionally, in a group of patients with neurocognitive disorder (Alzheimer disease or other neurocognitive disorders), it was found that a cognitive rehabilitation model (using strategies such as remembering names) produced significant improvements on goal performance and satisfaction [11].

Life review therapy uses the normal reminiscing of aging to deepen the person's self-knowledge often with exercises such as photo scrapbook review, memoir writing, and pilgrimages to childhood sites [29]. For instance, one method to facilitate the emergence of positive memories is the use of narration and creative writing, stimulated by a specific prompt such as "tell us about your favorite teacher" or "describe a joyful family vacation."

Reminiscence psychotherapy is about focusing more on positive memories in group settings to improve self-esteem and social cohesiveness; people choose to tell the stories that help them present themselves [29]. In addition, involving others in our life stories as we review them allows for new perspectives. Inpatient settings can provide the team with a reasonable sample of potential participants. Therefore, reminiscence and life review therapy should be encouraged.

18.2.6 Group Psychotherapy

A distinct advantage of inpatient settings is the presence of various staff at all times who can co-facilitate group sessions. It is more efficient to do psychoeducation and discuss general lifestyle principles to a group rather than one-on-one.

Certain group interventions, particularly CBT, appear promising for use with depressed older adults. It is a great format to counteract certain negative experiences stemming from loneliness and grief. It helps build a sense of solidarity and gives an opportunity for practicing interacting with others in the case of patients who are about to be discharged to transitional living or a nursing facility. It may offer significant advantages to older people: it is less expensive than individual treatment and the social network provided by group therapy presents potentially superior therapeutic benefits for a cohort dealing with various losses. ACT has also been modified to include group formats. The addition of group therapy to individual sessions dramatically expands the contexts in which processes (e.g., mentalizing) can take place [32]. Certain forms of art therapy are also more stimulating in groups.

18.2.7 Art, Music, and Exercise

Modalities which engage the senses should be encouraged; for instance, the graphonomic process of Chinese calligraphy integrates mind and body. Hearing or playing a musical piece that lifts one's mood is a universal experience. Crafts, painting, and pottery can also procure the satisfaction of producing and restore self-esteem. Body-based therapies, e.g., yoga, tai chi, and mind-body techniques, can increase mindfulness and physical fitness, thereby improving health outcomes. They can also be used with patients with more severe symptoms like psychosis (see Introduction section). Figure 18.4 provides a summary of cost-effective modalities to engage geriatric inpatients.

Expressive art also intersects with life review modalities described above; e.g., creative writing presents the advantages of changing the narrative of a difficult life by allowing transformation and at the same time providing a product the patient can be proud of, a legacy for future generations. Painting can serve a similar function by using visual arts and symbolism, which is especially

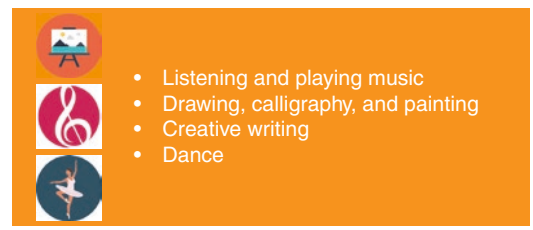

Fig. 18.4 Cost-effective modalities to engage geriatric inpatients

useful when some traumatic memories cannot be easily processed verbally. Esthetics is also part of the healing environment. Studies have shown that exposure to nature can improve health outcomes. When nature is not directly accessible, artwork can be displayed in care facilities. Realistic images are preferable to ambiguous artworks, which bear the potential for negative interpretations, especially in stressful environments like hospitals [33].

These modalities listed above are easily accessible and require inexpensive materials. In every inpatient unit, even in this digital era, it is easy to find a piece of paper and a pen. Handwriting and drawing are complex human activities that entail an intricate blend of cognitive, kinesthetic, and perceptual-motor features. The act of writing itself engages motor circuits and coordination, which can facilitate retention, hence an additional advantage for people suffering from memory decline. The meaningful coupling between action and perception during handwriting establishes sensory-motor memory traces. Intellectual leisure activities in later life, including calligraphy, may also delay cognitive deterioration.

A pilot study investigated the effects of calligraphy on cognitive function in older Hong Kong Chinese people with mild cognitive impairment, and it showed that calligraphy therapy can enhance spatial ability and sense of control [34]. There is some clinical evidence that Chinese calligraphic handwriting can help with behavioral change and emotional stability in patients with depression or cancer [35]. In a study by Kao et al., 8 weeks of Chinese calligraphic handwriting training had a significant attenuating effect on physiological parameters of arousal and its effectiveness compared favorably with that of meditation [35]. In summary, the act of calligraphic writing may train people's attention and concentration and result in relaxation and emotional stabilization. It can also improve the writer's cognitive activity [35, 36].

Different forms of artistic expression (e.g., dancing, drama, music) have positive effects on relaxation and emotional expressiveness [35]. Music has also been shown to help adults with major NCD. It is a universal language (through its symbolism and affect-laden qualities), which can stimulate non-language dominant structures, which generate the implicit self, the structural system of the human unconscious [37]. Direct access to these implicit processes by both patient and therapist is central to effective treatment.

Art therapy might also facilitate some transgenerational connections to one's ancestors and optimize the life review or reminiscence approaches. Again, therapy in general and art therapy in particular solicit mostly the right hemisphere and could help access collective, intuitive, holistic knowledge. The ability to mentalize is correlated with an interest in art, maybe because high levels of mentalization are associated with a sense of internal freedom to explore thoughts, feelings, desires, and experiences [32].

If possible, incorporation of physical exercise may augment the benefits of psychotherapy. Jacquart et al. found that psychiatrically hospitalized older adults with depression who participated in an exercise plus psychotherapy intervention had significantly lower scores on the GDS (Geriatric Depression Scale) than those patients who received psychotherapy only. Participants in the experimental group walked in the facility for 30 minutes daily. During that 30-minute walk, each participant received 20 minutes of validation therapy (preceded by a 5-minute warm-up and followed by a 5-minute cool-down) [38].

A review article by Theleritis et al. revealed that simulated presence (a personalized audiotapes approach) improved withdrawn behavior 69% of the time; it increased the level of interest more than placebo and usual care [39]. The stimulated retreat model of care (interdisciplinary care, activity programming, and family support) increased external engagement. Mindfulness-based stress reduction might also be effective for apathy [39]. Table 18.1 summarizes many of the aforementioned psychotherapeutic interventions which can be helpful for geriatric inpatients.

Apathy is a symptom of particular concern in the geriatric inpatient, because participation in treatment and the social environment is essential to overall well-being. Non-pharmacological treatments for apathy are quite safe and

Table 18.1 Psychotherapeutic interventions with potential benefit to geriatric psychiatry inpatients

Psychotherapy modality	Adaptable to group settings	Available as modules or manualized	Suitable for patients with psychotic disorders	Suitable for patients with cognitive impairment	Requires a trained therapist	Can be taught by staff/ volunteers
Cognitive behavioral therapy	Potentially	Yes	Maybe	No	Yes	No
Behavioral activation	No	No	Yes	Yes	No	Yes
Cognitive simulation	No	Yes (worksheets)	Maybe	Yes	No	Yes
Acceptance and commitment therapy	No	Yes	Yes	No	Yes	No
Interpersonal therapy	No	Yes	No	Maybe	Yes	No
Social problem-solving therapy	Yes	Yes	Yes	Yes	Maybe	Maybe
Reminiscence therapy & life review	Yes	Yes	Yes	Yes	No	Yes
Group psychotherapy	Yes	Depending on modality	Maybe	Yes (mild impairment)	Yes	Maybe
Art & music and exercise	Yes	Depending on modality	Yes	Yes	No	Yes

well-accepted. Per Cohen-Mansfield et al. (as quoted by Theleritis et al. [39], the person's attributes, environmental factors, and stimulus characteristics all contribute to the level and nature of engagement; therefore, the approach should be individualized. Caregivers may directly prompt patients to initiate activities, using visual cues to behaviors and setting up routines for daily activities. Education is a necessary element to assist families in understanding apathy and its mechanisms in major neurocognitive disorder (MNCD) since patients with MNCDs might be misperceived as lazy or oppositional [37]. Table 18.2 provides a summary of non-pharmacological interventions for apathy.

18.3 Summary

Psychotherapies and related interventions can benefit the geriatric psychiatry inpatient. Cognitive behavioral therapy (CBT) and acceptance and commitment therapy (ACT) have been studied in aging adults and have been associated with improvements in symptomatology, particularly depression. Due to time constraints on the inpatient unit, it may only be possible to give a patient a limited number of sessions of any modality, perhaps not enough for full therapeutic benefit. However, that experience can form a primer for entry into longer therapeutic experiences in other settings. Life review, for example, starts during the data-gathering process in the inpatient unit, which lays the foundation to more extensive reminiscence work at home or in an outpatient setting.

It is important to consider the patient's level of cognitive impairment in the choice of therapeutic modality. For those patients with cognitive impairment, more active and guided approaches; e.g., cognitive stimulation, behavioral activation, social problem-solving therapy, reminiscence therapy, life review, and art/music/exercise may be beneficial. Loss and role transition, nearly ubiquitous experiences for older adults, may be addressed through interpersonal psychotherapy. In settings with limited access to trained therapists, mental health workers, students, interns, or volunteers may be trained in the basics of the interventions and/or manualized techniques as described previously.

Table 18.2 Psychotherapeutic interventions for apathy in geriatric inpatients

Non-pharmacological interventions	Parameters of apathy that were improved
Stimulation retreat model of care	All 5 improved lack of interest and lack of initiative
Multisensory stimulation	
Kit-based activity intervention	
Live interactive music	
Brief emotional shaping	
Simulated presence	Improved withdrawn behavior
	Increased level of interest
Mindfulness-based stress reduction	Improved lack of interest
Recreational activities	Improved emotional blunting
Music, art therapy, psychomotor activity	Improved engagement
Reminiscence group treatment	In 1 group, it improved lack of interest and emotional blunting; in another study, it improved behavior, cognition subscales of AES (Apathy Evaluation Scale)
Progressive muscle relaxation	Improved interest, volition, and social relationships

Take-Away

- Geriatric patients accept psychotherapeutic interventions.
- The inpatient unit can be an excellent setting in which to introduce the geriatric patient to psychotherapeutic interventions.
- CBT may be better suited for patients without cognitive impairments, whereas problem-solving therapy might be more accessible for patients with cognitive impairment.
- When cognitive deficits make CBT a less suitable option, reminiscence therapy and life review, which have been developed initially for older populations, are recommended. They can enhance cognitive stimulation by facilitating the access to biographical memories.
- Brief group mindfulness interventions are well-tolerated and often suitable for acutely hospitalized psychiatric patients, including those with acute psychosis.
- Staff interactions and psychotherapeutic interventions may be tailored to each patient's specific needs.

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Part III

Special Topics

19 Medical Nursing Care and Communication Barriers

Andrea Graci, Erin Hachez, and Daniel Lavin

19.1 Introduction

The geriatric patient admitted to an acute psychiatric inpatient unit may have subacute or chronic medical conditions that require ongoing care. Yet the behavioral/psychiatric symptoms, for which they were admitted, can interfere with the delivery of such care. Conditions which impede medical treatment can include cognitive impairment, apathy, agitation, psychomotor retardation, wandering, aggression, refusal of care, denial of medical problems, psychotic symptoms, suicidal impulses, and reduced motivation.

In addition, communication problems can disrupt delivery of medical care. Cerebrovascular events, progressive neurodegenerative diseases, and neurological disorders result in dysfunctions such as anosognosia (denial of illness), dysarthria, aphasia, speech apraxia, and other language deficits. These conditions, as well as impairments in hearing and other senses, interfere with a patient's understanding or ability to comply. Identification of a patient's medical nursing needs early in the hospitalization, and recognition of the barriers to delivery of appropriate care, is crucial. Understanding recommended practices and consensus guidelines for specific medical nursing interventions can facilitate the treatment goals. Figure 19.1 summarizes the medical nursing issues in geriatric inpatient settings.

19.2 Vignette

A 77-year-old woman presented with psychomotor retardation, a profoundly depressed mood, and apathy, all of which developed within 2 weeks prior to the psychiatric inpatient admission. The patient had immigrated from an underdeveloped country to be with her children 3 years earlier. She only spoke her native language. She had difficulty adapting to the new situation, was often home alone, and became demoralized. She did not eat or drink regularly and had less apparent energy. She developed the fixed delusion that the "spirit" of a prior neighbor, who was always malicious in her original country, had followed her here and was poisoning her.

About 1 year prior to admission, she had suffered a left-sided hemispheric stroke and subsequently an apraxia of speech. She could no longer

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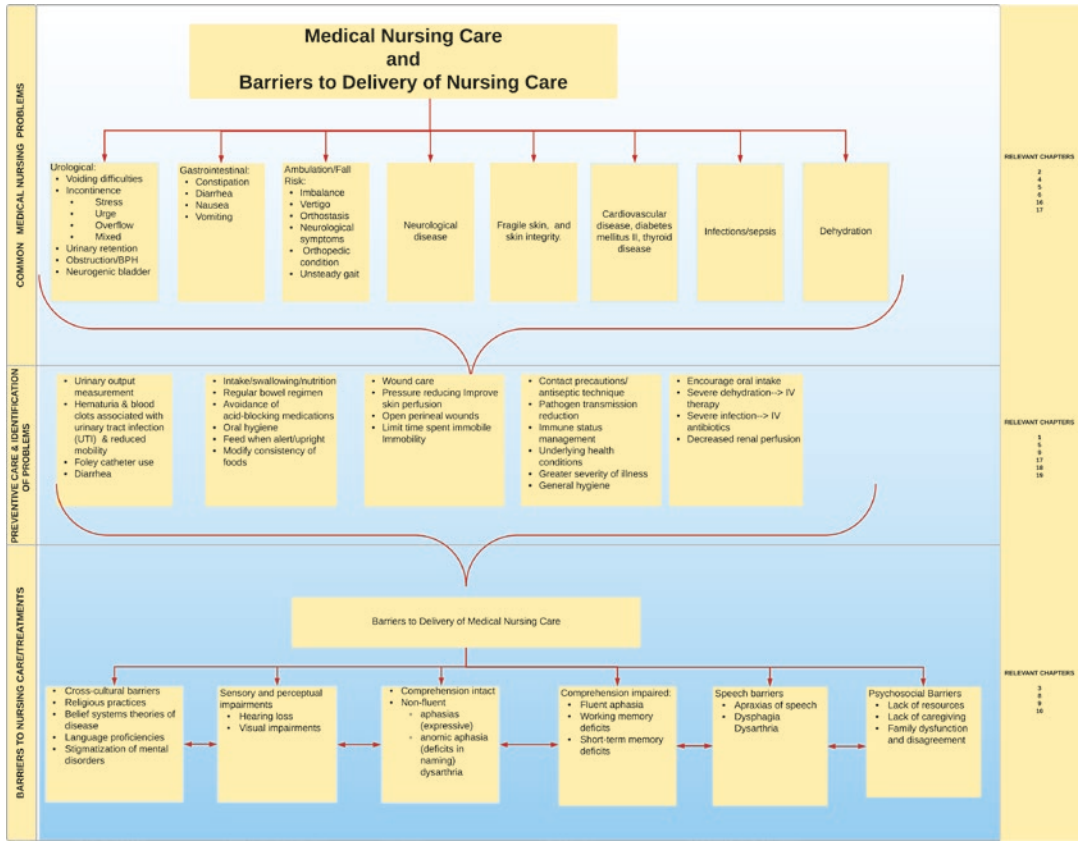

Fig. 19.1 Medical nursing issues in geriatric inpatient settings

articulate her needs and became apprehensive and agitated. The patient had never been admitted to a hospital. In outpatient medical appointments, she was prescribed temazepam 15 mg at bedtime to minimize agitation at night and to help initiate sleep. When her adult children brought her to psychiatric inpatient care, she was so dehydrated that IV fluids were indicated. She had been incontinent for the prior year, though this was never evaluated.

In the inpatient unit, the patient became agitated, pushed away any attempt to give her IVs, and whispered to her daughter that the IVs contained "a potion." Some of the nursing staff *could* speak her language, but they had difficulty understanding the patient due to her recent language/speech deficits. Nursing staff became increasingly frustrated because the patient could not articulate or explain her physical symptoms, nor cooperate with a clean-catch urine or a Foley catheter. Three days after admission, she developed fever and had a generalized seizure, during which she aspirated the little food she had ingested. She developed an aspiration pneumonitis and passed away within the next 3 days. A post-mortem examination found a chronic UTI, a pyelonephritis, and potential decubitus ulcer and skin breakdown, due, in part, to constant skin pressure and urine.

19.3 Incontinence

The prevalence of urinary incontinence in aging adults is common, estimated to be 11–33% in men and 17–55% in women [1], with the risk doubling if a patient has a diagnosis of diabetes mellitus [2]. Many factors can contribute to urinary incontinence in the geriatric patient. Figure 19.2 summarizes the risk factors for

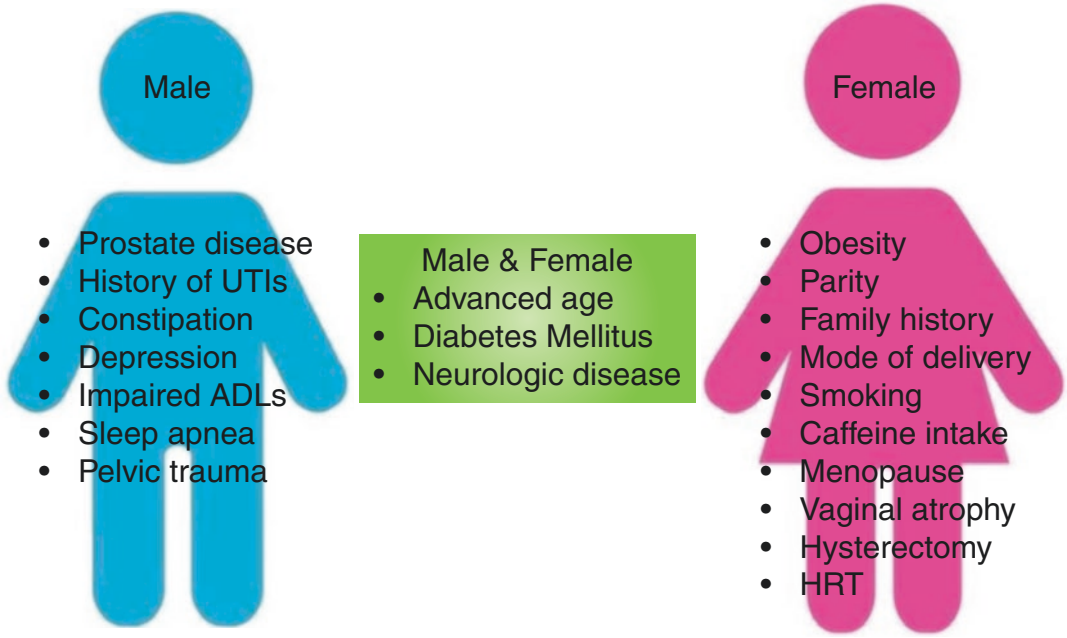

Fig. 19.2 Male and female urinary incontinence risk factors

urinary incontinence in men and women. A targeted health history, from either the patient or a reliable informant, can identify the pathogenesis and guide appropriate intervention. To consider:

- Details of onset, acute or chronic.
- The type of incontinence (see Table 19.1).
- Factors which precipitate the incontinence (e.g., coughing, laughing, medications) [3].
- Data surrounding the history of the symptoms to support the diagnosis of the urinary incontinence.
- An appropriate physical exam, including pelvic exam.
- An assessment for fluid overload for atypical symptoms, diagnostic uncertainty, or failure of initial treatment strategies.
- If symptoms of urinary incontinence present with sudden onset, a neurologic evaluation to assess for neurologic disease is recommended [3].
- Urine and blood tests to evaluate for infection, metabolic causes, renal dysfunction, and/or B12 deficiency [3].

Table 19.1 Classification of urinary incontinence by symptoms

Type of incontinence	Symptoms/signs
Stress	Associated with urine loss with an increase in intra-abdominal pressure
	Often occurs with laughing, coughing, or sneezing
	No urge to urinate prior to leaking
	Volume of urine: small or large
Urge	Usually described as "overactive bladder"
	Associated with frequent small volume voids
	May interrupt sleep at night
	Strong urge to void; possible inability to reach bathroom
Overflow	Can be secondary to detrusor muscle underactivity or secondary to urinary outlet obstruction
	Underactive detrusor muscle causes urine loss usually with change in position
	Obstruction causes intermittent or slow stream, hesitancy, and sensation of incomplete emptying
Mixed	Associated with both urgency and exertion (sneezing, coughing etc.), as seen in stress incontinence

Table 19.1 summarizes the subtypes of urinary incontinence. Functional impairments, comorbid medical conditions, and medications, in addition to cognitive deficits, can all precipitate or contribute to urinary incontinence. Antimuscarinic agents should be used cautiously, if at all, due to the risk of interfering with full voiding [3] (Chap. 6: Major Neurocognitive Disorder with Behavioral Disturbance and Chap. 3: Pharmacological Overview). For patients with mild or major neurocognitive disorders (MNCD), non-pharmacological approaches, e.g., prompt voiding and scheduled toileting, are preferred.

Incontinence and risk to skin Incontinence causes a moist environment, increases friction, and contributes to shearing, thus making breakdown of the skin more likely. In addition, urine and stool contain chemicals that may further irritate the skin. Skin must be protected from constant exposure to urine and feces with the goal of keeping skin clean and dry. Use of incontinence pads combined with consistent skin cleansing are often adequate for managing incontinence. A condom catheter or indwelling catheter may be needed when treating a skin ulcer [4].

Non-pharmacological treatment of incontinence All contributing factors, such as medical conditions and medications, should be addressed before starting any treatment for urinary incontinence [5]. Non-pharmacological therapies, including lifestyle modifications, bladder training, biofeedback, and pelvic floor muscle exercises, have demonstrated efficacy in women, yet the evidence is lacking for men. Given their safety, low cost, and proven efficacy in women, it is reasonable to try non-pharmacological therapies for men as well, prior to pharmacological interventions [6]. Treatment with these conservative interventions can usually continue for up to 6 months before initiating pharmacological therapies.

19.4 Foley Catheters

Among hospitalized geriatric patients, urinary catheterization has often been used excessively, with incontinence the most common inappropriate indication. Recurrent or inappropriate use of catheterization can be detrimental, leading to urinary tract infections (UTIs), and therefore, this should be used only when clinically indicated.

UTIs associated with urinary catheters are the leading cause of secondary health care-associated bacteremia and can lead to acute or chronic pyelonephritis [7]. Bacteriuria in patients with indwelling catheters occurs at a rate of approximately 3–10% per day of catheterization; duration of catheterization is a risk factor for catheter-associated bacteriuria and UTIs and a major target for prevention efforts [8, 9].

Other risk factors for catheter-associated bacteriuria and UTIs include female sex, older age, diabetes mellitus, bacterial colonization of the drainage bag, and errors in catheter care (e.g., inappropriate sterile technique, not maintaining a closed drainage system) [10]. In addition to urinary catheters as a major cause of infection, they also act as tethering devices, which can make ambulation more difficult for aging patients. Catheters are associated with increased rates of delirium, infections, and falls [11].

Catheters should also not be used to obtain a urine sample for testing if the patient is capable of voiding spontaneously and is reliable to collect urine for output monitoring. Often catheters are used to measure residual bladder volume, yet the use of portable bladder ultrasounds is the preferred method. These devices correctly estimate the bladder volume greater than 50 mL in over 90% of the patients and do not show the side effect profile of catheters [7, 12]. Table 19.2 provides a summary for the use of Foley catheters. During daily bathing, cleaning around the catheter with soap and water is adequate for ongoing maintenance. Sterile gloves should be used when

Table 19.2 Indications for use of Foley catheters [7, 12]

	Urinary retention
	Daily urine output monitoring for fluid management or diagnostic test (if patient not reliable to measure independently)
	Hematuria with clots
	Neurogenic bladder/spinal cord patients
	Assistance in healing of open sacral or perineal wounds in incontinent patients
	Comfort in context of end-of-life care

the catheter or drainage system is manipulated, and gloves should be disposed of immediately to limit the transfer of pathogens from patient to patient. The bag should be emptied regularly, and separate collection containers should be used for each patient. If leakage occurs around the catheter, it should be changed using a catheter that is larger by at least 2F. The collection system should be placed below the level of the bladder, and the catheter tubing should be securely fastened by tape or straps to the patient's lower extremity to prevent trauma from tugging. When a urine sample is needed for Gram stain or culture, the specimen should not be collected from the drainage bag. Indwelling catheters should not be replaced routinely, although catheters with mechanical problems should be changed [7].

19.5 Dehydration and Intravenous Therapy

Dehydration is a result of fluid and electrolyte imbalance and contributes to significant mortality rates [13]. Factors which place the aging adult at higher risk of dehydration include decline in renal function, identified by glomerular filtration rate (GFR) <60 mL/min/1.73 m^2, sensitivity to antidiuretic hormone (ADH), reduced thirst, chronic disease, depression, apathy, and loss of interest in self-care [13, 14]. In patients who can follow recommendations, encouraging oral intake is the preferred method of improving hydration, with a recommended daily oral intake not to be less than 1600 mL in a 24-hour period [13]. This is best accomplished by presenting fluids directly into the hands of the patient every 1.5 hours during waking hours [15].

In severe dehydration, intravenous (IV) therapy can be used for rehydration. However, a recent study compared acceptance, feasibility, and adverse effects of subcutaneous (SC) rehydration versus IV rehydration in dehydrated aging adults; both interventions were found comparable, safe, and effective [16]. Subcutaneous (SC) fluid replacement can be considered a superior method of rehydrating, compared to IV therapy, in those individuals who are confused and/or for whom IV access is difficult [16]. Table 19.3 summarizes the types of dehydration. It may be necessary to enlist consultation with a hospital ethics committee in cases where the patient remains resistant to care (Chap. 13: Involuntary treatment).

19.6 Wound Care

Pressure-induced skin and soft tissue injuries are common among hospitalized older adults, and identifying at-risk patients is central in preventing these injuries. A comprehensive history and physical examination can identify potentially correctable predisposing factors. In addition to an initial history and physical exam, regular follow-up is required to identify changes in the patient's clinical condition, and daily skin inspections should be performed to detect early evidence of pressure-induced skin damage [17]. Nutritional status, incontinence, immobility, neurological

Table 19.3 Categories of dehydration [13]

Categories of dehydration	Mechanisms
Isotonic	Balanced loss of solutes and water
	Can occur during compete fast or during episodes of vomiting and diarrhea
Hypotonic	Sodium is lost at a greater rate than water
	Results in low serum sodium levels
	Can occur from overuse of diuretics
Hypertonic	Water loss exceeds sodium loss
	Often termed hypernatremia
	Associated with serum sodium levels of greater than 145 mmol/L
	Due to excessive water loss when individuals are febrile or have decreased fluid intake

impairment, and reduced skin perfusion are all factors that increase the risk of developing ulcers during hospitalization.

19.7 Poor Nutritional Status

Poor nutrition for hospitalized geriatric patients may result from several factors, including failure to thrive, delirium, impaired cognition, poor appetite, difficulty in self-feeding, and lack of access to dentures. It has been associated with pressure injuries. In evaluating malnutrition, a history of changes in weight, physical examination, and/or dietary intake should all be assessed, as well as a serum albumin level. Cross-sectional studies have suggested that patients with pressure injuries are more likely to have hypoalbuminemia, and prealbumin levels are often used in clinical practice as a nutritional measure to predict risk of injury [5]. The useful measure of nutritional status is whether or not the patient has adequate dietary intake and is able to maintain body weight. Interventions, such as assistance with getting out of bed and feeding during mealtimes, are simple and can improve nutritional intake during hospitalization. A referral to the dietician or nutritionist may help identify nutritional deficiencies and facilitate future monitoring by family and/or caregivers [18, 19].

19.8 Immobility

Improved mobility during hospital stay has been linked to decreased risk of death at 2 years [6], which raises the need to minimize immobility. Some conditions call for absolute bedrest, such as unstable fractures and certain critical illnesses, yet the majority of conditions do not necessitate immobility. Unless absolutely medically required, bedrest should be avoided, and patients should be out of bed, in a chair at least twice daily with meals, and should be encouraged to walk when able. Apathy and psychomotor retardation which interfere with mobilization can be addressed with non-pharmacological and pharmacological interventions (Chap. 18: Psychotherapies and non-pharmacological Interventions).

Referral to specialists, such as a physical therapist, may be needed for patients with difficulty ambulating and/or who pose a significant fall risk [5]. Neurologic conditions which may impair full mobility include motor neuron disease, spinal cord injury, and neuropathy; all are important risk factors for the development of pressure-induced skin and soft tissue injury. Sensory loss is common, suggesting that patients may not perceive pain or discomfort arising from prolonged pressure [5]. Immobility is also a significant factor that contributes to the development of pressure-induced skin and soft tissue injury.

19.9 Reduced Skin Perfusion

Common comorbidities in aging adults, such as hypotension, volume depletion, vasomotor failure, vasoconstriction, peripheral artery disease, are all factors which contribute to reduced skin perfusion. When vital organs such as the kidneys and the gastrointestinal tract are not adequately perfused, blood flow to the skin will also be decreased, which increases the risk of pressure-induced injuries. In a patient with decreased perfusion, pressure applied to the skin for less than 2 hours may be sufficient to cause severe damage [20]. Ulcers are impossible to heal in the setting of decreased skin perfusion, yet efforts must be made to improve the quality of skin perfusion, such as treating and stabilizing hypotension, improving cardiac contractility, limiting vasoconstrictive agents, or revascularization for patients with severe peripheral artery disease [20].

Risk assessment for skin ulcers includes a history and physical assessment, including regular assessment with the Braden Scale, for predicting pressure sore risk [21, 22, 18]. The scale includes items which should identify patients at risk for skin and soft tissue breakdown and those patients who would benefit from preventative measures as well as potentially correctable factors. Figure 19.3 summarizes indicators which the Braden Scale relies upon to assess risk for skin ulcers or pressure sores.

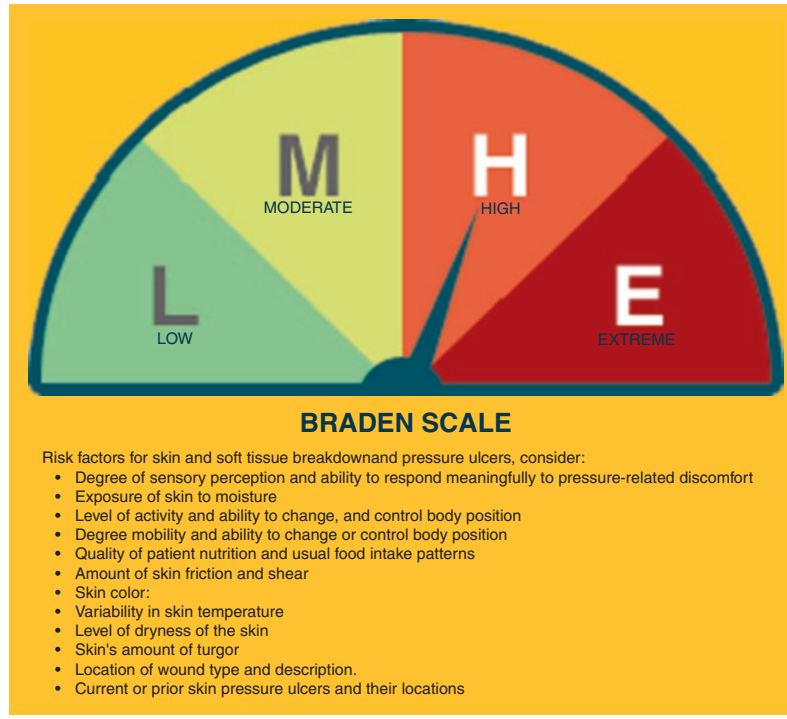

Fig. 19.3 Braden Scale—risk factors for soft tissue breakdown/ pressure ulcers

19.10 Prevention of Skin Breakdown

Redistribution of pressure is the most vital intervention to prevent skin and soft tissue breakdown/injury. Patients who are bedbound should be repositioned at least every 2 hours, with proper technique to minimize shear forces [17]. The skin should be clean and dry while avoiding excess dryness and scaling, using pressure-reducing products [4, 17]. Even though preventative practices have shown to decrease the rate of pressure-induced skin and soft tissue injuries, there are patients who remain at such high risk due to heart failure, imminent death, and critical illness that skin breakdown cannot be prevented. Table 19.4 provides a summary of skin care interventions to reduce this risk.

Table 19.4 Skin care intervention to reduce breakdown

	Assess skin daily, recording temperature, color, turgor, moisture status, and integrity
	Keep skin clean and dry
	Avoid excess dryness – to protect against friction and pressure
	Use lotions containing fatty acids, which can help protect the skin and reduce hyper-proliferative growth
	Use pH-balanced cleaning agent to minimize irritation when cleansing skin
	Avoid hot water
	Cleanse at routine intervals to avoid excess moisture, precipitation, and wound drainage
	Avoid vigorous massage over bony prominences

19.11 Infection Control

The risk of transmitting infections within the hospital setting is substantial. Pathogens are transmitted by hospital personnel, other patients, and the environment [23]. The risk varies and depends on an individual's immune status, yet underlying health conditions, poor nutrition status, and greater severity of illness contribute to increased rates of hospital-acquired (or nosocomial) infections in older patients [24]. Common nosocomial infections include:

Table 19.5 Standard infection control precautions

	Standard precaution	Description
	Hand hygiene	Before touching a patient
		After touching patient
		Before clean/aseptic procedures
		After body fluid exposure, or risk of body fluid exposure
		After touching patient surroundings
		Either soap and water or alcohol based disinfection can be used for most circumstances
		Alcohol-based disinfection is more efficient
		Handwashing with soap and water should be used when caring for patients with c. diff or norovirus since alcohol based disinfectant does not kill c. diff spores or norovirus
	Masks	Staff: when caring for patients with respiratory secretions
		Staff: when performing sterile procedures
		Patients: who are coughing or having respiratory symptoms wear when leaving their rooms
		Masks: distinct from respirators used to prevent transmission of airborne droplet nuclei
	Gloves	A protective barrier for the healthcare provider and the patient
		Worn when exposure to blood or body fluids is possible
		Non-sterile examination gloves are generally used
		Gloves do not replace proper hand hygiene
		Change in between patient encounters
		Change contaminated gloves – even for a single patient – to prevent contamination of medical equipment or cross-contamination of different body sites

- *Clostridium difficile* is a gram-positive anaerobic bacterium that causes antibiotic-associated colitis and is the most frequent cause of nosocomial diarrhea and a significant cause of morbidity and mortality among hospitalized older patients. This bacterium colonizes the human intestinal tract after the normal flora has been altered following antibiotic therapy [25]. Contact precautions have been found to help prevent the spread of *Clostridium difficile* spores and should be used in patients with both suspected and proven *Clostridium difficile* [26].
- Hospital-acquired pneumonia (HAP) is pneumonia that occurs 48 hours or more after admission [27]. Patients with major neurocognitive disorder, severe Parkinson's disease, or other neurologic conditions are at high risk for aspiration pneumonia, and those treated with antipsychotics are also at an increased risk.
- Urinary tract infection (UTI) associated with urinary catheters is the leading cause of secondary nosocomial bacteremia, which is associated with high mortality among hospitalized older adults [2]. To prevent or minimize urinary infections, avoid unnecessary catheterization, and prompt removal of catheters when no longer indicated [28]. Table 19.5 summarizes the components of standard precautions.

19.11.1 Standard Precautions

Standard precautions are recommended in the care of hospitalized patients to reduce the risk of infection, even when the presence of infection is not apparent: strict hand hygiene before and after contact; wearing gloves, gown, and eye protection when there is potential for exposure to body fluids; safe injection practices; and the disposal of sharp instruments into impervious containers [23].

Isolation precautions may be used to interrupt or reduce the risk of transmission of pathogens.

Table 19.6 Isolation precautions

	Isolation precaution	Description of precautions
	Contact	Protection from contact with patients who have multidrug-resistant bacteria, enteric and viral pathogens
		Private room or in a shared room with other patients – who have the same indication for isolation
		Hand hygiene and gloves prior to entering the room – even if no direct patient contact is anticipated
		Gowns – even if direct patient contact is not anticipated
		Medical equipment dedicated to a single patient when possible
		Medical equipment NOT dedicated to a single patient should cleaned and disinfected before reuse
	Droplet	Droplets: particles of respiratory secretions which remain suspended in air for limited periods
		Transmission is associated with exposure within 1–2 meters of the source
		Within 6 feet of patient, provider wears a surgical mast and has contact precaution/ personal protective gear (gown and gloves): for patients with suspected or confirmed infections of influenza, Neisseria meningitides, parainfluenza, pertussis, rubella, H. Influenza, mycoplasma pneumonia, other infections spread by droplets
	Airborne	Airborne droplet nuclei are particles of respiratory secretions that can remain suspended in the air for extended periods of time which make them a risk of inhalation exposure for susceptible individuals. This precaution indicated for patients with confirmed or suspected tuberculosis, varicella, measles, smallpox, and severe acute respiratory syndrome (SARS)
		Private room with negative air pressure of at least 6–12 air changes per hour
		Doors to the isolation room remain closed
		All individuals enter the room must wear a respirator with a filtering capacity of 95 percent, and a tight seal over the nose and mouth
		Gown and gloves

Three isolation precautions prevent major modes of pathogen transmission in the hospital setting: contact, droplet, and airborne precautions. Isolation precautions are marked outside a room with a sign that indicates the instructions for the isolation and the precautions that must be observed. Infection control supplies made available outside the room ensure proper compliance with the isolation [23]. Table 19.5 lists the standard infection control precautions. Table 19.6 lists three different types of isolation precautions.

19.12 Barriers to Communication

19.12.1 Language Proficiency

Delivery of optimal psychiatric care may be impacted by limited language proficiency [29]. Use of a language or dialect which is not fully understood by both provider of care and the patient may obscure important diagnostic clues, such as bizarre thought content, thought disorder, and perceptual disturbances. Early in a hospitalization, it is crucial to identify mutual limitations in language and to procure competent interpreters. Several studies have linked limited English proficiency with poorer access to health care, lower quality care, medical errors, and poor satisfaction with care [30]. Research suggests that psychiatric assessments conducted in non-native languages are less reliable [31].

In emergent situations, or if a professional interpreter is not feasible, untrained interpreters, occasionally family members, may be the only available resource. But these untrained interpreters contribute to a greater frequency of translation errors [32], as well as to the risk that sensitive matters may be withheld, particularly if the interpreter is a family member [32]. Although interpreter-mediated interactions are also prone to errors, professional interpreters

facilitate improved disclosure, crucial to accurate psychiatric assessment [33]. Sadavoy, J et al., 2004, revealed that geriatric patients place importance on interpreters who are committed to confidentiality and have an understanding of mental health issues. The study showed that seniors are hesitant with family members as interpreters, as it impacts their ability to disclose private information [34].

19.12.2 Speech and Language Impairments Due to Neurological Disorders

The vignette patient who sustained a stroke and a post-stroke depressive disorder also developed an impairment of verbal communication. The inability of a patient to clearly express needs, or to follow exact instructions, can result in frustration and strained interactions. In the geriatric population, neurological conditions, such as major neurocognitive disorders and stroke, can cause deficits in expressive language (e.g., nonfluent aphasia) and/or receptive language (e.g., fluent aphasia), both of which have been associated with behavioral disturbance [35].

To improve communication and minimize exacerbation of behavioral disturbance, it is useful to recognize two conditions which can interfere with meaningful, expressive speech: *dysarthria* versus *speech apraxia*. Neurological consultation is crucial to a definitive distinction between these conditions. A Speech-language pathologist (SLP), or a professional with similar expertise, may also help with this distinction.

- *Dysarthria:* a result of neuromuscular impairment of muscles involved in speech, often accompanied by struggling to produce the correct sounds. A behavior described as "articulatory groping" is a marker: the patient attempts different facial and oral maneuvers (e.g., grimacing), when asked to perform movements, such as opening the mouth or lips.
- *Speech apraxia:* the result of a neurological event which disrupts neural pathways involved in *language* production. The patient shows no apparent *effortful* struggle but may be unable to express substitutions of phonemes or has difficulty repeating multisyllabic words.

The patient with *dysarthria* who is asked to repeat his/her requests may struggle, with little improvement, due to neuromuscular impairment. In *dysarthria*, deficits in motor control of the tongue, lips, and mouth needed to produce speech are *less* likely to improve with repetition. Repetition and practice are especially ineffective and counterproductive for some forms of *dysarthria*, e.g., flaccid dysarthria of myasthenia gravis and amyotrophic lateral sclerosis (ALS). Figure 19.4 provides one approach toward these etiologies of disordered speech.

The patient with *speech apraxia* has a *language* impairment and demonstrates difficulty finding the appropriate words or phrases but may improve with repetition. Table 19.7 summarizes the different speech impairments, associated symptoms, and suggested adaptations.

In both conditions, an unrecognized neural pathway deficit can be a source of frustration for the patient with communication deficits due to a stroke, neurodegenerative condition, or brain injury. Clinicians may misinterpret the patient as uncooperative, oppositional, depressed, or cognitively impaired. This can lead to escalation of anger, agitation, social withdrawal, and/or demoralization.

19.12.3 Aphasias/Deficits in Language

Aphasia connotes the loss of ability to produce and/or understand language. Any insult or pathologic process in neural circuits that results in damage or dysfunction of the language network may cause aphasia. It can manifest both as difficulty speaking or understanding spoken language, but reading (alexia), writing (agraphia), and use of numbers (acalculia) can also be impacted. Aphasia can interfere with the use of manual sign language and Braille. The most common etiology of aphasia is an ischemic stroke. Other causes include hemorrhagic stroke,

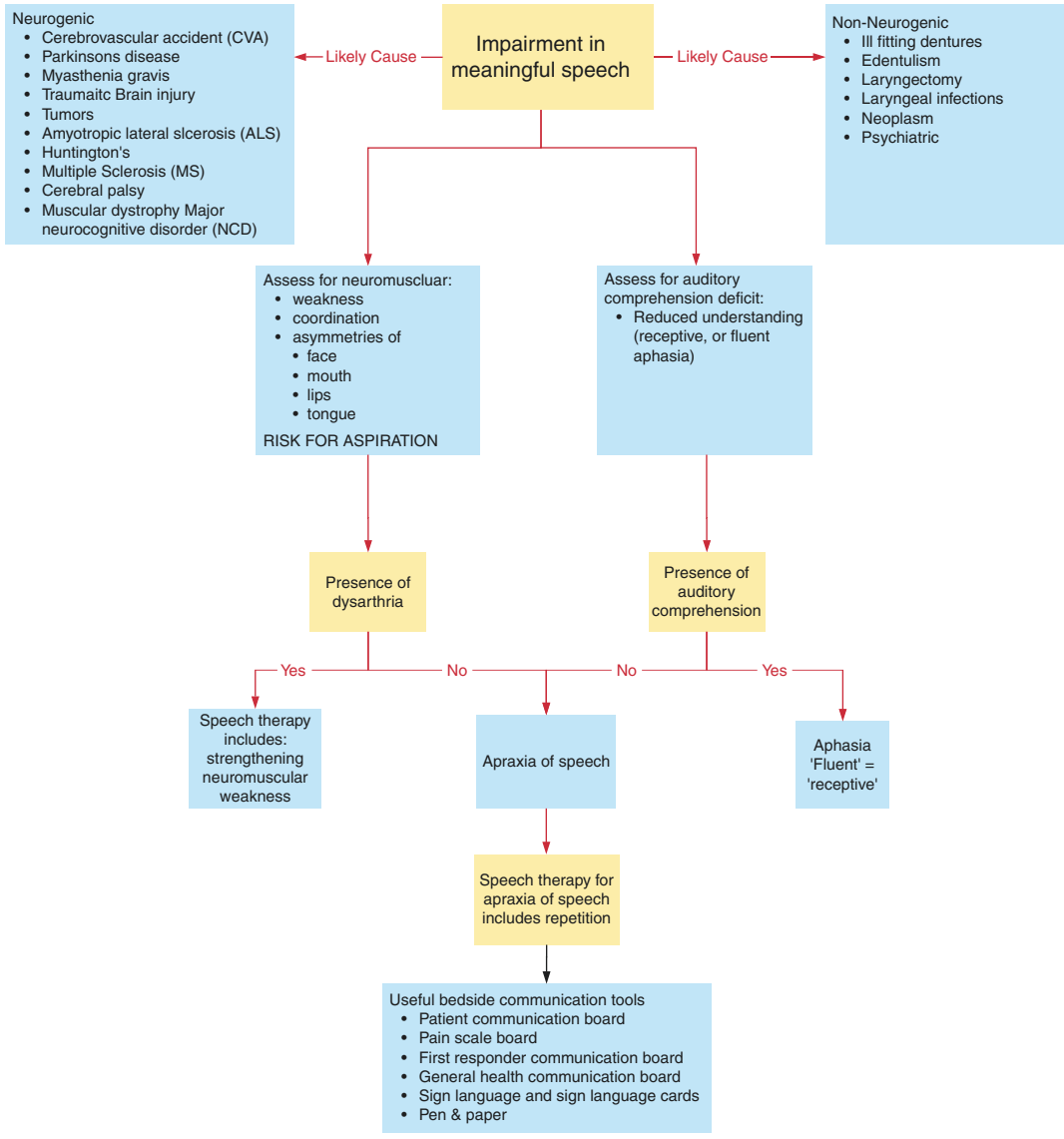

Fig. 19.4 Impairment in understandable speech

Table 19.7 Characteristics of *non*-fluent speech

	Sparse output (decreased number of words per minute)
	Shortened phrases (5 words or less)
	Agrammatism: the omission or substitution of words (prepositions, conjunctions) or suffixes (e.g., '-ed' for past events), which are grammatically awkward or idiosyncratic. This pattern is the most specific feature of dysfluency. Poor language skills from someone raised in another language may be misinterpreted as agrammatism
	Effortfulness: hesitations and disruption of normal melodic rhythm
	Breakdown of speech praxis: the ability to coordinate the articulatory movements required for comprehensible speech (40)

traumatic brain injury, neoplasm, cerebral abscess, encephalitis, or central nervous system infections [36]. Multiple sclerosis (MS) is an uncommon, but a reported, cause of aphasia [37]. In the late stages of Alzheimer's disease, aphasias can result in a significant loss in language ability, with use of nonsensical jargon words [38].

Aphasias include many variants, depending upon the neural networks disrupted. The broadest dichotomy is between fluent or non-fluent aphasia. Fluency is assessed by listening to the patient's spontaneous speech. Characteristics of non-fluent speech are listed in Table 19.7.

Bedside examination may be sufficient to assess aphasia, where verbal fluency tasks provide a means for rapid assessment of working knowledge and verbal executive functioning [39]. There is limited evidence that therapeutic interventions are effective for verbal apraxia or non-fluent aphasia, yet involvement of a speech language pathologist may be beneficial [39]. Neuropsychological testing may also help delineate the types of aphasia and language deficits (Chap. 2: Neuropsychological assessment). Aphasia may also resolve spontaneously.

Apraxia is an impairment of motor *programming,* rather than weakness or paralysis of the structures needed for speech. Apraxia of *speech*, or *verbal apraxia*, is discussed above. In *verbal* apraxia, there is no difficulty in producing automatic, or *spontaneous*, speech, but there is impairment in *novel* verbal output. The results may be misarticulations, accompanied by frustration. If the frustration increases, it can escalate to behavioral disruption, impacting the relationship with caregivers, nursing staff, and physicians [40].

19.12.4 Aspiration

Many neurological deficits in speech are the result of disrupted neuromuscular pathways, which can also affect the swallowing mechanism. The presence of speech and language impairments should raise awareness of the need for precautions against aspiration. Table 19.8 summarizes the various deficits associated with speech, some of which may also increase the risk of aspiration.

Table 19.8 Speech deficits and aspiration risks

Deficit	Characteristics
Dysarthria	Disorder of articulation, due to Impaired muscular control of speech mechanisms; involves central nervous system (CNS) or peripheral nervous system (PNS) damage→ paralysis, poor coordination, or weakness. Severe: anarthria – inability to articulate any meaningful speech
Dysphagia	Difficulty swallowing liquids or solids due to motor neuron disease of muscles related to swallowing. Results in weakness and aspiration; not due to aging per se
Verbal apraxia	Apraxia: impairment of motor *programming*, not due to motor neuron disease nor manifested in weakness or paralysis of muscles. Verbal apraxia: disorder of motor sequence which impairs ability to *program* muscles needed for speech in order to produce recognizable sounds
Hypophonia	Low tone or soft speech, common in Parkinson's disease – associated with poor coordination of vocal chords

Early signs of swallowing dysfunction such as coughing when eating or drinking should be identified, so immediate intervention can take place to prevent aspiration, acute respiratory distress syndrome, and aspiration pneumonia. Preventative measures include avoidance of acid-blocking medications, attention to oral hygiene, and feeding only when the patient is alert and able to sit upright [41, 42]. Modifying consistency of foods, formal swallowing assessment, and assistance with feeding are measures warranted in older adults who show impairment in swallowing [41].

19.12.5 Mutism

Mutism, or absence of speech, can arise within several contexts:

- Catatonic mutism is defined as the lack of verbal response in the absence of an established aphasia [39].
- *Akinetic* mutism manifests with immobility, mutism, and waxy flexibility [28]. It is not

responsive to benzodiazepines, and neuroimaging will demonstrate a lesion in the prefrontal area [43].
- *Locked-in syndrome* is characterized by immobility; neuroimaging will reveal a brain stem lesion [44]. These patients are typically able to communicate with eye movements and blinking [44].
- *CVA* mutism in the context of a CVA is associated with focal neurological deficits [45, 46].
- *Selective mutism* is the failure to speak in an otherwise vigilant patient, meaning that he/she electively chooses not to speak [47]. This is typically accompanied by a personality disorder or other evidence of poor coping, dissociation, acute social stressors, and no other signs of catatonia [38].

Impaired speech output, such as hypophonia, is *not* mutism and is seen in Parkinson's, major neurocognitive disorder (MNCD), and delirium [48]. As with all barriers to communication, it is important to identify the cause and, when possible, treat reversible causes. Despite the many etiologies of mutism, some general principles apply in approaching the mute patient. Communication may rely solely on non-verbal cues such as hand gestures and facial expression. Depending on the etiology, written communication may provide an alternative to the speech communication barrier [44, 45].

19.12.6 Sensory Impairments

The effectiveness of communication can affect outcome and satisfaction with the healthcare experience [49]. Sensory impairment represents a significant, perhaps underappreciated, barrier to delivery of quality healthcare and psychiatric care.

In the United States, hearing loss affects an estimated 9% of the general population and up to 24% of residents in long-term care facilities [50]. Hearing loss is the third most common chronic health condition in the United States [51]. It is estimated that 1–3 of 1000 children are born deaf or with significant hearing impairment [52, 53]. In patients aged 6–19, the prevalence of hearing loss can be as high as 15% [54]. In the geriatric population, an estimated 25% of adults age 60–69 have significant hearing loss; this figure approaches 80% of adults by age 80 [55, 56]. Common causes of hearing loss in the geriatric population include both reversible and non-reversible conditions (Table 19.9). In all, hearing impairment affects an estimated 48 million patients in the United States [57].

Despite its prevalence, hearing loss is likely under-recognized, as it is often not readily apparent to the casual observer or clinician. While an estimated 97% of newborns in the United States are screened for hearing impairment at birth, the percentage of patients screened for hearing loss within the primary care setting can be as low as 15–18% [51, 56]. Patients with hearing loss are significantly more likely to be of lower socioeconomic status, have lower overall levels of education, have higher rates of unemployment, and are more likely to be victims of sexual abuse [51, 52]. In general, they have a greater risk of developing chronic mental illness and may be at higher risk for social isolation, low self-esteem, and poorer quality of life [51, 57, 58]. Patients

Table 19.9 Most common causes of hearing loss

Causes of hearing loss	Description
Cerumen	Wax build up in the outer ear canal. Represents a reversible condition
Otosclerosis	Immobilization of the stapes, sometimes correctible through surgery
Noise-induced hearing loss	Result of excessive noise exposure, irreversible
Prebycusis	Age-related hearing loss, irreversible. Multifaceted problem involving heterogenous pathologic condition. Represents the most common cause

with hearing loss have reported approximately 10% lower rates of satisfaction with their level of patient-physician communication and 6% lower satisfaction rate with their healthcare experience as compared with patients without hearing impairment [49]. Some studies have found that patients with hearing impairment tend to have longer lengths of hospital stay, up to twice as long as patients with normal hearing [50]. For these reasons, it is important to consider hearing loss in the workup of a geriatric patient.

Hearing impairment exists along a spectrum, but patients can generally be categorized into one of the three groups: (1) hard-of-hearing patients, (2) deaf patients who communicate orally, and (3) deaf patients whose primary means of communication is sign language [50]. Patients in each of these three groups may have different methods of preferred communication. While recommendations for improving communication among these groups differ, in general, simply asking the patient about their preferred method of communication can be the most helpful.

Group 1: Hard-of-hearing patients are those whose impairment occurred after the linguistic development period (age 5) [52]; they depend on oral communication as their primary means of communication [50]. Patients within this group may minimize their degree of hearing loss or impairment to the point that it is not obvious to the examiner [51]. Techniques for improving communication with hard-of-hearing patients include minimizing background noise, using a room with adequate lighting, using assistive listening devices, and writing notes when necessary [50]. Hard-of-hearing patients may depend more on non-verbal visual cues to improve their communication. It is important that the examiner's face is well lighted and the mouth is unobstructed—in some cases, shaving or trimming of facial hair can be helpful [50, 59]. Suggestions for interacting with such patients are summarized in Table 19.10.

Table 19.10 Tips to assist with hearing deficits

Face the patient directly
Speak at normal volume and enunciate without exaggerated lip and mouth movement
Do not obstruct the patient's view of your mouth
Minimize background noise
Use a private room
Hearing aids and assistive devices work by amplifying sound across a range of frequencies; they do not correct hearing deficits
Rephrase sentences instead of repeating them
Label the chart to alert staff of hearing deficits
Ask patient what communication methods work best
Verify that information regarding treatments or procedures has been understood; provide written materials as indicated
Ensure that hearing aids are in place and working properly
Ensure that glasses are worn when needed
Discuss plans or procedures face to face prior to donning personal protection

Group 2: Deaf patients who communicate verbally include patients who became deaf later in life or who were educated in a setting wherein the primary means of communication was oral [50]. Having been exposed to oral communication, specifically spoken language, makes it much easier for patients to adapt to the traditional clinic or hospital environment. With the exception of assistive listening devices, the recommendations for improving communication in this group are similar to the hard-of-hearing group. In addition to the techniques listed above, it is recommended that the examiner periodically summarize the patient's responses and confirm an understanding of the patient's responses is accurate [50].

Group 3: Deaf patients who communicate using *sign language* primarily are specific in their communication preferences and needs. In general, these patients acquired their disability prior to the period of linguistic development in early childhood [57]. This group deserves special attention, as they typically *do not* benefit from techniques described above. Group 3 may *not* use English as their primary language and may rely upon American Sign Language (ASL) as well as other sign-based languages. It should be noted that ASL does not have the same grammar, vocabulary, or syntax as spoken English, and written communication may be less helpful.

Hearing impairment has implications for the interpretation of results of clinically validated screening tools such as the Mini-Mental State Examination (MMSE), Montreal Cognitive

Assessment (MoCA), Patient Health Questionnaire-9 (PHQ-9), and other similar instruments. The results and diagnostic reliability of these instruments may be skewed in deaf patients reliant upon sign language [51]. It is recommended that the examiner use a qualified ASL interpreter when speaking with these patients. Family members of the patient should not be used as their presence may inhibit the candor of the patient or present a biased interpretation of the patient's responses [58]. The examiner should focus on the patient and not the interpreter.

Visual aids, including illustrations, diagrams, and videos, may also improve communication [50]. Additionally, assistive technologies including video-based interpretation, text-to-voice, TTY, TDD, and captioned telephone services should be used as appropriate [51]. Lastly, it is important to remember that the deaf community is culturally distinct and may not see their hearing deficit as a disability. As with all cultures, the deaf community has its own values and cultural norms. The examiner needs to recognize that the norms of this unique subculture may affect communication in ways not readily apparent. Tips for assisting with hearing deficits are listed in Table 19.10.

19.12.7 Vision Impairment

As with hearing loss, visual impairment and blindness affect a significant number of patients in the United States who have psychiatric illness. Visual impairment is generally defined as having 20/50 or poorer best corrected visual acuity in the better-seeing eye as measured using a Snellen eye chart [60]. As of 2006, an estimated one in six US residents aged 70–79 was affected by a visual impairment; this number doubles in individuals 80 years or older [61]. In 2010, the American Community Survey reported that approximately 7% of the adult population, an estimated 22 million people, reported having blindness or significantly reduced vision that was not improved with corrective devices [61]. Of these patients, an estimated 18% are classified as completely blind or sensitive to light perception only [61]. It is important to note that, like hearing loss, visual impairment exists along a spectrum.

Some impairments, for example, those caused by early cataracts, may be less of an issue in a well-lighted office environment as compared with an individual with macular degeneration and significant loss of central vision. Patients with visual impairment, and in particular aging adults with the onset of visual impairment later in life, are more likely to socially isolate themselves, which increases the risk of depressive disorders, suicide, falls, and cognitive decline [62]. Patients with visual impairment are more likely to experience visual hallucinations (i.e., Charles Bonnet syndrome), which may be misconstrued for primary psychiatric illness. In one study, up to 1/3 of patients with visual impairment endorsed experiencing visual hallucinations—a number of these patients had not previously reported these visual disturbances or did not understand their etiology [62]. Obviously, not recognizing visual hallucinations as a result of visual impairment could be highly distressing to the patient.

Various techniques may be used to improve communication among patients with visual impairment. Most patients should be asked if they prefer assistance, and help should be individualized, e.g., offering to guide the patient through an unfamiliar office or exam room or describing the office environment including furniture and other people present [62]. For written materials, including screening instruments and discharge instructions, special care should be given to the font size and contrast. In general, a larger font size is recommended; 18-point font is considered the minimum size [61]. Colored text (e.g., red, blue, green) should be avoided in materials given to the patient [61].

Additionally, the psychiatrist and staff should be aware of the difficulties the patient may have with medication compliance due to difficulty reading prescription labels. Enlisting the help of a reliable family member, magnification device, or other assistive technology such as radio-frequency identification (RFID) tagging or text-to-speech apps may be helpful [61]. Lastly, it may be helpful to the patient to take extra time to read aloud medication dosage and other therapeutic instructions.

19.13 Legal Issues

Psychiatrists and allied mental health clinicians in the United States are legally obligated to accommodate patients with disabilities, including those that affect communication. The Americans with Disabilities Act (ADA) of 1990 and Section 504 of the Rehabilitation Act of 1973 mandate that healthcare providers accommodate patients with disabilities, including assuming the financial responsibility of hiring a qualified sign language interpreter if needed [63].

Take-Away

- Anticipate common medical nursing needs by reviewing history from medical records, patient, and family.
- Determine and institute appropriate medical nursing interventions based upon consensus guidelines.
- Identify, as early as possible, with history, diagnostic assessment, consultations, and testing results, any communication barriers which are likely to impede delivery of optimum care.
- Suspect speech, cultural, sensory, and language proficiency deficits when instructions are not fully followed or communication appears difficult.
- Consult neurology and speech-language pathologist (SLP) to identify and ameliorate communication barriers.
- Do not assume personality or psychiatric factors are responsible for disruption of communication until completion of an adequate assessment to rule out other etiologies.

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Telemedicine and IT: Use of Digital Technology on Inpatient Units

20

Donald M. Hilty, Andreea L. Seritan, and Terry Rabinowitz

20.1 Introduction

Patient-centered care offers quality, affordable, and accessible/timely care in a variety of settings. The proportion of older adults is growing faster than any other age group due to longer life expectancy and declining fertility rates [1]. Provider shortages limit mental healthcare access in many regions of the world, and new technologies like telehealth leverage scarce resources (e.g., psychiatrist time). Access to high-quality psychiatric inpatient and emergency services is a priority, and staffing around-the-clock is rarely possible and is costly/not cost-effective. Rural psychiatrists are isolated and have trouble obtaining coverage for vacations and unexpected illness, leading to increased turnover of providers [2]. Some patients report a preference for telepsychiatry (TP) over in-person care [3]. This includes, but is not limited to, child and adolescent patients with autistic spectrum disorders [4], rural inpatients [2], and veterans with PTSD [5, 6]. Patients attribute this to physical distance, less anxiety, and a greater sense of control leading to empowerment [7, 8].

Healthcare systems are trying to increase clinical operating efficiency by integrating care and providing care at multiple points of service and use it to leverage interdisciplinary team members' clinical, administrative, and other care coordination expertise [3]. Participants, both patients and providers, as well as loved ones, are highly satisfied with assessment, consultation, and a range of treatments in many populations (e.g., adult, child, geriatric), settings, and cultures [3, 9]. Figure 20.1 summarizes the aspects of telemedicine and information technology for geriatric inpatient care.

The evaluation of telepsychiatry and telemental health (TMH) has gone through three phases. First, TMH was found to be effective in terms of increasing access to care, acceptance, and good educational outcomes. Second, it was noted to be valid and reliable compared to in-person services. In addition to comparison (or "as good as") studies, telepsychiatric outcomes are not inferior to in-person care (i.e., non-inferiority studies). Third, frameworks are being used to approach complex themes like costs and models.

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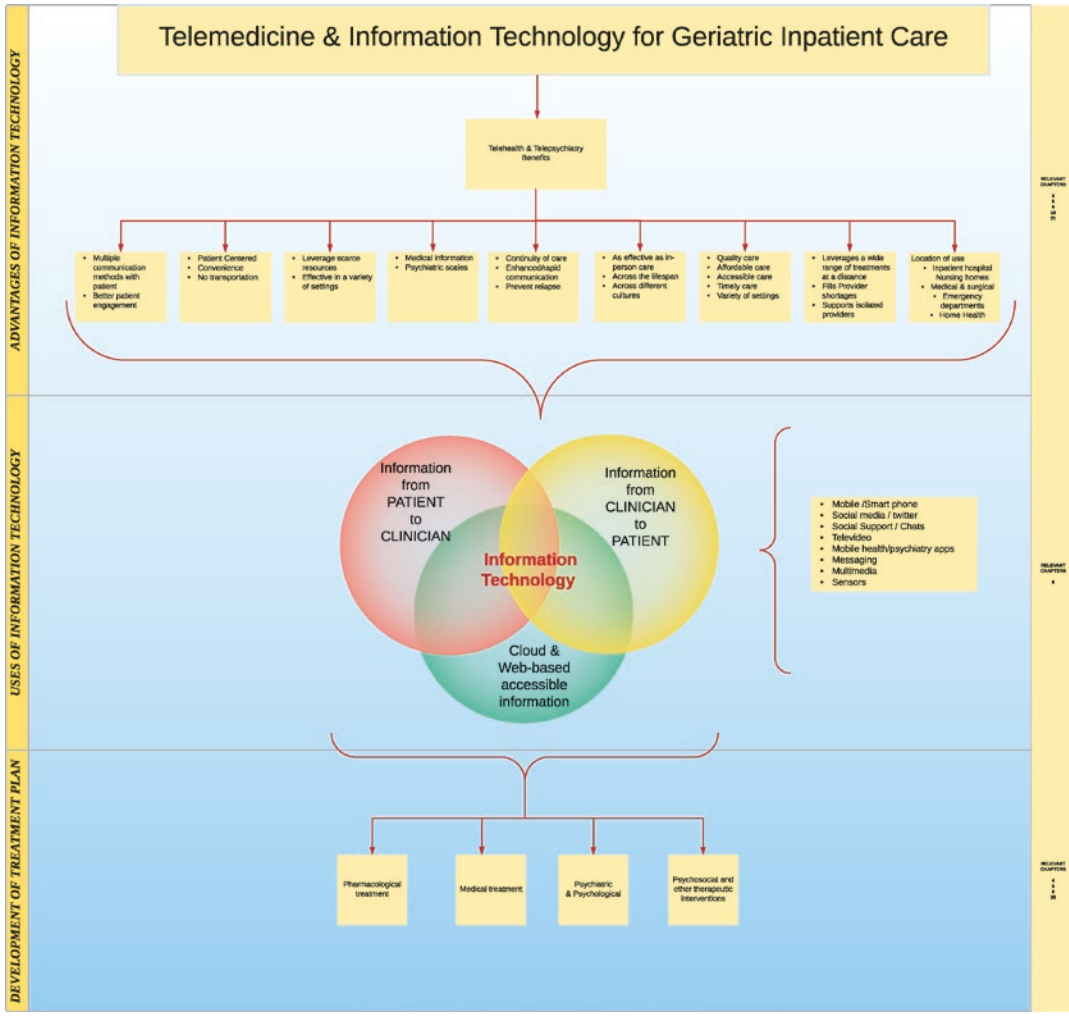

Fig. 20.1 Flowchart of telemedicine and information technology for geriatric inpatient care

TP outcomes are comparable to in-person care, and it has been used with a variety of models of care (i.e., collaborative care) [3, 10].

20.2 Clinical Vignettes

Vignette 1 Geriatric with Parkinson's disease, agitation, and confusion.

A 72-year-old man with Parkinson's disease for approximately 10 years was living at home with his wife and daughter. The family reported to the neurologist that the patient has been getting fearful and agitated several times a day. At the next neurology visit, the patient reported that "he" won't let him speak—the patient then became silent. Later in the visit, the patient resumed speaking but appeared intermittently confused. Asked to elaborate, the patient said, "he" is a man who visits him and controls his actions; "he" can make him stop talking or moving. He saw this man in his house for the past several months and even heard him ring the doorbell once and was frightened. The neurologist started quetiapine 25 mg at bedtime.

Over the next 2 weeks, the family called the neurologist's office almost daily, reporting episodes of agitation—two to three times daily. Quetiapine was gradually titrated to 25 mg in the morning, 25 mg at noon, and 50 mg in the evening, with minimal improvement.

Table 20.1 Summary of evaluation of telepsychiatry

Evaluation	Description
Effectiveness	Increasing access to care
	Acceptance
	Good educational outcomes
Comparability	Valid and reliable of compared to in-person services
	Outcomes are not inferior to in-person care
Cost and models of care	Comparable costs
	Used in a variety of care models (e.g., collaborative care)

The family then reported the patient began pushing his family members when they try to give him his medications; he once grabbed his daughter's arm and pushed her to the ground. Patient was admitted to an inpatient psychiatric facility, wherein quetiapine was titrated to 50 mg three times a day and pimavanserin 34 mg daily added. After 2 weeks, the psychotic symptoms diminished and he was discharged home.

The patient's home is 200 miles away from a large academic center, without any local psychiatrists contracted in his insurance plan. He was referred to a geriatric psychiatrist at the academic center, who offered telemedicine appointments for patient, wife, and daughter. The family was relieved to continue psychiatric care and satisfied without the need to drive, especially given Mr. A's mobility impairments. Over the next 6 months, the psychosis fully resolved and the patient remained stable psychiatrically. After 6 months, the neurologist took over prescribing the antipsychotic medications, with instructions to consider a careful taper of quetiapine within the next year, if psychotic symptoms remained under control. Table 20.1 provides a summary of the evaluation of telepsychiatry.

20.3 E-Health and E-BH Technologies' Impact and Opportunity

Technology needs to be matched with outcome targets, patient preferences, setting, service, and model of care to be "effective." The "what" for "whom" and "when" requires that the technology is chosen specific to the objectives [1]. TP is most often compared to in-person care, but it is one end of a *spectrum of e-mental health (e-MH) care* that has emerged. The continuum of technology is as follows: Internet information—self-help/support groups to psychoeducation classes—self-assessment and care (e.g., depression)—clinician professional education to informal online consultation—informal asynchronous clinician communication (e.g., e-mail/text, social media/networking)—purposeful asynchronous care coordination (e.g., e-mail/text, mobile health and apps, ATP)—STP/video and hybrid models with combinations to complementing in-person and/or TP services [11]. E-MH options may provide resources, connections, and meaningful activities for people facing particular obstacles to care such as geographic distance from services, special needs (i.e., autism spectrum, sensory), and immobility (i.e., housebound due to physical disabilities or MH problems such as panic disorder or phobias) [11].

Data on TP care for older adults are emerging, including inpatient, medical/surgical, nursing homes, and other settings [12]. This paper will help the reader to:

1. Learn how TP is used to provide psychiatric care across age, population, and medical and mental health settings.
2. Apply findings, models, and suggestions about TP to inpatient psychiatry and other acute care settings (e.g., medical/surgical and emergency settings).
3. Incorporate information technology, e-health, and e-behavioral health innovations to patient care for psychiatric inpatients and outpatients.

20.4 TP/TMH Across Age, Population, and Medical and Mental Health Settings

20.4.1 Effectiveness and Outcomes

Effectiveness should be considered for the patient, provider, program, community, and society. The only previous review of effectiveness considered it effective in terms of providing access, providing positive patient outcomes, and being well-accepted [3]. Effectiveness from the Latin origin of the word is defined as "having the

power to produce an effect … a decisive effect; efficient; as, … an effective … remedy." (TMH) initially compared it to in-person care and opined on quality of care, clinical outcomes, and costs [13–15], but the new research agenda [16] is pushing the evaluation forward rigorously. More information has been needed on the model of telepsychiatry used [10], other populations (e.g., child, geriatric), settings (e.g., emergency departments, home health), and services (e.g., cognitive behavioral therapy (CBT)). TP's effectiveness has been measured with regard to: diagnosis (reliability/validity) or assessment of disorders; populations (child, geriatric and ethnic); new models/settings (i.e., collaborative care, asynchronous, emergency, home health); costs; and other outcomes.

TP improves access to care, leverages a wide range of treatments at a distance, and provides outcomes as good as in-person care [3, 17] for geriatric patients [18] (Table 20.2). Comparison and non-inferiority studies show TMH is as good as in-person care in terms of diagnosis and treatment [3]. Guidelines and systematic reviews for therapy by TP exist [23, 24]. Telepsychiatry has been studied in culturally diverse populations including Hispanics/Latinos, Asians, Native Americans, Eastern Europeans, and other populations (e.g., those using sign language) [3, 9]. Acceptance of TMH is generally quite high, and it was better for older adults than adults in one study treating depression in rural populations [25].

Table 20.2 summarizes telepsychiatric clinical/outcome studies for geriatric patients. Table 20.3 provides tips on clinical, program, and system issues and provides outcomes and evaluation as it relates to new technology options.

Table 20.2 Summary of telepsychiatric clinical/outcome studies for geriatric patients

	Study	N	Study origin	Technology	Description	Comments/results
Nursing home	Jones [19]	2	USA	ISDN 128 KBS	Case reports	Able to provide care sooner and staff felt supported
	Lee et al.	140	South Korea	T1	Prospective over 2 years: CDR, SBT	TP = in-person; nurses satisfied; caregiver distress reduced;
	Tang et al.	45	Hong Kong	ISDN 512 KBS	Prospective over 1 year	Satisfaction high with learning curve; some savings in costs
	Johnston et al.	40	USA	ISDN 128 KBS	Descriptive study: MMSE	Satisfaction high; efficient use of psychiatrist's time
	Lyketsos et al.	–	USA	Standard telephone	Descriptive study	Reduced hospitalization rate compared to past
	Rabinowitz et al.	24	USA	ISDN 334 KBS	Pilot study: DCM	Satisfaction high; communication between providers and staff good
	Yeung et al.	9	USA	ISDN 384 KBS	Descriptive study: CGI-I	Satisfaction high: significant improvement in 6/9
	Rabinowitz et al.	106	USA	ISDN384 KBS	Descriptive study	Cost and time savings exceeded the start-up costs

Table 20.2 (continued)

	Study	N	Study origin	Technology	Description	Comments/results
Other	Montani et al.	15 medical inpatient	USA	Coaxial cable	TP vs. in-person: MMSE, clock drawing	Nearly equal, with 0.95 correlation: all preferred in-person, though
	Menon et al.	24, medical inpatient	USA	Standard telephone	In-person (twice) vs. in-person/ video:	TP as reliable as in-person
	Grob et al.	27, veterans home	USA	ISDN 384 KBS	In-person (twice) vs. in-person/ video:	TP as reliable as in-person
	Saligari et al. [20]	20, primary care	USA	ISDN 384 KBS	TP vs. in-person: MMSE, GDS	Equal, with MMSE 0.9 and GDS 0.78 correlation
	Shore et al. [21]	16, veterans home	USA	T1	TP vs. in-person: DSM-IV, clock drawing	TP equal to in-person
	Loh et al.	20, community	USA	ISDN 384 KBS	TP vs. in-person: MMSE, GDS	Nearly equal with 0.8 correlation for dementia
	Cullum et al. [22]	33, primary care	USA	ISDN 384 KBS	TP vs. in-person: MMSE, clock	High correlations (> 0.60) for all, though only 0.48 for clock drawing
	Turvey et al.	118, home	USA	Home monitoring system	Screening for depression with PHQ-2	95.6% completed the screen; helped with triage and treatment
	Fortney et al.	395, primary care	USA	–	TP vs. in-person (matched site)	For depression, TP patients more likely to respond at 6 months and
	Sheeran et al.	19, home	USA	ISDN 384 KBS	Descriptive: DCM, English & Spanish	For severe depression, all patients improved to mild depression
	Gellis et al.	102 (51 usual, 51 tele),	USA	Home monitoring system	Telehealth care for chronic illness & depression	Telehealth patients had significantly improved problem-solving skills & self-efficacy in managing their medical condition
	Egede et al.	204 (102 as usual; 102	USA	Standard telephone	TP vs. in-person: psychotherapy	TP vs. in-person; psychotherapy
	Vahia et al.	22, home	USA	DSL 512 KBS	P vs. in-person: neurocognitive testing	TP as good as in-person (5panish-speaking patients)
	Castanho et al.	69, home	Portugal	Skype	TP vs. telephone: cognitive testing	TP testing: sensitivity 87.8%, specificity 86.4%
	Amirsadri et al.	1, home	USA	10-inch tablet	Case report	Diagnostic clarification; treatment plan modification

Footnotes: *KBS* kilobits per second, *CDR* Clinical Dementia Rating, *SBT* Short Blessed Test, *BDS* Blessed Dementia Scale, *CGI-I* Clinical Global Impressions-Improvement Scale, *MMSE* Mini-Mental State Examination, *DCM* Depression Care Management module, *PHQ-2* Patient Health Questionnaire-2, *HDRS* Hamilton Depression Rating Scale, *GDS-15* Geriatric Depression Scale-15, *BPRS* Brief Psychiatric Rating Scale

Table 20.3 Tips on clinical, program, and system issues: outcomes and evaluation related to new technology options

Fundamental issues and components of evaluating care	1. Keep it simple: 1–2 foci to evaluate (e.g., depression as a diagnosis and a mobile app).
	2. Use a known standard of evaluation (i.e., Patient Health Questionnaire-9; PHQ-9 for depression; adapt a telepsychiatric satisfaction instrument for a mobile app)?
	3. Customize patient outcome targets (e.g., social engagement impact on depression,
	4. Measure satisfaction with an existing 5- to 10-item survey for regular care and one technology options (e.g., a chat room or a diary for depression),
	5. Contextualize the evaluation with a specific population or clinical setting
	a. Age or population (e.g., for patients over 60; outpatient; use of substance by screening with the Alcohol Use Disorders Identification Test (AUDIT)
	b. Disorder-specific (e.g., plan for tracking suicidal ideation for a depressed patient, in general, or if a teenager due to high risk)
	6. Employ a log/diary by the patient and the clinician about
	a. The experience, overall
	b. How and what technology was used and the relative frequency, too (e.g., texting 3 times/week)
Questions, reflections, and considerations for patients	7. What am I seeking when I choose to view a website, visit a chat room, get an informal suggestion, or work with a clinician directly?
	8. What are my means: time, $, and other resources?
	9. What is my learning style: alone vs. group of learners, reading versus doing something, prefer a little versus a lot of instruction?
	10. Do I experience my provider the same or differently at a distance, and what expectations did I have that I was not fully aware of?
	11. How intensive of a treatment do I want and how much should I "connect" in-person and on-line?
	12. How do I choose a clinician based on information on the Internet, screening them by phone or in meeting them?
	13. How do I pick the "best" technology option?
Clinical care issues for the provider related to patient care	14. Do the new technologies and associated behaviors affect the therapeutic relationship, clinical approach and treatment plan?
	15. Is tradition care complemented by technology-based options that are patient-driven?
	16. Is there a shift in my action (e.g., am I doing things 'outside' the regular 'hour?'), is it paid for, and what are the unanticipated consequences?
	17. Did I do things better/worse than expected, what are my technology-based strengths and did I have any unusual reactions?
	18. Did the patient and I talk about the options, work together to select the plan, and how should be continue to discuss this?
Questions for clinical, program and system administration	19. Are we using a standard approach or was it left to chance or played out spontaneously?
	20. What are we measuring and what is the best way to do it?
	21. How often are participants 'checking in' off-line, is it spontaneous/cued, is it tracked/reviewed, and are the important points fed back into the process of care?
	22. What are the outcomes are we measuring for patients, family, clinicians, and systems?
	23. Can the technology help us use resources better, as interdisciplinary teams help us in providing a range of services in stepped care?
	24. How does technology affect folks from the care coordinator to those with the most complex clinician roles and responsibilities
	25. What additional resources (i.e., time, $, staff/manager/medical director/ administrative director, trainings) are necessary to use new technologies?

20.5 Geriatric TP

Geriatric data are emerging, but more studies are needed related to access to service, functional challenges, and challenges, and primary care provider (PCP) attitudes [12]. There are many descriptive, non-randomized nursing home TP studies with positive outcomes, usually for depression or major neurocognitive disorder (dementia), and these show that consultant time is efficiently used (Table 20.2) [26]. Other assessment, cognitive intervention, and outcome studies—many done in medical settings—have direct in-person comparison groups with outcomes being equal [12]. In order for a TP program to be embraced, outreach to local PCPs, nurses, and other community healthcare workers is needed; a long-standing relationship between PCPs and a local geriatric MH outreach team is helpful [27]. A Canadian national survey of TP programs found that the number of geriatric consultations was low relative to adult and child/adolescent consultations [28].

A good geriatric mental health history not only includes the patient's point of view but also collateral information from all other stakeholders and medical providers—largely dependent on where the patient generally resides and is cared for (e.g., home, family and caregiver, nursing home, staff, and others). Cognition, pain severity, physical/other limitations, and environmental factors that may affect assessment are important to understand. Screening of geriatric patients via self-report questionnaires or clinician-rated instruments is virtually the same as for in-person assessments (e.g., Patient Health Questionnaire, 9 items; Geriatric Depression Scale, 30 items).

Additional items to keep in mind when using TP include:

- Informed consent: verbal or written, depending on the state.
- Pre-visit event summary: an accounting of general events and the patient's attitude, comments, complaints, sources of information, and clinician observations (e.g., olfactory/vision/hearing limitations, gait/balance problems, other) need to be communicated before the patient enters the room.
- The clinical examination, in general: this may require staff assistance (often a nurse facilitator) to complete, particularly if a patient is delirious, combative, or agitated, has a low level of formal education, or suffers from aphasia, poor hearing, or vision impairment.
- Cognitive examination: may require item substitution if clock drawing or sentence writing cannot be uploaded to see or held visually in the camera; again, staff are better in assisting here so as to not answer questions for the patient.
- Physical examination: camera control at the far end enables easy wide angle, close-up, and focused viewing to detect tremors, micrographia, and other abnormalities, but staff may need to be trained to check for extrapyramidal side effects (EPS) like cogwheel rigidity.
- We encourage family member to attend in general and when there is significant cognitive impairment, as this enhances patient acceptance. Families are very welcoming of TP interventions and are grateful for the extra time and effort put forth to facilitate a TP encounter [26].
- We recommend that most or all TP encounters for nursing home residents or elders in similar environments include a member of the social work staff to give input on family of origin, family dynamics, and past family and social history [26].

20.6 Models of Care for TP

Models of TP care may be organized into low-, moderate-, or high-intensity levels of care based on intensity of care intervention, technology complexity, specialist time, patient acuity, and use of other resources [10, 29] as updated from a previous formulation (Table 20.4) [29]. Services, models, and outcomes can be stratified by who is designated the primary service and the secondary consultant. Some of the first e-models of care were based on telephone and e-mail curbside consultations [10, 11, 30]. TP consultation care [3, 25], collaborative care [31–35], and

Table 20.4 Telepsychiatry models for psychiatry and primary care based on clinical outcomes, roles, and resource allocation

Intensity tier	Model method	Model examples and features
High	TP collaborative care	Versus in-person care in terms of diagnosis and treatment of depression in children and adolescents medication use for depression in children/ adolescents and adults
		Populations: adults with PTSD children/adolescents with ADHD
	Randomized controlled trials (RCT): TP with other methods	Synchronous TP (STP) and ATP for children with ADHD, with parents and teachers, with screening using a checklist, a diagnostic assessment instrument, rating scales for inattention, hyperactivity, role performance
	RCT disease management for depression	Improved TP and usual care outcomes over 12 months; the latter group benefitted from the Hawthorne effect and providers' application of skills from the intervention group
	Non-RCT, informal stepped care	Grant-funded e-MH service with a priori outcome evaluation with
		1. two-tiered triaging to
		2. therapy on site and/or initiation/change of antidepressant by PCP
		3. ongoing telepsychiatric consultation (by telephone, e-mail or video)
		4. continuing medical education
Mid	Initial +/− follow-up TP consultation	The consultation care or consultation-liaison (CL) model increases capacity of MH services7 with diagnostic assessment and medication changes in 91% and 57% of cases, respectively, leading to clinical improvements in 56% of cases
		PCP knowledge, skills, and complexity of questions improve over time, particularly rural
	Add geriatric nurse practitioner (GNP)	Adding a geriatric nurse practitioner (GNP) to an outpatient diagnostic multidisciplinary facility for patients with cognitive disorders
	Veterans affairs TP	Geographically dispersed population
	Non-RCT child and adolescent program to boost MH services	Contract for child and adolescent MH screening, therapy on site, telepsychiatric consultation (phone, email, or video), continuing medical education, and staff training improved patient outcomes and site-based
	Asynchronous telepsychiatry	This involves the PCP interviews a patient with video recording and sends the video with historical information for review by a distant psychiatrist; feasible, valid, reliable, and cost-effective in English and Spanish-speaking patients in primary care
Low	In-person and telephone doctor-to-doctor "curbside" consultations	Meet approximately 33% of informational PCPs' needs "in-time"
		Part of collaborative care
		Purposeful and timely
	Telephone or email doctor-to-doctor "curbside" consultations	A multi-specialty phone and email consultation system to PCPs for the care of adults and children across specialties with developmental disabilities
		TP consultation as part of a disease management to accelerate care; 1/3 did not need the TP
		Align PCPs' diagnoses and medication treatments and improve PCPs' knowledge and skills
		Improve nursing home TP care for depression or dementia Part of TP collaborative care and integrated
	Oversight of PCP for PCMH	Improve patient care and health. Desk-mounted video systems offer great convenience for therapy to cancer patients to avoid travel, but the cost used to be prohibitive for most consumers
	Case review with PCPs	Case review of diagnosis and follow-up to change treatment plans and improve knowledge Videoconference "virtual grand rounds" are led by a team of MH specialists to review and discuss cases, by Specialty Care Access Network-Extension of Community Healthcare Outcomes (SCAN-ECHO) model
	One-time cultural consultation	Cultural consultation to rural primary in order to match patients and specialists by culture, ethnicity, and language
	Distance neurocognitive assessment	Neurocognitive assessment via TP using a Spanish-language battery was comparable to in-person (IP)

asynchronous TP (ATP) care [36] models emerged and have significantly improved outcomes [34]. Hybrid care—combining in-person and TP care or TP with other technologies [37]—is increasing and referred to as "e-stepped care" and "e-integrated care" [10].

20.7 Findings, Models, and Suggestions for TP to Inpatient Psychiatry, Medical/Surgical, and Emergency Settings

20.7.1 Inpatient Psychiatry Issues

Inpatient psychiatry has high acuity, very ill patients, and complex treatment plans. TP may help prevent undesirable and longer hospitalizations for rural patients [38]. Interestingly, 77% of involuntarily committed patients said their TP provider understood them as well as or better than in-person and 63% thought the system was the same as or better; only 14% stated that they would be generally unwilling to use TP again [39]. Overall, TP appears effective and could provide flexibility and better leverage resources. It is important to understand interdisciplinary, team-based delivery models when using TP for any acute care settings, including inpatient settings [30, 40]. An appropriate care model includes care components like systematic assessment, biopsychosocial treatment, interdisciplinary planning, and an emergency in-person plan. If this program is continued, one clear upgrade would be "attending" the interdisciplinary treatment plan team meeting daily for communication and integration of data [40].

Vignette 2 A 79-year-old man seen in the ED by the on-call psychiatrist. The patient reported suicidal thoughts, reported "visions" of animals (i.e., visual hallucinations, VH) at home, and thought he heard auditory hallucinations (AH) telling him to do things. His mood was depressed, hopeless, and anxious, and he felt "overwhelmed." He appeared withdrawn and tired. He denied substance use. His hygiene was adequate, but his mouth was dry. He had short hair and a well-trimmed beard. His affect was flat. Thoughts were remarkable for being distractible. He reported VH, but it was unclear about AH—no SI, HI, or delusions. PE/Lab/Imaging: Elevated blood urea nitrogen and creatinine; no imaging was done. He was diagnosed with dehydration and possible altered mental status due to other conditions.

He was hydrated and supported overnight. On day 2, he was alert, was more responsive, and denied VH/AH. He reiterated depression for weeks and was diagnosed with depression, recurrent, severe without psychosis, and admitted to inpatient psychiatry unit. During day 1 of hospitalization, he was treated by exercise, diet, sleep, education, supportive therapy, and groups for coping skills. Fluoxetine was 10 mg started and seen in TP daily by the same psychiatrist; medication was increased to 20 mg on hospital day 4. Patient was discharged home on hospital day 7.

TP was preferred as a means to conduct interview by the same physician rather than be seen by another physician and the patient stated, "I just want to work with one doctor."

A prejudice remains that psychotic patients are not eligible for TP care and that they do not use technology. Initially, providers of TP hesitated with outpatient schizophrenia patients, as there was concern about delusions of surveillance would impede its success, but that has not been found. Other presumptions were stimulus overflow/inability to deal with the abundance of information, distraction or disorganization by psychosis, lack of energy, paranoid ideas, and/or fear of symptom provocation.

However, psychotic patients can successfully use the Internet for information related to their illness and medication (e.g., side effects and the hope of finding better medication) [41–43]. On the other hand, patients may feel the need to guard themselves against excess information that Internet frequently offers. Many psychotic and otherwise severely ill psychiatric patients have felt comfortable with TP, and some see advantages of using it over traditional in-person visits in this preliminary series.

20.8 Medical/Surgical Inpatient, Nursing Home, and Other Medical Settings

Initiation of TP may begin with determination of location of the patient, the model, the providers, and the actual provider of care. Ideally, MH service delivery (e.g., in primary care) has a continuum of providers: the care coordinator-medical assistant-social worker-nurse-primary care provider-MH clinician-telepsychiatrist [30] (Chap. 4: Interdisciplinary Roles). Patients learn to use care managers (to monitor follow-up plans, for interventions based on risk stratification, and for patient tracking). In optimal MH service delivery, the psychiatrist better attends to more complicated cases, provides clinical oversight, and reviews cases in team formats. This model ensures a range of effective, non-pharmacologic, health psychology-based treatments. The key questions:

- The patient and others.
  - The patient's setting: inpatient vs. nursing facility vs. emergency room vs. outpatient vs. home; as is relevant, MH vs. medical setting location informs the plan.
  - It is important to be aware of legal standing (i.e., surrogate decision-makers, power of attorneys, conservatorships, and other issues) (Chap. 5: Legal Aspects).
  - Who else is helping from the patient's end? Spouse/partner, child/children, other family/siblings, nurse, social worker, therapist or others may be involved.
- The model of care.
  - Low-intensity options: telephone doctor-to-doctor "curbside" consultations, typically; rarely by e-mail or text messaging.
  - Psychiatric care (freestanding, as if a patient came to a clinic). This involves follow-up plans, prescriptions, and contingencies for emergencies for which one is directly responsible.
  - Consultation care: consultation to a PCP or other medical provider, who provides the care. This involves follow-up plans, prescription instructions (with specifics, backup options), and contingencies for emergencies for which the PCP/clinic is directly responsible. It is helpful to be familiar with local MH standards, services, and expectations.
  - Collaborative care: joint provision of care by specialist and PCP. This involves a mixture of the two models above, often with care coordinators and other interdisciplinary providers helping.
- Legal/consultation-liaison issues for evaluation: civil commitments, forensic issues (e.g., capacity evaluations), or others (Chap. 5: Legal Aspects).

Many hospitals do not have regular access to psychiatric consultation services, particularly in a nonacademic community and smaller and rural hospitals [44]. Even some academic health centers (AHC) in urban settings struggle with this service due to cost issues, managed care influences, and other systems' pressures [45]. This service requires a skill set for diagnosis and co-management of concurrent medical/surgical and neuropsychiatric illness, as well as liaison functions to medical colleagues to better understand behavioral issues relevant to the presentation of medical and surgical inpatients. An AHC-based telemedicine-based care model delivered 30 inpatient psychiatry consultations to an affiliated, separate small hospital without on-site services within 24 hours; only 1 patient was unwilling to participate in the telemedicine interview.

Teleconsultation supports a focus in healthcare on cost analysis, integrated care, and quality/performance improvement (process of care, cost, communication, and other intangibles). Integrated models of in-person care usually stratify patients in quadrants—with the setting predetermining those quadrants—and co-locate psychiatric and medical services [46]. In *outpatient* setting, there are four quadrants: I, low medical and MH; II, low medical and high MH; III, low MH and high medical; and IV, high for both. In the medical *inpatient* setting, the quadrants are I, medium-to-high psychiatric acuity and none-to-low medical acuity; II, medium-to-high medical acuity and none-to-low psychiatric acuity; III, medium-to-high psychiatric acuity

and low-to-medium medical acuity; and IV, medium-to-high psychiatric acuity and medium-to-high medical acuity [47].

20.9 Using Information Technology, E-Health, and E-BH Innovations for Psychiatric Inpatients and Outpatients

20.9.1 TP to Geriatric Populations [18]

- Nursing home TP non-randomized studies have been effective for depression or major neurocognitive disorder (dementia), making evaluation easier and more efficient using consultant time [26].
- Assessment, cognitive intervention, and outcomes have been similar to in-person and a new development is telemonitoring of depression in the home, which facilitates connectedness [48].
- Neurocognitive assessment via TP using a Spanish language battery was comparable to in-person (IP) testing for rural Latino patients in a sample of Spanish-speaking older adults in a rural setting; the order of IP and TP test administrations [38].
- Adding a geriatric nurse practitioner to an outpatient diagnostic multidisciplinary facility for patients with cognitive disorders reduces subjective burden of the informal caregiver [49].
- Telehealth problem-solving therapy (tele-PST) for low-income homebound older adults in a 6-month, randomized controlled trial showed that both tele-PST and in-person PST were associated with reduced depression severity and disability, but tele-PST benefits lasted significantly longer than those gained through in-person PST [50].
- In elders with chronic illness and depression, compared to regular in-home nursing with education, a telehealth nurse intervention (daily telemonitoring of symptoms, body weight, and medication use; eight weekly sessions of problem-solving treatment; and communication with patients' PCPs prescribing antidepressants) significantly reduced depression scores (50% less at 3- and 6-month follow-up) and improved self-efficacy with care management [51].

20.9.2 mH Principles/Approach: mH

A review of mobile health [52] (mHealth) can be defined as the use of mobile computing and communication technologies in healthcare and public health [53]. A common low-cost feature is short message service (SMS) containing 160 characters or fewer between smartphones [54]. Messages can be sent in a standardized or individualized format and are available on all smartphones and other low-cost devices. Text message frequency (daily, weekly), text message interactivity (one-way vs. two-way), personalization (message content based on known characteristics, including patient's condition, history), and tailoring (message frequency, interactivity, and/or content matching each recipient's characteristics) [55]. SMS text messages can also be sent from Web-based platforms that allow for pre-scheduling of sending, automation, and better monitoring. Competencies for mobile health have been suggested [56].

Potential integration and components of a generic mobile health system potentially link four core pillars: (1) a national health network, (2) hospital and other acute care centers, (3) home-based care, and (4) mobile health devices [57]. The main functions of the smartphone that have enabled its clinical applications [57] include:

- Voice/video calling: convenient way for clinicians and patients to remotely communicate
- Short message services (SMS) and multimedia message services (MMS): transmit text messages and video clips/sound files as a cost-effective way to deliver education
- Multimedia functions: provide a range of learning opportunities.
- Inbuilt sensors: touch, motion, and GPS sensors that simplify clinical assessment and lifestyle and social activities
- Device connectivity: practical and less error-prone than manual data entry

20.9.3 Range of E-BH Options [11]

The mobile phone or tablet PC is the core device linking clinicians with patients in their own environment (Fig. 20.2). Smartphones have integrated virtually all the core functions of a modern computer, and the apps facilitate use of these functions in everyday lives. These functions help patients to self-manage their diseases and provide other paths to clinical interventions. Mobile devices serve as organizing hubs that link patients' health data to other health services; the bi-directional flow enables routine care or education from clinicians to patients in their own environment. Wireless monitoring devices gather data from sensors, input that data into a mobile medical app on the smartphone, and then relay the information to a centralized network. Patients with bipolar disorder, schizophrenia, and other serious mental illnesses are common users of mobile devices [58]. There is greater concordance between smartphone-captured mood ratings and clinician-rated affective symptoms than between paper-and-pencil mood ratings and clinician ratings [59]; the latter often includes incomplete data, whereas alarms used with the former are helpful [11].

Mobile apps offer (1) portability for access anytime, anywhere, regardless of patient geography and transportation barriers, (2) an inexpensive option versus traditional desktop computers, and (3) additional features (e.g., context-aware interventions and sensors [60] with real-time feedback). MH app demand is high across census-designated areas, generations, and, to a degree, age, with less use by older adults [60]. Stress reduction programs using a mobile phone app is an increasing field of interest [61]. With regard to mMH, a review of mobile phone and Web-based text messaging in mental health of 36 studies (out of a possible 677) showed that text messaging was used in a wide range of mental health situations, notably substance abuse (31%), schizophrenia (22%), and affective disorders (17%) [62]. Four ways were identified in which text messages were used: reminders (14%), information (17%), supportive messages (42%), and

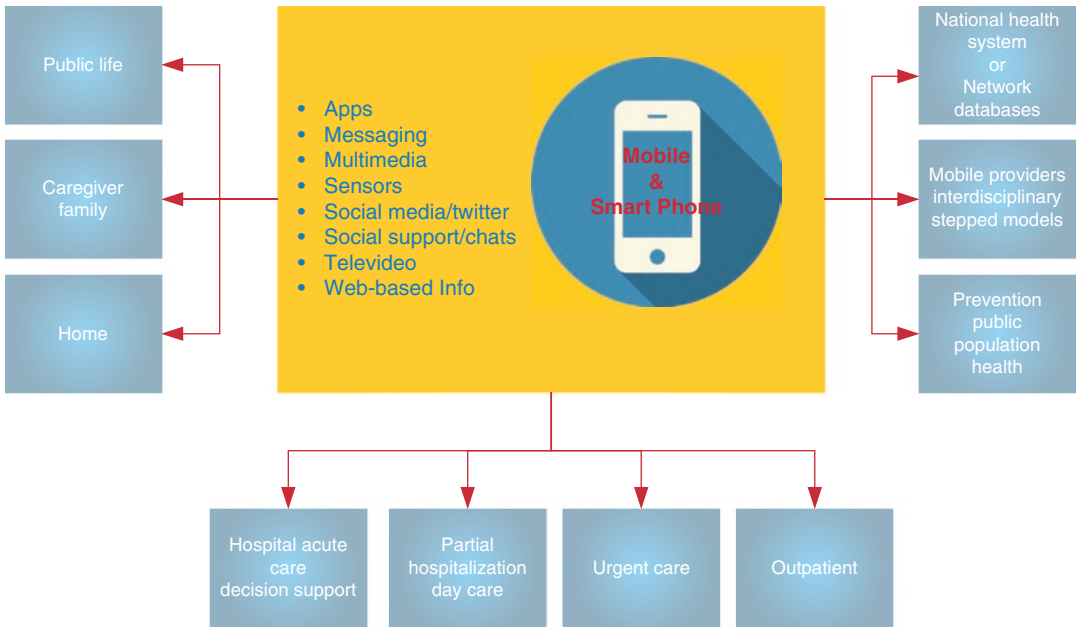

Fig. 20.2 How mobile health and apps integrate information in the digital age

self-monitoring procedures (42%); combination use was common, too.

Vignette 3 Use of video, telephone, and mobile apps in the inpatient-outpatient continuum.

A 65-year-old female with bipolar disorder, mania, and psychotic features was prepared for discharge on hospital day 9 (HD9). She had been on lithium 300 mg BID, but it was not tolerated (i.e., confusion) and at a low level. She was switched to valproate 500 mg BID and olanzapine 10 mg HS. The follow-up plan is with the inpatient psychiatrist as outpatient on day 14 (OD14). Her sister is helping, and they live together.

Outpatient course: The patient did well with mood, organization, and daily diaries on a hard copy mood log. Her sister called at OD5 due to the patient sleeping only 4–5 hours/night.

- Decision point: (1) short in-person visit, (2) telephone call, or (3) brief Webex or other private link for video. Selection: video to assess for relapse and literally see eye contact, focus, and affect. Results: the patient engaged visually with eyes, answered questions, and had full range of affect. Weighing medication options and ease of planning, olanzapine was raised to 15 mg HS.

The patient seen OD14: mood good, sleep okay, and weight gain from #120 to #125 (5′2″).

- Decision point: diet education, walking 30 minutes for 3 days/week and (1) follow-up OD42 or (2) OD42 and implement mobile app for walking, calorie intake, and weight *sent weekly*.

The patient remained stable though at #132 at O28. Psychiatrist called: patient and sister worried.

- Decision point: cut bread and carbohydrate snacks from diet, walking 30 minutes/day, and follow-up OD42; mobile app data *sent weekly* to psychiatrist.

The patient seen OD42: mood good, sleep okay, weight stable at #132. Plan: continue regimen.

20.10 Discussion

Today, TP services are unquestionably effective in most regards, although more analysis would be helpful for some subpopulations. They are effective for diagnosis and assessment, across many populations (adult, child, geriatric, and ethnic) and in disorders in many settings (emergency, home health) and are comparable to in-person care and complement other services in primary care. Additional evaluation (i.e., randomized trials, lack of inferiority designs) would be helpful for some treatments (e.g., psychotherapy), populations (e.g., child and adolescent, geriatric), disorders (e.g., anxiety, substance use, psychotic), and settings (e.g., ER, schools, home MH). In any regard, a sound basis of leadership, administrative coordination, modern equipment, and "good" clinical skills is necessary. Overall, evaluation with formal measures (i.e., randomized trials, lack of inferiority designs) and predictors of outcomes and analysis of variance are moving forward.

Several findings from the evidence base of studies are quite interesting. First, it is clear that TMH is a versatile way to increase access and empower patients—similarly when applied to systems of care, it helps providers and administrators integrate care. Second, TMH can be done in a variety of e-models (e-mail, telephone, video, and other asynchronous options) and it can facilitate clinical care models (e.g., collaborative care into services in primary care settings). Care more thoughtfully conducted—with attention to culture, diversity, and language, "better" care at a distance nationally and internationally—this is now within reach. Finally, though inconceivable to all of us in the 1990s, when systematic application and evaluation of TMH began, we may be at a tipping point in which all the "little" things that TMH makes

possible start adding up—and changing our framework and approach to healthcare—as we move from a new way to practice and a new standard of practice, in which we can better disseminate evidence-based treatments and new modalities of treatment for a number of psychiatric disorders delivered at a distance. A new way to practice is "hybrid models care," which employs in-person and technology-delivered care [63] and by implication, multiple levels of technological complexity (i.e., from low-intensity e-mail and phone to high-intensity videoconferencing).

Research and data will show if hesitation to use TP for acute care (ED) and other settings is prudent caution and/or unnecessary worry. TP liked is liked, though, for many reasons:

- Novelty (i.e., fun, exciting, more visual, video game-like, and, as a result, less threatening).
- Focus/direction, as those with autism or inattention feel "directed" by the technology, making the interview go more smoothly [4].
- Less stigma (e.g., chemical-dependent patients more freely talk about their issues as they felt less judged).
- Authenticity of the interaction to the home or school facilitates better multiparty interaction in more naturalistic setting [40, 64].

Three key questions remain about inpatient TP: (1) Is it really TP that is the limitation or how it is being used? (2) Should we shy away from using it in the sickest patients, particularly those who have delusions, paranoia, and other symptoms that may be theoretically worsened by telemonitoring? (3) How is TP best used—or with what model—in acute psychiatric and/or medical/surgical settings?

Clinicians/faculty are at the crux of these technological paradigm shifts, since they supervise trainees and oversee care. Residents have interests in learning the technology [65], yet there is little in the form of systematic education of TP in residency training [66]. The need for technological competencies led to the TP competencies [67], which used the Accreditation Council of Graduate Medical Education [68] domains of Patient Care, Communications, System-Based Practice, Professionalism, Practice-Based Improvement, and Knowledge; it also was influenced by the CanMEDS competencies [69]. One on technology operational skills was added symbolically. A succinct overview of the TP competencies (Table 20.5) shows three levels based on the Dreyfus model for learners (Level 1, novice; 2, advanced; 3, competent; 4, proficient; and 5, expert) [70]. Live teleconsultations, brief didactic teaching, and case-based learning vignettes are suggested [67].

Since then, TBH [71] competency domains across professions have been added at novice, proficient, and authority levels: (1) Clinical Evaluation and Care, with three subdomains addressing Assessment and Treatment, Cultural Competence and Diversity, and Documentation and Administrative Procedures; (2) Virtual Environment and Telepresence; (3) Technology; (4) Legal and Regulatory Issues; (5) Evidence-Based and Ethical Practice; (6) Mobile Health and Apps; and (7) Telepractice Development. Social media [72, 73] and, finally, mH competencies with domains of patient care, medical knowledge, practice-based learning and improvement, systems-based practice, professionalism, and interpersonal skills and communication—with evidence base, decision support, mobile health, and risks of using smartphones/devices/apps subdomains—were published [56].

The limitations of this chapter are many. First, it only briefly summarizes the existing evidence bases in TP, service delivery models, and mH options to help older adults in a variety of clinical settings. Second, this chapter cannot cover the breadth and depth of the existing databases. Third, some conclusions are based on informal consensus of institutions and authors, rather than on empiric data or a Delphi consensus process; it is based on collaborative work

Table 20.5 Abbreviated telepsychiatric (TP) competencies for patient care [67]

	Area/topic	Novice/advanced beginner	Competent/proficient (e.g., advanced resident/graduating resident/faculty/attending)	Expert (e.g., advanced faculty/attending/interdisciplinary team member)
Patient care	History-taking	Standard history	Informed consent for telehealth Contextualized history (e.g., aware of geographic and cultural specificity)	Address informed consent problems In-depth, well-paced, and concise interview
	Engagement and interpersonal skills	Therapeutic alliance with trust and rapport	Identify and manage problem(s) Adjust interview to technology	Resolve problems and adjust assessment contextually
	Assessment and physical examination	Stratify risk and protective factors based on epidemiology Learn tools (e.g., MMSE)	Assess danger risk and adjust follow-up plan vs. in-person Ensure full MSE or alternative Administer tools with adjustments	Synthesize information Adjust tools contextually (e.g., substitute score item) Teach on distance MSE vs. in-person
	Management and treatment planning	Treatment plan based on who will provide care follow-up with others (e.g., PCP)	Contextualize to patient and PCP Awareness of care continuum Medication recommendations (i.e., side effects) with PCP instructions to initiate, titrate, and augment Follow-up with PCP by TP or phone	Tailors recommendations to resources, culture, and patient preference Engages patient and referring doctor Select "best" mode: e-mail, telephone, or other (and if it changes the process) Refines medication recommendations
	Documentation	Draft TP note hard copy or rudimentary EHR draft	Initial/revised draft with consultation model; complex EHR (e.g., epic)	Balance findings /detail with plan; uses variety of documentation options
	Privacy and confidentiality	Learn in-person basic regulations	Be aware of regulations; TP context Cell HIPAA limitations	Practice within all standards; be aware of technology option limitations; make recommendations to optimize these parameters
Communication	Communication	Clear communication with patient and professionals	Amplify communication based on TP	Trouble-shoot communication difficulties; optimal telepresence; ability to teach/enhance others' telepresence
	Cultural, diversity, and social determinants	Consider diversity of oneself and patients	Adjust to patient culture and preference	Follow cultural formulation frameworks Adjust interview, assessment, and care
	Language/interpreter ability	Use the interpreter	Manage time and pick best option (e.g., professionals > staff and family)	Verbal and non-verbal dimensions

(continued)

Table 20.5 (continued)

	Area/topic	Novice/advanced beginner	Competent/proficient (e.g., advanced resident/graduating resident/faculty/attending)	Expert (e.g., advanced faculty/attending/interdisciplinary team member)
Systems-based practice	Outreach to community	Participate and engage	Identify relevant resources and needs within community	Visit, establish, and maintain relationships Integrate in-person and TP care events
	Interprofessional education issues	Participates in and experiences different roles	Work with IPE team and begin to teach within IPE framework	PE provider and teacher Support interdisciplinary team care Expert level knowledge of the extant
	Care models	Grasp care provider vs. consultant role	Employ traditional referral (i.e., management), and consultation TP Begin to learn collaborative care	Has facility with models of consultation, integrated, stepped and hybrid care; practices with one that fits context
	Rural health	Learn about rural access,	Learn rural health basics	Practice and role model
	Special populations	Learn differences (e.g., veterans, child/adolescent/parent/family, geriatric)	Recognize differences and adapts assessment and management	Practice and role model
	Licensure regulations for TP and model used	Learn in-person regulations and that states differ	Be aware that in-person and TP regulations may/may not differ	Practice within TP regulations state-to-state or within system (e.g., VA)
Professionalism	Attitude	Learn/be open to	Openness to technology, IPE and and consultation process	Lead in groups/teams
	Integrity and ethical	Demonstrate behavior	Role model	Role model and give feedback
	Scope	Become aware of scope issues	Practice within scope(s)	Provide feedback on scope and boundary issues; trouble-shoots problems
Practice-based learning	Administration	Learn basics of in-person care	Be aware of important differences between in-person and telemedicine care	Practice with adjustments to telemedicine care
	Safety and quality improvement (QI)	Systematic assessment; learn how to participate in QI	Identify risks; apply QI information to cases and system	Adjust treatment plan; analyze QI options, select and evaluate
	Teaching and learning	Participates and contributes	Organize and further education	Provide context and steps for learning
Knowledge	Knowledge	Relevance, history	Relevance, history, and evidence base	History, and evidence base, and clinical guidelines
Technology	Adapt to technology	Project self 15% more when using TP; realize non-verbal	Plan for differences, identify barriers, and put patient at ease	Additional ways to engage and express empathy
	Remote site design	Observe	Identify problems and solutions Add toys or furniture for child TP	Pre-planning: iterative improvement Modification based on care options
	Technology operation	Microphone, camera, and other	Operate hardware, software, and accessories; basic trouble-shooting	Optimize components based on context and manage all trouble-shooting

with many primary care systems and input from healthcare delivery model experts, though. Fourth, not all findings apply to all locales or settings therein. Fifth, the landscape of healthcare is rapidly changing; with consumer/patient use of technology, the field will be hard pressed to keep up. Finally, the evidence base on acute care setting and TP needs to improve—for all ages of patients.

20.11 Summary

Telepsychiatry (TP) is as effective as in-person care in terms of outcomes. It leverages a wide range of treatments at a distance to clinics, nursing homes, and patient homes. Geriatric TP is more similar to than different from in-person care and TP for other population ages. The clinician needs to consider the patient population, setting, service provided, model of care, and type of technology to achieve good outcomes. There is steady growth of the TP evidence base; geriatric patients are as open to using TP as other populations and report high satisfaction. TP may be especially useful to expand provision of care to geriatric patients, due to shortage of subspecialists in geriatric psychiatry and geriatric medicine.

TP leverages psychiatric care across age, population, and medical and mental health settings, including inpatient settings. Other information technologies can also provide a variety of educational, self-help, and interactive strategies for patients and clinicians. E-health and e-behavioral health innovations are underutilized but may increase points of entry, enhance patient-provider communication, and help continuity of care—all to prevent illness recurrence. More research is needed on effectiveness and outcomes related to inpatient TP and information technology applied to health services in geriatric patients. Combining technology options with team, interdisciplinary, and interprofessional models is suggested.

Take-Away

- Telepsychiatry (TP) has been shown to produce treatment outcomes as effective as in-person or direct care.
- The use of telepsychiatry for geriatric patients has not yet been studied extensively, but evidence to date from younger populations suggests it will also be applicable to older populations, and this age group appears to like it.
- The potential advantages of information technology applications and telepsychiatry to geriatrics include minimizing transportation time and inconvenience, expansion of professional geriatric expertise to settings wherein it is not available, and increased access to timely patient information.
- Continuity of care with one consistent provider over time, through TP, is often preferable to face-to-face care with several different providers.
- Information technologies and telepsychiatry may help inpatient teams develop treatment plans earlier, by providing more accurate and fresh information from the outpatient settings.

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Placement, Coordination, and Follow-Up

21

Debra Bakerjian, Eric Vanraay, and Bianca Ferris

21.1 Introduction

According to the 2014 National Hospital Ambulatory Medical Care Survey (National Center for Health Statistics), there were 65.9 million visits to physician offices for mental disorders and 5 million emergency department visits with a mental health primary diagnosis in the United States; 15% of those visits were by patients aged 65 or older. In Canada, over 1.8 million people over the age of 60 have a mental health illness—excluding those people with dementia [1]. The number of people over age 65 with mental health or substance abuse problems in the U.S. is expected to reach between 10.1 and 14.4 million by 2030 [2].

In North America, decades of deinstitutionalization and/or defunding for long-term, state-funded psychiatric facilities was not offset with a commensurate increase of outpatient and inpatient mental healthcare resources. There was a 14% decrease in the number of state psychiatric beds between 2005 and 2010 [3]. This imbalance may become even more problematic for the aged patient, as the proportion of the geriatric population expands, along with its psychiatric conditions. Of geriatric patients seen in the ED for mental health issues, 20% are eventually admitted to the hospital, but geriatric patients with mental health issues often have long lengths of stays in the ED waiting for beds (13% of patients in the ED with a medical diagnosis also have longer lengths of stay due to difficulty finding community-based care). Judicious use of available inpatient resources and better coordination between the community services and inpatient services can help alleviate this problem [4, 5].

Aging adults with chronic medical and psychiatric conditions move often between inpatient and community-based healthcare systems. This can strain resources and exert pressure on discharge planners to return patients to community settings without a comprehensive plan [6]. For geriatric patients, an abbreviated discharge process can contribute to decompensation and early readmission to acute inpatient care [7].

Geriatric patients and families are most concerned about who will accept the responsibilities for care [6], Appropriate planning for follow-up must therefore take into account the need for regular medical and psychiatric follow-up, the possibilities of functional decline, the need for transportation and logistics, cognitive limitations, and whom to contact in emergencies or other support needs. It is best for one professional to take

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full responsibility for coordination among all required follow-up systems [8, 9]. Discharge planning meetings during hospitalization can help patients, caregivers, and family prepare for the discharge [8]. Effective discharge planning can help anticipate gaps early in an admission and pursues solutions (Chap. 4: Interdisciplinary roles). This coordination of patient and family education about diseases, symptoms, and recovery can foster self-management [10] with regard to:

- Medications (timing, dosage, side effects).
- Illnesses (identifying symptoms, risks of exacerbations, and coping strategies) [11].
- Knowledge of community resources for additional support [12].
- Actions to take in case of a medical or psychiatric decline, or emergency (e.g., if a urinary catheter appears to cause pain) [10].
- Anticipation of unexpected crises can reassure and avert negative consequences.
- Potential financial barriers.
- Deficiencies in the physical environment or in the home and help for adaptive mobility.

21.2 Vignette

A 77-year-old homeless man was brought to the emergency department by local police after he was found wandering, disheveled, and confused. He was admitted to the acute geriatric psychiatry unit. Over the next 24 hours, he became increasingly disoriented and developed symptoms consistent with acute alcohol withdrawal and Wernicke's encephalopathy. A 50-year history of severe alcohol abuse and alcohol use disorder was obtained.

Treatment for alcohol withdrawal with the Clinical Institute Withdrawal Assessment of Alcohol Scale, Revised (CIWA-Ar) withdrawal protocol, and IV thiamine was instituted. His sensorium cleared, and he became oriented and affable. His cognition showed adequate concentration and attention (Chap. 10: Alcohol and Substance Intoxication & Withdrawal). Compared to his mental status on admission, he appeared personable, cooperative, and engaged in his own care. No family could be found to help plan the discharge. The treatment team became convinced he could live independently due to his rapid recovery and demeanor. There was also a demand for hospital bed space. In order to expedite discharge, neuropsychological testing was not pursued. The inpatient team provided written materials with information about housing, including shelters and vouchers, as well as medical care. The patient agreed with suggestions and repeated the instructions, although sometimes he rambled and gave repetitive comments. He agreed to follow-up at local Alcoholics Anonymous (AA), meetings, which he had attended intermittently in the past.

Within 48 hours after discharge, he was found by police wandering in a local park, disoriented, disheveled, and dehydrated. Upon readmission to the inpatient unit, the patient did not demonstrate any signs of alcohol use or withdrawal. He also did not remember that he was hospitalized 2 days earlier, nor did he appear to recognize any staff, nor could he repeat any of the discharge plans. A thorough cognitive assessment found that the patient could not retain any new information beyond 1 minute, although he often could repeat information immediately. He confabulated, was socially appropriate, was pleasant, and gave well-rehearsed answers to questions. He had limited executive functioning, nor could he articulate any future plan of action (Chap. 2: Neuropsychological Assessment).

At this second admission, the patient was diagnosed with Alcohol induced (major neurocognitive disorder), amnestic-confabulatory type, and alcohol use disorder, severe. No family or others could be found to help with placement. The discharge planner encouraged and initiated the pursuit of a conservatorship for grave disability due to an irreversible mental illness and the inability to provide shelter for himself. A temporary public guardian, with surrogate decision-making power over housing and medical treatment, was appointed. The conservator mandated a structured living situation, with close observation of medical and psychiatric care, and no access to alcohol. The patient was discharged to a long-term, sober living facility. The patient continued to wander off grounds, looking for alcohol.

After about 1 month, the patient appeared at an in-court hearing and presented well. The court found that evidence was insufficient to support a finding of grave disability and the conservatorship was not extended. Alternative housing was offered, but the patient refused; he began drinking alcohol again, and 2 months later, he was hit by a car while crossing the street intoxicated.

The vignette highlights multiple system issues that can contribute to readmission, in spite of adequate effort toward good discharge planning and placement (Chap. 4: Interdisciplinary Roles, Chap. 2: Neuropsychological testing). As in many best practice guidelines, system issues must be addressed in order for appropriate discharge and placement to succeed.

21.3 Best Discharge Practices: Overview and Guidelines

Research has shown that comprehensive discharge planning (as depicted in Fig. 21.1) leads to fewer readmissions and to better outcomes [8–10]. As a result, many healthcare systems have developed evidence-based guidelines when planning transitions. Health Quality Ontario, National Institutes of Health in the United States, and Care Excellence in the United Kingdom, Department of Veterans' Affairs in Australia and the World Health Organization provide examples of standards for care transitions [13–16].

Discharge guidelines cite the principle of "patient-centered care"; clinicians should involve the geriatric patient, family, and/or caregivers

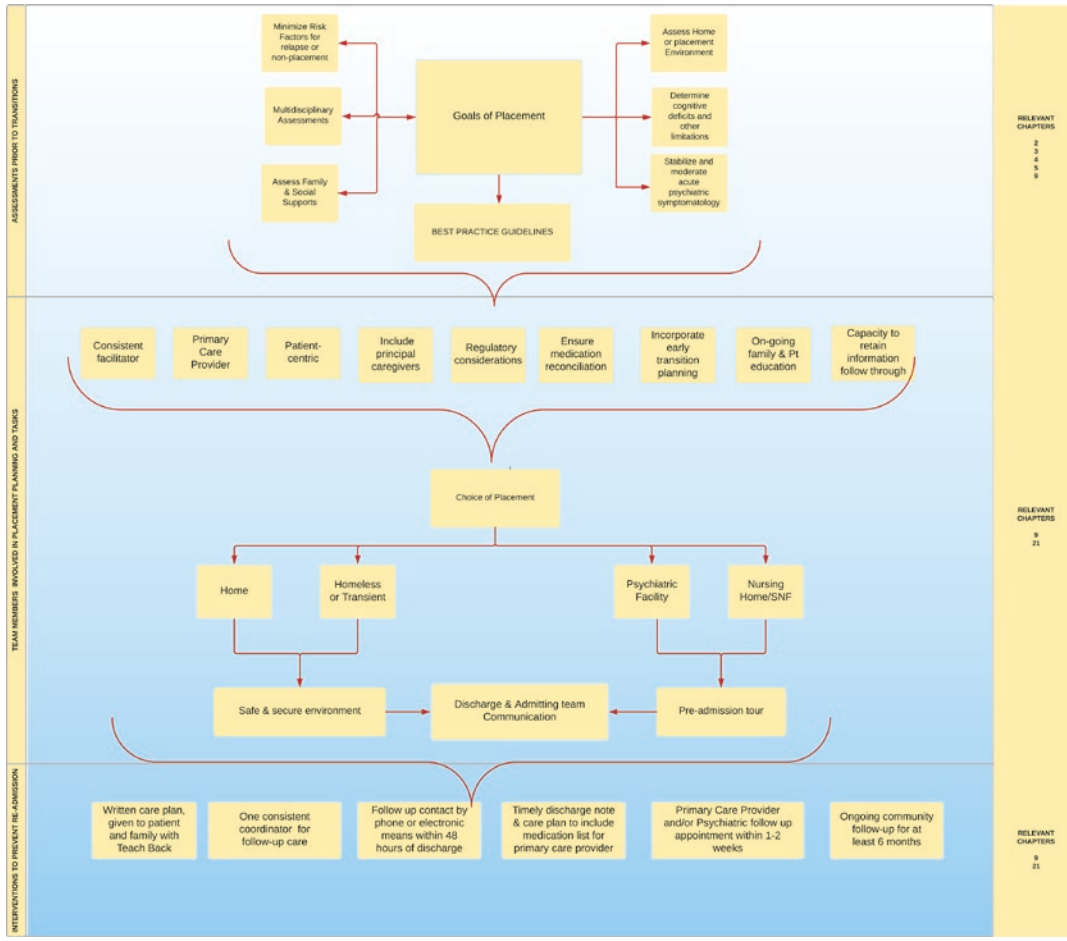

Fig. 21.1 Flowchart of the placement process

[13–15]. Early planning identifies specific patient needs and potential gaps that may impact the transition of care to the community. Recommendations include:

- Delineate in detail the current support network most appropriate for the patient [13, 14].
- Facilitate contact between the discharge planner and outpatient provider that is best face-to-face, either through telepsychiatry or in person (Chap. 20: Telemedicine and Information Technology).
- Encourage the patient to make her/his needs known, minimize dependency, and plan self-management upon discharge [10]. Patients who are involved in the discharge planning meetings feel more prepared [10].

Usually the discharge planner takes responsibility for facilitating the coordination and information-sharing between healthcare professionals [9] who may include physicians, nurses, social workers, pharmacists, occupational therapists, and physiotherapists [15, 16]. Engaging a pharmacy team may also be valuable for family education, to help anticipate future medication questions and avoid crises [11].

It may be a challenge to coordinate professionals and providers from various practice settings. A direct contact is optimal to review major procedures in the hospital, develop the current care plan, detail the current medications, and review care strategies which worked [16]. However, the goal of involving many professionals in face-to-face discharge planning is not an end in itself. Effective planning can also happen with the digital participation of a few professionals who have worked closely with the patient and/or have specific and unique contributions.

Current information technologies can enhance the ease of communication between discharging physician/teams and receiving primary care providers/teams (Chap. 20: Telemedicine and Information Technology). Sharing a thorough review of a hospital course, along with the effective strategies/medication for management of symptoms and behaviors, provides an intrinsic reward for such communication [14]. Figure 21.2 illustrates some basic components of discharge planning.

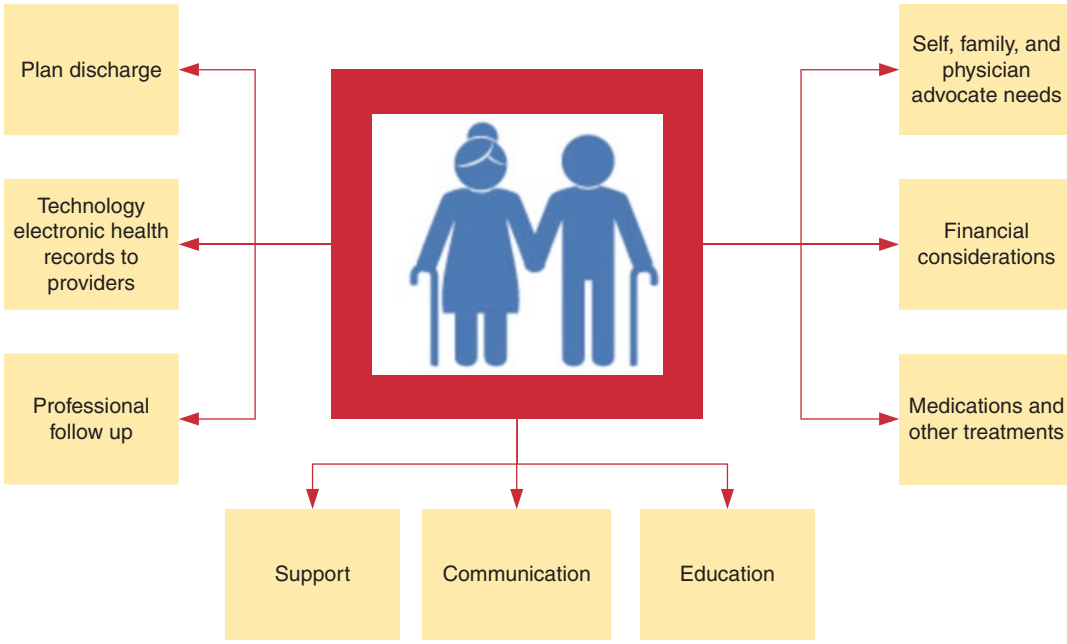

Fig. 21.2 Components of discharge planning

21.4 Challenges and Limitations of Community Placement

Studies in both the United States and Australia identified poor communication between patients, family/caregivers, and health professionals as one of the primary obstacles to discharge planning [22–24]. Geriatric patients require more complex care and discharge planners may not always be able to arrange for appropriate services, which can place larger responsibility on family/community caregivers to provide post-discharge care [25]. Lack of information can leave families/community caregivers feeling unprepared to take on their new role in post-discharge care which can subsequently lead to higher readmission rates [22–27]. Research illustrates the importance of family/community caregiver inclusion in discharge planning as an essential component of developing quality transitional care. To improve communication between healthcare staff and family/community caregivers, strategies to enhance discharge effectiveness include:

- The discharge process should begin within the first 24–48 hours of the geriatric patient's admission [24, 27, 28]. Early and active engagement enables family/caregivers to guide the process, providing families/community care providers with a sense of autonomy while also creating the opportunity to identify caregiver and patient needs [29–35].
- Detailed information should be communicated to both the family and community caregivers about the patient's medical and/or psychiatric condition and prognosis, signs of complications and decline and what to do if those situations arise, physical care requirements, medications, availability of and access to community supports, as well as the roles of the various health professionals involved in the patient's circle of care [26, 36].
- Care-related information is often overwhelming to aging patients and their family members. However, with effective and ongoing caregiver-staff communication, discharge planners can empower families/caregivers to feel confident in the discharge plan, as well as in their ability to take on their new and/or additional roles. When all care providers are in consensus and verbalize understanding of the discharge plan, they can more adequately respond to the patient and family needs.

21.5 Specific Tools/Strategies for Discharge

Promote the following tools or strategies to improve the discharge process:

- Meetings with all involved parties (patient, family, primary care provider, community support professionals) may be a reasonable goal but are often not possible. A discharge planner may use information technology (IT) and other communication modalities to ensure members of the discharge team and follow-up team understand her/his role and responsibilities during the patient's transition [6] (Chap. 20: Information Technology).
- Detailed information in written, email, or other formats are provided to the patient, caregivers, and family prior to discharge [7].
- Names and contact information of care providers, as well as what professional or system is responsible at each level of care (home, another hospital, or nursing home).
- Key strategies and activities to enhance self-management in written form.
- The teach-back method can help assess the patient's (and family's) understanding of the information provided [17].
- Medication reconciliation prior to a discharge is crucial [13, 15, 16]. The physician, registered nurse, or pharmacist reviews current medications to reconcile with the regimen prior to hospitalization. The patient and family can then be included to assess their understanding of the medications and provide the opportunity for questions. Medications taken only "as needed" are especially problematic to

transfer from inpatient care to outpatient settings.
- A Microsoft Excel spreadsheet, the electronic health record (EHR), or other software can help organize the discharge plan, saving unnecessary meeting time.
- An assessment of cognitive function provides perspective on the geriatric patient's ability to understand the symptomatology and follow care plan strategies.
- A list or chart of the discharge plan so that the clinical team can provide a consistent message to patients and family members [13].
- An accurate, detailed medication list for patient as well as the primary care provider [14, 16].
- A list of preferences on ways to receive answers to questions after discharge such as telephone, email, or other technologies [Chap. 20: Information Technology] along with timely follow up with community support after discharge can help ensure the success of transition back home.
- A phone call or email to patient/caregiver/family within the days of discharge to assess the patient's progress and to determine whether support services are addressing needs and reinforcing the care plan [13–15].
- If an aspect of the transition plan has not been completed, the discharge planner can follow up with the appropriate community agency.
- A scheduled appointment with a primary care provider within 2 weeks of discharge; information technologies, telemedicine, and other technologies may bridge gaps if this is not feasible.
- A timely discharge summary to the follow-up team with accurate medication/treatment regimens [14, 16]. To avoid errors, direct communication between the hospital physician/provider and community primary care provider is recommended [14].
- If a community case manager is involved, a follow-up visit with the patient is suggested to occur within the first week of discharge. This support should continue for a period of at least 6 months [11].

21.6 Discharge to Specific Settings

21.6.1 Home, Assisted Living, or Residential Care Home

Discharge planning ideally includes patient, family, and care providers [6, 13, 18]. Inpatient discharge planners and providers may encourage the patient to include key people, since it is in the patient's interest. If there is an objection about confidentiality, specific privileged medical information can be excluded (Chap. 5: Legal Aspects).

To minimize unsuccessful placement to Assisted Living or similar settings, ensure the following interdisciplinary assessments to identify patient risk factors and impairments: [16, 18].

- Ability to manage activities of daily living (ADLs) such as bathing, dressing, feeding, mobility
- Ability to manage instrumental activities of daily living (IADLs); e.g., managing finances, making meals, and organizing transportation
- Whether or not family members or caregivers can provide assistance for ADLs or IADLs, or if additional support needed
- Potential abuse from care providers (Chap. 5: Legal Aspects)
- Decision-making ability and manipulation of information, such as finances, healthcare

The discharge planner facilitates a coordinated plan with supporting agencies [9]:

- Home healthcare, providing physical care, nursing care, physiotherapy
- Community mental health for ongoing case management
- Primary care provider or healthcare system
- Accessible transportation if independent mobility is a problem
- Meal delivery services if meal preparation is impaired
- Medication delivery
- Senior-specific daycare programs or exercise programs
- Outpatient therapy or support groups
- Respite care if needed for family or caregiver

Education for the geriatric patient and family about the illnesses and management strategies [7, 9, 10] encourages self-management and a sense of well-being [10]. Geriatric patients may otherwise adopt a "patient role," allowing others to speak or do many things for them, leading to helplessness and a decreased ability to cope. Training can help geriatric patients invest in their own care [10].

Once the various supports have been arranged and a discharge date set, the social worker/planner will coordinate a meeting or a means for direct contact between key care providers, patient, and family. Key agenda items include a review of which agency will provide support for what, in written and verbal form [7, 13]. Some guidelines recommend a phased return to community [14], which can allow the geriatric inpatient to spend periods of time out of the hospital and at home to try care strategies, identify unforeseen barriers, and build confidence in the transition plan [14].

When at home, follow-up support will vary depending on the complexity of the needs. For example, in-home care services need to be available the day of discharge, such as nursing care, meal delivery, and medications. Within a period of 48 hours, the hospital discharge planner should connect with the patient and/or caregiver/family to ensure planned services have been implemented [13, 15]. It is ideal if outpatient mental health support can provide an in-home visit within the first week of discharge from hospital [11], as well as a consistent contact thereafter. An appointment with primary care provider or psychiatric provider as soon as possible after hospital discharge is also important [15].

21.6.2 Skilled Nursing Facility (SNF)

Multidisciplinary assessments during hospitalization may indicate the need for an increased level of care upon discharge such as an SNF; likewise, a patient coming into the hospital from an SNF before hospital admission may need to return to the SNF. At issue is whether the SNF can provide the level of medical and psychological care needed, can manage behavioral issues, and has access to in-house medical and psychiatric support.

Patients who require SNF care likely have significant functional or cognitive impairment and multiple medical co-morbidities. Care strategies developed in the hospital should be shared in detail with the SNF staff to ensure they can be safely implemented in the SNF [19]:

- Specific wound care management guidelines
- Recommendations for maintaining or improving mobility
- Behavioral management strategies for physical care, providing medications, managing meal times, hygiene, baths, and sleep
- Behavioral strategies to manage agitation and physical or verbal aggression toward staff or co-residents

Once a patient is ready for discharge, it is helpful to know the wait time before a bed will be available. If there is a wait for a bed at an SNF, then discharge planners must continue to function under the philosophy of "patient-centered care." Many hospitals have developed policies and procedures to prevent patients from staying in the hospital while awaiting an SNF bed. These policies include "Home First Policy" (returning home with enhanced in-home supports), transfer to a retirement home with increased support services, or recommending patients pick SNFs with short or no wait lists, which may mean they are located further away from the patient's home community [20]. Ultimately, patients and families need to be fully informed of the options and their rights to make informed decisions about these alternatives [20]. Healthcare professionals also have the responsibility of recommending options that are in the patient's best interests. If a patient is deemed appropriate or needing the level of care provided at an SNF, then it is the responsibility of the clinical team to ensure any intermediate placements meet the same level of care required.

21.6.3 Acute Inpatient Psychiatric Unit: Transfer from Other Settings

A geriatric patient may need to be transferred to an inpatient psychiatric unit for further evaluation once acute medical issues have been resolved. Chronic medical conditions, pain, or physical decline resulting in loss of function and independence can all be contributing factors for anxiety or depression. Geriatric patients with a major neurocognitive disorder (MNCD), especially those with behavioral disturbances, may have developed confusion, agitation, or non-cognitive symptoms within the medical setting. These conditions may require treatment on an inpatient psychiatric unit before community discharge is possible (Chap. 6: Major Neurocognitive disorder with behavioral disturbance).

As mentioned, early communication with the patient and family is necessary when discussing any planned transfer [8]. If the patient is willing to transfer to acute psychiatry, a voluntary admission can be arranged. If the geriatric patient is ambivalent or unwilling, and the controlling legal requirements are met, an involuntary transfer may be pursued (Chap. 5: Legal Aspects; Chap. 13: Involuntary Treatment). The patient and family should be fully informed of their rights and available options. To facilitate a smooth transition, both inpatient teams should communicate between themselves, the patient, and the family [14].

21.6.4 Homeless or Transient Patients

When a homeless patient is capable of making shelter decisions, discharge planners can help find housing appropriate for the care needs [21]. This is dependent on finances as well as available social support in community. When the patient is not capable of making decisions about shelter due to cognitive impairment or psychiatric symptomatology, as in the vignette, a surrogate decision-maker (guardian, family member, or government-appointed trustee) may be sought. Hopefully, a practitioner for medical care can be found who is willing to provide care within a system of healthcare delivery. Discharge planning for aging adults who are homeless includes outpatient psychiatric support and coordination with community resources to help the patient to live as independently as possible [21]. Needs may include resources for transportation, assistance with money management, medication delivery, or in-home nursing support. Discharge to a homeless shelter is not optimal, although this may be the only option.

21.7 Mitigating the Risk of Rehospitalization

Risk factors which predispose older adults to readmission have been identified. These factors may be due to decline in functional status, cognitive changes, the numbers and types of medications, and medical co-morbidities (Fig. 21.3):

- Poor functional status prior to admission (ability to perform ADLs), length of stay in hospital, and history of depression.
- Cognitive impairment (delirium and major neurocognitive disorder), malnutrition, low socioeconomic status, and lack of a social network/support system [17].
- Lack of family involvement and/or absence of social support network to provide caregiving can hinder the recovery process [38]. Intensive Geriatric Service Worker (IGSW) programs, developed in Ontario, Canada, can also help to alleviate caregiver burden, improve health outcomes at home, enhance system effectiveness, and build community capacity to achieve a sustainable health system [39]. IGSWs work collaboratively with emergency department nurses and the Community Care Access Center (CCAC) to develop a plan to keep seniors at high risk for readmission out of hospital. Services may include home visits and phone calls to ensure safety, education, and information to promote treatment compliance and connecting older adults to needed services and supports such as home care, transportation, food, housing, and geriatric outreach.

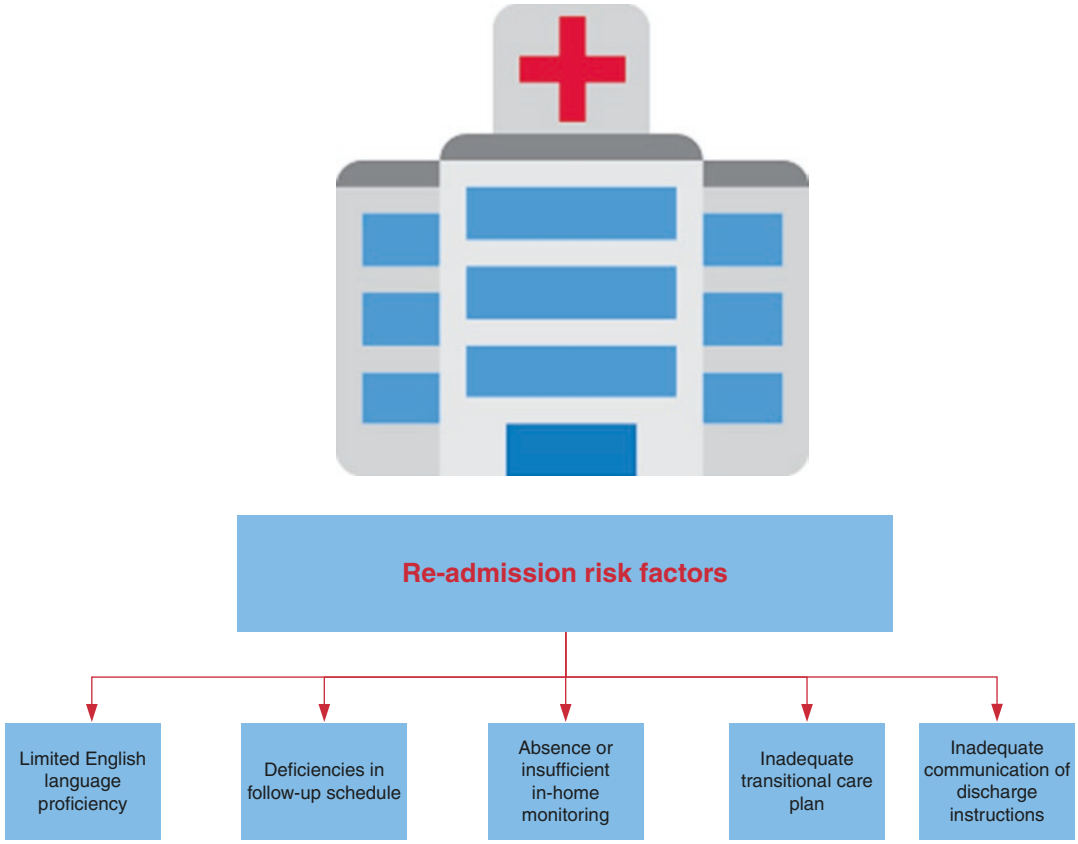

Fig. 21.3 Risk factors for hospital readmission

- Programs which provide behavioral support, such as in Ontario, Canada, offer innovative solutions: A unique initiative called Behavioural Support Ontario (BSO) provides enhanced family/caregiver support in the community, in long-term care or wherever the patient and/or caregiver(s) reside. The program strives to effectively address the complex and ongoing care needs of the geriatric population, particularly those with complex and responsive behaviors associated with major neurocognitive disorder, other mental health conditions including substance use, and/or other neurological conditions.
- Research has offered recommendations on how to better sustain and enhance the health and wellness of seniors: a geriatric interdisciplinary consult team in the emergency department to help with early identification for high-risk patients [39, 40], a systematized program such as the Hospital Elder Life Program (HELP) to better treat delirium and functional decline [41], a specialized unit for the management of responsive behaviors due to major neurocognitive disorders and an Acute Care Unit (ACU) for patients above a targeted age [42, 43], Specialized Geriatric Services (SGS) to assist with co-management of patients across specialities, a comprehensive nutrition support program, an elder-assist program, assistance with appropriate decision-making, and enhancing links between the acute care hospital and the community [44]. The Collaborative Care Model, developed in the United States, provides complex medical and psychiatric outpatient care by a physician, physician assistant, or nurse practitioner in collaboration with a mental health professional and has shown improved outcomes and greater satisfaction [45].

21.8 Summary

Impediments to discharge planning and optimal placement stem from both systemic/ organizational and personal/individual factors. Barriers to discharge at the organizational level include:

- A shortage of community resources/supports
- A limited understanding of the special needs of seniors by health professionals
- Disrupted continuity of care
- Poor communication between seniors and health providers and among health providers
- Difficulty accessing care due to transportation obstacles
- Long wait times for services
- Cost of services [37]

The modern healthcare system does not fully address the complex and ongoing needs of the geriatric population. An "Age-Friendly Hospital" (AFH) and specialized geriatric training for all healthcare professionals is one possible solution [44]. Guiding principles include a favorable physical environment, zero tolerance for agism throughout the organization, an integrated process to develop comprehensive services using the geriatric approach, assistance with appropriateness decision-making, and fostering links between the hospital and the community [44]. By incorporating the "geriatric approach" into practice standards for all care programs, geriatric patients can receive quality care regardless of where they are in hospital and who is treating them. Other barriers that seniors are likely to face due to the nature of the aging process include difficulty adhering to treatment recommendations, language and cultural barriers, social isolation, ineffective self-management, poor health behaviors, and having limited knowledge into their illness [37].

Discharge planning and quality transitional care is a multifaceted process that has become increasingly complex on both a large and small scale. Unfortunately, there is no easy solution to improve the efficacy of care transitions, particularly with the shortages at all levels of mental healthcare workforce. By establishing the "geriatric team-based approach" as a best-practice method, it is hoped that more geriatric patients will be able to age in place longer or become established in a safe, secure, and appropriate setting after an inpatient psychiatric hospitalization.

Take-Away

- Discharge planning should start early in the admission.
- Early and ongoing communication with patient and family/caregiver needs is crucial in discharge planning.
- Assessments by the following disciplines all promote the development of quality transitional care plans: nursing, physical and occupational therapy, geriatric psychiatry, speech and language pathology, dietary/nutrition, primary care, neurology and other medical specialties, neuropsychology, and social work.
- Input from these disciplines aids in the development of a coordinated and integrated treatment plan for long-term follow-up.
- Evidence suggests that tailored discharge plans which integrate both formal and informal supports and address complex needs, are associated with the prevention of emergency department revisits and readmissions [38].
- Accurate delineation of patient needs and limitations, cognitive and physical, is crucial for effective discharge planning and prevention of early readmission.
- Detailed information about the patient and direct communication from inpatient staff to follow-up providers is crucial.
- Anticipation of crises, recognition of gaps in care/follow-up, and limitations of care can prevent early readmissions.
- The geriatric patient is especially vulnerable during transition to new or unfamiliar settings.

- Direct communication between the discharging healthcare providers, discharge planning staff, and the receiving provider team is optimal.
- Person-centered care includes attention to medical, psychological, emotional, cognitive, and social situations as well as special considerations for the discharge destination.
- Follow-up after discharge can correct errors and fill gaps in service, in order to head off an early readmission.

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