Intensive Care Med (2001) 27: 1297–1304
DOI 10.1007/s001340101017

ORIGINAL

Marc-Jacques Dubois
Nicolas Bergeron
Marc Dumont
Sandra Dial
Yoanna Skrobik

Delirium in an intensive care unit: a study of risk factors

Received: 11 April 2000
Final revision received: 1 November 2000
Accepted: 14 May 2001
Published online: 17 July 2001
© Springer-Verlag 2001

M.-J. Dubois · Y. Skrobik (✉)
Division of Critical Care,
Université de Montréal,
Montréal, Canada
E-mail: skrobiky@total.net
Phone: +1-514-2523400ext.6229
Fax: +1-514-9398891

N. Bergeron
Department of Psychiatry,
Université de Montréal,
Montréal, Canada

M. Dumont
COREV, Canada

S. Dial
Department of Critical Care
and Respirology, McGill University,
Montréal, Canada

Address for correspondence: Y. Skrobik,
Soins Intensifs, Hôpital Maisonneuve-
Rosemont 5415 Boulevard de l'Assomption, Montréal, Québec, Canada H1T 2M4

Abstract *Objectives*: (1) To establish risk factors for the development of delirium in an intensive care unit (ICU) and (2) to determine the effect of delirium on morbidity, mortality and length of stay.
Design: Prospective study.
Setting: Sixteen-bed medical/surgical ICU in a university hospital.
Patients: Two hundred and sixteen consecutive patients admitted to the ICU for more than 24 h during 5 months were included in the study.
Interventions: Medical history, selected laboratory values, drugs received and factors that may influence patient psychological and emotional well-being were noted. All patients were screened with a delirium scale. A psychiatrist confirmed the diagnosis of delirium. Major complications such as self-extubation and removal of catheters, as well as mortality and length of stay were recorded.
Results: Forty patients (19%) developed delirium; of these, one-third were not agitated. In the multivariate analysis hypertension, smoking history, abnormal bilirubin level, epidural use and morphine were statistically significantly associated with delirium. Traditional factors associated with the development of delirium on general ward patients were not significant in our study. Morbidity (self-extubation and removal of catheters), but not mortality, was clearly increased.
Conclusion: Predictive risk factors for the development of delirium in studies outside the ICU may not be applicable to critically ill patients. Delirium is associated with increased morbidity. Awareness of patients at risk may lead to better recognition and earlier intervention.

Keywords Delirium · Agitation · Risk factors · Critical care · Intensive care unit · Length of stay · Morbidity

Introduction

Agitated behaviour is a common and worrisome problem in the intensive care unit (ICU). Its differential diagnosis is extensive. One of these diagnoses, delirium, is of concern to the intensivist because of its associated mortality and morbidity in other populations [1, 2, 3]. This reversible organic mental disorder is thought to be a reflection of underlying medical illness and not a cause-and-effect of being in the ICU [4].

Data on the incidence of delirium in the intensive care setting are rare and, when reported, a wide range is cited (13–70%) [5]. Existing descriptions have two major limitations. There is methodological discrepancy between studies, as pointed out in an elegant review of post-cardiotomy delirium studies [5]. Different types of study design were used; some lack control groups; explanations on how studied factors were selected are missing. The second major drawback is the absence of diagnostic criteria for delirium. Agitation and "ICU psychosis" have no operational definition and psychiatric DSM criteria are usually not applied [6]. Sessler, in an abstract, reported medical ICU patients who were mildly, moderately and severely agitated, but not nec-

essarily delirious, in 32%, 20% and 5%, respectively [7].

Prospective trials on risk factors for developing delirium in the medical or surgical ICU setting have not been published. The risk factors provided in most standard critical care textbooks for the development of delirium are based on studies describing patients outside the ICU [4, 8]. Our study's goal was to identify risk factors associated with the development of delirium in a critical care setting and, secondarily, to evaluate the occurrence of complications and length of stay in patients who developed delirium.

Methods

Pilot study

A pilot screening evaluation of 99 patients was carried out before we conducted the study in order to identify potential risk factors relevant to the ICU population. Medical information (diagnosis on admission, past medical history, habits) and 11 laboratory variables classically associated with the development of delirium were collected [9]. We identified delirious and non-delirious patients in this preliminary screening and compared variables between the two groups. Univariate analysis allowed us to retain laboratory variables of statistical significance ($p < 0.05$) and clinical relevance in an ICU population: sodium, glucose, creatinine, urea, bilirubin, albumin and calcium, PCO_2 (data not shown). This patient population was not considered in the prospective study described below, as not all patients were consecutively admitted, not all delirium patients were seen by a psychiatrist and not all data were systematically collected to describe complications of delirium (such as removal of central lines).

Risk factor study

From November 1998 to April 1999 we conducted a prospective study in a 16-bed medical and surgical ICU. All consecutive patients aged 18 or more, admitted for more than 24 h, for whom direct or surrogate consent was obtained and who were deemed likely to survive 24 h by the staff physician, were included.

For each patient the following data were collected:

- Demographic and admission data (predisposing factors): age, sex, admission diagnosis, APACHE II (Acute Physiology And Chronic Health Evaluation) [10] and Glasgow scores, medical history (diabetes, hypertension, coronary disease, chronic obstructive pulmonary disease (COPD, defined by PFT or clinical criteria) [11], renal failure, central nervous system disease, stroke and dementia); psychiatric history; visual and auditory impairment; current active smoking history; alcohol [12] and drug abuse.
- Daily recorded data during the first 5 days in the ICU (precipitating factors): bed location in the ICU (the ICU has two types of room: open with windows or closed without windows); family visits lasting 2 h out of 24 in total or more; endotracheal intubation; total number of catheters and tubes; fever; infections; laboratory values (sodium, glucose, creatinine, urea, bilirubin, calcium, albumin, PCO_2); drug use : analgesics (morphine, fentanyl or other), sedatives (lorazepam or other benzodiazepine, propofol), antipsychotic medication (haloperidol or other), corticosteroids; and the use of epidural analgesic administration. The use of restraints was not documented as it is difficult to document reliably, varies widely within our ICU between individual nurses and is influenced by work load and staffing issues.
- Complications during the first 5 days in the ICU (self-extubation, removal of catheters and death) and length of stay.

The diagnosis of delirium was made by the intensivist and confirmed by a formal psychiatric assessment. In addition, in order to enhance the identification of delirious patients, each patient was screened for the presence of delirium using the 8-item Intensive Care Delirium Screening Checklist (Appendix I). For each abnormal item, a score of one was given. The study describing the screening tool reports a sensitivity of 99% when a total score cut-off of 4 points was used [13]. A nurse completed the checklist at the end of every 8-h shift for a maximum of 5 days on each patient admitted. Any patient scoring 4 points or more on the scale underwent a psychiatric assessment, whether or not the attending intensivist made a diagnosis of delirium. The hospital ethics and research committees approved the study protocol.

Statistics

Four subsets of factors were submitted to the analysis : (1) sociodemographic and medical history; (2) laboratory data; (3) drugs received; (4) environmental factors. For laboratory data, we used the proportion of days with abnormal values (for each measured laboratory variable) from admission to the day delirium occurred (labelled as delirium phase).

Analgesic and sedative medications were evaluated by product group, and also respectively transformed as parenteral morphine and lorazepam using equivalent dosage scales [14]. For delirious patients, the mean daily dose of equivalent dosage drug was calculated during the delirium phase. For non-delirious the mean dose was calculated using all 5 days.

Univariate analyses (likelihood ratio chi-square, Mann-Whitney U test or *t*-test) were first used for preliminary selection of model variables, based on a 0.15 alpha level. Stepwise block logistic regression was then applied to model the risk of delirium using significant univariate predictors. Odds ratios and their 95% confidence interval were used to assess independent contribution of significant factors.

Results

Two hundred sixteen patients were recruited for the study. Nine patients were admitted with delirium and excluded for the determination of risk factors in the univariate analysis. Due to missing data, multivariate analysis was performed on data from 198 patients (160 non-delirious and 38 delirious) and length of stay documented for 209 patients. Complications and mortality were assessed on the total sample (216 patients). During the time period described 353 patients were admitted to the ICU. The remaining 137 patients were admitted for brief surveillance (62%), moribund (7%), missed from screening (11%), under 18 (2%) or did not give consent for data to be gathered.

Delirium developed in the first 36 h following admission in 78% of our delirious patients and within 72 h in

Table 1 General characteristics of patients

	Delirious patients ($n = 38$)	Non-delirious patients ($n = 160$)
Age, mean (SD)	63.2 (13.9)	66.3 (13.4)
Sex (M/F %)	55.3	44.7
APACHE II, mean (SD)	15.2 (7.9)	15.2 (6.9)
Medical patients	17 (16%)	89 (84%)
Surgical patients	21 (22.8%)	71 (77.2%)

Diagnoses: COPD/respiratory failure 31, asthma/respiratory failure 2, pneumonia 14, sepsis/septic shock 18, necrotising fasciitis 1, CHF 9, arrhythmia 3, post-CPR 3, CVA or intracranial haemorrhage 14, brain abscess or tumour 5, seizures 4, subarachnoid haemorrhage 4, Guillain–Barré 2, myasthenia gravis 1, meningitis 1, hypothermia 1, coma NYD 3, trauma 1, hip fracture/postoperative complications 5, GI bleed 4, liver failure 1, placental detachment 1, acute renal failure 5, overdose 3, renal cancer 2, liver cancer 2, laryngeal implant 2, facial or mandibular reconstruction 2, neck haematoma 1, epiglottitis 1, intestinal resection/obstruction 12, peritonitis 5, cancer gastrectomy 2, eventration 1, aortic aneurysm repair 16, other vascular procedures 12, thoracic surgery or gastric pull-up 9

93.7% of them. Of the cases, 98.6% developed within 5 days; all cases of delirium developed within 13 days.

Table 1 shows patient characteristics. Age, sex, severity of disease (APACHE II score), type of admission (medical or surgical) and whether or not general anaesthesia had been administered were not different between delirious and non-delirious patients. Type of surgery (neurosurgical, vascular or general; and emergency or elective) did not show a difference in the risk for developing delirium (data not shown). Agitation, a common feature of delirium, was not always present; one-third of our patients never manifested agitation but were considered delirious after psychiatric assessment.

Univariate analyses

Univariate analyses (Table 2) allowed us to identify 12 factors associated with delirium. A history of hypertension increased the risk of delirium two-fold; none of our patients presented with uncontrolled hypertension on admission or during the study. COPD, alcohol abuse [12] or smoking (minimum 20 cigarettes a day until admission, independent of COPD) roughly doubled the risk of delirium. Rooms with windows protected against delirium occurrence, while windowless ones doubled the risk. Endotracheal intubation and total number of catheters did not increase risk for delirium. For each day where sodium, glucose and bilirubin values were in the abnormal range, the risk of developing delirium increased. Individual benzodiazepines did not increase the risk for delirium; when grouped in dose-equivalents, only the highest benzodiazepine dosage was linked to delirium. Individual opiates were not associated with a risk for delirium when compared with each other. Morphine-equivalent dosage, however, was strongly associated with delirium, even with lower daily dosages (expressed as three homogeneous categories compared to a "non-user" category). The use of epidural analgesia had a similar effect. The use of corticosteroids was not linked to delirium.

Table 2 Univariate analysis

Variable	Odds ratio	95% Confidence interval	*p* value
Hypertension	2.1	1.03–4.15	0.04
COPD	1.7	0.82–3.41	0.15
Alcohol abuse	2.8	0.94–8.14	0.08
Smoking history	2.2	1.07–4.51	0.03
Sodium level[a] (% of days with abnormal level)	1.1	0.99–1.23	0.07
Glucose level[a] (% of days with abnormal level)	1.1	0.95–1.35	0.18
Bilirubin level[a] (% of days with abnormal level)	1.1	1.01–1.33	0.04
Epidural use	2.2	0.90–5.25	0.09
Morphine (mean daily dose)[b]			
0.01–7.1 mg	4.7	1.22–18.3	0.02
7.2–18.6 mg	8.1	2.16–30.5	0.002
18.6 mg-331.6 mg	5.4	1.4–20.5	0.01
Lorazepam (mean daily dose)[b]			
0.01–0.68 mg	1.6	0.58–4.19	0.38
0.69–1.82 mg	1.1	0.36–3.45	0.85
1.83–14.75 mg	3.3	1.31–8.04	0.01
Rooms without windows	2.2	0.88–5.55	0.11
Rooms with windows	0.5	0.25–1.11	0.10

[a] Odds ratio expresses a risk increase for each 10% increased of days with abnormal laboratory value

[b] Analgesic and sedative medications were respectively transformed as parenteral morphine and lorazepam using equivalent dosage scales [12]

Table 3 Multivariate analysis

Variable	Odds ratio	95% Confidence interval
Hypertension	2.6	1.14–5.72
Smoking history	2.2	0.94–4.94
Bilirubin level[a] (% of days with abnormal level)	1.2	1.03–1.40
Use of epidural	3.5	1.20–10.39
Morphine (mean daily dose)[b]		
0.01–7.1 mg	7.8	1.76–34.4
7.2–18.6 mg	9.2	2.17–39.0
18.7–331.6 mg	6.0	1.41–25.4

[a] Odds ratio expresses a risk increase for each 10% increased of days with abnormal laboratory value

[b] Analgesic and sedative medications were respectively transformed as parenteral morphine and lorazepam using equivalent dosage scales [14]

Table 4 Morbidity and mortality associated with the development of delirium (numbers in parenthesis represent percentage)

Event[a]	Delirious patients	Non-delirious patients	*p* value
Self-extubation	4 (10)	4 (2.3)	0.02
Removal of catheters	8 (20)	10 (5.7)	0.003
Mortality	6 (15)	24 (13.6)	0.82
Length of stay (days)	9.3 ± 12	7 ± 7.9	0.14

[a] Self-extubation, removal of catheters and mortality were calculated on 216 patients; length of stay was calculated on 209 patients

Multivariate analysis

Table 3 illustrates the five factors that were retained in the optimised multivariate model (model chi-square = 31.4, $p < 0.00001$). Hypertension history increased by 2.6 (CI 1.1–5.7) the risk of developing delirium; smoking increased the odds by a factor of 2.2 (CI 0.94–4.90); COPD was not associated with smoking in this analysis. Bilirubin abnormality, the only significant laboratory variable, was modestly associated with delirium (OR 1.2, CI 1.0–1.4). The epidural route of analgesia was also associated with delirium occurrence (OR 3.5, CI 1.2–10.4). Morphine, in all dosages used, was linked to the development of delirium (OR between 6 and 9.2); the highest OR was associated with an intermediate dosage of opiates.

Consequences of delirium

Table 4 shows the complications recorded for delirious and non-delirious patients. Removal of catheters (20%) and self-extubation (10%) were much more frequent in the delirious population. Mortality was not different between the two groups (15% for delirious patients and 13.8% for non-delirious patients, $p = 0.84$). Length of stay, although not statistically different, showed a trend toward longer stay for delirious patients (9 days versus 7 days, $p = 0.14$).

Discussion

The ICU is a unique hospital setting, not only because of its physical environment but also because the patients are unstable and submitted to important physical and psychological stresses, which may have an important impact on the development of delirium. We systematically recorded elements of potential interest in the development of delirium in the eligible patients admitted.

The possible risk factors data we collected can be categorised into four broad sections : medical history-related risk factors, laboratory abnormalities, drugs received and factors which may influence the psychological and emotional well-being of the patient, such as bed location and availability of family.

Medical history risk factors

History of hypertension was strongly linked to the development of delirium in our population. A published review of the neuropsychological effects of chronic hypertension describes studies where hypertensive patients perform poorly compared to normotensives, especially for memory, attention and abstract reasoning [15]. Other studies link hypertension with memory impairment [16] and diminished psychomotor speed [17]. Such premorbid abnormalities may be the expression of fragility. Hypertensive patients, when subjected to important physical or psychological stresses present in the ICU, may thus be more vulnerable to develop delirium.

Vascular disease, a feature of hypertension, puts patients at higher risk for cerebral hypoperfusion and possibly for cerebral cellular hypoxia. Hypoxia, in a broader sense, is associated with the development of delirium [18]. Our clinical experience is that patient blood pressure in the ICU is often low compared to their usual value due to the use of sedatives and shock states. Such low blood pressure could impair cerebral perfusion in hypertensive patients. These underlying facts could explain the statistical association we found.

Smoking history was also linked to delirium. It is not known to be related to the development of delirium except in two case reports of dying cancer patients, which suggest that nicotine withdrawal was associated with delirium [19]. Cigarette smoking effects are believed to be the result of nicotine inhalation. Nicotine induces neuroadaptive changes in the brain of the chronic smoker. The hallmark of neuro-adaptation to nicotine is thought to be upregulation of true nicotinic acetylcholine receptors, possibly due to nicotine-induced desensitisation [20].

The effects of nicotine in the central nervous system appear to be complex; among others, nicotine increases dopaminergic and acetylcholinergic transmission. Abrupt cessation of smoking may create an imbalance in neurotransmission particularly related to acetylcholine and dopamine, both of which have been hypothetically linked to neurotransmitter hypotheses of delirium [21]. There are thus possible common pathological neurochemical pathways shared by delirium and nicotine withdrawal. In chronic smokers, abrupt nicotine withdrawal due to hospitalisation could be viewed as an additional stressful biochemical event that could favour the emergence of delirium. Neither COPD nor the use of corticosteroid drugs accounted for the smoking/delirium association; despite the fact that the time of year the study was conducted provided many smokers who were COPD patients.

Alcohol abuse, a classic factor associated with delirium [18], was linked to delirium only in the univariate analysis. Several historical factors were not linked to the development of delirium, contrasting with published risk factors in other populations [9]. Age was not associated with the development of delirium. Many critical care textbooks list older age as a risk factor for the development of delirium [4, 8] based on studies performed outside the ICU setting. Types of surgery (emergency or elective) did not influence this risk. Our centre is not a tertiary trauma centre, and cardiac surgery is referred to a nearby specialised institution. Previous studies suggested that the cardiac surgery population is at risk to develop delirium [5]; however these data should be assessed with more recent perspectives on post cardiac surgery cognitive dysfunction in mind [22]. Clinical evidence of infection, renal failure and history of dementia were not associated to delirium. The severity of illness (APACHE II) was also not a risk factor.

Laboratory abnormalities

Bilirubin abnormalities (above the normal range) were detected as significant in our analysis. The relationship between hepatic dysfunction with cirrhosis and delirium is known [23], but isolated elevation of bilirubin is not a likely explanation for it in adults. Neuropsychiatric manifestations with an isolated elevation of bilirubin are described exclusively in newborn babies [24].

Sodium abnormality (above or below the normal range) was associated with the development of delirium in univariate analysis. Abnormal sodium has been associated with encephalopathy, especially in the postoperative period, with abnormal sodium levels due to pharmacological agents and heart failure [25].

Finally, glucose abnormalities (above or below the normal range) were linked to delirium in the univariate analysis. This finding is supported by a thorough review on the psychiatric symptoms found in patients with abnormal glucose, mainly in hypoglycaemic patients [26]. However, our diabetic patients were usually hyperglycaemic.

None of the other laboratory values we recorded (BUN, creatinine, calcium, albumin, PCO_2) were associated with the development of delirium.

Drugs received in the intensive care unit

Common sense dictates that proper analgesia and sedation should put the patient at less risk of developing delirium [27]. Patients at our institution are carefully evaluated for sedation or analgesia needs using Ramsay and visual analogue scales.

The use of opiates as analgesics was strongly related to the development of delirium regardless of the dosage. Morphine as well as fentanyl, which are widely used in our unit, have anticholinergic properties that may favour the development of delirium [28]. The different routes of administration were not evaluated because we used equivalent dosage scales. Epidural catheter use for drug administration was also linked to delirium. A case-control study on postoperative delirium showed that meperidine was associated with delirium, but only when administered by the epidural route [29]. In our unit meperidine, but also fentanyl, are used as epidural agents.

Benzodiazepines and propofol are used frequently as sedative agents in the ICU and they were not associated with the development of delirium in our multivariate analysis. High dosage of lorazepam increased the risk significantly in the univariate analysis. Delirious patients may receive benzodiazepines as adjuvant in the treatment of delirium [18] or as medication to control delirium prodromal symptoms, resulting in an association. Those results are in contrast with a prospective study that showed that the uses of benzodiazepines, regardless of dosage, were considered a risk factor for the development of delirium [30]. The population described in this study, elective postoperative patients, is clearly different from ours, which included elective, but also emergency, postoperative and medical patients.

Steroids proved to be neutral. It is common lore that steroids can induce neuropsychiatric manifestations but no prospective studies support this assertion, which is based on case reports. A review of the relevant literature describes the relationship between psychiatric symptoms and administration of corticosteroids as poorly understood; only 5 % of steroid-treated patients will develop severe reactions and most of them suffer from underlying affective or psychotic disorders [31].

Environmental factors

Several factors could threaten the physical, psychological and emotional integrity of patients. The ICU has

two types of rooms: open with windows and closed without windows. Windowless rooms increase the risk of developing delirium and open with windows decrease it in the univariate analysis. Environment modification may be associated with a better control of delirium in the elderly, without necessarily preventing it [32, 33]. This notion is in contrast with a recent publication in the geriatric population that showed no benefit from environmental intervention once delirium is established [34]. The common notion that environment itself exacerbates, or even precipitates, delirium is unfortunately not based on scientific grounds [18].

The presence of a family member at the bedside 2 h or more a day was not protective; we did not qualify the interaction of the family with the patients. Visual and auditory impairment were also not linked to an increased risk of developing delirium.

Consequences of delirium

Complications associated with the delirium are manifold. We found an increased rate of self-extubation and catheter removal among delirious patients. Self-extubation is associated with an increased morbidity, prolonged length of stay and mortality [32]. An APACHE score was determined for each patient within the first 32 h of admission to the ICU. Sequential daily scoring (such as the SOFA score [33]) which reflects the patient's severity of illness, was not performed in this study. It is unlikely that this is a major shortcoming, since the onset of delirium occurred within 36 h in 80 % of patients.

A trend toward an increased length of stay in the ICU was seen in delirious patients. The 2.3-day difference between the two groups did not reach statistical significance, but this trend is in agreement with studies in general medical ward patients [34]. Mortality during the ICU stay did not differ between delirious and non-delirious patients. This result is in contrast with studies linking the development of delirium to an increased mortality [2].

Study limitations

Several limitations deserve mention. The diagnosis of delirium is difficult in a critical care setting. Some patients may have been either misdiagnosed or missed in our evaluation. We believe, however, that the systematic and frequently applied use of our screening tool reduced the risk of missing delirium. The psychiatric diagnostic confirmation we used was the best available "gold standard". We screened all patients systematically only during the first 5 days; subsequent episodes of delirium were diagnosed by the treating clinician, thus making underestimation of late delirium diagnoses a possibility.

We did not qualify the interaction of the family with the patients. This, and our difficulty in evaluating the physical restraints in these patients, constitute additional limitations. Comfort from a caregiver familiar with the patient's likely fears has been described as decreasing the incidence of delirium [35]; physical restraints are said to increase agitation in elderly patients [36]; both of these elements would have been important to document. Benzodiazepines were pooled to assess their impact on the development of delirium. Lorazepam and diazepam, the two most commonly used benzodiazepines in our ICU, have different metabolic pathways. This, as well as renal and hepatic dysfunction and its effect on the metabolism of these drugs, was not considered in the analysis.

We wished to design a risk factors study and not a natural history study. We did not follow patients on the ward or record the length of hospital stay. Duration of delirium episodes was, therefore, not recorded. Such data would have been interesting. Our study did not encompass a whole year, so seasonal variability in the risk factors for the development of delirium cannot be assessed.

In conclusion, our study addresses a common and important problem facing every ICU patient. Delirium was diagnosed by the intensivist, a simple and easy-to-use screening scale was routinely applied in all patients and the diagnosis was clinically confirmed by a psychiatrist. We prospectively identified risk factors for the intensive care patients and found that delirium is associated with increased morbidity and a trend to longer ICU length of stay. Most traditional risk factors for delirium were not found to be significant in our study. Identifying risk factors is the initial step of a strategy that could lead to earlier diagnosis and intervention, which could contribute to better and more cost-effective management of our patients.

Acknowledgements We would like to thank Lise Gaucher R.N., helped by Nicole Bouchard R.N., Johanne Harvey R.N., Ginette Régnier R.N. and Huguette Ruel R.N., who collected most of the data.

Appendix I

The Intensive Care Delirium Screening Checklist

The scale is completed based on information collected from each entire 8-h shift or from the previous 24 h. Obvious manifestation of an item = 1 point. No manifestation of an item or no assessment possible = 0 point. The score of each item is entered in the corresponding empty box and is 0 or 1 (Fig. 1).

PATIENT EVALUATION	DAY 1	DAY 2	DAY 3	DAY 4	DAY 5
Altered level of consciousness* (A-E)					
If A or B do not complete patient evaluation for the period					
Inattention					
Disorientation					
Hallucination - delusion – psychosis					
Psychomotor agitation or retardation					
Inappropriate speech or mood					
Sleep/wake cycle disturbance					
Symptom fluctuation					
TOTAL SCORE (0-8)					

Level of consciousness*		**Score**
	A: no response	none
	B: response to intense and repeated stimulation (loud voice and pain)	none
	C: response to mild or moderate stimulation	1
	D: normal wakefulness	0
	E: exaggerated response to normal stimulation	1

SCORING SYSTEM :
The scale is completed based on information collected from each entire 8-hour shift or from the previous 24 hours. Obvious manifestation of an item = 1 point. No manifestation of an item or no assessment possible = 0 point. The score of each item is entered in the corresponding empty box and is 0 or 1.

1. Altered level of consciousness:
A) No response or B) the need for vigorous stimulation in order to obtain any response signified a severe alteration in the level of consciousness precluding evaluation. If there is coma (A) or stupor (B) most of the time period then a dash (-) is entered and there is no further evaluation during that period.
C) Drowsiness or requirement of a mild to moderate stimulation for a response implies an altered level of consciousness and scores 1 point.
D) Wakefulness or sleeping state that could easily be aroused is considered normal and scores no point.
E) Hypervigilance is rated as an abnormal level of consciousness and scores 1 point.

2. Inattention: Difficulty in following a conversation or instructions. Easily distracted by external stimuli. Difficulty in shifting focuses. Any of these scores 1 point.

3. Disorientation: Any obvious mistake in time, place or person scores 1 point.

4. Hallucination, delusion or psychosis: The unequivocal clinical manifestation of hallucination or of behaviour probably due to hallucination (e.g. trying to catch a non-existent object) or delusion. Gross impairment in reality testing. Any of these scores 1 point.

5. Psychomotor agitation or retardation: Hyperactivity requiring the use of additional sedative drugs or restraints in order to control potential dangerousness (e.g. pulling out iv lines, hitting staff). Hypoactivity or clinically noticeable psychomotor slowing. Any of these scores 1 point.

6. Inappropriate speech or mood: Inappropriate, disorganised or incoherent speech. Inappropriate display of emotion related to events or situation. Any of these scores 1 point.

7. Sleep/wake cycle disturbance: Sleeping less than 4 hours or waking frequently at night (do not consider wakefulness initiated by medical staff or loud environment). Sleeping during most of the day. Any of these scores 1 point.

8. Symptom fluctuation: Fluctuation of the manifestation of any item or symptom over 24 hours (e.g. from one shift to another) scores 1 point.

Fig. 1 The Intensive Care Delirium Screening Checklist

References

1. Rabins SV, Folstein MF (1982) Delirium and dementia: diagnostic criteria and fatality rates. Br J Psychiatry 140: 149–153
2. Weddington WW (1982) The mortality of delirium: an under-appreciated problem? Psychosomatics 23: 1232–1235
3. Cole MG, Primeau FJ (1993) Prognosis of delirium in elderly hospital patients. Can Med Assoc J 149: 41–46
4. Heckers S, Tesar GE, Stern T (1999) Diagnosis and treatment of agitation and delirium in the intensive care unit patient. In: Irwin RS, Cerra FB, Rippe JM (eds) Intensive care medicine, 4th edn. Lippincott-Raven, Philadelphia, pp 2393–2401
5. Dubin WR, Field HL, Gastfriend DR (1979) Postcardiotomy delirium: a critical review. J Thorac Cardiovasc Surg 77: 586–591
6. American Psychiatric Association (1994) Diagnostic and statistical manual of mental disorders, 4th edn (DSM-IV). APA, Washington, DC
7. Sessler CN, Rutherford L, Best A, Hart R, Levenson J (1992) Agitation in a medical intensive care unit: prospective analysis of incidence and risk factors. Chest 102: 191 S
8. Wise MG, Cassem NH (1997) Behavioral disturbances in the intensive care unit. In : Civetta JM, Taylor RW, Kirby RR (eds) Critical care, 3rd edn. Lippincott-Raven, Philadelphia, pp 2017–2029
9. Trzepacz PT (1996) Delirium. Advances in diagnosis, pathophysiology and treatment. Psychiatr Clin North Am 19: 429–448
10. Knaus WA, Wagner DP, Draper EA, Zimmerman JE (1985) APACHE II: a severity of disease classification system. Crit Care Med 13: 818–829
11. American Thoracic Society Guidelines (1995) Am J Respir Crit Care Med 152:S77
12. O'Connor PG, Schottenfeld RS (1998) Patients with alcohol problems, N Engl J Med 338 (9):592–602
13. Dubois MJ, Bergeron N, Dial S, Skrobik Y (1999) ICU delirium screening checklist: a new tool. Intensive Care Med 25 [Suppl 1]: S160; Intensive Care Med online April 2001
14. Cammarano WB, Drasner K, Katz JA (1998) Pain control, sedation and use of muscle relaxants. In: Hall JB, Schmidt GA, Wood LDH (eds) Principles of critical care 2nd edn. McGraw-Hill, New York, pp 87–109
15. Waldstein SR, Manuck SB, Ryan CM, Muldoon MF (1991) Neuropsychological correlates of hypertension: review and methodologic considerations. Psychol Bull 110: 451–468
16. Madden DJ, Blumenthal JA (1989) Slowing of memory-search performance in men with mild hypertension. Health Psychol 8: 131–142
17. King HE, Miller RE (1990) Hypertension: cognitive and behavioural considerations. Neuropsychol Rev 1: 31–73
18. American Psychiatric Association (1999) Practice guideline for the treatment of patients with delirium. Am J Psychiatry 156: 1–20
19. Gallagher R (1998) Nicotine withdrawal as an etiologic factor in delirium. J Pain Symptom Manage 16: 76–77
20. Ochoa ELM (1994) Nicotine-related brain disorders: the neurobiological basis of nicotine dependence. Cell Mol Neurobiol 14: 195–225
21. Trzepacz PT (1994) The neuropathogenesis of delirium. Psychosomatics 35: 374–391
22. Newman MF, Kirchner JL, Phillips-Bute B, Gaver V, Grocott H, Jones RH, Mark DB, Reves JG, Blumenthal JA for the Neurological Outcome Research Group and the Cardiothoracic Anesthesiology Research Endeavors Investigators (2001) Longitudinal assessment of neurocognitive function after coronary-artery bypass surgery. N Engl J Med 344: 403–409
23. Carroll RE, Hayes PC, Ebmeier KP (1991) Regional blood flow and cognitive function in patients with chronic liver disease. Lancet 1: 1250–1253
24. Gourley GR (1997) Bilirubin metabolism and kernicterus. Adv Pediatr 44: 173–229
25. Fraser CL, Arieff AI (1997) Epidemiology, pathophysiology and management of hyponatremic encephalopathy. Am J Med 102: 67–77
26. Fishbain DA, Rotundo D (1988) Frequency of hypoglycemic delirium in a psychiatric emergency service. Psychosomatics 29: 346–348
27. Shapiro BA, Warren J, Egol AB, Greenbaum DM, Jacobi J, Nasraway SA, Schein RM, Spevetz A, Stone JR (1995) Practice parameters for intravenous analgesia and sedation for adult patients in the intensive care unit: an executive summary. Society of Critical Care Medicine. Crit Care Med 23: 1596–1600
28. Marcantonio ER, Goldman L, Mangione CM, Ludwig LE, Mucara B, Haslauer CM, Donalson MC, Whittemore AD, Sugarbaker DJ, Poss R, Haas S, Cook EF, Orav EJ, Lee TH (1994) A clinical prediction rule for delirium after noncardiac surgery. JAMA 271: 134–139
29. Herrick IA, Ganapathy S, Komar W, Kirkby J, Moote CA, Dobkowski W, Eliasziw M (1996) Postoperative cognitive impairment in the elderly. Choice of patient-controlled analgesia opioid. Anaesthesia 51: 356–360
30. Marcantonio ER, Juarez G, Goldman L, Mangione CM, Ludwig LE, Lind L, Katz N, Cook EF, Orav EJ Lee TH (1994) The relationship of postoperative delirium with psychoactive medications. JAMA 272: 1518–1522
31. Lewis DA, Smith RE (1983) Steroid-induced psychiatric syndromes. A report of 14 cases and a review of the literature. J Affect Disord 5: 319–332
32. Atkins PM, Mion LC, Mendelson W, Palmer RM, Slomka J, Franko T (1998) Characteristics and outcomes of patients who self-extubate from ventilatory support: a case-control study. Chest 113: 1429–1430
33. Vincent JL, De Mendonca A, Cantraine F, et al. (1998) Use of SOFA score to assess the incidence of organ dysfunction score/failure in intensive care units: results of a multicenter, prospective study. Crit Care Med 26: 1793–1800
34. Stevens LE, De Moore GM, Simpson JM (1998) Delirium in hospital: does it increase length of stay? Aust N Z J Psychiatry 32: 805–808
35. Lazarus HR, Hagens JH (1968) Prevention of psychosis following open-heart surgery. Am J Psychiatry 124: 1190–1195
36. Cole MG, Primeau FJ, Bailey RF, Bonnycastle MJ, Masciarelli F, Engelsmann F, Pepin MJ, Ducic D (1994) Systematic intervention for elderly in-patients with delirium: a randomised trial. Can Med Assoc J 151: 965–970