



C. Miller Fisher and the Comatose Patient

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Abstract

Neurologic examination of the comatose patient has gradually matured. Less than 50 years ago, neurological examination in coma became a regular part of textbooks with separate chapters devoted to the topic but many were deficient in detail. In 1969, C.M. Fisher published an extraordinary 56-page paper on the examination of the comatose patient. The paper—one of Fisher’s gems—is not well known and infrequently cited. The many new observations collected in this comprehensive paper are reviewed in this vignette, which highlights not only how these contributions shaped our thinking on coma but also questioned shaky concepts.

Keywords Coma · Bilateral or unilateral decerebrate posturing · Cheyne–Stoke respiration · Brain stem lesions

Introduction

Attempting to categorize coma in the first half of twentieth century, neurologists and neurosurgeons faced a textbook case of nebulosity in terminology. Describing an unresponsive patient ranged from drowsy to deep coma—with semi-coma thrown in somewhere. Lack of a comprehensive clinical neurological assessment of a comatose patient was a notable failing of medical texts of clinical neurology. *DeJong’s The neurological examination (1950)* was possibly the first English-language text that devoted a specific chapter to several degrees of coma and included details on cranial nerve examination that emphasized the value of eye signs in localization and a comprehensive section on differential diagnosis. Specific localizations were suggested, but DeJong concluded painfully that “there is no incontrovertible evidence, however, that one site is essential and consciousness is probably a function of the entire organism [1].”

The understanding of the mechanism of coma does not require full understanding of consciousness, but discovery of the ascending reticular formation and experiments studying the effects of neurosurgical decompression all

eventually and slowly merged with clinical practice. Subsequently, separate chapters emphasizing the neurological examination in coma became a regular part of textbooks in [2].

In 1969, C.M. Fisher published an extraordinarily detailed 56-page paper. Fisher wrote, “From the standpoint of neurophysiology, it is not exaggeration to say that the examination of the human comatose patient has been almost totally neglected,” followed by “nowhere in the literature could we find a comprehensive, detailed discussion of the neurologic findings in coma, although papers dealing with individual aspects of the problem were not rare.”

In addition to his acts of bravery in World War II and his major cerebrovascular contributions (e.g., lacunar syndromes, transient ischemic attacks, symptomatic carotid stenosis), the neurologist C. Miller Fisher (Fig. 1) is also known for a number of insightful articles of interest to neurointensivists [3–6], his several eponyms [7], and—for those who knew him closely—the so-called Fisher rules [8].

Here I revisit his manuscript on coma—now classic but less well known outside certain circles (Fig. 2). Generally, Fisher was not unduly interested in publishing in high-impact journals (“if it is interesting, it will get noticed,” he used to say), and the opportunity afforded by this arguably lower-profile journal to extensively record a catalog of observations may have suited him well. Fisher’s contributions, therefore, included the first full clinical description of

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Fig. 1 C. Miller Fisher (Massachusetts General Hospital, Archives and Special Collections, used with permission)

the comatose patient, the first classification based on CSF results, the first serious questioning of the clinicopathological correlation with brain herniation, and the first major emphasis on relevant eye findings in coma. Previously, only pupils were considered relevant, and unfortunately some physicians still limit their practice this way.

The Examination of Coma

The number of observations collected in this comprehensive paper is staggering. Fisher emphasized the general examination of skin color and texture, fingernails, mucous membranes, respiration patterns, autonomic stability, vital sign measurements, and general posture of the patients could be important pieces of the overall clinical picture. “A comfortable-looking patient curled up with legs crossed will prove to be in light coma at the worst.” He said, “The odor of breath may bespeak alcohol, acetone, cholemia (feter hepatis) or uremia.” He also noted that ingestion of vodka would not cause an alcoholic odor. Fisher particularly highlighted the correlation of fever with acute

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The Neurological Examination of the Comatose Patient

C. M. FISHER

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Fig. 2 Cover page of the paper, © John Wiley & Sons A/S. Published by John Wiley & Sons Ltd (used with permission)

brainstem disease, particularly with the presence of shivering and the absence of sweat. On the other hand, he noted that hyperthermia could be due to an acute disorder of temperature regulation due to a brain lesion when sweating was present without shivering. Blood pressure was low in systemic illnesses and not due to neurologic involvement, and elevated blood pressure could reflect a hypertensive disease but also a response to massive subarachnoid hemorrhage from a ruptured aneurysm, but he saw it rarely in brainstem infarction.

Fisher described ocular bobbing, which he correlated with pontine pathology. He reported the 1½ syndrome, “wrong-way eyes,” pontine miosis, doll’s eyes, eye closure and blinking in coma, and reflex blepharospasm. He questioned as unproven the dictum that dilatation of one pupil means herniation. “An isolated third-nerve deficit in the uncus syndrome certainly suggests a peripheral insult. Yet what is the mechanism in the early stages before herniation occurs?” He also noted “cerebellar coning has probably been overemphasized as a mechanism of disastrous events.”

Fisher pointed out that the eyelid tone and length of time the eyes remain open after being opened by the examiner are both findings that indicate the depth of the coma. His emphasis of eye and motor responses, the use of pain and voice as stimulus were all forerunners of the Glasgow Coma Scale (Fig. 3). He described bilateral decerebrate posturing resulting from acute lesions involving the supratentorial motor system; previously in humans, it had only been described with brain stem lesions. He also newly attributed unilateral decerebrate posturing to an acute hemispheric lesion.

Fisher’s general observations also included a classification of the comatose state by cerebrospinal results and symmetry (Table 1). Although such a classification has been superseded by the CT scanner, it is a valid attempt to focus the possible causes of coma by combining laboratory tests and neurologic findings.

He also suggested that rapid-onset coma without prompt death indicated a basilar artery thrombus, massive trauma and anoxia or barbiturate intoxication combined with alcohol or cerebral ischemia after a long “Stokes-Adams attack.” Seizures or hypoglycemia also should be considered. Of particular interest is his description of short-cycle Cheyne–Stoke respiration in intracranial bleeding, indicating a rapid, progressive, and downhill course. Periodic breathing could include very short breathing and apneic phases—3 or 4 rapid, deep breaths—while the waxing-and-waning phase might consist of only 1 or 2 breaths. He noted that fish-mouthing, “where the lower jaw is depressed with each inspiration representing one of the least organized forms of breathing and is an ominous sign.”

Fisher noted that bilateral extensor posturing resulting from acute lesions involving the supratentorial motor system in humans was only described with brainstem lesions. With extensive posturing, he noted the thumb could lie in a

Table 1 Fisher’s classification of causes of comatose-drowsy state

Normal cellular content in cerebrospinal fluid
Symmetrical neurologic signs
Asymmetrical neurologic signs
Gross or microscopic blood in cerebrospinal fluid
Symmetrical neurologic findings
Asymmetrical neurologic signs
Leukocytosis in cerebrospinal fluid
Symmetrical neurologic signs
Asymmetrical neurologic signs

palm clasp by the fingers, which was known previously as “Sugling faust” (baby’s fist).

A Fallout?

It should not be surprising that neurointensivists found Fisher’s observations invaluable to their practice. There are no specific methods to the paper or any indication as to exactly how many patients with each finding he described; yet many of his observations have stood the test of time.

In a memoir published decades later, Fisher indicated that after he had discussed some principles of this paper with another neurologist at an academic meeting, this person published a paper on the topic without acknowledging information from Fisher’s previously relayed experience. Without providing further details about this apparently disturbing experience, he “decided to let the matter rest. [9]” (Details about this academic accident have not been forthcoming.) C.M. Fisher published a classic, extraordinarily detailed paper, and such a purely clinical description in this age of advancing technologies (which

Fig. 3 Critical role of eye examination in determining degree of coma, © John Wiley & Sons A/S. Published by John Wiley & Sons Ltd (used with permission)

Table 1. Drowsy—Comatose States.
(Eyes persistently closed and often unblinking).

1. Drowsiness	<ul style="list-style-type: none"> – command elicits motor response – question elicits speech – pain elicits speech or groan – voice elicits opening of eyes or stirring – threat with own arm elicits blink or greater response 	}	light coma
2. Stupor	<ul style="list-style-type: none"> – pain elicits voluntary response – pain in arm elicits grimacing – pain elicits opening of eyes and blinking 	}	moderate coma
3. Coma	<ul style="list-style-type: none"> – pain elicits elementary reflex responses – pain elicits no reaction 	}	deep coma

some may consider detrimental to examination and observation skills) is a rarity today.

In 1962, McNealy and Plum [10] published their observations of clinically deteriorating patients and outlined two brain herniation syndromes. One year later, an anesthesiologist, Eckenhoff, published one of the first papers that focused on the clinical care (rather than the diagnosis) of the comatose patient [11]. Eckenhoff's paper is not been widely known and may have been overshadowed by Plum and Posner's [12] monograph on the diagnosis of stupor and coma in 1966. Their monograph organized the approach of comatose patients in an unprecedented way, but it is no heresy to note that the clinical observations that Fisher provided in this landmark paper provided a solid foundation for generations of future neurologists when approaching, describing, and examining, a comatose patient.

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