

## PART 1

# Central nervous system disorders

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## Section A: Altered consciousness: confusion, delirium, and unresponsiveness; agitation, hallucination, and abnormal behavior

These are some of the “altered states” that prompt neurology consults. Patient problems rather than specific, prepackaged “diagnoses” generate consults. Hence, clinical training rather than standard texts is the major source of learning the physician’s approach to managing *problem-oriented* questions.

Unfortunately, the causes (or diagnoses) underlying a particular complaint are legion—consider the potential causes of “dizziness,” for example, low blood pressure or neurilemmoma, migraine or brainstem stroke, low blood sugar or otolith disease, and multiple sclerosis or Meniere’s disease.

Clearly the constellation of symptoms and signs (and those absent) from the patient’s description of clinical features (the syndrome) will pare down the possibilities and direct the diagnostic evaluation and investigation. Excellent texts are available that can address “lists” of probable alternatives to particular complaints. Maybe the future will lie in the use of a palm-held computer into which the complaint/symptom will be logged, followed by associated (or not) clinical features, resulting in the generation of a “probability list,” which can be used even while one is rounding on patients.

In this section, we address certain states of altered consciousness or behavior that fall short of coma. Locked-in states, minimally conscious states, akinetic mutism, and vegetative states are a different order of “unresponsiveness,” and are found in their own section further on. Those examples contained here involve acute or subacute global diminution in the level of consciousness, vigilance, memory, and cognitive processing in keeping with encephalopathies (“altered mental status”) or “acute con-

fusional states” due to toxic/metabolic, infectious, or ictal disturbances.

Some definitions in current use are as follows:

*Delirium*: An acute alteration in cognitive function with impaired short-term memory, sleep cycle inversion, sometimes with increased motor activity in the form of agitation and tremulousness (think withdrawal or delirium tremens), often with amnesia.

*Confusion*: A general term that usually needs further definition. Often, however, it is used to refer to a state of impaired language output, orientation, the ability to follow commands and to retain information.

*Altered mental status*: This could subsume the above. Also a non-specific term, which could apply to psychosis, coma, or dementia. It also needs further specification.

*Encephalopathy*: A Greek-derived term for diffuse brain dysfunction—also non-specific. But then globally confused patients are often perforce “nonspecifically” cognitively impaired (a clue in itself).

Or there may be a clinical question at the outset: Is this nonconvulsive status epilepticus (NCSE)? This is specific and provable one way or the other. One might consider the variety of clinical features seen with NCSE and obtain an EEG.

So where to go? Once the probable type of higher cortical disturbance has been tested, for example, with a mini-mental status examination, more detailed testing of the patient’s orientation, language, memory, ability to follow commands, to interpret events (the “cookie thief” picture), and then a probability list of diagnoses

## 2 Section A: Altered Consciousness

can be produced. This might include a consult with the following:

***Possible toxic/metabolic encephalopathy. Suggest the exclusion of systemic infection in this patient with chronic diminished tolerance to the many causes of encephalopathy (e.g. cerebral atrophy; dementia). Consider also investigation of ictal/post-ictal possibilities (with an EEG).***

If in the course of investigating altered consciousness or abnormal behavior in a patient, the EEG reveals an epilep-

tiform abnormality, turn then to the section on seizures (Part 2) for further electroclinical correlations and suggestions.

The easier questions to answer are often those centered on a request for *prognosis*. In particular instances such as after anoxia, "ball-park" answers can be provided, or even some highly exact ones. For example, the prognosis in a lethargic patient 3 days after CRA can be given with much support from the literature, and from EEG and SSEPs (somatosensory evoked potentials). For these types of questions and for those patients in coma, locked-in states, and vegetative states, please refer to Part 3 on these disorders. A brief overview on prognosis and evaluation can also be found in the section on Evoked Potentials in Consultative Neurology.



## 1. Diffuse and frontal fast activity—beta

MICU, CICU, NICU, SICU, WARD, ER

**CLINICAL CORRELATES:** A patient may have been referred to the electrophysiology laboratory for one of several clinical reasons, and the EEG reveals medium to high-voltage diffuse beta frequencies. In a patient with little history, it would suggest drug intoxication and the need for a toxin screen. The patient may be normal, drowsy, or rarely agitated.

**ETIOLOGY:** Benzodiazepine, chloral hydrate, or barbiturate treatment or intoxication. Occasionally, sedative withdrawal states. With high medication doses, the patient may be sedated to the point of unarousability (beta coma, usually  $>30 \mu\text{V}$  on EEG). It can occur with brainstem injury [4].

**CLINICAL EVALUATION:** Record all medications to which the patient has access. Look for medication/sedative effects; alternately, the patient may be agitated rarely with delirium.

**ANCILLARY TESTING:** Toxin screen for barbiturates or benzodiazepines. MRI of brainstem structures.

**DIFFERENTIAL DIAGNOSIS:** For the EEG pattern, it may occur with benzodiazepines, barbiturates, sedative withdrawal, childhood mental retardation and cerebral palsy, brainstem injury.

**PROGNOSIS:** There is little dependable literature on the significance of this finding. The prognosis/reversibility, when this is due to medications, is excellent. In children there is a report of continuous beta spindling in cerebral palsy and mental retardation (extreme spindles). The spindle beta patterns are associated with a good prognosis regardless of etiology, with the exception of children not on barbiturates or benzodiazepines.



This EEG shows a medium- to high-voltage diffuse fast beta pattern. In this case, it is prominent anteriorly, particularly in light sleep and following arousal. Occasionally, it may show a spindling pattern. On EEG, in general, there are beta frequency bands typically seen at 18–25 Hz, less frequently at 14–16, and in one report at 35–40 Hz. It is considered high voltage when it exceeds 25  $\mu$ V [1–4]. It was originally, probably incorrectly, believed to be associated with epilepsy, minimal brain dysfunction, dyslexia, hyperactivity, or other behavioral dysfunction. Conversely, this pattern is typical of a medication effect.

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