

VIEWPOINT



# Taking a History in Neurocritically Ill Patients

Eelco F. M. Wijdicks\*

© 2020 Springer Science+Business Media, LLC, part of Springer Nature and Neurocritical Care Society

## Abstract

A clinical history leads to an examination, tests and a diagnosis. This time-honored sequence in medicine remains valid in critical illness, but in the heat of the moment there is a quickly appearing inevitable sketchiness. Intensivists should never be too unquestioning, too comfortable with incomplete information, or too unwilling to start over if information is muddled or contradictory. No scale in neurology looks at history. There is no tool or requirement to provide a standard system of communication. I review the essentials of history taking in a neurocritically ill patient. Examples of the value of a good medical history are shown but also the familiar biases when asking questions. There are obstacles, errors of commission and omission, and the importance of recognition of a clinical trajectory.

**Keywords:** History, Clinical course, Diagnosis, Bias, Intensive care unit

An established component of neurocritical medical evaluation is, of course, taking a history (known also as anamnesis). This is a formidable task in critical illness and especially taxing when patients are confused, aphasic, or, worse, sedated and intubated. The information-seeking healthcare provider relies on accompanying persons (ideally, close family members), but these often must travel separately, arriving significantly after the patient. Recounting the circumstances of the ictus and clinical trajectory mostly falls to others; the narrative may understandably be somewhat emotive. We can expect to miss important points; they accumulate quickly in the heat of the moment.

Few textbooks address clinical history taking in critically ill patients and none, I believe, comprehensively. However, intensivists should not be too comfortable with incomplete information or too unwilling to start from square one if information is muddled or contradictory.

Several clinical situations are unique to neurointensive care: the comatose patient found down, rapidly progressive weakness, respiratory failure without obvious pulmonary or cardiac triggers, and, of course, mysterious, progressive encephalopathy with abnormal cerebrospinal

fluid (CSF) and hard-to-pinpoint magnetic resonance imaging (MRI) abnormalities. Often, in retrospect, a clinical diagnosis becomes obvious with a better history. We all are reminded of the CSF we should have requested to diagnose meningitis if we had known of the looming infection and fever. There is the MRI of the spine we should have ordered to diagnose an epidural abscess, which we might have considered if we had queried the patient about his unremitting back pain before he lapsed into unresponsive septic shock. Acute double vision may be painful and indicates an immediate need for vascular studies and contrast-enhanced MRI. We expect cerebral vasospasm after a ruptured aneurysm, but it may come earlier than expected if we are unaware that the presenting headache was already a rebleed. Unexplained respiratory failure becomes clearly neurologic if we elicit a prior history of progressive dysphagia, muscle mass loss and twitching (motor neuron disease), diplopia, and weakness increasing with exercise or repetitive use but with day-to-day variation, yet typically strong after a good night's rest (myasthenia gravis).

This paper addresses the obstacles, competencies, and recognition of a clinical trajectory. Examples of the value of a good medical history are shown but also the familiar biases, prejudices, and potential lost focus when asking questions. We can decry the loss of clinical skills—and it is true—but let us start with what comes first. Even the

\*Correspondence: wijd@mayo.edu  
Division of Neurocritical Care and Hospital Neurology, Mayo Clinic, 200  
First Street SW, Rochester, MN 55905, USA

most skilled clinical neurointensivist must work from a solid history.

### Obstacles and Competency

Obtaining a reliable history requires the collaboration of a bystander (ideally) or a close family member. Clinicians must appreciate the tremendous burden of the witness, often summarizing a medical history for the first time. Vague histories are often linked—derisively—to the so-called poor historians. This label is often applied when families or patients provide vague descriptions, imprecise explanations (“doctor so-and-so said”), and mixed-up timelines. Moreover, neurologists recognize that families using words such as “numbness” may actually be describing weakness, that “seizures” often indicate a vasovagal collapse, and that “confusion” may be aphasia. More than in other, less acute neurology settings, families are distraught and overwhelmed, with impaired observational skills. This often causes significant communication lapses, but language and ethnic barriers and communication style may pose other problems. Unfortunately, the hectic intensive care unit (ICU) environment may result in disjointed information and often hearsay. The time needed for a carefully reconstructed timeline is infrequently available; it often occurs after the fact and, sometimes, just does not make sense. Particularly worrisome is the current “cut-and-paste” environment of electronic records and histories that are never questioned or confirmed.

A patient’s history may be presented as a handoff, which we may trust too easily. Some handoff histories may have been generated when there were continuous interruptions. Moreover, miscommunication-related medical errors have been linked to poor handoffs with communication interruptions [1, 2]. These interruptions lead to poor recall and disagreement on what is the most important piece of communicated information.

There is no tool, requirement, or system to provide standardized communication. No scale in neurology looks at history. In addition, there is little training in how to pick up cues with attentive listening, how best to communicate, how to avoid distractions in an urgent environment, and how to ask open-ended questions. In legal circles, these are known as errors of commission, which include trusting faulty memory, obtaining erroneous information. Errors of omission include failure to resolve contradictory statements in records and to review prior medical records. Urgent cases require quick histories, but sufficient information gathering (and, if needed, correctives) should come later, when the dust has settled. We tend to forget and move on to the next patient.

Taking a good history depends on experience—knowing what to ask and how to interrupt and guide

when needed. It also requires knowing what can be ignored or minimized and what is crucially important. Some elements of the history are exceedingly important, and one must inquire about certain circumstances, onset, and progress of symptoms (Table 1). In patients found comatose, drug overdose must be considered if there are no neurologic signs other than coma and CT brain is normal. In addition to medication prescribed for the patient, neurointensivists must consider (and test for) other medication or drugs to which the patient might have had access.

### Biases and Pattern Recognition

There have been many situations when the team looks at an MRI and concludes that it “does not make sense.” A clinical history may be incompatible with neurologic examination or tests results. Examples of discrepancies abound. Red flag symptoms might be missed in history taking and may include failure to inquire about fever, thunderclap onset of headache, intravenous drug abuse, empty medication bottles, or prolonged use of corticosteroids (Table 2). An important part of the history is to know what medication the patient was taking and what was recently (either deliberately or accidentally) discontinued. Some drugs are notoriously toxic, and others cause serious withdrawal symptoms; therefore, medication reconciliation is absolutely essential for a full history (Table 3). Inaccurate history leads to failure to order appropriate diagnostic imaging and missing the diagnosis.

**Table 1 Deconstructing a clinical history**

Baseline function (and recent change)	Memory Productivity Mobility Responsibilities Wandering and unsafe
Prior hospitalizations or ED visits	Presumptive diagnosis
Drugs for infection	Antibiotics
Prior vices	Drinking habits Illicit drugs Over the counter
Prior psychiatry	Suicide attempts or considerations
Family history of predisposition	Aneurysms /AVM
Found down	Scene description Outside temperature Visible trauma Need for CPR Prior diabetes and insulin use Stroke clues (atrial fibrillation)

**Table 2 Urgent medication reconciliation**

Drugs	Consequences
Anticoagulation (Lovenox, NOACs)	Urgent reversal
Antibiotics (Flagyl, cefepime)	Neurotoxicity
Withdrawal (baclofen; levodopa with carbidopa, opioids)	Treatment for rhabdomyolysis or seizures
Antiepileptics (levetiracetam, phenytoin)	Withdrawal or toxicity
Antidepressants (SSRI)	Serotonin syndrome

NOAC New oral anticoagulants; SSRI selective serotonin reuptake inhibitors

And then there is a touchy issue. Although the young and less experienced are not bothered by arrogance of their seniors, we are never absolved from making gross regrettable errors. Arguably, errors will become less common as available knowledge increases—but not always. Even a seasoned, highly active, involved physician may be fooled by a slightly different presentation. Much of what we do in medicine is pattern recognition, and knowing the patterns is critical to avoiding errors. Clinical experience does not reduce error rate, and physicians can easily repeat the same mistake time and again. The most common bias is the so-called representativeness restraint. Physicians may fail to perceive important signs and try to force everything into a more recognizable scenario. History taking is subject to confirmation bias, which reflects the tendency to seek out data to confirm one's original idea rather than to try to account for contradictory data and also to jump quickly to a diagnosis [3]. Confirmation bias seeks confirmation rather than discerning other possibilities. With that, also comes “anchoring,” when a history is cemented too early. (Table 4) Often, it is useful

not to get distracted by incongruities, but sometimes an important observation requires explanation. Errors are often in judgment and not exclusively procedural [4].

### Construct a Clinical Trajectory

In the ICU, all must seem acute. But there are problems. Adjectives such as acute, hyperacute, subacute and rapidly progressive are commonly used, but likely mean something different to each person. However, it is useful to view illness in definable events such as time of onset, time to nadir, time in nadir, or time with stable deficit and rate of recovery. Certain neurocritical disorders have predictable patterns in their clinical trajectory. Before the recent luxury of detailed neuroimaging, neurologists predicted a diagnosis on the clinical trajectory and findings on examination. Many of us still practice a priori estimations, and neuroradiologists also use these when interpreting images and estimating a posteriori probabilities.

A good history can provide a clinical trajectory; several examples are shown in Fig. 1. Acute occlusions of the middle cerebral artery often suddenly present with a major deficit—but not always. Some patients improve substantially only to worsen when the collateral circulation fails [5, 6]. Occluded vertebrobasilar arteries often fluctuate markedly in posterior circulation signs such as ataxia, dysphagia, and dysarthria. Although a clot can dissolve and fully resolve [7–9], there is consensus that it is better to retrieve the clot when there are residual symptoms; clinical history has taught us in the past to anticipate worsening later. Recognition that fluctuations and major improvement of clinical signs may not be fully reassuring becomes important in decisions on endovascular treatment in acute stroke.

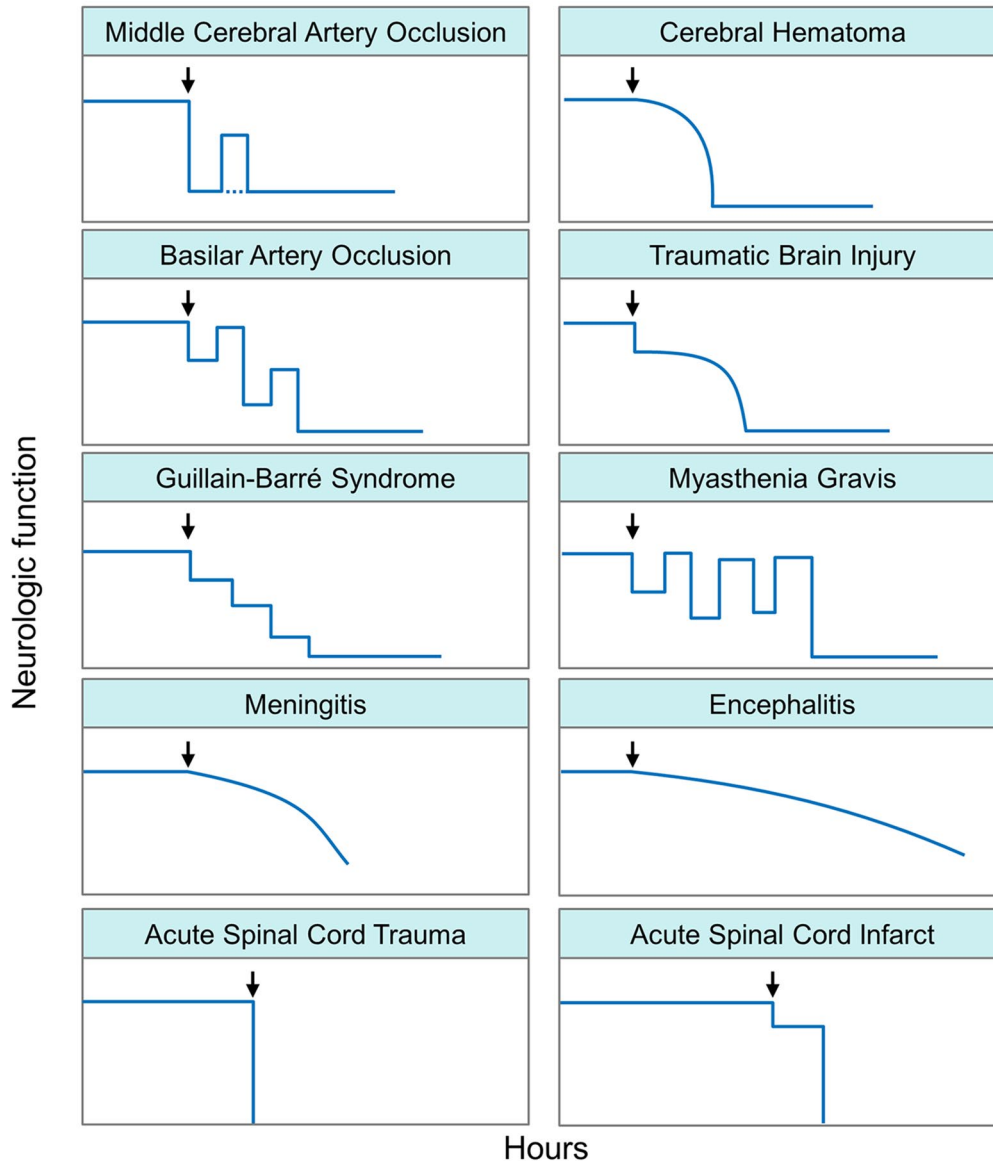
**Table 3 Red flags in history taking**

Fever (meningitis, encephalitis, epidural spinal abscess)
Immunosuppression and intravenous drug use (fungal or parasitic infections)
Comorbidity (medication errors due to failure of dose adjustment)
Failure to inquire about sphincter syndromes and loss of sensation (signs of cauda equina syndrome)
Failure to appreciate recent use of NMJ blockers (presenting intubated comatose patients)
Failure to appreciate alcohol intoxication (presenting intubated comatose patients)

**Table 4 Biases in taking a history**

Anchoring	Stuck on features of the patient's presentation too early and failure to adjust when other information becomes available
Availability	The disposition to judge things as being more likely or frequently occurring, if they readily come to mind
Premature closure	The diagnosis is accepted before it has been fully verified
Representativeness restraint	Pattern recognition and failure to think out of the box (atypical variants)
Unpacking principle	Not getting all information to establish a differential diagnosis
Context errors	Too much information and losing track of the most important facts

## ACUTE TRAJECTORIES IN NEUROCRITICAL ILLNESS



**Fig. 1** Clinical trajectories for common neurocritical disorders in the first hours/days (not including recovery trajectories)

The clinical trajectory in cerebral hemorrhage often involves more gradual symptoms than ischemic stroke. Small arterial bleeds lead to growing volume under pressure, damaging other arteries when they spread into the perivascular spaces, tearing penetrating arteries or veins along the way. This domino effect progresses until platelet plugs appear surrounded by walls of red blood cells and fibrin—Fisher's fibrin globes [10]. This pathophysiology has a clear clinical correlate. Although hemorrhages can be suddenly catastrophic (and lethal), neurologic deficits typically appear more gradually with worsening

weakness and new deficits in a number of hours. In some patients, these may culminate in a further decline in consciousness and an abrupt change in breathing (from early mass effect and brain tissue shift). This gradual course is more common in putaminal and thalamic hemorrhages than in cerebellar hemorrhages because the compartments are larger and more accepting of newly increased volume.

The course in traumatic brain injury is also markedly determined by presence of extracranial blood in either the subdural or epidural compartment. The lucid interval



after trauma has been considered **pathognomonic** for **epidural hemorrhages**, but blossoming contusions may be more common than has been truly appreciated. Traumatic brain injury commonly correlates with alcohol intoxication or drug use, thus easily confounding and masking the typical trajectory. **High blood alcohol levels** correlate with an **increased expansion rate** [11].

The **two most common acute neuromuscular disorders** in the neurointensive care unit are **Guillain-Barré syndrome (GBS)** and **myasthenia gravis** but with **markedly different presentations**. **GBS proceeds in a stepwise fashion, using several days to reach a nadir**. **Myasthenia gravis, however, fluctuates within a day** and may show both extremes with near-normal examination to marked weakness. Recognition of the trajectory as a result of fatigable weakness often clinches the diagnosis.

Traumatic spinal cord injury is acute and complete, although symptoms may improve when the spinal shock phase resolves [12]. Spinal cord infarct is also acute, but may be **gradual** before a **very sudden loss of motor function**, which occurs up to **12 h after initial onset** [13].

Central nervous system infections usually progress gradually with a much steeper slope in acute bacterial meningitis. Nevertheless, patients appearing mildly lethargic or obtunded on initial presentation may become deeply comatose within hours.

After the acute phase, the clinical course in patients with acute brain injury is determined by a number of events. First, **obstruction of the CSF flow** causes further deterioration unrelated to the primary event. Examples are aneurysmal subarachnoid hemorrhage, thalamic hemorrhages trapping the ventricle, cerebellar hematomas obliterating the 4th ventricle, or simply hemorrhage breakthrough into the ventricular system **blocking outflow**. Second, **shift from expansion due to cerebral edema** and often driven by increased intracranial pressure can worsen the clinical picture until patients need intubation for airway protection. Clinical course in many patients is determined by whether a neurosurgical intervention is performed. Evacuation of a hematoma or ventriculostomy placement often interrupts a downward spiral. Later clinical trajectories are often predicted by the aggressiveness of interventions and not by the natural history alone.

These specific clinical trajectories can often be obtained after a careful history and significantly add to a full description of the clinical picture. It is anticipatory information for attending neurointensivists—forewarned is forearmed.

## Conclusion

Taking a clinical history takes time and, for some, just too much time. There is often no time to listen carefully;

cell phones or pagers predictably interrupt the encounter. Physicians often redirect opening statements of patients and families, but also direct questions toward a specific concern; we have no patience and redirect already after 20 s on average [14, 15]. Failures of information gathering and integration are due to insufficient time and being pressed for time. **We must force the mind to slow down**. Most errors are related to process breakdowns in the patient–practitioner clinical encounter.

We understand why there may be complacency that is difficult to address. Generally, physicians generate a working diagnosis almost immediately upon hearing a patient's initial symptom presentation, and there is a tendency to seek a familiar pattern. **A decision may be reached without exploration of other possibilities**. *Mostly*, the diagnosis is correct, appropriate tests are ordered, and effective treatment begins. This pattern is common, difficult to change, and, counterintuitively, reassuring to families. However, exceptions must always be considered. According to Berner and Graber, physicians know that diagnostic error exists, but **think that the likelihood of error is less than it really is** [16]. As Vickrey advises, deliberately pursue another angle: **"Let's play devil's advocate"** or **"Let's rereview elements of the history"** [3].

A deliberate consideration of a differential diagnosis is an essential part of diagnostic reasoning prior to the final decision. The most common breakdown points are test ordering and interpretation, performance of the medical history and physical examination, and initiation of consultations. We should praise independence, but we should also, once in a while, seek opinions from colleagues. **Failure to order appropriate tests was the most frequent breakdown**.

Getting the history right is a core principle. A phone call to family members or direct communication is essential to provide a sense of the time course and urgency. Seemingly acute conditions may actually be chronic and vice versa as more information becomes available.

In the new era of intensive care imaging with handheld and smartphone-connected devices, the clinical history may be sidelined even more. Who wants to know what really happened if we can see it already? Are examination and imaging not enough? Without diminishing these parts of the clinical assessment, we should approach them cautiously, with the intent to achieve accuracy. If pressed for time, it may be preferable to tailor the examination and spend more time taking a history. Information gathering starts within the first moments of an encounter. Do not assume or trust anything; verify everything.

Future machine learning may enhance knowledge, but ambiguous, "out-of-the-blue" statements by those providing the history will remain challenging. Stories seldom occur in a vacuum; every story of acute illness

involves human interactions, decisions, and varying circumstances. The attending will still make multiple phone calls to obtain information. Nothing can be easily compartmentalized in bits or algorithmic modules. For a while, we may still have to endure Siri proclaiming “sorry, I didn’t quite get that.” Taking a history and owning the conversation remains the most critical skill of a neurointensivist.

#### Conflict of interest

The author declares that he has no conflict of interest in relation to this manuscript.

#### Publisher’s Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Published online: 28 April 2020

#### References

1. Gandhi TK. Fumbled handoffs: one dropped ball after another. *Ann Intern Med.* 2005;142(5):352–8.
2. Riesenberger LA, Leitzsch J, Massucci JL, et al. Residents’ and attending physicians’ handoffs: a systematic review of the literature. *Acad Med.* 2009;84(12):1775–877.
3. Vickrey BG, Samuels MA, Ropper AH. How neurologists think: A cognitive psychology perspective on missed diagnoses. *Ann Neurol.* 2010;67(4):425–33.
4. Rolston JD, Bernstein M. Errors in neurosurgery. *Neurosurg Clin N Am.* 2015;26(2):149–55.
5. Antunes Dias F, Castro-Afonso LH, Zanon Zotin MC, et al. Collateral scores and outcomes after endovascular treatment for basilar artery occlusion. *Cerebrovasc Dis.* 2019;47(5–6):285–90.
6. Hernandez-Perez M, Perez de la Ossa N, Aleu A, et al. Natural history of acute stroke due to occlusion of the middle cerebral artery and intracranial internal carotid artery. *J Neuroimaging.* 2014;24(4):354–8.
7. Cornelius JR, Zubkov AY, Wijdicks EF. Following the clot in spectacular shrinking deficit. *Rev Neurol Dis.* 2008;5(2):92–4.
8. Lee VH, John S, Mohammad Y, Prabhakaran S. Computed tomography perfusion imaging in spectacular shrinking deficit. *J Stroke Cerebrovasc Dis.* 2012;21(2):94–101.
9. Minematsu K, Yamaguchi T, Omae T. ‘Spectacular shrinking deficit’: rapid recovery from a major hemispheric syndrome by migration of an embolus. *Neurology.* 1992;42(1):157–62.
10. Fisher CM. Pathological observations in hypertensive cerebral hemorrhage. *J Neuropathol Exp Neurol.* 1971;30(3):536–50.
11. Carnevale JA, Segar DJ, Powers AY, et al. Blossoming contusions: identifying factors contributing to the expansion of traumatic intracerebral hemorrhage. *J Neurosurg.* 2018;129(5):1305–16.
12. Zalewski NL, Rabinstein AA, Krecke KN, et al. Characteristics of Spontaneous Spinal Cord Infarction and Proposed Diagnostic Criteria. *JAMA Neurol.* 2019;76(1):56–63.
13. Atkinson PP, Atkinson JL. Spinal shock. *Mayo Clin Proc.* 1996;71(4):384–9.
14. Beckman HB, Frankel RM. The effect of physician behavior on the collection of data. *Ann Intern Med.* 1984;101(5):692–6.
15. Marvel MK, Epstein RM, Flowers K, Beckman HB. Soliciting the patient’s agenda: have we improved? *JAMA.* 1999;281(3):283–7.
16. Berner ES, Graber ML. Overconfidence as a cause of diagnostic error in medicine. *Am J Med.* 2008;121(5 Suppl):S2–.