The Accuracy of Bedside Neurological Diagnoses

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The accuracy of bedside diagnoses was prospectively studied in 100 consecutive patients admitted to the neurology service at New England Medical Center, Boston. Each patient was evaluated independently by a junior resident, a senior resident, and a staff neurologist, who were required to make an anatomical and etiological diagnosis based solely on the history and physical examination. Fourteen patients were excluded because their diagnoses were known before admission. Of the remaining 86 patients, it was possible to confirm anatomical and etiological diagnoses in 40 by matching the clinical syndromes with highly specific laboratory findings. In the other 46 patients, the diagnoses could not be confirmed because the laboratory studies (including magnetic resonance imaging) were negative or nondiagnostic. In the 40 patients with laboratory confirmed final diagnoses, the clinical diagnoses of the junior residents, senior residents, and staff neurologists were correct in 26 (65%), 30 (75%), and 31 (77%), respectively. There was a trend for error rates to be higher among junior residents than staff (p = 0.06). The errors by the junior residents, [senior residents], (staff) were attributed to incomplete history and examination in 4 [1] (0), inadequate fund of knowledge in 4 [3] (3), and poor diagnostic reasoning in 6 [6] (6). These results indicate that technology is not a panacea for our diagnostic difficulties and that there is room for improvement in our clinical skills, especially diagnostic reasoning.

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Formulation of an accurate bedside diagnosis is essential because it determines the efficiency of patient care and is important for initiating appropriate treatment. Studies on the accuracy of clinical diagnoses evaluated at autosy [1–3] and in living patients with specific clinical syndromes (e.g., stroke [4], gastrointestinal bleeding [5]) indicate that clinical diagnoses are frequently incorrect; however, there is no prospective validation of these results in a living patient population with a wide spectrum of neurological diseases. This study of the accuracy of bedside neurological diagnoses was undertaken to determine (1) the frequency of and reasons for diagnostic errors made by residents as opposed to staff neurologists; and (2) whether the neurological clinical method has become relatively obsolete in this era of high technology [6].

Material and Methods

One hundred consecutive patients admitted to the neurology service at New England Medical Center, an acute care, referral center in Boston, were prospectively studied. Patients with a wide spectrum of neurological conditions are seen at this institution, which has subspecialty services in stroke, neuromuscular diseases, epilepsy, and neurooncology. Patients entered into the study were admitted through the emergency room or outpatient department. All admission diagnoses were hidden from the physicians participating in the study. Each patient was evaluated independently by a junior resident, a senior resident, and a staff neurologist within 24 hours of admission. Unless emergent diagnostic procedures or treatment were necessary, communication between the study physicians regarding the patient's preliminary diagnoses was not permitted until all the study physicians had evaluated the patient. If the preliminary diagnosis became known to a study physician before the patient was seen, the patient entered the already known category.

Each physician was required to make a specific anatomical diagnosis and an etiological differential diagnosis for each patient based solely on the history and physical examination. Only one anatomical diagnosis was permitted unless the clinical syndrome could be explained by a lesion in more than one location, e.g., pure motor hemiparesis, which can be caused by a pontine or capsular lesion [7]. The etiological differential diagnosis was arbitrarily limited to the three most likely mechanisms to prevent inclusive lists of all potential diagnoses. The diagnoses were required to be quite specific—it was unacceptable to provide generic diagnoses, such as left hemisphere stroke or peripheral neuropathy. Acceptable precise descriptions would be left middle cerebral artery (MCA) territory infarct from a cardiogenic embolus, or diabetic polyradiculopathy, respectively.

The final diagnosis was agreed upon by all the study physicians participating in the patient's care and was determined by matching the clinical syndrome with the results of labora-

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tory and radiological studies. The final anatomical and etiological diagnoses were considered (1) *confirmed* if the clinical syndrome could be matched with highly specific laboratory or radiologic findings; (2) *unconfirmed* if the clinical syndrome was typical but special investigations did not confirm the diagnosis; or (3) *uncertain* if neither of the above conditions applied.

Bedside diagnostic accuracy was determined by the percentage of correct clinical diagnoses made by the residents and staff in the patients with laboratory confirmed and unconfirmed final diagnoses. A clinical diagnosis was considered correct if the anatomical diagnosis and any one of the three etiological diagnoses were correct.

If an error in bedside diagnosis was made, the reason for the error was determined by reviewing the diagnostic process with the physician involved. Errors were classified into three broad groups. Those related to (1) *an inadequate data base,* defined by incomplete or incorrect collection of the historical and physical findings; (2) an *inadequate fund of knowledge,* defined by a sufficient data base but insufficient knowledge of the spectrum of clinical manifestations associated with the patient's disease; or (3) *reasoning errors,* defined by a sufficient data base, adequate knowledge of the clinical manifestations of the patient's disease, but incorrect analysis of the data. If more than one factor contributed to the error, the reason for the error was assigned to the factor considered most important.

The focus of this study was to evaluate the frequency and causes of errors in clinical diagnosis; no effort was made to determine the effects of such errors. Therefore errors with significant clinical consequences were not differentiated from those with little consequence.

Results

One hundred consecutive patients were evaluated in the study. Each patient was seen by two residents and one staff neurologist. All eleven residents and nine staff members in our department saw at least one patient.

In 14 of the 100 patients the diagnoses were already known. Of the remaining 86 patients, it was possible to confirm diagnoses in 40, establish probable but unconfirmed diagnoses in 18 (Table 1), and the diagnoses remained uncertain in 28. In the 40 patients with laboratory confirmed final diagnoses, the clinical diagnoses of junior residents, senior residents, and staff neurologists were correct in 26 (65%), 30 (75%), and 31 (77%), respectively. There was a trend for error rates to be higher among junior residents than among staff neurologists ($p = 0.06$, binomial test) despite the small sample size of patients with confirmed diagnoses. Assuming that the 18 unconfirmed diagnoses shown in Table 1 were correct, the clinical diagnoses of the junior residents, senior residents, and staff neurologists were accurate in 10, 15, and 16 of these 18 patients, respectively. Therefore, the overall clinical diagnostic accuracy rates of the junior residents, senior residents, and staff, in patients with confirmed and unconfirmed diagnoses, were 62% (36/58), 77% (45/58), and 81% (47/58), respectively. The difference in overall error rates between junior residents and staff was highly significant ($p = 0.006$, binomial test).

Table 1. Unconfirmed Diagnoses in 18 of 100 Study Patients*

Diagnosis	No. of Patients
Somatoform disorder	6
Lacunar stroke	4
Classic migraine variants (Raeders' syndrome [1], vertebrobasilar migraine [1], occipital migraine [1])	3
Alzheimer's disease	1
Shy-Drager syndrome	1
Schizophrenia	1
Traumatic concussion	1
Primary lateral sclerosis	1

*Established by typical clinical presentation with negative or non-diagnostic laboratory and radiological studies.

In the patients with confirmed diagnoses, all 10 diagnostic errors made by the senior residents were made by the junior residents, who made an additional 4 errors. Eight of the 9 staff errors were also made by both junior and senior residents; however, in 1 case (patient 26, Table 2), only the staff neurologist's diagnosis was incorrect. The 14 [10] (9) errors made by the junior residents, [senior residents], (staff) were incorrect anatomical diagnoses in 4 [2] (3), incorrect etiological diagnoses in 4 [4] (3), and both in 6 [4] (3). These errors were attributed to incomplete history or examination in 4 [1] (0) (Patients 1, 5, 15, 61 in Table 2), inadequate fund of knowledge in 4 [3] (3) (Patients 2, 18, 27, 37 in Table 2), and poor diagnostic reasoning in 6 [6] (6) (Patients 17, 26, 52, 56, 59, 72, 77 in Table 2). A summary of the errors made in patients with confirmed diagnoses is provided in Table 3. The following case studies illustrate some of the errors in diagnostic reasoning.

Patient 17

A 59-year-old man with a history of a partially resected pituitary adenoma experienced sudden onset of horizontal diplopia, dizziness with nausea and vomiting, bifrontal headache, and difficulty walking. The diplopia resolved after an hour. He was able to walk by holding on to walls. He spent the next two days in bed before being admitted to the hospital. There was no history of hypertension, diabetes, or cardiac disease.

On examination he was obese and slightly lethargic but easily arousable. His blood pressure was 150/85, his pulse 85 and regular, and he was afebrile. His cognitive function was normal. The optic disks were sharp and flat without hemorrhages. The right palpebral fissure was 4 mm, the left was 5 mm. The pupils were

Table 2. Diagnostic Errors Made by Residents and Staff in Patients with Confirmed Diagnoses

Patient No.	Presentation	Clinical Anatomical Diagnosis	Clinical Etiological Diagnosis	Final Anatomical Diagnosis	Final Etiological Diagnosis	Major Cause of Error	Error Made By
1	Weakness, atrophy, and occasional fasciculations in UE; spastic gait	Diffuse involvement of anterior horn cells and corticospinal tracts	ALS	Cervical myelopathy with C5-C6 radiculopathies bilaterally	Cervical spondylosis	Failure to recognize radicular pattern of weakness and sensory loss in UE and markedly diminished biceps reflexes relative to brisk triceps and finger flexors	JR,SR
2	Weakness and pain of left foot	Sacral plexopathy	Tumor invasion, diabetes	Polyradiculoneuropathy	Vasculitis	Inadequate knowledge regarding atypical presentation of vasculitis	JR,SR,S
5	Sudden left arm and leg incoordination and sensory loss; headache; left arm drift with eyes closed, but normal strength of left arm with eyes open and fixating on arm	Right frontoparietal lobes	Infarct due to cardiogenic embolus or right carotid occlusion	Right thalamus	Hypertensive hemorrhage	Incorrectly attributed left arm and leg dysfunction to weakness rather than hemisensory loss	JR
15	Confusion with language disturbance after coronary bypass surgery	Diffuse bilateral cortical involvement	Hypoxic-ischemic encephalopathy	Left temporal-parietal lobes	Infarct caused by cardiogenic embolus	Incomplete aphasia testing	JR
17	Dizziness, diplopia, gait disturbance	Pituitary gland	Infarct or hemorrhage	Right cerebellar hemisphere	Infarct caused by cardiogenic embolus	Reasoning error (see text)	JR,SR,S
18	Rash, fever, headache, confusion preceding weakness of LE by a few days	Meningoencephalitis and bilateral L3-L4 radiculopathies	Lyme disease	Meningoencephalitis and bilateral L3-L4 radiculopathies	EBV infection	Inadequate knowledge regarding (1) atypical presentation of EBV infection; (2) expected time course of meningitis following ECM in Lyme disease	JR,SR,S

Table 2. (Continued)

Patient No.	Presentation	Clinical Anatomical Diagnosis	Clinical Etiological Diagnosis	Final Anatomical Diagnosis	Final Etiological Diagnosis	Major Cause of Error	Error Made By
26	Walking into objects on the left, behavioral change, hemianopia of which patient was unaware	Right occipital and cerebellar lesions	Infarction caused by cardiogenic embolus or basilar thrombus	Right parieto-temporal lobes	Infarct due to right inferior division MCA stenosis	Reasoning error (see text)	S
27	Nonfluent aphasia, hemianopia (no weakness)	Territory supplied by superior division left MCA	Cardiogenic embolus	Territory supplied by inferior division left MCA	Cardiogenic embolus	Lack of knowledge regarding occurrence of nonfluent aphasia and hemianopia without hemiparesis in inferior division left MCA infarcts [27, 28]	JR
37	Sudden onset of flinging movements of right arm	Left thalamus or subthalamic nucleus	Lacunar infarction; hemorrhage	Left parietal lobe	Hemorrhage	Inadequate knowledge regarding atypical presentation of parietal lobe lesion	JR,SR,S
52	Episode of right TMB and left arm weakness after jarring neck while chopping wood. One day later: confusion, hemianopia, and mild left hemiparesis	Right parietal lobe supplied by inferior division of right MCA	Atherosclerotic right carotid occlusion	Right parietal lobe supplied by inferior division of right MCA	Right carotid dissection with artery to artery embolus	Reasoning error—failure to associate neck movement with disease mechanism	JR,SR
56	Inability to use left arm to command though spontaneous movement normal; halting speech	Left frontal lobe and corpus callosum	Ischemic infarct, tumor		Hysteria	Reasoning error—failure to explain absence of other expected findings for proposed lesion	JR,SR,S
59	Persistent right IIIrd nerve palsy preceding bilateral LE weakness by several weeks	Right IIIrd nerve palsy and bilateral lumbosacral radiculopathies	Diabetes (despite history of normal glucose tolerance test)	Right IIIrd nerve palsy and bilateral lumbosacral radiculopathies	Lymphoma of nasal sinuses with lymphomatous meningitis	Reasoning error—failure of proposed diagnosis to explain normal glucose tolerance and time course of illness	JR,SR,S

Table 2. (Continued)

Patient No.	Presentation	Clinical Anatomical Diagnosis	Clinical Etiological Diagnosis	Final Anatomical Diagnosis	Final Etiological Diagnosis	Major Cause of Error	Error Made By
61	Bilateral asymmetrical leg pain and weakness	Polyneuropathy	Diabetes	Lumbar polyradiculopathy	Diabetes (amyotrophy)	Predominantly proximal nature of symptoms and signs not recognized	JR
72	Right leg weakness and incoordination of right arm	Left anterior cerebral artery territory	Cardiogenic embolus; carotid occlusive disease	Left corona radiata	Lacunar infarct	Reasoning error—failure to consider subcortical location in differential diagnosis	JR,SR,S
77	Confusion, left arm and leg numbness, neglect of left visual field	Right inferior division MCA territory	Coagulopathy; dissection; embolus of unknown source	Right inferior division MCA territory	Cardiogenic embolus secondary to SBE	Reasoning error (see text)	JR,SR,S

UE = upper extremities, LE = lower extremities, ALS = amyotrophic lateral sclerosis, EBV = Epstein-Barr virus, ECM = erythema chronicum migrans, MCA = middle cerebral artery, TMB = transient monocular blindness, SBE = subacute bacterial endocarditis, JR = junior resident, SR = senior resident, S = staff.

Table 3. Summary of Bedside Diagnostic Errors in Patients with Confirmed Diagnoses

	Junior Residents	Senior Residents	Staff
Number of correct diagnoses/patient encounters (%)	26/40 (65)	30/40 (75)	31/40 (77)
Number of errors	14	10	9
Type of error			
Incorrect anatomical diagnosis	4	2	3
Incorrect etiological diagnosis	4	4	3
Both	6	4	3
Reason for error			
Incomplete history or examination	4	1	0
Inadequate fund of knowledge	4	3	3
Poor diagnostic reasoning	6	6	6

3 mm, equal and reactive to light. Visual fields and acuity were normal. Extraocular movements were full without nystagmus, and the corneal reflexes, facial sensation and strength, gag reflex, and tongue strength were normal. He had normal strength, tone, sensation, and coordination in all extremities. The tendon reflexes were 2+ throughout; both plantar responses were flexor. His gait was slightly unsteady, and he was unable to perform tandem walking. Romberg's sign was absent.

The initial clinical diagnosis was pituitary apoplexy. A cranial computed tomography (CT) scan revealed a large right cerebellar infarct. Vertebral angiography showed a right posterior-inferior cerebellar artery occlusion, and an echocardiogram revealed a dyskinetic left ventricle.

In this patient both the anatomical and etiological diagnoses were incorrect. The physicians were misled by the presence of a preexisting disease. They made the diagnosis of pituitary apoplexy despite their knowledge that confusion, ophthalmoplegia, and visual field deficits [8] are expected findings in that condition and were absent in this case.

Patient 26

A 72-year-old woman suddenly began to bump into things on the left side when she walked. The same day she developed a bifrontal headache that persisted for the next five days. During this period she intermittently felt nauseated and vomited on two occasions after taking codeine. Her family noted that she was unusually irritable and impulsive. The patient admitted

making "nasty comments to family members in situations that ordinarily I would attempt to say something nice." Her past medical history was significant only for hypertension, which was well controlled with treatment.

On examination, her blood pressure was 130/80, pulse 80 and regular, and she was afebrile. There were no carotid or ocular bruits. Cardiac examination was normal. She was alert, intelligent, and astute about current and past events, yet she was unconcerned about her health. She copied poorly, and when she read, she began a new line in the middle. She failed to point to objects on the left side of a picture when asked to identify what she saw. Cranial nerve examination was normal except for a dense left homonymous hemianopia, which she was unaware of. She had normal strength, sensation, and coordination. The reflexes were 3+ bilaterally, and both plantar responses were flexor. She was a little unsteady when she walked but there was no obvious ataxia. Romberg's sign was absent.

The clinical diagnosis was a right occipital infarct to explain her hemianopia and possibly a cerebellar infarct to explain the nausea, vomiting, and mild unsteadiness. A cardiogenic embolus to the posterior circulation was considered the likely mechanism. The cranial CT scan revealed a right parietotemporal infarct in the territory of the inferior division of the MCA. A right carotid angiogram revealed stenosis of the proximal inferior division of the MCA with a missing angular branch more distally.

The diagnostic error was failure to explain her behavioral changes, poor copying, and neglect of her dense field cut, which are characteristic of right inferior division MCA infarcts [9] but are unexpected manifestations of a right occipital infarct. Despite knowledge of this MCA syndrome, the physician was misled by the less specific symptoms of nausea, vomiting, and mild unsteadiness.

Patient 77

A 35-year-old right-handed internist was referred for evaluation of left hemisensory dysfunction. He awoke the morning before admission feeling confused. He dressed and went to work but noticed that he could not feel things well on his left side and was not thinking clearly. An hour later he had a right supraorbital headache that worsened over the day. He denied any weakness, dysarthria, loss of consciousness, or visual disturbance. There had been no transient neurological spells in the past, nor had there been any head or neck trauma. His general health had been excellent, although in the preceding month he had noticed a 3 × 3 cm red macule on his right ankle which resolved spontaneously over a week. Additionally, he had been given a steroid injection for a painful, red, swollen lesion in the palm of his right hand in the region of the third metacarpal phalangeal joint. The evening before admission he had a low grade fever. His past medical history was negative for hypertension, diabetes, or heart disease.

On examination, blood pressure was 130/88, he was afebrile, and his pulse was 76 and regular. His neck was supple and there were no bruits. There was a soft systolic ejection murmur over the aortic area. He was alert with an obvious right gaze preference but could look fully to the left. Speech was normal, and he was aware of his sensory dysfunction. He copied well. He had left visual neglect on double simultaneous stimulation but he perceived a single stimulus to the left. Left optokinetic nystagmus was diminished. Sensation was diminished on the left side of his face. He had a left hemisensory loss to pin, touch, cold, and proprioception. Strength, reflexes, and gait were normal. The plantar responses were flexor.

The clinical diagnosis was an infarct in the territory of the inferior division of the right MCA. The mechanisms considered included a carotid or intracranial dissection, coagulopathy, or embolus of unknown source. The cranial CT scan confirmed a right parietal infarct, and a right carotid angiogram showed occlusion of the inferior division of the right MCA. Heparin was begun but was discontinued later that night when he developed a fever of 39°C. Blood cultures taken at that time grew *Streptococcus viridans.* An echocardiogram revealed a bicuspid aortic valve with a vegetation on the valve, confirming subacute bacterial endocarditis (SBE).

In this patient the anatomical diagnosis was correct but the etiological diagnosis was incorrect. Review of the diagnostic process showed that the physicians collected sufficient clinical data and knew that fever, skin lesions, and embolic stroke are possible features of SBE, yet they did not generate this hypothesis (i.e., a reasoning error).

Discussion

Evaluation of the clinical method is difficult because methodologies for studying complex skills such as clinical reasoning and judgment have not been established. This may account for the paucity of data on this subject. To our knowledge, this study is the first to measure the accuracy of bedside diagnoses in patients with a wide spectrum of neurological diseases. The diagnostic accuracy rates of residents and staff neurologists in this study should be interpreted cautiously, however, because the methodology we used has certain limitations. First, arbitrarily limiting the etiological differential diagnosis to the three most likely disease mechanisms forced us to label the etiological diagnosis as incorrect even if the correct diagnosis was listed

below the top three. Second, the simple binary (i.e., correct or incorrect) system used in the study for scoring the accuracy of diagnoses has practical limitations because it fails to distinguish degrees of diagnostic errors and does not differentiate errors with serious implications (Patient 59) from those with little consequence (Patients 27, 61). Despite these limitations, these results provide an estimate of the accuracy of bedside neurological diagnoses for residents and staff neurologists at our institution, and are similar to the accuracy rate of 69% for neurologists attempting to differentiate stroke mechanisms [4].

Analysis of the diagnostic errors made in patients with confirmed diagnoses suggests that history taking and physical examination skills depend on clinical experience, whereas reasoning skills do not. Although there was no difference between residents and staff in error rates related to inadequate knowledge, the method we used for categorizing reasons for errors masks the impact that fund of knowledge has on data collection. For example, it is likely that some of the residents' data collection errors were partly due to insufficient knowledge of clinical syndromes. Furthermore, in the inadequate fund of knowledge category all the errors made by the staff were due to unfamiliarity with atypical presentations of certain diseases (Patients 2, 18, 37), whereas the junior residents made an additional error related to inadequate knowledge of a relatively common disease presentation (Patient 27). We suspect that in a larger sample of patients, the difference in error rates between residents and staff in the inadequate fund of knowledge category would become evident, especially in the subcategory related to more common disease presentations.

Previous studies have also found that certain reasoning errors are independent of clinical experience. Voytovich and associates [10] reported that premature diagnostic conclusions ("premature closure") occurred with equal frequency in a group of physicians, residents, and students. Friedlander and Phillips [11] found that experienced physicians may be more susceptible to "anchoring" errors [12] (i.e., retaining an initial diagnostic hypothesis despite subsequent evidence to suggest another). Several types of reasoning errors were identified in this study: anchoring (Patient 17); failure to distinguish the cardinal symptoms and signs from less specific, potentially misleading ones (Patient 26); errors of omission [10] in which important clues are simply ignored (Patients 52 and 77); and generating a hypothesis that is logically inconsistent with the facts (Patients 56 and 59).

How can these errors be prevented in the future? Closer observation of residents' history taking and examination skills by the staff will help to correct deficiencies in data collection [13]. The accumulation of additional clinical experience and knowledge will presumably prevent some errors related to inadequate fund of knowledge. Errors of reasoning constitute a large proportion of the errors made by the residents and staff combined in this study. Clinical reasoning involves generating and testing sequential hypotheses and requires creative and analytical skills. Although there is growing interest in this subject [10, 11, 14–22], most medical schools have not developed curricular strategies to teach these skills but expect students to acquire them during their clinical clerkships [23]. Some authors, however, have suggested that reasoning skills should be specifically taught and practiced [22, 24]. In this regard, Kassirer [22] has suggested a novel way of conducting attending rounds: A resident who has seen the patient acts as the source of all the patient's clinical data. The chief complaints are presented, and then other residents/students who have not seen the patient inquire about the key historical facts needed to generate or narrow the list of hypothetical diagnoses. Each question needs to be justified by explaining the reason for the question and the hypothesis being considered. The questioner also must interpret the information elicited by the question, and if necessary, change the diagnostic hypothesis. The same process of questioning, justification, and interpretation is continued for the physical examination and investigations. The advantages of this approach are that residents/students learn to generate diagnostic hypotheses, to accumulate data efficiently, and to interpret findings as they arise, as opposed to the inefficient method of collecting data in a stereotypical fashion and analyzing it once the clinical encounter is complete.

Some might argue that with the ready availability of advanced technology there is less need to emphasize the clinical method. Certainly technology has played a remarkable role in permitting more accurate neurological diagnoses. In this study the technology clarified 14 [10] (9) diagnostic errors made by the junior residents, [senior residents], and (staff). Technology is not, however, a panacea for our diagnostic difficulties, since it was nondiagnostic in 46 of the 86 patients whose initial diagnoses were unknown. Although negative laboratory studies are sometimes important for supporting certain diagnoses (see Table 1), in this study the diagnostic burden still fell on the clinician in more than 50% of cases.

A corollary of these results is that as clinicians we should strive to improve our interview and examination skills, clinical knowledge, and reasoning abilities because they remain our most powerful diagnostic tools [25]. As Sir Francis Walshe [26] wrote in 1933, "There is no mechanical substitute for the use of a trained intelligence . . . . The truth is that it is the clinician's business to be the master of all available

weapons, and to know the use and the proper occasions of each but not to be the slave of any one of them."

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