The need to build a strong foundation: a return to the basics

Introduction

Recent years have seen considerable advances in diagnostic investigations. However, there is often misuse of these techniques and resources, and this is reflected in persistently high rates of misdiagnosis, iatrogenic illness and rising health-care costs.

This article discusses three cases that demonstrate clinical errors resulting from a neglect of basic bedside skills and an over-reliance on diagnostic test results. It is hoped that these cases serve to demonstrate that detailed history taking and examination remain the most important diagnostic tools. The escalating demands of acute medical care, the increasing complexity of hospital medicine caseloads and the expanding range of diagnostic investigations available to the clinician (with their attendant cost and potential harm) mean that these skills are more vital now than ever.

The authors hope that Modernising Medical Careers and the new foundation training programme will be viewed as an opportunity to improve basic clinical skills and reinvigorate the practice of bedside teaching.

Case 1

A 45-year-old woman presented to casualty with left-sided weakness and ataxia. She had no previous medical history and was taking no medication. She was a non-smoker, physically active and worked in a local nursery. Neurological examination showed decreased power in the left and right arm with increased reflexes and a positive Babinski sign on the left. There were no other abnormal findings and routine blood tests were normal

A computed tomogram (CT) scan was arranged. This showed patchy low density in the peri-ventricular white matter of both cerebral hemispheres and a prominent low density area in the right deep white matter (*Table 1*).

A diagnosis of ischaemic stroke was made and the patient was commenced on aspirin, an angiotensin-converting enzyme (ACE) inhibitor and a statin. She underwent a rehabilitation programme and was discharged with lifestyle advice.

The patient represented 6 months later with an extension of her left-sided weakness. Detailed history taking revealed that she had complained, 6 years ago, of diplopia and nystagmus at which time a provisional diagnosis of depression had been made. She also complained of intermittent gait ataxia, vertigo, dysarthria and visual loss over several years.

Fundoscopy on this admission revealed optic neuritis. She had gross movement ataxia with evidence of nystagmus and left-sided upper motor neurone signs. A clinical suspicion of demyelination was made. This was confirmed on magnetic resonance imaging (MRI) (*Table 1*). Visual evoked responses were abnormal and there were oligoclonal bands on CSF examination. A diagnosis of relapsing-remitting multiple sclerosis was made and she was treated with pulsed methyl-prednisolone and interferon.

Dr SA Summers *is Specialist Registrar Nephrology and General Internal Medicine, Royal London and St. Bartholomew's Hospitals, London E1 1BB,* **Dr C Laing** *is Specialist Registrar Nephrology, Critical Care and General Internal Medicine, Royal Free Hospital, London,* **Dr PA Kelly** *is Consultant Cardiologist, Southend General Hospital and The London Chest Hospital, London, and* **Dr D Marshall** *is Specialist Registrar Cardiology and General Internal Medicine, Southend General Hospital, Southend-on-Sea, Essex*

Correspondence to: Dr SA Summers

Table 1. Radiology reports for case 1

CT scan	There is patchy low density seen in the deep/peri-ventricular white matter of both cerebral hemispheres. There is a fairly prominent low density area seen in the right deep white matter which could represent a recent watershed infarct. No other abnormalities detected
MRI scan	There are fairly well-defined oval high signal lesions seen in a peri-ventricular location, the largest of the confluent lesions seen in the right parietal lobe, there are also innumerable scattered small high signal foci seen in the white matter tracts of both cerebral hemispheres including sub-cortically. The larger peri-ventricular lesion, however, has a relatively central low density and a mildly hyperintense rim on the T1w image
	Following contrast there is no definite enhancement noted. There is a single lesion identified in the left middle cerebellar peduncle. Both lateral and third ventricles are minimally prominent and magnetic resonance appearances are suggestive of a demyelinating process such as multiple sclerosis. The magnetic resonance appearances do not suggest vascular aetiology

CT = computed tomogram; MRI = magnetic resonance imaging

Case 2

A 73-year-old woman seen in the outpatient clinic complained of malaise and dyspnoea. Her exercise tolerance had decreased significantly over the last 6 months, to the extent that she struggled to walk up three flights of stairs. Her previous medical history included diabetes mellitus and mild hypertension. She was a non-smoker.

Examination revealed a mild elevation in blood pressure (150/90 mmHg) and a regular heart rate (80 beats/min). Further clinical examination revealed only a 'systolic murmur'.

The patient was reviewed 4 months later. Her dyspnoea had worsened to the extent that she struggled to walk up one flight of stairs. She was also noted to be in atrial fibrillation, clinically rate controlled with a blood pressure recorded at 130/70 mmHg. The patient was referred to an anticoagulation clinic and an echocardiogram was booked to investigate the 'systolic murmur'. A follow-up appointment was booked for 3 months.

When seen 3 months later the patient had deteriorated clinically to the extent that she could only walk 20 yards on the flat. She had lost weight and her mobility was poor. Unfortunately, she had not yet had her echocardiogram and was referred to a cardiology clinic.

When seen 4 months later in cardiology clinic she was noted to have severe paroxysmal nocturnal dyspnoea and was now housebound. Further clinical examination revealed tachycardia at 120 beats/min and a slow-rising pulse. The measured blood pressure was 90/60 mmHg. She was admitted and underwent emergency echocardiography. This confirmed a diagnosis of severe aortic stenosis with a dilated, poorly contracting left ventricle. She was deemed unfit to undergo cardiac surgery. She was treated medically and counselled regarding her poor prognosis.

Case 3

A 35-year-old man presented to his local accident and emergency department following a grand mal convulsion. He had a prolonged period of drowsiness following the seizure and was sedated and intubated for an emergency CT scan of the brain. This showed evidence of subarachnoid haemorrhage.

He was loaded with phenytoin and commenced on nasogastric nimodipine. He was then transferred to the regional neurosurgical intensive care unit and was listed for urgent cerebral angiography. After 12 hours his sedation was stopped to assess his neurological status. His Glasgow Coma Scale remained significantly reduced at 7/15 and, following a period where he appeared grossly encephalopathic, he had a further grand mal convulsion and required further sedation.

His CT scans were reviewed. It was felt that the small amount of subarachnoid blood present would not account for the severity of his illness. Further information was obtained from his partner. She gave a clear history of progressive headaches and vomiting over several days before his presentation. He had become confused 24 hours before his seizure.

Further diagnoses were considered, including viral encephalitis, bacterial meningitis, cerebral venous thrombosis and cerebral vasculitis. An urgent MRI scan and magnetic resonance venography of the brain demonstrated thrombosis of the entire cerebral venous system. His supratentorial venous drainage was being achieved entirely through facial collaterals.

It was felt that extensive cerebral venous thrombosis was the cause of his illness and the small amount of subarachnoid blood was secondary to a minor head injury sustained during his seizures. He was commenced on intravenous heparin, eventually extubated and made a full recovery.

Discussion

In case 1 an inadequate history failed to determine that there had been significant previous symptomatology with probably multiple neurological lesions dissociated in time and location over several years. This was highly suggestive of demyelination. It is also highly likely that significant neurological examination findings were not detected. In practice there is often a tendency to frame the clinical assessment around whether a test is indicated on the mistaken assumption that once performed the test (in this case CT brain) will be diagnostic. Neurological signs such as internuclear ophthalmoplegia or optic atrophy are not only missed but are often never sought in the first place. Without the complete clinical information the CT findings were interpreted as showing ischaemic stroke and treatment (steroids and interferon) was delayed.

Case 2 demonstrates how an inadequate clinical assessment may have led to a poor outcome for this patient. The aortic stenosis was diagnosed late and the severity of the lesion was not appreciated. As a consequence by the time echocardiography was performed curative surgery was no longer possible as a result of a loss of general condition and poor cardiac function. Echocardiography services consistently have long waiting lists; a survey of four neighbouring hospitals showed the average waiting time for routine echocardiography to be 5 months.

Cardiovascular examination skills (developed in the pre-echocardiography days) are still invaluable. An examination cannot be limited to merely determining that a systolic murmur is present. The pulse character and volume, cardiac palpation, and the site, character and radiation of the murmur should be determined. Not only can proper clinical assessment determine whether a valvular or other cardiac abnormality is likely to be present (and hence the appropriateness of echocardiography) but can determine the severity of the lesion and the urgency of the investigation.

In case 3 a decision was made that a CT brain was indicated on the basis of a new onset of epilepsy. When this was reported to show a small amount of subarachnoid blood this was assumed to be the primary event. In fact there was a clear history of progressive headaches over several days with associated confusion, which would be highly atypical for subarachnoid haemorrhage. Furthermore the severity of the illness was out of proportion to the relatively minor amount of haemorrhage which was present. This should have led the admitting team to seek out a different primary diagnosis. The failure to do so resulted in inappropriate treatment, an unnecessary and potentially hazardous inter-hospital transfer to a neurosurgical unit and a delay in appropriate investigation and management.

The further fallibility of relying on tests was demonstrated in a publication looking at the routine use of serum troponin when assessing chest pain. In the publication Burness et al (2005) discuss the implications of an erroneous diagnosis of myocardial infarction for the patient and for the health services.

British medicine has always been dependant on thorough history taking and good clinical examination. This is in contrast to other developed countries where health-care economics are more liberal and greater onus is placed on diagnostic investigation. One of the limitations for doctors in the UK is that a lower per capita expenditure on health care in Britain compared to other countries means that there are often waiting lists for investigations (Anderson et al, 2003).

Increasing clinical and research commitments among senior doctors has reduced the amount of individual attention afforded students. Although university trusts are rarely shy about publicizing their teaching hospital status, basic medical education is rarely a priority within busy clinical services. Bedside teaching, being time consuming, has declined in particular, and this has negatively affected clinical skills development. Research continues to document serious deficiencies in clinical skills

among students and junior doctors (Holmboe, 2004).

Previously medical teams admitted patients with several grades of cover. This provided opportunities for students and junior doctors to undertake the initial assessment of new cases and have their findings reviewed and discussed by more senior members of the team. The imperatives of the European Working Time Directive, and a drive to cut salary costs and waiting time targets have resulted in most admission duties being covered by a skeleton crew of middle-grade doctors. While this may be satisfactory for hospital management and individual patients in the short term, it risks a long-term decline in the adequacy of clinical training and the competency of the senior doctors of the future.

There is consensus that true adherence to basic clinical teaching and personal values may avoid significant pitfalls in patient care (Schattner and Fletcher, 2004). This has been addressed in the USA by the development of an integrated developmental curriculum that emphasizes bedside teaching and role modelling, focusing on enhancing fundamental clinical skills and professionalism (Goldstein et al, 2005).

Foundation training, recently introduced in the UK, offers a chance to include comprehensive curricula with goals and objectives, problem-based learning, a renewed emphasis on physical diagnosis, evidence-based medicine, patient-centred interviewing, ambulatory education, computer-based learning and new methods of assessment (Jotkowitz, 2004). The Royal College of Physicians has produced *Laying The Foundations for Good Medical Practice* (Mann et al, 2003), to help junior doctors develop generic skills and knowledge required for good medical practice.

Models developed in the USA, including direct observation, mini clinical evaluation exercise, 360° appraisal and directly observed procedural skills, have been adopted for training purposes in the UK. These tools will be used to evaluate the new trainees (Davies et al, 2005) and ensure they develop predetermined skills.

KEY POINTS

- Recent advances in radiology and pathological services have advanced diagnostic accuracy in clinical medicine.
- Over-reliance on these tests can lead to incomplete or incorrect diagnoses.
- Clinical skills including detailed history taking and thorough examination are essential in reaching a diagnosis.
- There has been a decline recently in clinical skills, probably related to less emphasis on teaching these skills.
- Bedside teaching from senior doctors is essential in the development of good clinical skills for junior doctors in training.

Such changes seek to provide systems to counteract the decline of the more informal apprenticeship model that served medicine well in the past. The authors welcome such change; however, more is needed than just paying lip service to educational theory and methods of assessment imported from the commercial sector to rescue declining clinical competence. The direct acquisition of clinical skills through interaction with patients at the bedside must be the bedrock of training. This requires time, patience and accommodation from not only clinical tutors but also health-care organizations as a whole. Students and junior doctors must be allowed to participate intensively in the assessment of patients in a supervised fashion. Above all, bedside teaching must be viewed as the starting point for improved medical education. **BJHM**

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