Letter from... Chicago

Rounds

George Dunea

From time to time on rounds I discuss with the new students and residents the principles of history taking. I talk about the great Sir William Osler, hero of all bedside pedagogues, who urged his students to “listen to the patient, he is telling you the diagnosis.” I mention Sir Thomas Lewis, also great though lacking name recognition nowadays, who expected his interns to know how to examine a patient but not as yet to have enough experience to obtain a good history. Then I refer to the late Michael Balint, the psychiatrist, who wrote, “If you ask questions you will get answers but precious little else.”

Next I try to bridge the gap between theory and practice, for instructors must be men of the world to command the residents’ respect. I concede that life is too short for mere listening and grunting encouragingly—except for the psychoanalyst. Betraying my age, I refer to Paul Woods’s introduction to his 1955 book, in which he recommends the skilful use of leading questions, such as “Does the pain tingle?” I also mention Sam Levine’s technique of asking the opposite of what he expected to hear—such as “Is your chest pain worse when you rest?” or “Is your joint stiffness worse in the evening?” Ideally, I say, there should be only one history, not seven specialists’ subjective perspectives like in the Japanese tale of Rashomon.

Drawing from Alvan Feinstein’s observations but leaving out the Greek, I make a distinction between the main problem and what took the patient to the doctor—what he called the isotropic stimulus. In the manner of Gibbon I suggest that the failure of the once popular problem oriented record might be due not to who gave it but to whom it was given. I ask instead that progress notes be short and to the point, emphasising assessment of the clinical state and plans for the future. I warn against trying to write down the history in its final form while taking it to avoid breaking the flow with interruptions. I advise the students to construct complex histories not so much along symptoms as along the sign posts that patients are likely to remember: being admitted to a hospital bed, cut by a surgeon, or seen in a doctor’s office. I talk about the need to sometimes go into great detail, referring to the once obligatory exercise of writing 15 page case histories and I quote Voltaire’s dictum about doctors giving medicines they know nothing about to patients they know nothing about. At this juncture I stop short of items such as empathy, sympathy, putting yourself in the patient’s shoes—which is just as well because the students are getting restless and anxious to get to the noon lecture on the prostaglandin cascade.

We meet again at 2 pm for rounds. The intern is puzzled at my insistence on hearing the history at the bedside. Their previous attending physician, he says, first wrote his notes in the conference room while the interns presented their findings—a compromise, I reflect, between Feinstein’s abstract and veterinary forms of rounds. The first patient is an anxious 66 year old woman with oxygen prongs up her nose, being “stepped down” after three days of ruling out myocardial infarction in the intensive care unit. She is “an unstable angina,” explains the intern; she also has hypertension and congestive heart failure.

When I ask how long she has had hypertension he turns to the woman and asks “How long have you had high blood pressure?” When I ask about previous hospital admissions he says that she has had a “cardiac cath” and that he will send off for the records. Trying to practise what was preached in the morning, we learn that she spent two weeks in intensive care 20 years ago for an alleged heart attack after her dentist discovered high blood pressure. Suspicious and somewhat peeved at first at being asked so many details, she remembers having had a cardiac catheterisation in 1980 and being told she had a funny valve. Chest pains began after a car accident in 1984, usually starting in the middle of the back and radiating round both sides to the front. Her right shoulder also hurts. It hurts to turn over in bed, she says.
The student intervenes, protesting that this must be angina because she gets the pain after walking eight blocks and after lifting weights. By now quite relaxed, the woman volunteers that she brings up mouthfuls of acid and often feels something bubbling in her stomach. A doctor once told her that she had a hole in the stomach. "You never told me that," says the student, quite vexed. Visibly pleased, she now says that she had suffered from pains all her life, "Even when I was a little girl my legs hurt so much that my mother used to carry me around."

One pain or two?

The resident grudgingly concedes that this is most unlikely to be angina. She has a tender spot over her dorsal spine at the point where the pain originates. The heart is normal on auscultation, on x-ray examination, on electrocardiography, and on echocardiography, but the rate is 50 from atenolol. The sedimentation rate is normal. We decide to send her home and watch her for a while on analgesics and perhaps on H2 blockers. We agree that we do not know the exact diagnosis; it could be spondylosis, it could be oesophageal. We do not think that she has porphyria, or myeloma, or an aortic aneurysm. The intern wonders if she shouldn't "work up" the spine and the oesophagus while she is here. The students are puzzled—does she have one pain or two different ones—and the empirical approach makes them uncomfortable. The resident agrees that we can always do a barium swallow and a spine x-ray examination later.

As we move on I refrain from starting a discussion on the failings of our health system. Why, might I have asked, does this woman not have a primary care physician or general practitioner? Why must she always be seen by new doctors, invasive cardiologists, hurried emergency room physicians, angiography oriented intensivists, all too busy to engage in old fashioned symptom analysis?

The next patient is 75 years old and has precordial pain relieved by nitroglycerin. The cardiologist wants to do a stress test—presumably looking for a correctable lesion. Next we see a young patient with cirrhosis who has been drinking for 15 years and has a belly like in a full-term pregnancy—a candidate for the rapid paracentesis protocol. Two pneumonias, a stroke, and an addict with fever come next. Then we sit down for an extended presentation on strategies for stratifying patients with angina. It sounds simple. You operate those with intractable pain and leave alone those who don't seem to have angina, such as the lady we just saw. The middle group get a stress test, also an ejection fraction determination. If this is more than 50% they should take nitroglycerin, etc; if it is under they may need further tests, for it is in this group with impaired ventricular function that surgery may prolong life. The reference comes from the very journal founded by Sir William Osler.1

Then we see the patients with AIDS. Alas, we have all become very experienced. If the enzyme linked immunosorbent assay is positive you confirm the result with a western blotting. If the CD4 cell count is under 500 you consider zidovudine. Diarrhoea, usually caused by cryptosporidium, is treated symptomatically with loperamide because nothing else works. Pneumonias are usually caused by pneumocystis and require co-trimoxazole or pentamidine. For cytomegalovirus retinitis you give ganciclovir, which is quite toxic; for herpes use acyclovir. Every patient seems to have candida esophagitis and needs ketoconazole, but for histoplasmosis you double the dose or try fluconazole. When the kidneys fail you start dialysis. For Mycobacterium kansasi you give three antituberculous drugs and ciprofloxacin. But Mycobacterium avium-intracellulare usually requires a bone marrow biopsy for diagnosis and five or six drugs to treat it: ciprofloxacin, ethambutol, amikacin, rifampicin, clofazimine, and perhaps rifabutin (ansamycin). It is all very simple, truly a primary care disease.2

---