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POSTMORTEMS: AN EDINBURGH PERSPECTIVE

The editorial in the last issue (Horowitz RE. Autopsies: an exercise in futility? *J R Coll Physicians Edinb* 2009; 39:194–5) was provocative, valuable and of immediate concern. In addition it is worth recording the researches carried out in Edinburgh by HM Cameron, at that time a senior lecturer in the University Department of Pathology. He and his colleagues, McGoogan, Clarke, Wilson and Watson, provided an exhaustive and thorough enquiry on the role of the autopsy; an enquiry noteworthy for its close collaboration with consultants from four medical and two surgical units in Edinburgh.

In one of their series[1] of 154 autopsies they found that 15% of main diagnoses were not confirmed and 42% of causes of death were not confirmed. These results were deemed by pathologists and clinicians to be clinically significant. Consequently they were not convinced that the clinician was always well placed to judge the potential value of an autopsy. In a prospective study[2] of 1,152 hospital autopsies, there were many examples of overdiagnosis and underdiagnosis, all encountered in a routine hospital autopsy service. They also noted that death certificates were unreliable as a source of diagnostic data, the major diagnosis being wrong in about a quarter of cases.[3]

An editorial in the *Journal of the Royal Society of Medicine* questioned if we really can accept that modern clinical techniques are so definite as to be regarded as supporting the demise of the autopsy.[4]

Although there are many reasons for the decline of the autopsy, one factor is the sordid and depressing environment of many postmortem facilities. A significant rise of interest in Edinburgh occurred when the late Sir Alastair Currie improved accommodation, introducing air-conditioning, audio-visual aids and closed circuit television; specimens were dissected by junior staff and comments made by experienced consultants.

The idea of Dr Horowitz of a central or regional facility divorced from the hospital pathologist who then would be freed from the chore of the autopsy is indeed radical. It seems to me like the creation of a ghetto of the dead. The autopsy and the diagnostic biopsy are intrinsically linked, each supporting the other. The quality of both is improved by combination. After all, what is clinical medicine but pathology on the wing?

Professor Angus E Stuart
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Author's reply

I thank Professor Stuart for his comments. The discrepancies between clinical and autopsy diagnoses and the inaccuracies of death certification as cited by Cameron et al. are well known[1] and bear reiteration.

The centralisation of autopsies may be a practical approach to a thorny problem. Ideally, every hospital would have a decedent affairs programme which assures a high autopsy rate; there would be a modern autopsy facility staffed by expert prosectors, supervised by knowledgeable and interested pathologists, all paid for by 'the healthcare system'. The attending clinician would attend the autopsy and gain immediate knowledge and benefit. In most American hospitals that ideal is simply not available.

The regionalisation of autopsies in a single facility under the auspices of a medical school or medical examiner's (coroner's) office would optimise the utilisation of scarce resources. One university medical centre in the United States has already done this: it contracts with 23 community hospitals, both locally and up to 200 miles away. The community hospitals' pathologists do not do autopsies, the community hospitals do not need to invest in space and personnel and these savings are passed on to the university as payment for performing the autopsy. The university thus gets sufficient numbers of autopsies for medical student and resident training, the autopsies are performed by people who are skilled and interested in doing them and there is sufficient material for research and publication, allowing the university to attract pathologists dedicated to the autopsy. With modern information and imaging systems, autopsy reporting is prompt and the distances are no problem.

Is such centralisation ideal? No! But is it a potential viable alternative? Yes! Is it a 'ghetto of the dead'? Perhaps, but with a marquee that reads: *Hic locus est ubi mors gaudet succurrere vitae* (This is the place where the dead give sustenance to the living).

Professor RE Horowitz

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OTHER CORRELATES OF THE DECLINE IN AUTOPSY RATE

The decline in the autopsy rate (Horowitz RE. Autopsies: an exercise in futility? *J R Coll Physicians Edinb* 2009; 39:194–5) might well be a manifestation of the increasing reliance on technological advances in imaging so as to arrive at a diagnosis. Although imaging techniques have led to huge improvements in the quality of healthcare, the paradox is that there has not been a corresponding improvement in the quality of the content of the undergraduate medical curriculum because the decline in autopsy rate has resulted in an atrophy of the skills of clinico-pathological correlation, the latter skills being ones traditionally taught in tandem with skills in eliciting physical signs.

Even bedside teaching of clinical skills is 'unfortunately in steady decline',[1] and it has now also become a 'badge of honour' to arrive at a diagnosis without the benefit of performing a clinical examination.[2] The latter development is a fortunate one for the National Health Service because in the new era of nurse-led consultations (a third of consultations in general practice in England in 2008 were with nurses[3]) it is much less labour-intensive to bypass the clinical examination altogether when teaching diagnostic skills to prospective nurse practitioners. Only in non-Western rural societies such as that in the Eastern Cape province in South Africa, where I practised medicine for seven years, was there an expectation among members of the public that the ingredients of the 'complete package' of a medical consultation should comprise, in this order, history taking, clinical examination, diagnosis (articulated in terms which the patient could understand) and treatment. Fortunately, highly sophisticated patients in the West have no such expectations when they attend clinics in primary as well as in secondary and tertiary care, where stripping down to the waist to have a full clinical examination has become increasingly anachronistic, thanks to the advent of high-tech imaging.

The 'downside' is the inappropriate use of laboratory tests and imaging, as shown in a study where 'selecting tests based on history and [clinical] examination and prioritising less expensive and higher yield tests' would have been more cost-effective.[4] In that study postural blood pressure recording, performed in only 38% of episodes, had a higher yield with respect to affecting diagnosis and management and determining aetiology of the syncopal episode than head computed tomography scans, which were performed in as many as 63% of instances.[4] The wastefulness (in this era of economic stringency) of bypassing the clinical examination is compounded by the scant respect with which the results of high-tech tests are themselves treated, exemplified by the traditional disorder prevailing in the health record, where 'anything' is filed 'anywhere' and 'anyhow',[5] and no audit is made either of the quality and content of record-keeping or of the quality and content of discharge summaries and clinic letters.

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THE ROLE OF VENOMETERS IN THE DIAGNOSIS OF DEEP VEIN THROMBOSIS

I read with interest the recent paper entitled Venous thromboembolism: the role of the clinician (N Curry, D Keeling. *J R Coll Physicians Edinb* 2009; 39:243–6). It does not highlight the role of the venometer in the diagnosis of venous thromboembolism.

Clinical diagnosis for deep vein thrombosis (DVT) is notoriously unreliable because the external symptoms of a DVT are so equivocal. The venometer is a simple bedside non-invasive test that uses automated strain-gauge plethysmography to detect DVT.[1,2] Although there have been no randomised controlled trials to support the role of venometers, they play a major role in the safe screening of patients and are used successfully in the DVT protocols of many UK hospitals, providing an efficient and a cost-effective way to manage DVT.

The definitive characteristic of a DVT is that it blocks normal blood flow. This feature is exploited by the venometer. It determines the likelihood of a thrombosis by objectively measuring the rate at which blood drains under gravity from the patient's calf. The venometer automatically calculates the likelihood of a DVT based on the measurement taken by the sensor on the calf, while a blood pressure cuff is inflated on the thigh during this brief test. The results, either positive or negative, can help in the diagnosis of venous thromboembolism.

A portable device operated by a trained nurse or technician, the venometer is suitable for use in medical admissions units, community health centres or patients' homes. The test is quick and simple, taking up to 15 minutes for the complete interpretation. The venometer software is extremely user-friendly and guides the operator precisely through every step in the test sequence.

Venometers decrease the risk and cost of unnecessary anticoagulation and reduce waiting times and the

number of ultrasound tests, thus providing 24-hour DVT care in the NHS.

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Authors' reply

We agree that there are little data on the venometer. A recent *Health Technology Assessment* concluded that plethysmography techniques appear to have a limited role in the diagnosis of clinically suspected DVT.[1] The authors estimated that strain-gauge plethysmography has a sensitivity and specificity of 83% and 81% and that there was some evidence that diagnostic performance depended on the prevalence of DVT in the cohort and the setting for recruitment. They recommended the evaluation of the role of plethysmography, its interaction with other diagnostic tests, outcome of low-risk patients with negative plethysmography and measurement of the costs of providing plethysmography as areas for research but concluded that diagnostic algorithms based on a combination of Wells score, D-dimer and ultrasound (with repeat if negative) are feasible at most UK hospitals and are among the most cost-effective.

Dr David Keeling, Dr Nikki Curry

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JOHN FOTHERGILL MISIDENTIFIED

I read with interest the article by MJ Eadie on men of science and the migraine aura (Hubert Airy, contemporary men of science and the migraine aura. *J R Coll Physicians Edinb* 2009; 39:263–7). Unfortunately there is a serious misidentification of the Dr John Fothergill whose paper on the subject is referred to in reference 10. There was no such individual as Dr John Fothergill of Manchester in 1784. The individual who wrote about the migraine aura was Dr John Fothergill, the famous Quaker physician of London. An Edinburgh graduate of 1736, he was the first graduate of the Edinburgh Medical School to be licensed by the Royal College of Physicians of London, wrote a classic book on the malignant sore throat and was the first to recognise the relationship between angina pectoris and disease of the coronary arteries. He was a close friend and physician to Benjamin Franklin. His paper on the sick headache was read to a Society of Physicians in London, of which he was then President, on 14 December 1778. After Fothergill's death in 1780, his pupil John Coakley Lettsom wished to publish this paper among his works but was refused permission by his executors. He therefore had to wait until it was published in the sixth volume of the *Medical Inquiries and Observations* in 1784, four years after Fothergill's death. Fothergill was one of the most distinguished graduates of the early Edinburgh Medical School. He was also a Fellow of the Royal Society and a member of the American Philosophical Society as well as being a strong supporter of the American colonists in their struggle for independence.

Sir Christopher Booth
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Author's reply

I am very grateful to Sir Christopher Booth for detecting my mistake in stating that Fothergill's city of residence was Manchester rather than London, and apologise to any readers who may have been misled by my error. As published in *The Journal*, the reference to Fothergill's account of his migraine aura is correct, but Sir Christopher's letter has explained the circumstances of its delayed appearance in print.

Professor Mervyn Eadie

INSULIN PEN DEVICES

In their recent article (Insulin delivery devices. *J R Coll Physicians Edinb* 2009; 39:146–50) Dr Graveling and Dr McIntyre state that insulin pen devices were developed by Dr John Ireland in 1981.

The first pen device was actually designed jointly by John Paton of the Department of Clinical Physics and Bioengineering (DCP&B) in Glasgow, the late Dr John Ireland and myself.[1]

After the initial trials with the prototype pen, further devices were made by the DCP&B, and then Hypoguard was licensed to produce the first commercial insulin pen with the royalties being paid to the Greater Glasgow Health Board (GGHB), which held the patent on our behalf.

Following this agreement, Michael Forsyth and other MPs tabled an Early Day Motion in the Commons congratulating John Paton, John Ireland and myself on our invention and on this example of co-operation between the NHS and private industry.[2] Later, the patent was sold to Novo by GGHB. None of the three inventors received any financial reward.

This letter is not in any way intended to minimise the contribution made by Dr John Ireland to this device,

which is widely used today, but simply to set the record straight. I hope that this information will be of interest to readers of *The Journal.*

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PAIN PALLIATION FOR BONE METASTASES

This refers to the symposium report 'Palliative care in your hospital' (Buchanan D. *J R Coll Physicians Edin* 2009; 39:252–6). In his discussion of new interventions Dr Edwards makes no reference to an important strategy for pain palliation due to skeletal metastases. I am referring to systemic radiotracer therapy. It may not be considered a 'new' way as the first therapy was performed in 1941.[1] There has been significant progress in systemic radiotracer therapy since then with the focus on developing specific and less toxic compounds. While phosphorus-32 has been in use for many decades and revisited,[2] newer compounds such as Sr89,[3] Sm153 EDTMP,[4] Re186 HEDP and Sn117m pentetate[5] have been successfully employed for pain palliation.

Targeted radiotracer therapies can ameliorate pain in 40–80% patients. Therapy is done on an outpatient basis. Onset of relief may take five days. The patient can be pain-free for about three months. Repeat therapies are indicated in patients who benefit from the first dose. Improved performance and quality of life has been reported in several studies. Patients with osteoblastic lesions show good response. Bone marrow suppression is a known toxicity but is usually mild and reversible in the majority. The cost of newer compounds limits frequent use. However, it is worthwhile when one takes into consideration the cost of alternate intervention and the potential benefit to the patient.

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MORE ON THE ASSOCIATION OF GASTRIC AND COLONIC NEOPLASMS

The report of the association of synchronous gastric polyp and colon cancer in the last *Journal* issue (Cooper H, Dhar A. Synchronous gastric polyp and colonic cancer. *J R Coll Physicians Edinb* 2009; 39:218–20) has, as its corollary, the occurrence of metastases to the stomach from primary adenocarcinoma of the colon.[1] In the latter series there were five such cases, one identified by endoscopy, and four by autopsy. The five cases comprised 7% of 67 examples of primary tumours metastatic to the stomach.[1] The reverse scenario is exemplified by primary gastric adenocarcinoma metastasising to the colon. In the latter instance, the metastases presented as multiple colonic polyps.[2] Apart from the two-way neoplastic traffic between stomach and colon,[1–2] colorectal cancer can also metastasise to the small bowel.[3]

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Author's reply

We thank Dr Jolobe for his comments. The association of colorectal metastasis from gastric cancer has been well known for many years and the occurrence of gastric metastasis from colonic cancer is less common. But the metastatic spread of dysplastic cells from polyps in one site to the other site has not been described. Further, to attribute the development of a frank cancer at one site to the metastatic seeding of dysplastic cells from a polyp at the other site, and subsequent progression to cancer, has also not been described. Most occurrences of cancer at one site and polyp at the other have therefore to be regarded as synchronous rather than metastatic.

Dr Anjan Dhar