Letters to the Editor

"Medical audit of rectal biopsy diagnosis of inflammatory bowel disease" is not medical audit

Dr Morson kindly sent me a pre-publication copy of this article. It is an important paper, in which the authors studied the prognostic reliability of biopsies and the biological behaviour of colorectal disease. This is not remotely an approach to medical audit as suggested in the last paragraph.

In chemical pathology we already go three stages further towards medical audit: (a) in (internal) quality control a laboratory repeatedly analyses the same specimen to see if it gets the same answer; (b) in (external) quality assurance the same specimen is analysed by many different laboratories, and comparison is made of their results; (c) an advisory panel is available to give confidential help to laboratories that are persistently poor performers. There are similar procedures in haematology and in medical microbiology.

Histopathology has barely started on such assessments, and I think the authors missed an opportunity. Why did they not (i) have their staff look at the same specimens several times, without knowing that these were the same specimens? (ii) send the specimens to a number of unselected different histopathologists, and compare their reports? These two procedures could have been a beginning of medical audit applied to histopathology.

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References


Drs Morson and Frei reply as follows:

Professor Baron's comment fails to recognise the essential difference between histopathology and other branches of laboratory medicine particularly chemical pathology. Identical aliquots, objective instrumental evaluation, mean and standard deviations do not exist for pieces of tissue as they do for tubes of blood. We studied the consistency of written reports, not the specimen itself. In other words, we examined the implications of reporting rectal biopsies in inflammatory bowel disease as they were documented in the departmental records. It was a retrospective research into the accuracy of reporting in the hope that further study will lead to improved performance. Professor Baron proposes an altogether different study involving review of specimens rather than records.

But what constitutes medical audit? We are not qualified to disagree with Professor Baron's definition of how audit should be conducted for chemical pathology, but he must accept that it is at present defined differently to suit the varying circumstances of medical practice. The general objectives of medical audit are to investigate standards and improve the performance of individual practitioners. In the first place, it is essentially a comparison of what has happened with what ought to happen. But medical audit is not complete unless, as a second stage, it leads to an appropriate change for the better.

We maintain that our article is essentially a first stage audit and that hopefully further research will lead to higher accuracy rates for rectal biopsy diagnosis in non-specific inflammatory bowel disease. In histopathology audit can be prospective but more often retrospective, as in clinical medicine, and relies on a degree of uniformity in departmental record systems. It is little different in our opinion from what is generally included under the heading of 'clinical research' except that medical audit should be a continuing and organised curiosity into how we are performing on both an intradepartmental and interdepartmental basis. A good example of the latter was reported recently and subsequently discussed in an editorial in the British Medical Journal.

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Possible diurnal variation in 2,3-diphosphoglyceric acid

The importance of 2,3-diphosphoglyceric acid (2,3-DPG) as a regulator of overall oxygen affinity of haemoglobin (Hb) in human erythrocytes is well established, and it is an important mechanism by which the body controls the oxygen supply to the tissues.

The purpose of the present study was to investigate whether 2,3-DPG concentrations show any diurnal variation which would need to be considered in future work; no information on this point could be found.

SUBJECTS AND METHODS

Five apparently healthy, non-smoking male medical students volunteered to take part in this study. Blood samples were withdrawn by venepuncture into heparinised vacuum tubes at 08.00, 12.00, 17.00 and 00.00 hours.

Samples were assayed for 2,3-DPG according to the ultraviolet enzymatic method of Sigma Chemical Co; the method uses 2-phosphoglycolic acid as a stimulator, and is similar to that of Lowry et al, but the reaction is monitored spectrophotometrically instead of fluorometrically: 3-phosphoglycerate phosphokinase (PGK) and glyceraldehyde-3-phosphate dehydrogenase (GAPD) are coupled to convert 3-phosphoglycerate (3-PGA) to glyceraldehyde-3-phosphate (G-3-P), with simultaneous formation of NAD from NADH. The concentration of 2,3-DPG is proportional to the amount of NADH oxidised; calculations are based on the molar extinction coefficient of NADH.

In this procedure, the test is performed on a protein-free supernatant; this was prepared as quickly as possible, using cold 8% trichloroacetic acid (TCA), and stored frozen until analysed. Samples for each subject were assayed in a single batch;