et al., 1979), base their findings on analytical coefficients of variation of 2% for calcium and of 3% for albumin, which they found at normal serum concentrations. These give a coefficient of variation for their 'adjusted' total calcium concentrations of 2.4% (Sanderson, personal communication). Now most of their data refer to patients whose calcium and albumin levels are far removed from normality, and yet no figures are given for the analytical coefficients of variation at these abnormal levels. One can, therefore, only assume that they have extrapolated their findings obtained in the normal to the abnormal situation without modification—a somewhat dangerous practice I would have thought! However, I feel that this uncertainty at least entitles me to say that the coefficient of variation for their 'adjusted' total calcium determinations in the patients they discuss is 2.4% +.

Now Mitchell (1978) states that 'for diagnostic purposes, calcium requires to be measured in serum with a high degree of precision and accuracy—($\pm 1\%$ is generally considered to be desirable) —...

In my opinion, there is a marked difference between 1% and 2.4% +, which leads me to ask just who is right?

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ness depends on the overall variation in calcium concentration in the groups that are being compared or in the individual in whom a change is being sought and not on the analytical variation alone.

Now, overall variance = biological variance + analytical variance. When calcium is adjusted for albumin there is a reduction in overall variance because there is a reduction in the biological variance which is greater than the increase in analytical variance. In the group of patients studied by Payne *et al.* (1973), the range of values for total calcium was 1.78–2.70 mmol/l whereas after adjustment for albumin the range was reduced to 2.25–2.60 mmol/l. Similarly, in our paper which Mr Sanderson criticises (Payne *et al.*, 1979), we showed that adjustment for albumin reduced the within-patient SD from 0.148 mmol/l to 0.100 mmol/l. We commented in our paper: 'These reductions in variability are the more striking because the values for adjusted calcium concentrations are based on independent single measurements of calcium and albumin, each of which has its own analytical error'.

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1978). The mean value was found to be at the upper limit of the overall reference range for the method. Taking into account variation in enzyme activity in relation to age and sex, 28 patients (14%) were found to have raised enzyme levels. One of these patients had Paget's disease of bone.

More detailed biochemical investigations were carried out in nine of these patients with raised serum alkaline phosphatase activity. The enzyme was estimated in a different laboratory using the SMA 12/60 system (Morgenstern *et al.*, 1965) and was found to be still elevated in eight (the values ranged between 3% and 42% above the upper limit of the reference range; mean rise 21%). Serum alkaline phosphatase isoenzyme fractionation (performed by electrophoresis on both agarose and polyacrylamide gels) showed that the liver component was raised in all eight, and, in addition, one had a small elevation in the bone component. Serum γ -glutamyl transferase (EC 2.3.2.2) was raised in two to about twice the upper limit of the reference range, and three had high-normal or slightly raised results. Only one patient had marginally raised serum transaminases (aspartate transaminase EC 2.6.1.1 and alanine transaminase EC 2.6.1.2), and none had raised serum lactate dehydrogenase (EC 1.1.1.27) or bilirubin. Serum calcium corrected for albumin (Kennedy *et al.*, 1975) was normal in all eight, and serum phosphate was normal in seven and only marginally raised in one.

Our results suggest that ankylosing spondylitis may not be as frequently accompanied by elevated serum total alkaline phosphatase as has been suggested by Kendall *et al.* (1973). Our findings of a raised liver fraction in all specimens from patients with raised total enzyme activity is also at variance with Kendall's finding of predominance of elevation of bone fraction. Only one of our patients had a slightly raised bone fraction in addition to the raised hepatic component. Our findings of raised γ -glutamyl transferase in two patients and border-line results in another three further support the contention that the biochemical abnormalities in these patients are mainly of hepatic rather than of bone origin. Golding (1973) has also stated that raised serum alkaline phosphatase in ankylosing spondylitis is not generally accepted to be the result of bony ankylosis.

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The authors have commented as follows:

Mr Sanderson is worried that the analytical variation of calcium adjusted for albumin is greater than that of calcium alone. This is, of course, inevitable because the analytical variance of adjusted calcium is the sum of the analytical variances of total calcium and of albumin. However, diagnostic usefulness

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Serum alkaline phosphatase in ankylosing spondylitis

As part of a survey of over 200 patients with ankylosing spondylitis attending the Centre for Rheumatic Diseases, Glasgow, the serum alkaline phosphatase (EC 3.1.3.1) activity was estimated. Details of the method have recently been published (Gardner and Scott,

All our patients were taking non-steroidal anti-inflammatory drugs. We are, therefore, clearly unable to exclude drug therapy as a cause of the abnormalities in liver function tests. Clarification of this aspect would require monitoring of the above biochemical parameters in patients not receiving drug therapy.

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Anaphylaxis and systemic mastocytosis

We were interested by the report of what appears to be the first case of fatal anaphylaxis in a patient with systemic mastocytosis (Dodd and Bond, 1979) since we recently observed a similar case.

This 50-year-old man had had urticaria pigmentosa for 20 years and intermittent flushing of the face for four years. The diagnosis of systemic mastocytosis was based on typical bone lesions, invasion

of bone marrow by mature mast cells as shown on biopsy, hepatosplenomegaly, and a leucoerythroblastic blood picture. There was a threefold increase of blood and urine histamine levels.

The patient died shortly after a bee sting. No previous history of insect hypersensitivity was known. He was in circulatory collapse with a low central venous pressure; haemolysis and disseminated intravascular coagulation were documented. As pointed out in the report of Dodd and Bond, a fatal outcome of systemic mastocytosis is rarely related to anaphylaxis. Although the association of systemic mastocytosis and fatal insect hypersensitivity in our patient may be merely coincidental, the dramatic outcome suggests that in some cases an offending agent, such as a bee sting, a drug reaction, or an infection, may precipitate mast cell degranulation and death in systemic mastocytosis.

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Priority test request form

In the February issue of the *Journal of Clinical Pathology* a special form was described for requesting results to be telephoned from the laboratory (Henderson, 1979). This form included the name and telephone number of the requesting doctor only as an optional detail. Telephoning results disrupts the work of a laboratory, and we present data showing that it can also introduce errors. The use of forms that would increase the number of telephoned results might well be a disservice to both the laboratory and the patient.

In 1976 it was the practice in this laboratory to telephone results, on demand, to a number of wards and clinics in the hospital. The technician giving the results would read them in the laboratory, and a nurse or doctor would write them down on pre-printed pads. The recipient of the results was asked to read back the transcribed data before they were agreed to be correct.

We examined 172 sets of haematology results which had been telephoned to either the haematology ward or the intensive care unit in this hospital. These telephoned copies were compared with the result recorded on the back-copy of the appropriate laboratory report. Only 169 telephoned ward copies had the patient's name clearly recorded and only 13 included the patient's case record number (CRN). The CRN is a particularly important identifier when neither the surname nor address can distinguish individuals clearly. One hundred and forty-one telephone copies bore the correct date. There were 12 instances in which the copy of the results recorded at the ward differed from that given on the laboratory report (Table). Some of these differences were trivial but at least five were substantial.

Comparison of telephoned results recorded at the ward with the correct laboratory report

Test	Ward telephone copy	Laboratory report
Haemoglobin (g/dl)	13.4 15.3 10.9	10.8 15.2 10.5
White cell count ($\times 10^9/l$)	17.5 38.0 43.0 6.4 8.9 13.0	7.5 3.6 4.3 6.5 7.4 13.9
Haematocrit (%)	39 31	37.9 32.1
BCR	6.8	1.8

That no harm befell those patients whose results were mistranscribed may have been fortuitous. It was more probably an indication that these results were either not looked at critically or, because they were telephoned, not taken seriously. In either case there was every reason to discontinue the practice of regularly telephoning results. We believe that this has saved the laboratory from wasting time and has protected the patient. In those cases where it is necessary, rather than convenient, for results to be telephoned the likelihood of significant error being introduced must be considered by both the donor and recipient of the data.

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