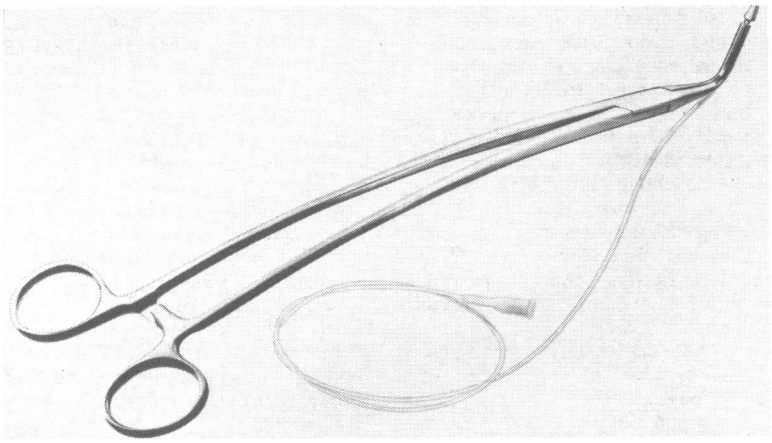

The grooved jaws of the introducing forceps should be placed on the Stoke-on-Trent cannula about 3-4 mm from its expanded end (Fig.). After the cystic duct has been dissected clear of surrounding structures, a ligature is tied round it close to the gall bladder. The duct is incised immediately distal to this ligature, and may have to be dilated gently with a probe or small bougie before cannulation is attempted. Now the expanded end of the cannula is introduced with the forceps, but only the tip of the instrument itself should be inserted into the duct. The cannula is secured in position with a second ligature drawn round the duct, and tightened just beyond the jaws of the forceps. The forceps can then be released, and withdrawn from the duct without fear of dislodging the cannula. The procedure of operative cholangiography is continued as described previously.[1]

The instrument is available from Down Bros. and Mayer and Phelps, Church Path, Mitcham, Surrey. My thanks are due to Mr. A. J. G. Percy, Home Sales Director of Down Bros.

—I am, etc.,

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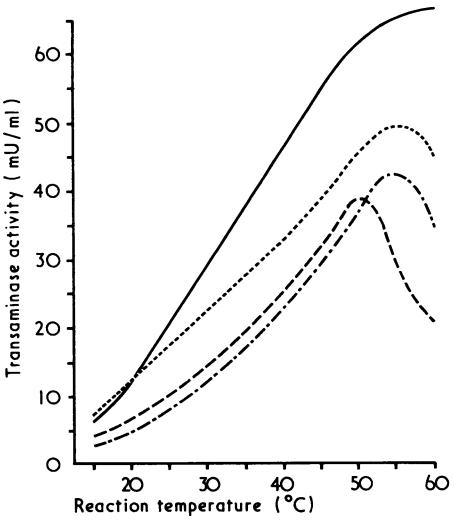
City General Hospital,
Stoke-on-Trent

1 Buchanan, J. McK., *British Medical Journal*, 1969, **1**, 706.

Normal Range for Serum Transaminase

SIR,—We were most distressed to read the letter by Professor D. N. Baron and others (4 September, p. 583), not only because of the misleading information therein but also the overt lack of appreciation of the limited validity of temperature correction factors. King[1] has shown that since the optimal reactant concentrations for serum lactate dehydrogenase vary with temperature and the temperature-activity relationship alters with the optimal conditions, temperature correction factors are only valid over a narrow temperature range.

In the Boehringer Corporation transaminase kits the reactant concentrations have been increased in the "optimized" packs, and the typical effect of this on the temperature-activity relationship is illustrated in the Figure. This clearly demonstrates the greater thermostability of alanine transaminase and the thermal protection given by the increased concentration of substrate in the "optimized" assays. This in turn indicates that while the standard methods for both enzymes have reasonably similar conversion factors up to 40°C those for the "optimized" procedures differ from this and from each other. Although Professor Baron and colleagues do not state their method of conversion it can readily be appreciated that if the factors for the standard assays were applied to the optimized procedures this would result in fictitiously low values for the latter, particularly in the case of aspartate transaminase.



However, we are at a loss to understand why one should wish to convert to 25°C the results of assays performed at 35°C. Like all LKB Reaction Rate Analyzers in Scotland and an increasing number in England our instrument is set to the logical temperature of 37°C, and our normal ranges for both Boehringer standard and optimized transaminase assay were determined[2] and are reported at this temperature. The statement that the procedures of Henry *et al.*[3] are the most widely used and accepted non-kit spectrophotometric assays of the transaminases requires supportive evidence and only confuses matters further since these workers employed the bizarre reaction temperature of 32°C.

Finally, we are perplexed by the upper limits given for the standard "UV test" kits, that is, alanine transaminase 17 mU/ml and aspartate transaminase 12·5 mU/ml at 25°C. This is contrary to our own experience and contradicts the findings of most other workers, which indicate that these upper limits are either the same or that that of aspartate transaminase is marginally higher.

—We are, etc.,

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1 King, J., *Clinical Biochemistry*, 1967, **1**, 42.
2 Griffen, D., *Journal of Medical Laboratory Technology*, (in press).
3 Henry, R. J., Chiamori, N., Golub, O. J., and Berkman, S., *American Journal of Clinical Pathology*, 1960, **34**, 381.

Lymph Nodes and Gastric Cancer

SIR,—It is disappointing to see an inaccurate belief perpetuated in the *B.M.J.*, especially in a leading article (9 October, p. 67). I would therefore like to challenge a statement made by the anonymous author of "Prognosis of Gastric Cancer." It reads "metastasis [to lymph nodes] is associated with a greatly decreased life expectancy," and though this is prefaced by a vague reference to the "extent of involvement of the lymph nodes," the inference is that patients with any lymph nodes involved have a prognosis totally different from those with all nodes free of metastases. Pygott's figures[1] which were quoted from other surveys, put patients into two categories, lymph glands free (LG −ve) and glands invaded (LG +ve). Many other writers have assumed that this generalization is adequate. Hawley, Westerholm, and Morson[2] have basically used the same approach though they do report some modification and their results showed that patients with few metastatic nodes did better than those with many secondaries. Pack and McNeed,[3] however, showed that 30·8% of their five-year survivors had histological evidence of lymphatic metastases at the time of operation. This LG +ve: LG −ve division may therefore be an oversimplification.

From the results of my own study[4] it was apparent that gastric cancer patients whose metastases involved less than half of the regional lymph nodes achieved a survival rate that was statistically no different from those with all nodes free. Full details were available from the records of 165 patients suffering from gastric carcinoma, including surgical findings, histology of primary and lymph nodes, and five-year progress postoperatively. I divided the series into three groups: those who had no histological evidence of nodal metastases (OX), those with less than half of the lymph nodes invaded (LTH), and those with more than half of the nodes containing metastases (MTH). The five-year survival rates for different combinations of these groups were:

OX	—	46%	survived 5	years
OX+LTH	—	41%	,,	,,
LTH	—	35%	,,	,,
LTH+MTH	—	14·6%	,,	,,
MTH	—	8%	,,	,,

Statistically (using χ^2 test and Yates's correction) the difference between OX and LTH is highly significant ($P<0{\cdot}001$). This evidence supports the hypothesis that survival is related to the proportion of local nodes invaded, and is not a function of the absolute presence or absence of secondary spread to lymph

nodes. This may well link up with the work done by Hawley *et al.*[2] and Paile[5] suggesting that some patients may do better because their cellular immune mechanisms are putting up resistance to the primary, and they may do well even if some metastases have reached a few local nodes.

This is an important problem, because the surgeon at laparotomy who finds some palpable nodes near a resectable gastric tumour may well decide against radical local dissection on the basis of an apparently hopeless prognosis ("glandular metastases"). My study showed that where surgeons reported "malignant glands present," the histological findings in no less than 1:3 patients were of OX or LTH status (and therefore a better prognosis). In other words, there are two reasons to continue with radical lymphatic surgery, even with palpable nodes. Firstly, they may be benign, and secondly, they may represent the minimal invasion and quite good prognosis situation. For those who can stand awful doggerel this may be summarized:

"All that bulge are not maligne,
Even be some so, do not pyne."

May I also point out the fact that early diagnosis of gastric cancer, and greatly improved results in treatment, have been taken a stage further by the use of gastric cytology, with or without fibrescope collecting methods.[6 7]—I am, etc.,

E. G. CANTRELL

Medical Faculty,
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1 Pygott, F., *Gut*, 1964, **5**, 118.
2 Hawley, P. R., Westerholm, P., Morson, B. C., *British Journal of Surgery*, 1970, **57**, 877.
3 Pack, G. T., McNeer, G., *Surgery*, 1948, **24**, 769.
4 Cantrell, E. G., *British Journal of Surgery*, 1971, **58**, 384.
5 Paile, A., *Annales Chirurgiae et Gynaecologiae Fenniae*, 1971, **60**, Suppl. 175.
6 Cantrell, E. G., *Quarterly Journal of Medicine*, 1971, **40**, 239.
7 Kasugai, T., *Gastroenterology*, 1970, **58**, 429.

Antileprosy Drugs

SIR,—In "Today's Drugs" (17 July, p. 174) it is mentioned that it is doubtful if psychosis can be accepted as a toxic effect of dapsone. W. H. Jopling, to whom this statement is attributed,[1] was not in on the early treatment with dapsone in which high dosage was given to large numbers.

In 1950 I was in charge of Oji River Leprosarium, Nigeria. We treated all 15,000 patients with hydnocarpus oil and scarcely ever had any mental trouble. Within six months we changed the 1,800 settlement and 8,000 of the clinic patients to dapsone. Having been warned by the initial toxicity in a much smaller series under Dr. J. Lowe at Uzuakoli receiving a dose of 300 mg/day (1,800 mg/wk), we adopted a dose of 200 mg/day (1,200 mg/wk) in the settlement and 400 mg twice weekly in the clinics. Our idea of the lower dose in the clinics together with a slower induction was to minimize the serious reactions where fewer and less skilled staff were able to cope with the consequences.

We also induced treatment more slowly than at Uzuakoli (12 weeks instead of six weeks to reach the maximum), but still we were faced with many cases of acute psychosis with violence, delusions, and visual and auditory hallucinations together with two suicides within the first few months. Most of these were in the settlement where we had a higher dose and more rapid induction, so we soon changed our treatment to twice weekly, lowered the maximum, and induced more slowly. In addition, we were alerted to early signs of mental disturbance and took immediate action, thus reducing the rate of psychotic and other drug reactions to a much lower figure.

Dr. Jopling started treatment on much smaller numbers, used lower dosage, and increased more slowly. Modern low dosage treatment must also contribute to minimize this complication.

There was no statistically organized test but I believe the sudden outcrop of psychosis on dapsone treatment can be explained on no other hypothesis.—I am, etc.,

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Reepham,
Norfolk

1 Joplin, W. H., *Handbook of Leprosy*, London, Heinemann, 1971.

Blood Flow in Ischaemic Feet

SIR,—We would like to respond to one or two points which arise in the letter from Dr. V. C. Roberts and others (9 October, p. 114) commenting on our recently published findings (24 July, p. 220).

They would doubtless agree that there is not necessarily a discrepancy between their observations in atherosclerotic subjects of a lower than normal total limb perfusion as measured in the great vessels and our observations of a higher than normal resting foot blood flow. There may well be a fundamental difference between proximal and peripheral perfusion under these pathological conditions. Our findings and those of Yao[1] indicate that there is a low systolic blood pressure and peripheral resistance in apparently ischaemic feet and therefore the level of femoral artery or vein blood flow is not necessarily a reliable indication of the level of foot blood flow in atherosclerotic subjects. There may, of course, be a simpler explanation for the difference between our findings and those of Dr. Roberts. Our measurements were made in controlled resting conditions where the normal exposed foot vasoconstricts in response to room temperature around 22°C. There is no information about environmental conditions in Dr. Roberts's letter and, therefore, it is difficult to comment on the relative significance of their observations. However, it is certain that both anaesthesia and warm operating theatre conditions would have completely altered our results.—We are, etc.,

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1 Yao, S. T., *British Journal of Surgery*, 1970, 57, 761.

Predicting Fetal Maturity

SIR,—In their article (25 September, p. 736) Dr. Rosemary A. Underhill and others state that liquor studies were inexact when compared with ultrasound cephalometry and radiology. This is unfortunate. The scoring by Brosens and Gordon[1] should have attempted a two-week interval in the significant 34-38 weeks period of gestation. Had this been practised, liquor studies would have probably fared better by comparison. Ultrasound studies are more useful in the early stages of pregnancy and liquor studies in the last 6-8 weeks, when more critical decisions are imposed upon the obstetrician.

More recent experience with cytology[2-4] does suggest a better scoring system can be evaluated by utilizing not only the percentage of orange-staining cells but also the size of clusters, the presence of turbidity and debris, the existence of orange-staining globules, and unstained flakes. Not taking these into account may be responsible for certain spurious readings. As in other investigations, there is a tendency to give them a percentage of accuracy rating which may only reflect the need to improve the technique or interpretation thereof. We feel that in practice the clinician may utilize with benefit the augmented information resulting from a combination of investigations.—We are, etc.,

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London S.W.10

1 Brosens, I., and Gordon, H., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1966, **73**, 88.
2 Sharma, S. D., and Trussell, R. R., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1970, **77**, 215.
3 Parkin, F. M., Lind, T., and Cheyne, G. A., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1969, **76**, 673.
4 Husain, O. A. N., and Sinclair, L., *Proceedings of the Royal Society of Medicine*, 1971, in press.

Fingerprint Changes in Dermatitis Herpetiformis

SIR,—Dr. T. J. David and colleagues (5 December 1970, p. 594) in a study mainly in adults reported the common occurrence of epidermal atrophy to actual loss of fingerprint patterns occurring on finger digits in coeliac disease, though one of us (Dr. J. Verbov, 2 January, p. 48) advised caution in interpretation of such fingerprint changes and Dr. W. M. McCrae and colleagues (10 July, p. 109) did not find ridge atrophy in six children with untreated coeliac disease.

The occurrence of jejunal mucosal abnormalities in dermatitis herpetiformis was first reported by Marks *et al.*,[1] and Shuster and Marks[2] found from published studies that two-thirds of patients with dermatitis herpetiformis have the enteropathy (coeliac syndrome) and in some cases there is intestinal malabsorption. More recently, Brow *et al.*,[3] using a multiple biopsy technique, have found the enteropathy to be almost invariably present. The enteropathy usually responds to a gluten-free diet.

Fingerprint changes in dermatitis herpetiformis are obviously of interest in view of the above and we report some preliminary findings. So far, fingers and fingerprints have been examined in 37 patients with dermatitis herpetiformis (12 women and 25 men). The age range of patients was 21-73 years and the mean age was 47·3 years. Intentionally, patients have been examined and prints analysed without prior knowledge of any jejunal biopsy findings. Minor degrees of ridge flattening with some white lines in prints were common, but did not appear to