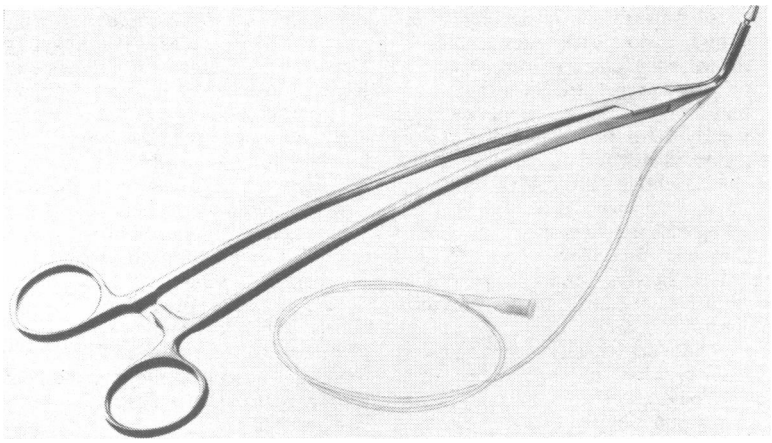

The grooved jaws of the introducing forceps should be placed on the Stoke-on-Trent cannula about 3-4 mm from its expanded end (Fig.). After the cystic duct has been dissected clear of surrounding structures, a ligature is tied round it close to the gall bladder. The duct is incised immediately distal 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 The forceps can then be released, and withdrawn from the duct without fear of dislodging the cannula.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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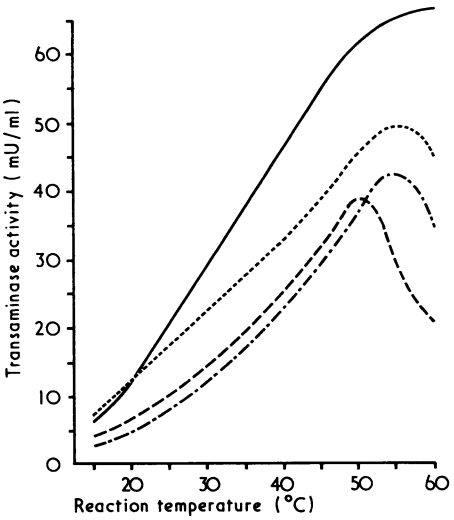
1 Buchanan, J. McK., *British Medical Journal*, 1969, **1**, 706.

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 ($P<0.001$). 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