Obesity and Smoking Habits

SIR,—I have read with great interest the article by Dr. T. Khosia and Professor C. R. Lowe (2 October, p. 10) on smoking and obesity.

Our results at the Institute of Directors Medical Centre (Table) do not show the same relationship. There is no convincing difference between any of the smoking classes. The figures have not been broken down into age groups, but we have previously shown that in our population age has little effect on the number of smokers and the amount they smoke.[1] There is a tendency for absolute weight to increase with age,[2] but this does not seem to be true for relative weight, except perhaps for the over 60's.[3]

	Fewer Cigarettes	Given up Cigarettes	1-19 Cigarettes a Day	20 or more Cigarettes a Day	Total
Number	677	388	424	511	2,000
% more than 10% underweight	7	9	14	13	11
% Average weight	65	61	60	57	61
%10-19% Overweight ..	18	20	18	21	19
% 20% or more Overweight	10	10	8	9	9

Average weight for the patients age and height calculated from the tables of the Metropolitan Insurance Company of New York.

In view of the large numbers in both series it is not likely that these differences in trends are due to chance. The most obvious difference between the two series is one of social class, our patients being almost totally from class I or II, whereas the South Wales series is presumably almost entirely social classes IV and V.

Nicotine is known to raise the blood sugar, and it may be that this tends to lead to a lower intake of carbohydrate and hence lower weight as found by Dr. Khosla and Professor Lowe. We have found an association between heavy smoking and heavy drinking; overeating may also be part of a syndrome of general overindulgence. The increased financial opportunities for this sort of behaviour in our patients may account for the lack of difference in relative weight between our non-smokers and heavy smokers. Those who exhibit moderation in non-smoking (1-19/day) have indeed got the lowest incidence of overweight—in agreement with Dr. Khosla and Professor Lowe.

There is general agreement that stopping smoking usually leads to weight gain. In our population we have found that this is by no means a necessity. Of 222 men who had given up smoking at follow-up a year or more after their first visit 50% had the same weight (± 5 lb), 28% had gained weight, but 22% had lost weight. This does not agree with weight difference between smokers and non-smokers found by Dr. Khosla and Professor Lowe.

In future studies it would be of great interest to see if there is a true difference in the relationship between smoking and weight according to social class.—I am, etc.,

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1 Pincherle, G., and Wright, H. B., *Journal of the College of General Practitioners*, 1967, **13**, 280.
2 Richardson, J. F., and Pincherle, G., *British Journal of Preventive and Social Medicine*, 1969, **23**, 267.
3 Pincherle G., and Wright H. B., *Practitioner*, 1970, **205**, 209.

Intravenous Practolol

SIR,—Continuous intravenous infusion of practolol was used in the treatment of lignocaine-resistant ventricular dysrhythmia which followed ventricular fibrillation in three patients. Before starting treatment the systemic arterial pressure recordings in the three patients were 70 mm Hg, 95 mm Hg, and 80 mm Hg respectively. All three patients received intermittent positive pressure ventilation to combat pulmonary oedema. In the first case the infusion rate was 30 mg an hour, amounting to 720 mg a day; the second patient required an infusion rate of 25 mg an hour, and the third patient received 1,050 mg of practolol over 50 hours. In all cases the intravenous infusion was subsequently discontinued and practolol was given orally.

Continuous infusion studies have been carried out using oxprenolol in the treatment of dysrhythmia after cardiac infarction.[1] In 59% of cases significant hypotension was reported. Despite the relatively high dose of practolol infusion we used, the systemic blood pressure showed no fall. In all our cases, after the dysrhythmia was controlled by the administration of practolol, the blood pressure started rising, the urinary output improved, and the heart failure was brought under control. Sinus bradycardia has been reported to occur after direct current shock in patients receiving the beta-blocking agent propranolol.[2] Two of our patients experienced a similar effect after direct current shock, but sinus rhythm was restored after a few seconds of external cardiac massage, and on neither occasion did we have to use atropine.

We confirm Jewitt's reports[3 4] on the effectiveness of practolol in the treatment of lignocaine-resistant ventricular dysrhythmia, as well as its less adverse haemodynamic effect when compared with other beta-blocking agents given intravenously.

We thank Dr. W. G. A. Swan and Dr. P. Szekely, consultant cardiologists, Dr. C. Strang, consultant physician and staff of the coronary care unit at the Newcastle General Hospital for their valuable assistance.

—We are, etc.,

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1 Sandler, G., and Pistevos, A. C., *British Medical Journal*, 1971, **1**, 254-257.
2 Szekely, P., Wynne, N. A., Pearson, D. T., Batson, G. A., and Sideris, D. A., *British Heart Journal*, 1970, **32**, 209.
3 Jewitt D. E., Mercer, C. J., and Shillingford, J. P., *Lancet*, 1969, **2**, 227.
4 Jewitt, D., Hubner, P., and Maurer, B., *Cardiovascular Research*, 1970, World Congress of Cardiology Supplement, **7**, 176.

Epitaph for the M.C.H.C.

SIR,—Dr. M. S. Rose states (16 October, p. 169) that neither the M.C.H.C. nor the M.C.H. conveys anything of particular clinical value any longer. However, he does not give an explanation nor does he take into account the following:

(1) The M.C.H.C. returns to normal on successful treatment of hypochromic anaemias.

(2) One cannot accept the red cell count even if estimated by a machine as 100% accurate. This is the basis on which the M.C.H.C. came into being. This applies equally to the M.C.V. particularly when there are striking variations in cell size and shape.

(3) Dr. Rose states "automatic apparatus is churning out normal M.C.H.C. to the accompaniment of hypochromic films" without any reference to the patients from whom these films were obtained. If these patients were hypochromic, then the machine is deceiving us, not the M.C.H.C.

As a haematologist and as I have pointed out in my book[1] I believe (contrary to Dr. Rose's view) that information of immense value (sometimes even a diagnosis) can be extracted by an experienced worker from a well prepared blood film. Even with the latest machine, the Hemalog, a blood film is invaluable in many circumstances.—I am, etc.,

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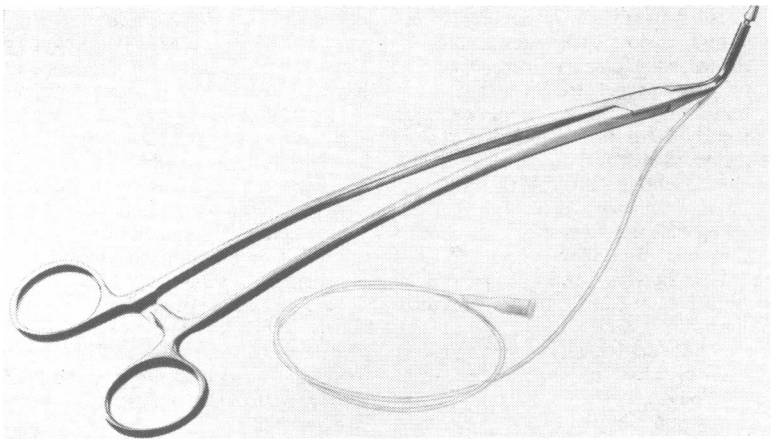
1 Nour-Eldin, F., *Haematology, Rudimental, Practical and Clinical*, London, Butterworths, 1971, in press.

Cholangiogram Cannula-introducing Forceps

SIR,—The Stoke-on-Trent cannula[1] was designed specifically for the purpose of carrying out primary operative cholangiography. The cannula is manufactured from flexible polyethylene tubing; because of this flexibility a valid criticism has been the technical difficulty of manipulating it into the cystic duct, particularly if the latter is narrowed. New introducing forceps have now been made to overcome this problem.

The overall length of the forceps is 27·5 cm. They have bow handles and their shanks are slightly curved so that, when held by the operator, his view of the cystic duct will not be obscured. They have a box lock fitting to ensure complete approximation of the jaws, which are 2·5 cm in length. The jaws are grooved and, when closed, they provide a channel with an internal diameter of 1·3 mm. This receives exactly the Stoke-on-Trent cannula, gripping it firmly without undue pressure which would otherwise reduce or obliterate its lumen. The external diameter of the closed jaws is the same as that of the widest part of the expanded distal end of the cannula. The jaws are angled at 80° to the shaft so that actual insertion into the cystic duct may be facilitated. Finally, the instrument has been given a matt finish to avoid glare and reflection.



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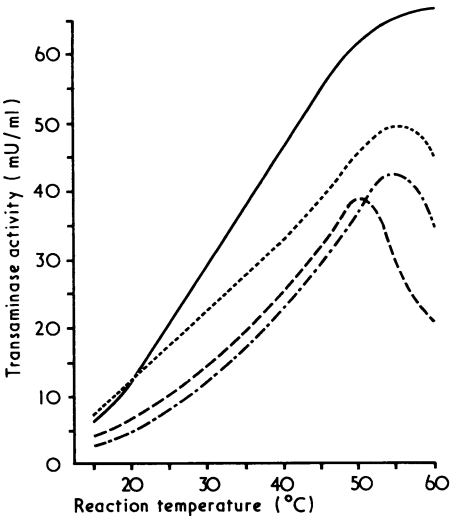
1 Buchanan, J. McK., *British Medical Journal*, 1969, **1**, 706.

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 ($P<0{\cdot}001$). 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