underestimated in the past. There are three main factors involved in this transfer of Phoenix/Delphi: (1) translation from one patois of Coral to another; (2) transfer from Modular One to a Ferranti Argus computer; (3) transfer from a teaching hospital to a district general hospital. Experience to date confirms that even this simple exercise needs close co-operation from both the hospital laboratories and the manufacturers. The task is intellectually demanding and requires properly trained and widely experienced computer scientists as well as enthusiastic support by the laboratory staff.

Once this transfer is complete there is no doubt that Phoenix/Delphi will be an economical and extremely useful adjunct to our regional pathology service and to anyone else who wishes to make use of it.

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Papilloedema

SIR,—Your leading article on papilloedema (4 February, p 263) is timely but ignores many recent reports and thus completely misrepresents current thinking on the subject. Even at its earliest stage of development disc swelling from raised intracranial pressure is due not to extracellular fluid accumulation but primarily to swelling of retinal ganglion-cell axons^{1,2}; it thus has a distinct pathology from that of "brain oedema." The axonal swelling is associated with obstruction of orthograde axoplasmic transport as demonstrated by autoradiography in experimental models^{3,4} and inferred from clinical observation⁵; this is not a "delayed effect" of papilloedema but is the crucial pathophysiological response of the disc tissue to raised pressure in the optic nerve sheath. Microvascular leakage around the disc in papilloedema appears to be a secondary event and is unlikely to be caused by pressure from axonal swelling on the central retinal vein; it may represent a local capillary reaction to the accumulation of axoplasm.

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¹ Hayreh, M S, and Hayreh, S S, *Archives of Ophthalmology*, 1977, **95**, 1237.

² Tso, M O M, and Hayreh, S S, *Archives of Ophthalmology*, 1977, **95**, 1448.

³ Minckler, D M, Tso, M O M, and Zimmerman, L E, *American Journal of Ophthalmology*, 1976, **82**, 741.

⁴ Tso, M O M, and Hayreh, S S, *Archives of Ophthalmology*, 1977, **95**, 1458.

⁵ McLeod, D, *Transactions of the Ophthalmological Society of the United Kingdom*, 1976, **96**, 313.

* "Over the past 120 years, views on the pathogenesis of papilloedema have changed again and again with the prevailing fashion."¹ This observation was made by S S Hayreh, whose own writings in this field have not been without conceptual tergiversations over the years. In 1964 he asserted categorically that "pressure in the subarachnoid space is transmitted into the optic nerve sheath where it presses upon the central retinal vein and produces oedema of the optic disc."² In 1977 he wrote that "raised CSF pressure in the sheath of the optic nerve produces axoplasmic flow stasis in the optic nerve head."³ Yet it must be accepted that severe papilloedema can occur when the optic nerve sheath is completely obliterated. As recently as 1975 Hayreh wrote concerning the part played by axoplasmic flow stasis in the production of papilloedema that it was based "on small,

tenuous, doubtful and often highly speculative shreds of evidence."⁴ In the circumstances it is difficult to accept the implication that Hayreh's writings—past or present—are representative of "current thinking on the subject."—Ed, *BMJ*.

¹ Hayreh, S S, *Survey of Ophthalmology*, 1975, **20**, 181.

² Hayreh, S S, *British Journal of Ophthalmology*, 1964, **48**, 522.

³ Hayreh, S S, *Archives of Ophthalmology*, 1977, **95**, 1553.

Erythematous oedema of the legs due to nifedipine

SIR,—I would like to report two patients with angina pectoris who were treated with nifedipine (Adalat) and who subsequently developed a marked erythematous oedema of both legs, mimicking erysipelas. Both women are in their early sixties, and after receiving nifedipine 10 mg three times a day for one week developed a well-marked erythema of both legs and ankles which subsequently became oedematous and very painful. The condition of the legs improved within a matter of three days after stopping the drug. One of the patients was rechallenged with the same dose of nifedipine and developed a similar reaction as before.

According to Lydlin *et al*¹ and Mostbeck *et al*² there is a well-marked increase in peripheral blood flow, as shown by plethysmography, to the legs after administration of nifedipine, and this probably accounts for the oedema.

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¹ Lydlin, H, *et al*, in *New Therapy of Ischemic Heart Disease*, Proceedings of the 2nd International Adalat Symposium. Amsterdam, Excerpta Medica, 1976.

² Mostbeck, A, *et al*, in *New Therapy of Ischemic Heart Disease*, Proceedings of the 2nd International Adalat Symposium. Amsterdam, Excerpta Medica, 1976.

Bran and HDL-cholesterol

SIR,—The Reverend H C Trowell (21 January, p 170) has pointed out two different lines of research which suggest that a high level of high-density-lipoprotein (HDL)-cholesterol¹ on the one hand and a high intake of cereal fibre² on the other afford protection against coronary artery disease. He concludes that it is important, therefore, to ascertain whether a diet rich in unrefined high-fibre foods can raise plasma HDL-cholesterol levels. Both of these variables have been recently studied in 16 medical students and physiotherapists aged between 20 and 25 years who added bran to their diets over a period of 13 weeks.

Each of the 16 subjects (10 men, 6 women) took two heaped tablespoonfuls of Allinson's Bran Plus daily during the initial six weeks and all but four subjects took three heaped tablespoonfuls thereafter. This would represent increases of about 8 and 12 g of unavailable carbohydrate respectively to the daily diet after Southgate's method of analysis.³ In addition, 10 of the 16 subjects ate bread made from wholemeal flour during the 13 weeks. Three control blood samples for estimation of serum HDL-cholesterol, total cholesterol, and fasting triglycerides were taken in the two weeks preceding the study, and similar analyses were made during each of the subsequent 13 weeks.

Average weight increased by 0.5 kg (1.1 lb) and women had higher initial serum HDL-cholesterol levels than men (mean $\pm$ SD, 1.62 ± 0.33 mmol/l (62.5 ± 12.8 mg/100 ml) compared with 1.33 ± 0.35 mmol/l (51.5 ± 13.6 mg/100 ml)), as has been shown previously.⁴ Neither difference, however, was statistically significant. Throughout the trial period there was no significant change in mean serum HDL-cholesterol, total serum cholesterol, or triglyceride concentrations. Statistical analysis of individual data confirms this. The finding that there was no reduction in either total serum cholesterol or serum triglycerides is in agreement with several other studies.⁵⁻⁷

In summary, then, this study shows that increasing daily dietary fibre intake by 12 g by taking bran does not raise serum HDL-cholesterol levels.

I would like to thank the 16 subjects who participated in this research and Professor I Macdonald for his valuable advice and support.

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¹ Miller, G J, and Miller, N E, *Lancet*, 1975, **1**, 16.

² Morris, J N, Mair, J W, and Claydon, D G, *British Medical Journal*, 1977, **2**, 1307.

³ Southgate, D A T, *Journal of the Science of Food and Agriculture*, 1969, **20**, 331.

⁴ Fredrickson, D S, Levy, R I, and Lees, R S, *New England Journal of Medicine*, 1967, **276**, 148.

⁵ Connell, A M, Smith, C L, and Somsel, M, *Lancet*, 1975, **1**, 496.

⁶ Durrington, P, Wicks, A C B, and Heaton, K W, *Lancet*, 1975, **2**, 133.

⁷ Truswell, A S, and Kay, R M, *Lancet*, 1976, **1**, 367.

ECT and lithium

SIR,—Electric convulsion therapy (ECT) has been shown¹ to be effective in the treatment of depressive illness. Recently it has been the subject of critical comment and the Royal College of Psychiatrists has laid down guidelines concerning the indications for ECT and the manner in which the treatment should be offered.² Diane Mallaby (28 January, p 234) raises important points of general interest and I should like to contribute to this discussion my findings on the effect of lithium on the amount of ECT given to patients.

In the process of an open study³ of lithium in the treatment of acute depressive illness it was found that of the 30 subjects successfully treated with lithium, 22 had been given ECT for previous acute depressive illness and only one of the 30 patients was given ECT when lithium was used in the treatment of the acutely depressed phase. A total of 293 ECTs were administered to the patients before lithium treatment; as these patients had suffered from recurrent depression lithium was continued following the remission of depressive symptoms and the drug has been continued for an average period of 2.8 years, during which time no ECT has been required. Continuous lithium treatment of the current depressive illness has dispensed with the need for maintenance ECT in this group of patients. A computerised analysis of the outcome of 169 patients treated with lithium showed that 162 had been either severely or moderately severely handicapped by their illness and had required repeated admission to hospital but that during lithium therapy only 32 patients were so affected. Double-blind trials⁴ have confirmed lithium's therapeutic action and my results demonstrate that the use of lithium