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

in acute and recurrent depressive illness can lead to a rapid return to health and dramatically reduce the need for ECT.

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- ¹ Greenblat, M, Grosser, G H, and Wechsler, H, *American Journal of Psychiatry*, 1964, 120, 935.
² Royal College of Psychiatrists, *British Journal of Psychiatry*, 1977, 131, 261.
³ Bennie, E H, paper presented at First British Lithium Congress, 1977.
⁴ Bastrup, P C, et al, *Lancet*, 1970, 2, 326.

The cancer patient: communication and morale

SIR,—While agreeing with Dr Thurstan B Brewin's thoughtful paper (24-31 December, p 1623) we would like to draw attention to one distortion of communication which can arise when we tell patients they have a malignant disease.

There is an understandable tendency to convey an over-optimistic prognosis. We have found that instilling a false sense of optimism can be as destructive as the earlier tendency to conceal the diagnosis.

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Active immunotherapy in malignant melanoma

SIR,—About a year ago¹ you published the preliminary results of a controlled trial of active immunotherapy in the management of stage IIB malignant melanoma, in which we showed that a postoperative vaccination with a mixture of BCG and autologous irradiated tumour cells resulted in death from metastases in half the patients evaluated at one year. In contrast, recurrence in the non-vaccinated controls appeared later and all the patients followed up for one year were still alive.

The period of observation in this study is now at least two years and the accompanying table shows the updated recurrence and death rates in the two groups of patients. Three of the non-vaccinated patients died during the second year of follow-up, while only one further vaccinated patient died in this period.

Recurrence and death rates in vaccinated and unvaccinated patients with stage IIB malignant melanoma (total numbers in parentheses)

Follow-up period	Recurrences		Deaths	
	Vaccinated patients (8)	Controls (7)	Vaccinated patients (8)	Controls (7)
12 months	4	3	4	0
24 months	6	5	5	3

Thus the difference in death rates between the two groups that was so striking at one year is now less obvious. The two vaccinated patients who remain tumour-free have been followed up for 34 and 36 months and the two non-vaccinated patients who remain tumour-free for 40 and 24 months respectively.

These additional results do not deny our original claim that active immunotherapy given on the 14th postoperative day may

enhance tumour growth in some patients with stage IIB malignant melanoma. However, the observation that two vaccinated patients remain tumour-free 34 and 36 months later indicates that tumour enhancement is not an inevitable consequence of such treatment. The extent to which this is likely to happen may be determined by the tumour load (albeit clinically undetectable) at the time of vaccination, and a sensitive tumour marker, if available, might identify patients in whom active immunotherapy may still be of some benefit.

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¹ McIllmurray, M B, et al, *British Medical Journal*, 1977, 1, 540.

The NHS 5-ml spoon

SIR,—In the letter from Dr S W V Davies (11 February, p 365) attention is again drawn to the inadequacy of the 5-ml NHS issue measuring spoon. This form of measuring device has been the subject of adverse criticism from time to time in the pharmaceutical press. Experience suggests that medicines are prescribed with equal frequency in two 5-ml doses, thus resulting in further error when the 5-ml spoon is used. Perhaps, therefore, consideration might be given to the production of a measure somewhat similar to that suggested by Dr Davies but made to contain 10 ml and graduated at the 5- and 10-ml levels.

Such a measure, of course, would need to be made in transparent material and is, in fact, provided by some manufacturers in the carton containing their proprietary medicine. The cost of this type of measure would be greater than that of the 5-ml spoon currently provided, but in view of an increasingly aging population, who find difficulty in using the latter, a reasonable increase in cost would surely be justifiable.

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Cytomegalovirus infection in a physician

SIR,—Your readers may be interested in the following case.

Early in February 1972 a 38-year-old consultant physician developed a sore throat and pyrexia. Lymphadenopathy developed after 10 days and he felt extremely tired. In the succeeding months he developed severe occipital headaches with meningism, photophobia, generalised muscle pains, and ulceration of the fauces. On 28 April the patient's serum was found to fix complement in the presence of cytomegalovirus (CMV) to a titre of 1/756. CMV was repeatedly grown from the urine and saliva and a significant level of IgM antibody was demonstrated (1/64). All other investigations were negative.

The illness progressed with repeated episodes of fever, usually following even the mildest of exercise, associated variously with fleeting lymphadenopathy, mouth ulcers and oedema of the lips, conjunctival ulcers, severe muscle pains with girdle pain, fleeting rashes, and headache with

occasional meningism. A presumed viral osteomyelitis of the distal phalanx of a finger occurred, diagnosed with the help of thermography. The phalanx fractured without trauma but healed after six months in a plaster splint. Marked malaise was present in the febrile episodes and the impairment of exercise tolerance was so severe that the physician was unable to continue his consultant post. After five years, however, he is relatively well and has other medical employment. The patient's previous medical history seemed to indicate that the common viral illnesses of childhood had been unduly severe, and at the age of 25 he had had a particularly severe attack of glandular fever and was off work for 4½ months, the lymphadenopathy, fever, and malaise lasting for three years.

It must remain extremely unlikely that the CMV infection in this patient was secondary to an underlying reticulosis or other sinister disorder as this would almost certainly have become manifest after five years.¹ There were numerous episodes, however, during the course of the patient's work when he was extensively splashed with fresh blood from neonates with congenital heart lesions, possibly related to CMV infections, and from young adults who were probably shedding virus at the time. It is most likely that the initial infection dated from one of these episodes. Infection with CMV is well recognised in patients who have undergone cardiac surgery² but should also be considered when medical and nursing staff³ looking after such patients fall ill.

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¹ Duval, C P, et al, *Annals of Internal Medicine*, 1966, 64, 531.

² Smith, D R, *British Medical Journal*, 1964, 1, 945.

³ Muller-Stamon, A, et al, *British Medical Journal*, 1974, 3, 609.

Traumatic external rectus palsy in a child

SIR,—Sudden onset of an external rectus palsy in a child raises the suspicion of serious neurological disease—raised intracranial pressure, an infiltrating glioma of the pons, and tuberculous meningitis being possibilities.¹ However, benign external rectus palsy can occur and it is now 10 years since Knox et al² reported 10 children aged 18 months to 15 years in whom benign sixth nerve palsy occurred 7-21 days after a mild upper respiratory tract infection. In nine of the 10 children spontaneous complete recovery took place within 10 weeks. The development of external rectus palsy following an otherwise uncomplicated head injury in childhood appears to be rare. Yarzagaray,³ in a study of 246 children with head injury, reported only 15 with cranial nerve palsies and there is no record that any of these were isolated lesions. Of the 15 children, nine had sixth nerve involvement (six bilateral and three unilateral), four had seventh nerve palsies, and two had third nerve palsies. If the palsy is isolated and the evidence for intracranial trauma minimal there may be a strong indication to undertake complete investigation in order to exclude other serious underlying causes. The following case illustrates the diagnostic difficulty and also emphasises that spontaneous complete recovery after trauma can occur.

A previously healthy, intelligent, 9-year-old