a difficult problem. This, it should be noted, is an isolated cognitive defect.

Few qualified doctors find the concept of acquired dyslexia, as for example the result of a cerebral vascular accident, difficult to accept, but strangely an inherited defect causing difficulty in learning to read in an otherwise normal child appears to be almost unbelievable. Were this not the case the term dyslexia would not be held in such suspicion and the powerful negating control that many educational psychologists hold over the whole problem of reading difficulties would never have eventuated.

One must sadly come to the conclusion that many doctors have abrogated their responsibilities for this important aspect of the school medical service. This has led to greater hardships for this group of children, who with their parents are in an impossible position to conduct their own defence. This is the chief cause for the shortage of specialised remedial teachers of which you make mention, whereas many other countries which recognise the true nature of the problem have no such deficit. For a more balanced view of the whole subject of the problem of children who cannot read I recommend a small book by MacDonald Critchley.¹

It is, of course, recognised that this group of children with a neurological defect are suffering from only one of a number of causes of reading difficulties. They are, however, quite separate from the continuum usually recognised by the educational psychologists, which range from intellectual inadequacy at one end of the scale to neurosis at the other. They are nevertheless all too often incorporated into this, and as a result spend their secondary school careers in the general remedial section undergoing the usual group therapy. It is generally recognised by experts that they respond poorly to this and need much greater specialised teaching.²

They need, and are worthy of, special consideration, and as a working rule it is most misleading to qualify the degree of their disability as "particularly likely to express the behaviour—poor reading—in an adverse environment (for example, a large family or a poor school)." Most of them come from good caring and loving homes with two or three children, and it has not been my experience that any school shows particular poorness by demonstrating a greater number than others.

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¹ Critchley, M, *The Dyslexic Child*, 2nd edn. London, Heinemann, 1970.

² Department of Education and Science, *Children with Specific Reading Difficulties*, Report of the Advisory Committee on Handicapped Children, p 4, para 15. London, HMSO, 1972.

SIR,—With regard to your leading article (1 July, p 3), it should also be mentioned that those who study the physiology of education will mostly agree that a dedicated mother or other adult will prevent all chance of future reading difficulty if she arouses a very early interest by the infant in all aspects of words—talking, reading, singing, drawing, writing, etc.¹⁻³ In support for this early start it is obviously very desirable that all television programmes for children should illustrate some enjoyable aspects of all the language skills. The current failure of our TV children's programmes to help adequately in this way is, I think, quite deplorable and positively

damaging to our national standards of education.

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¹ Brierley, J, *Growing and Learning*. London, Ward Lock Educational, 1978.

² Ibuka, M, *Kindergarten is Too Late*. London, Souvenir Press, 1977.

³ Russell, W R, *Explaining the Brain*. London, Oxford University Press, 1975.

Hypostatic ulceration and male sex chromosomal anomalies

SIR,—Dr R Howell (8 July, p 95) recently reported six cases of Klinefelter's syndrome suffering from ulceration of legs and collected from the literature 14 other cases of the same disorder. In this hospital we have, among 343 male mentally handicapped patients, the following sex chromosomal abnormalities: two with Klinefelter's syndrome, one with XXXXY syndrome,¹ and another with XYYY syndrome.² In 1968 we admitted a patient for assessment who had an XXXY/XXY sex chromosomal anomaly. All these patients suffer from varicose veins of various degrees and two (one with XXXXY and one with XYYY syndrome) from hypostatic ulceration of the legs.

As we have observed hypostatic ulceration in other clinical conditions and younger patients—for example, Prader-Willi syndrome³ at 12 years of age—we are planning to investigate all the hospital population for hypostatic ulceration and varicose veins and we would be grateful for any relevant observations on this subject from workers in mental handicap and, for comparison, from workers with the normal population.

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¹ Jancar, J, *Proceedings of the International Copenhagen Congress for SSMR*, 1964, vol 1, p 179.

² Jancar, J, *Lancet*, 1968, 2, 970.

³ Jancar, J, *Journal of Mental Deficiency Research*, 1971, 15, 20.

SIR,—The article by Dr R Howell (8 July, p 95) provides evidence that chronic leg ulcers appear more often than expected in men with Klinefelter's syndrome. Dr Howell suggests that this chromosomal anomaly should be considered in men presenting with hypostatic ulceration.

We wish to draw attention to the fact that not only supernumerary X chromosomes in men but also additional Y chromosomes seem to have this effect. Court Brown *et al*¹ found varicose ulcerations in two YY men in a series of 15. We have systematically examined 24 men with double Y chromosomes, cared for in mental hospitals, with respect to the tendency to varicose veins.² We recognised two grades, a severe and a milder. Subjects were assigned to the severe grade who, in addition to varicose veins, had complications in the form of attacks of thrombophlebitis or leg ulcers, either currently or in the medical history. Such a condition of severe varices with complications was present in five of our index cases (21%). By comparison with the prevalence figures given in the literature we considered it justified to assert that our figure shows that the chromosomal constitution 47,XXY carries with it an increased risk of varicose veins and hypostatic ulceration.

It is interesting that the two gonosomal

aberrations in males should have this effect in common.

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¹ Brown, W M C, Price, W H, and Jacobs, P A, *British Medical Journal*, 1968, 2, 325.

² Forssman, H, *et al*, *Males with Double Y-chromosomes*. Göteborg, Akademiförlaget, 1975.

Depot fluphenazine and tardive dyskinesia

SIR,—The view expressed in Any Questions? (8 July, p 114) that the risk of causing long-term dyskinesia with depot fluphenazine is small is probably optimistic. Although different workers have described greatly differing figures for the incidence of tardive dyskinesia (TD) in different studies, their criteria for diagnosis must have differed, and recent investigations suggest a high incidence of the condition in patients who have received neuroleptic drugs for many years. For instance Bell and Smith,¹ looking at the population in nine mental hospitals in Illinois and rating signs of all severity, found that 40% of patients exhibited probable evidence of TD and 26% showed undoubted signs. Most of these patients were on oral neuroleptics, but there is no reason to suppose that patients receiving fluphenazine by injection would be less likely to develop dyskinesia. Not only do they have less chance to be non-adherers to a treatment schedule, but the injection bypasses the vagaries of absorption of oral preparations.²

In this area we have nearly 400 patients on depot fluphenazine and flupenthixol and since 1973 63 of them have developed TD, though it is fortunately mild in the majority. All had had other neuroleptics before. Nevertheless, depot injections have allowed thousands of people to lead reasonably normal lives outside hospital and it is surely better to have involuntary movements of the lower face and tongue than to suffer the disturbing perceptions of uncontrolled schizophrenia; if we psychiatrists would use neuroleptics in the smallest effective therapeutic dose (half my patients on fluphenazine need 12.5 mg monthly) and eschew the use of the routine anticholinergic drugs the next generation of schizophrenics might have less trouble with TD than the present.

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¹ Bell, R C H, and Smith, R C, *Journal of Clinical Psychiatry*, 1978, 39, 39 and 46.

² Adamson, L, *et al*, *Diseases of the Nervous System*, 1973, 34, 181.

Prostaglandin F_{2α} and tumours of the female genital tract

SIR,—Williams *et al*¹ first reported elevated levels of prostaglandins (PG) in tumour tissues and in the plasma of patients with medullary carcinoma of the thyroid. Subsequently, raised blood PGE₂ and PGF_{2α} levels have been reported in association with a variety of human tumours, including phaeochromocytoma, carcinoid tumours, rectal carcinoma, Kaposi's sarcoma, neuroblastoma, bronchial carcinoma, islet cell tumours, and carcinoma of the breast.

Plasma PGF_{2α} concentrations in female genital tract malignancy

Patient No	Age (years)	Diagnosis	Plasma PGF _{2α} concentration (ng/l)
1	65	Carcinoma ovary stage III	88
2	41	Carcinoma ovary stage III	97
3	52	Carcinoma ovary stage III	70
4	58	Carcinoma ovary stage III	65
5	59	Carcinoma ovary stage III	45
6	16	Carcinoma ovary stage IV	61
7	76	Carcinoma ovary stage IV	92
8	62	Carcinoma ovary stage IV	59
9	65	Carcinoma endometrium stage I	34
10	64	Carcinoma endometrium stage I	39
11	57	Carcinoma cervix stage I	25
12	60	Carcinoma cervix stage IV	94
13	69	Mixed Mullerian tumour stage IV	94

Blood PGF_{2α} levels were measured by radioimmunoassay² in 13 untreated patients with gynaecological cancer presenting to this centre. The results are shown in the table and indicate a uniform elevation above the range (9-24 ng/l) established in our laboratory for normal adult women. A positive correlation with the stage of the disease is also apparent.

In common with other forms of malignant disease tumours of the female genital tract are associated with raised blood levels of PGF_{2α}. The mechanism of this association is unclear but most likely reflects abnormal PG production by neoplastic cells. One practical implication of this finding is the possibility that hypercalcaemia and osteolysis occurring in advanced malignancy³ may be caused by the osteolytic effect of PGF_{2α} and not be due to bone secondaries.

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¹ Williams, E D, Karim, S M M, and Sandler, M, *Lancet*, 1968, **1**, 22.

² Dray, F, Charbonnel, B, and MacLough, J, *European Journal of Clinical Investigation*, 1975, **5**, 311.

³ Powles, T J, *Proceedings of the Royal Society of Medicine*, 1977, **70**, 199.

Schizophrenia and neurosis

SIR,—Your leading article (8 July, p 76) on the predominance of neurotic complaints in a group of chronic schizophrenics treated in the community quotes, from Cheadle *et al*¹, surprise that this should be the case. It furthermore reiterates their assertion that neurotic symptoms are the major stumbling block to rehabilitation within the community.

Might I suggest that the work of Cheadle *et al* merely confirms something which has been known for the greater part of this century—that premorbid adaptation has a considerable bearing on recovery from schizophrenic breakdown? A 100-year review² of the literature on premorbid personality and schizophrenia illustrated well that in many

studies chronicity has a relationship to poor premorbid personality, and presumably the latter is a major determinant in so-called neurotic symptoms in the group studied.

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¹ Cheadle, A J, Freeman, H L, and Korner, J R, *British Journal of Psychiatry*, 1978, **132**, 221.

² Fritsch, W, *Fortschritte der Neurologie, Psychiatrie und ihrer Grenzgebiete*, 1976, **44**, 323.

Colonoscopy in the diagnosis of pseudomembranous colitis

SIR,—Dr A Kappas and his colleagues (18 March, p 675) are of the opinion that pseudomembranous colitis (PMC) is far more common than the sporadic published reports would suggest, a view which, as stated in your leading article in the same issue (p 669) has also been expressed by others.¹ Dr Kappas and his co-workers believe also that the only means of avoiding a high mortality rate is to establish the diagnosis promptly and give early supportive treatment. They conclude that careful, repeated sigmoidoscopy should be performed in all clinically suspected cases and in any patient with an unexplained complication after a colorectal operation.

But, as Bartlett and Gorbach in their excellent review of PMC pointed out,² one must also remember that the anatomical location of pseudomembranes includes virtually all portions of the intestinal tract. When the colon is attacked the more severe lesions occur in the proximal portion—the caecum and ascending colon. In patients with antibiotic-related disease the major impact of the disease is in the colon. This is evident also in some clinical studies. For example, Dr Kappas and his colleagues performed sigmoidoscopy on 20 patients with PMC but found membranes in only 13, while the appearances were normal in two. Price and Davies³ found typical plaques on sigmoidoscopy in only 14 out of 21 cases. Bearing in mind how serious a disease PMC can be, it does not seem to me that investigation by repeated sigmoidoscopy with biopsy and faecal toxin tests is sufficient to make an early diagnosis of PMC in all cases. Colonoscopy in skilled hands is safe,⁴ and I believe that the risk of perforation is not very great in the early stages of PMC with less severe mucosal changes. My own experience of colonoscopy for the diagnosis of PMC has been good, as the following case history shows.

A 27-year-old electrician was admitted with severe abdominal pain and bloody diarrhoea after three days' amoxicillin treatment (total dose 3.375 g). He had previously had abdominal pain and diarrhoea after penicillin treatment five years previously but had since been well except for symptoms of allergic rhinitis.

In the emergency room his temperature was 37.5°C, leucocyte count $21.9 \times 10^9/l$ ($29\,000/mm^3$), serum total protein 55 g/l. Sigmoidoscopy showed a totally normal rectal mucosa, but the contents of the sigmoid colon were brightly bloodstained. Next day I carried out a bedside colonoscopy with biopsy without x-ray or special bowel preparation. The rectum was normal, but at a height of 30 cm there was a heavily bleeding mucosa with gray plaques. The examination was stopped when it was seen that the whole descending colon was diseased except for some small areas with visible blood vessels. Although it was impossible to make a definite diagnosis because of the similarity of the colonoscopic picture to that of ulcerative

colitis and our case, a tentative diagnosis of PMC was made. The patient was treated with intravenous fluids and orally metronidazole 500 mg twice daily. The abdominal pain and bloody diarrhoea disappeared in five days and the patient made a full recovery.

The biopsy specimens taken at colonoscopy from diseased areas at the level of the sigmoid and ascending colon were later found to show the typical diagnostic features of PMC. After 10 days a control colonoscopy was performed, with normal findings. Faecal and biopsy specimen cultures taken during the first days in hospital showed only a growth of *Klebsiella pneumoniae* and *Pseudomonas maltophilia* with an otherwise normal flora. Unfortunately, no special investigations for the detection of *Clostridium difficile* or *Cl sordelli* were carried out.

I suggest that careful colonoscopy is indicated for the early diagnosis of potentially fatal PMC if the key investigation, sigmoidoscopy, gives negative results.

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¹ Tedesco, F J, Barton, R W, and Alpers, D H, *Annals of Internal Medicine*, 1974, **81**, 429.

² Bartlett, J G, and Gorbach, S L, *Advances in Internal Medicine*, 1977, **22**, 455.

³ Price, A B, and Davies, D R, *Journal of Clinical Pathology*, 1977, **30**, 1.

⁴ Britton, D C, *et al*, *British Medical Journal*, 1977, **1**, 149.

Sulphinpyrazone and prevention of death after myocardial infarction

SIR,—Your leading article (15 April, p 42) which discussed the report of the sulphinpyrazone trial¹ rightly calls for caution in accepting the favourable results without further information. One reason mentioned is the disparity between groups in the incidence and characteristics of arrhythmias. Another perhaps more obvious reason for doubting the results is the fortuitously high incidence of death in the placebo group (9.5%). For patients surviving the first month following myocardial infarction, especially when those with cardiomegaly were excluded, a death rate of 5% per annum or less might have been expected, more in line with the figure of 4.9% for the treated group in the trial. The data for the placebo group in the long-term practolol post-infarction trial² indicate the importance of heart size in prognosis (see table).

Death rate related to cardiac width (data from placebo group in Multicentre International Study¹)

Cardiac width (cm)	Deaths	No of patients	Death rate (%)
≤13	7	309	2.3
14-15	24	658	3.6
16-17	30	316	9.5
≥20	4	33	12.1

Armitage test for linear trend relating increasing death rate with increasing cardiac width significant at 1% level ($P < 0.001$).

A positive role for the platelet in the development of ischaemic heart disease seems very probable and the mode of action of sulphinpyrazone is therefore attractive. However, the long-term use of the drug following infarction should be postponed until a further large trial has confirmed benefit against placebo group figures which are more in accord with general experience. Perhaps