incidence of postoperative pulmonary embolism has also remained much the same. Is it possible that the radioactive fibrinogen test is too sensitive, and can any statement be made at present concerning the likelihood of pulmonary embolism from a series of localized "hot spots" on a patient's leg after surgery? Are Mr. Browse and Mr. Negus and the other investigators who have used this diagnostic method currently administering anticoagulants to all patients who have radioactive-fibrinogen-proved deep vein thrombosis? Should prophylactic intra- and postoperative anticoagulants be considered as a routine for all surgical patients, as the incidence of postoperative venous thrombosis is indeed approximately 25-30%, and even higher in patients of older age groups?—I am, etc.,

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- 1 V. V. Kakkar, C. Flanc, C. T. Howe, and M. B. Clarke, *Lancet*, 1969, 2, 230.
- 2 V. V. Kakkar, J. T. G. Renney, A. N. Nicolaides, J. R. Friend, and M. B. Clarke, *Lancet*, 1970, 1, 540.

Retirement

SIR,—I am surprised Dr. Clifford Allen takes such a dismal view of his retirement (10 October, p. 112). Surely his approach is the exact antithesis of that which is being widely suggested nowadays by such organizations as the Pre-retirement Association or the Heart and Chest Association.

Surely, Dr. Allen should be very considerably gratified by his not inconsiderable literary achievements in both medical and fictional fields. I personally feel that his philosophical and practical approach to his future years needs reappraisal.—I am, etc.,

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Driving and Epilepsy

SIR,—Dr. K. T. Farn's letter (12 September, p. 646) shows too much concern for man-made classifications and too little for a humanistic approach to the epileptic. The report on new regulations for issue of driving licences to epileptics (15 August, p. 362) represented progress. How long does the epileptic have to be treated with fear and restraint? The number of patients who have accidents as a result of a "heart attack" or a "black out" must certainly be more significant than those caused by an occasional epileptic having a seizure. Add to that the accidents caused by sedated or tranquillized patients and alcoholics and the rare accident caused by an epileptic becomes insignificant. The restrictions imposed on epileptics should be extended to include other conditions which are more prevalent and take a larger toll of life. Let us keep things in their proper perspective. I am not sure whom Dr. Farn is really trying to "protect."—I am, etc.,

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Fibrin Degradation Products

SIR,—Pre-eclampsia is a syndrome comprising three manifestations with a single aetiology. Further, the clinical signs disappear promptly on delivery, and therefore the single aetiological factor could not have produced an organic change. Dr. E. N. Wardle's attempt to separate and deal with a single item of this triad and ascribe to it an underlying organic change in the tissues (24 October, p. 245) is completely indefensible and should be so recognized by all obstetricians.

He has posed two questions. Does placental degeneration with fibrin deposition in the glomeruli cause renal hypertension? Or does hypertension provoke vascular damage by producing a low grade intravascular coagulation especially in the kidney? He proceeds to suggest that both possibilities stem from placental ischaemia, completely oblivious of the fact that the "ischaemic placenta" has long been shed when postpartum eclampsia supervenes, completely denying any responsibility it may have in the toxæmic aetiology. Further, the continuing presence of such a placenta in a uniovular twin pregnancy carried to term when one of the fetuses supplied had perished¹ denies it aetiological significance, for the fetal death of this twin caused a complete subsidence of severe pre-eclampsia, which did not recur during that pregnancy, which continued for several months thereafter with the placenta retained.

Dr. Wardle reports results based on fibrinogen experiments in pregnant rabbits² in which toxæmia had been induced by placental ischaemia. Experimental attempts on these lines have evoked unwittingly the utero-renal stimulus for they have compressed the area so included, dammed back its blood supply, distended the blood vessel supplying it, and in so doing stretched their sympathetic investments thereby provoking a stimulus to the kidney and ensuring a utero-renal reflex with all its consequential effects—including cortical necrosis which Franklin and Sophian's experiments³ had produced by equivalent experiments of faradic stimulation of the renal nerve. More recent investigation of these sections has revealed fibrin depositions in the glomeruli in which haemolysis has been shown to occur. Herein therefore lies the answer to the findings that Dr. Wardle seeks to explain. He has unwittingly proved that this occult stimulus of the utero-renal reflex "placental ischaemia" has caused hypertension—a stimulus that is known to evoke renin from the kidney. Once again, it should be emphasized that postpartum eclampsia completely excludes placental intrusion in the aetiology of pre-eclampsia.

Mention is made by him of placental abruption, but here again reference is incomplete, for it is the distension of the uterus by blood that accounts for both the shock and the anuric syndrome, which has been shown to be overcome by rupture of the membranes and conduction anaesthesia reaching the eleventh dorsal nerve, proving that a renal nervous vasopastic state—more recently confirmed in a parallel condition by Luke *et al.*⁴—existed. They proved this association to depend on oxytocic drugs and to be transitory. These latter findings explain both the cause and the rapid disap-

pearance of the signs of the syndrome on delivery.—I am, etc.,

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- 1 Brown, G. R., *Journal of Obstetrics and Gynaecology of the British Empire*, 1958, 65, 61.
- 2 Berger, M., and Boucek, R. J., *American Journal of Obstetrics and Gynecology*, 1964, 89, 230.
- 3 Sophian, G. J., *Nephropathy of Pregnancy*, in press, London, Butterworths.
- 4 Luke, R. G., Siegel, R. R., Talbert, W., and Holland, N., *Lancet* 1970 2, 750.

Myocardial Infarction and the G.P.

SIR,—I read with interest the letter of Dr. L. C. Bousfield and others (3 October, p. 54). Working in a coronary care unit, I realize that there may be a lack of specific treatment during the prehospital phase of acute myocardial infarction. The fact that in a recent symposium¹ on the prehospital phase of myocardial infarction no emphasis was placed on the general practitioner's contribution in reducing the early deaths following this condition on the one hand, and on the other, the lack of information regarding the general practitioner's treatment of myocardial infarction before the patient's admission to hospital, made me study the following:

- (1) The visit delay—that is, the delay between the onset of symptoms (the time of patient's call for the general practitioner) and the time of the visit of the general practitioner.
- (2) The diagnosis offered by the general practitioner.
- (3) The treatment given by the general practitioner in the patient's home, and
- (4) The information given, in the accompanying letter, about the patient's past and present history.

In a six-month period, 113 patients were admitted to the coronary care unit with the diagnosis of acute myocardial infarction. Of these, 41 were first visited by the general practitioner. The visit delay ranged from 5 minutes to 2 days; the mean visit delay was 3 hours and 48 minutes. The general practitioner made the diagnosis in 27 out of the 41 patients, and treatment was given to 20 patients, and in nearly all consisted of intramuscular pethidine or morphine. The general practitioner's accompanying letter contained useful information in only 5 out of the 41 visited patients.

The condition of the 41 patients on admission to the hospital is summarized in Table I. Complications of myocardial infarction may develop within a matter of minutes, and therefore the findings on admission might not be present at the time of the examination by the general practitioner in the patient's home.

TABLE I.—Condition on Admission

Condition	No.
In pain	34
Dysrhythmias and heart block	14
Left Ventricular Failure	13
Hypotensive (Systolic B.P. < 90)	4
Hypertensive (Diastolic B.P. > 100)	8
Known Diabetics	3
Ventricular Fibrillation on Arrival	1

Most patients developed more than one complication.

There is supporting evidence that bradyarrhythmias and ventricular fibrillation are the cause of most of the early deaths following infarction. In order to reduce this very high prehospital mortality, it is suggested that the general practitioner should visit the patient as soon as possible, cutting down the admission delay, and that he should try to treat not only pain and dysrhythmias, but also left ventricular failure with digoxin and frusemide if it is severe, or with frusemide alone if it is mild. The accompanying letter should contain information about the medical history of the patient, and the treatment given to the patient at home, with drugs, dosage, route of administration, and the exact time given.

I should like to thank Dr. C. S. McKendrick for permission to report this study, and Miss L. J. Bown for secretarial assistance.—I am, etc.,

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REFERENCE

- 1 *American Journal of Cardiology*, 1969, 24, 609.

Living it up with Concorde

SIR,—Squadron Leader A. N. Nicholson and Dr. F. S. Preston (24 October, p. 242) take your correspondent on time zone changes and supersonic flight (3 October, p. 53) to task for suggesting that safety in the air could be compromised by disturbances in the circadian rhythms of aircrew. They do not, however, provide very much evidence to substantiate their criticisms.

It is irrelevant to suggest that the difficulties of adaptation to a new time zone are of less interest to aircrew than to passengers. What matters is that the physiological and psychological rhythms of aircrew after long-distance flights across time zones are likely to be disturbed. We simply do not know at present the full extent or importance of these disturbances.

If, however, they are of importance it could well be that in this respect supersonic aircraft will be safer than present-day aircraft. Their very speed may make it possible to arrange aircrew schedules and duty hours in such a manner that disturbances in circadian rhythms are minimized.—I am, etc.,

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Choreoathetosis in Alcoholism

SIR,—Dr. P. J. Mullin and colleagues (31 October, p. 278) are to be congratulated for drawing attention to transient choreoathetosis occurring during the withdrawal from alcohol. I am surprised, however, that they regard the improvement of one patient with chlorpromazine as evidence that the previous administration of phenothiazines was an unlikely cause.

Oral dyskinesia when induced by phenothiazines is most marked when the drug is withdrawn and is controlled if the dose of phenothiazine is increased. In this connexion it is interesting that dopa induces oral dyskinesia as a direct effect, and it is dose-related. Phenothiazines also reduce dopa-induced dyskinesia, and it is possible that the antagonism of dopamine is closely related to the antipsychotic effect. Thus it seems likely that the alcohol-induced

dyskinesia is similar to that induced by phenothiazines and is not related to liver damage, as the authors postulate.—I am, etc.,

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Undiagnosed Abdominal Pain

SIR,—I read with considerable interest the letter from Drs. Eva A. Frommer and D. G. Cottom (10 October, p. 113) with their advice that some children with recurrent abdominal pain should be treated with antidepressant drugs. It is well and generally recognized that we do not know the correct aetiology or treatment for over 90% of such cases, but this can equally be said for many other simple common ailments of childhood. I think it is small reason, because we do not know these answers, to label some of these children "depressives" and to treat them with powerful and sometimes dangerous drugs. Undoubtedly emotional problems are a factor, but there can be very few severe enough to need more than simple psychotherapy to themselves or their parents. The symptoms put forward as suggestive of depressive illness could equally well be due to and not the cause of the abdominal pain, and I would like to see a little more diagnostic accuracy and commonsense being displayed before embarking on long and often disappointing psychiatric treatment which these small children in the main do not deserve and cannot protect themselves against.

The treatment of depression has made enormous strides in the past few years owing to such drugs, but let us be very sure that it is only those people who really need them who are given them. To embark on courses of treatment with tricyclics and monoamine oxidase inhibitors in childhood must be an indictment of the management of the parents, and surely must not be advocated for a very common and usually entirely benign condition in normal children.

I dispute the last sentence of the letter by Drs. Frommer and Cottom. I agree that missing a case of true depression is serious, but missing an acute abdomen can be fatal. The great danger of placing children on antidepressive therapy is that all abdominal pain is going to be attributed to the depression, and a perforated appendix only diagnosed when the child becomes seriously ill. The last sentence of your leading article (10 October, p. 66) precisely covers what I feel about the treatment of this condition.—I am, etc.,

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Pseudo-obstruction of the Large Bowel

SIR,—As can be seen from the references appended to your Current Practice article by Mr. P. K. Caves and Dr. H. A. Crockard (6 June, p. 583), as well as some of the ensuing correspondence (20 June, p. 732; 27 June p. 793; and 4 July, p. 47), this condition has been featured in the surgical literature from time to time over the past 20 or 30 years.

Yet if the instances described are examined, it is very rare indeed to find one that is not secondary to some other serious condition. In the half dozen or so cases that

I have seen over the past 18 years all have been related to either an intra-abdominal condition, such as acute cholecystitis, or to cardiorespiratory disease, notably bronchitis and emphysema, or recent coronary occlusion. Almost all these patients have had a raised blood urea.

Might I suggest that this condition of pseudo-obstruction of the large bowel, being merely symptomatic, would be far better termed "reactive megacolon." This removes the suggestion of obstruction, emphasizes its secondary nature, and has a precedent in such conditions as congenital megacolon (Hirschsprung's disease) and the toxic megacolon of ulcerative colitis.

I am sure that operation should be avoided in these cases if at all possible. Two of my cases suffered a burst abdomen—one did so four times. (On the fourth occasion, he was left to heal by second intention.) If surgical intervention is considered necessary, then a "blow-off" caecostomy should be all that is required.—I am, etc.,

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Handlebar Injury

SIR,—With reference to the medical memorandum on this subject (24 October, p. 222) I am stimulated to report a further mode of injury which can occur with this type of bicycle. Recently my own son aged seven years travelling at high velocity as described on a bicycle of the same type stopped abruptly and himself continuing forward from the saddle impaled his scrotum on the lever which is intended to lock the telescopic rise and fall of the handlebar.

After the accident his right testicle was absent from the scrotum having been propelled up to the inguinal canal into the abdomen in a similar situation to the undesecured testicle. Surgical opinion was sought urgently because of the risk of incidental torsion. One and a half hours later—following the ambulance journey and a great deal of distress—on arrival at the hospital the displaced gonad was found to have returned to the scrotum. All's well that ends well. But surely, as Mr. J. A. Sneddon suggests, the designers should improve their vehicles.—I am, etc.,

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Self-certification?

SIR,—I agree with Dr. J. D. E. Knox (7 November, p. 363) that short term absence is often a management problem. Most modern managements do not require any certificate for absence of less than three days. However, the various sick clubs run through the trade unions or by committees of management and employees do not have the same faith in their members, and in many cases private notes are required for these organizations.

For some years past in this division we have been telling employees that their National Health Certificate is sufficient evidence for sickness lasting over three days, and we even arrange to forward the certificate to