Flying Squads for Road Accidents

SIR,—There recently has been considerable controversy both public and private over the attendance of doctors at the site of serious accidents. In order to fulfil this gap in medical care (*Supplement*, 8 May, p. 91) several flying squads are in existence, and these fall into three large groups—general practitioner flying squads, casualty department flying squads, and hospital flying squads. The latter can be dismissed rapidly since they almost never function except in a major catastrophe.

There are several general practitioner flying squad services, and notably that in Richmond, Yorkshire, has demonstrated what can be done with enthusiasm and effort. They undoubtedly save lives and decrease morbidity, but in my opinion could give an even higher standard of care if they had the necessary resources.

In Derby a flying squad was originally founded in 1955, and has been functioning increasingly successfully since that time. Its organization, etc., has been previously described,[1] and this is basically unchanged, although it is being continuously improved. Briefly, it is an accident flying squad which is based on the casualty department at Derbyshire Royal Infirmary. It consists of a casualty surgeon, an anaesthetist, and a trained nurse, who are always available, with a comprehensive range of surgical, anaesthetic, and resuscitation equipment to travel to any emergency scene. Perhaps I can quote a recent patient who, but for the existence and efficiency of this squad, would inevitably have died before reaching hospital.

At 1.55 p.m. on 1 June the flying squad was called by the ambulance service to the scene of a road traffic accident since the patient was seriously ill with neck injuries. On arrival at 2.02 p.m. the patient was found to be a motor cyclist of 19 years who had collided with a plate glass window. He was lying on his back unconscious in a large pool of blood which was running down the gutter to a drain some 10 yards away. He was pale with no palpable pulses or audible heart sounds and no respiration; his pupils were small and unreacting; there was still profuse blood oozing from the gaping three inch laceration in his right anterior neck region.

A drip was rapidly inserted and plasma/macrodex infusion begun under pressure. Cardiac massage was started, and the patient intubated and ventilated with an Ambu bag (oxygen being entrained). A police car was despatched for some O Rhesus-negative blood. Every bleeding point in his neck was clamped with artery forceps (eight being eventually left in situ). After approximately 2 litres of fluid had been infused in under 10 minutes a slow pulse became palpable. The bradycardia speeded steadily until he had a good strong rapid pulse, and after some 20 minutes of resuscitation he began to breathe spontaneously. He was given 1 litre of blood and only after this time was he transferred to the ambulance and thence comfortably to the hospital casualty department.

On arrival his B.P. was 160/80 mm Hg and pulse 84/min, he was breathing spontaneously, and moving his right side but not his left. He was taken to theatre and his completely split jugular vein ligated, the carotid artery being intact. His right vagus nerve had been divided together with several small arteries. Some four days after the accident his only residue is a recovering mild left hemiplegia.

We are continually seeing such cases but often they are not so successful or clearcut as the above. Without our flying squad he could not have survived, and I feel he is the complete justification for our service. Medical personnel attending accident victims must be well equipped and well versed in the drastic resuscitation techniques necessary for the survival of such seriously injured patients.—I am, etc.,

KEITH LITTLE

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1 Collins, J., *British Medical Journal*, 1966, **2**, 578.

Infectiousness of Glandular Fever

SIR,—As glandular fever is usually considered an infectious disease it is surprising that there are so few reported cases occurring in both husband and wife. This happened recently within my practice in two normally healthy individuals. The three children aged 5, 2, and 1, have so far not been affected. Because of the difficulty of positively identifying a causal organism, achieving artificial transmission of the disease, detecting a carrier or an immune state, and establishing an incubation period, I feel that it is of interest to describe this "mini epidemic."

The husband, aged 31, attended surgery with a week's history of malaise. Four days later he was in bed with fever, and had developed a generalized lymph node enlargement. By then the laboratory had been able to report a leucocytosis with typical mononuclear cells and a positive Monospot test. After a short course of prednisone he quickly felt better and was able to return to work after 14 days, although there were still some palpably enlarged cervical nodes.

Just over seven weeks later I was called to his 28-year-old wife who had a high temperature, frontal headache, and sore throat of two days' duration. A diagnosis of frontal sinusitis was made and oral ampicillin started. After two days she was no better and she had an acute follicular tonsillitis. A course of twice daily penicillin injections was begun. However, after a further two days she was still acutely ill with a persistently high temperature. A throat swab taken two days previously revealed no pathogens, but she had developed an enlarged right tonsillor node on this the sixth day of her illness. Examination of the blood showed typical mononuclear cells and a positive Monospot test. Next day there was splenic enlargement with generalized lymphadenopathy and she required prednisone for several weeks to suppress her symptoms.

The interval between the husband's first symptoms and those of his wife was 63 days. I would be interested to have details of any other two similar cases occurring in close contacts which might help to establish an incubation period for glandular fever, otherwise known as infectious mononucleosis.—I am, etc.,

RICHARD M. WHITTINGTON

Four Oaks,
Warwicks

Eczema and Detergents

SIR,—I have recently read Dr. D. Blair Macaulay's booklet on allergy produced under the auspices of the B.M.A.[1] While there is much in this work to be commended it is unfortunate that in referring to detergents as a cause of eczema he implies that this eczema is due to an antigen-antibody mediated allergic response. That this is done repeatedly implies a lack of understanding of the conceptual differences between contact irritancy and contact allergy.

True cutaneous allergy to detergents is, of course, very rare indeed, a fact that I trust will not surprise Dr. Blair Macaulay one jot.—I am, etc.,

F. A. IVE

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Dryburn Hospital,
Durham

1 Macaulay, D. B., *You and Your Allergy*. London, Family Doctor, 1971.

Deaths from Tuberculosis

SIR,—Your leading article (22 May, p. 419) on "Deaths from Tuberculosis" is timely, considering the implementation of the Local Authority Services Act (1970). Good results in the control of tuberculosis are still dependent on fully trained health visitors, especially in areas where there are high rates of immigration, bad housing, etc. Their local knowledge is invaluable in seeking out infectious patients and their contacts from obscure lodging houses; their skill in supervising effective chemotherapy at home and in ensuring regular follow up at chest clinics is proverbial. In my opinion, any reduction in their numbers or quality would be premature.

The Report of the Research Committee of the British Thoracic and Tuberculosis Association on tuberculosis mortality in Britain[1] showed that a high proportion of deaths was due to failure to follow straightforward clinical and administrative procedures. I am reminded of how the breakdown of communications delayed successful therapy in another type of mycobacterial infection, as related in the fifth chapter of the Second Book of Kings, verses 1-14. This account is a classical example of the tactful approach, which chest clinics still possess by virtue of their health visitors.

With the concept of the "community physician" playing an increasing part in the coordination of health services, I believe that it behoves chest physicians to try to make sure that the needs of the more vulnerable sections of the population are not neglected for the sake of administrative unity.—I am, etc.,

R. A. L. AGNEW

Liverpool Central Chest Service,
Liverpool

1 British Thoracic and Tuberculosis Association, *Tubercle*, 1971, **52**, 1.

Serum Transaminases during Salicylate Therapy

SIR,—I found the observations of Dr. A. S. Russell and others (22 May, p. 428) that prolonged salicylate therapy may cause an increase in some serum enzymes most interesting.

I have recently measured the serum

aspartate aminotransferase (SGOT) and alkaline phosphatase in 16 adults with serum salicyclate greater than 35 mg/100 ml as a result of single-dose self-poisoning. In only one patient was the SGOT minimally increased to 44 units. This was an apparently healthy 24-year-old man with a serum salicyclate of 80 mg/100 ml four and a half hours after taking aspirin tablets; his alkaline phosphatase was normal. A 60-year-old man with a serum salicyclate of 131 mg/100 ml eight hours after ingestion was the only patient with a raised alkaline phosphatase (16 King-Armstrong units); his SGOT was normal

These findings tend to support the implied conclusion of Dr. Russell and colleagues that, age apart, increases in these enzymes in serum are probably related to prolonged administration of salicylates.—I am, etc.,

M. LEE-JONES

Dudley Road Hospital,
Birmingham

E.E.G. and Anticonvulsants

SIR,—While I must agree with Miss Coleen Darby and Dr. D. Fung (8 May, p. 341) that alteration of the state of awareness may influence the E.E.G., such marked slowing of the E.E.G. as I described (24 April, p. 207) is most unusual in normal states of diminished awareness (for example, drowsiness or mild confusion), and is seldom seen in cases of drug intoxication. In the case of anticonvulsant toxicity reported the patient was alert enough to open her eyes to command as shown (Fig. 1), and was also able to perform three minutes of hyperventilation with only a single encouragement. No specific response was seen to arousal stimuli ("K" complex), nor were sleep spindles seen as might be expected in such loss of awareness resulting in the widespread delta activity illustrated, if this were due to drowsiness or sleep. Thus the E.E.G. changes shown are presumably the result of the excessive drug therapy and are present as a complication, with the low serum folate, of the medications. At the time of the second E.E.G., eight days after the initial tracing, the patient was clinically much improved, up and about the ward, and was no longer confused or disorientated.

It is well known that very many factors may be associated with slowing and subsequent return to normal of cerebral rhythms. I reported this case to illustrate the association of a low serum folate in anticonvulsant intoxication with E.E.G. change, and to stress the value of the investigation in assessment and follow up of such cases.—I am, etc.,

W. I. M. DOW

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Southampton General Hospital,
Southampton

Diet and Diverticulosis

SIR,—Mr. N. S. Painter and Mr. D. P. Burkitt (22 May, p. 450) argue from clinical records that the increasing incidence of diverticulosis and diverticulitis in man is due to eating diets deficient in roughage.

Many years ago, when I was on the staff of the Rowett Institute in Aberdeen, I took part in a long-term experiment with rats on a human dietary.[1] A large colony of rats was kept for several years on a diet which mimicked accurately the foods eaten by the people of Peterhead, a coast town in Aberdeenshire. One group of rats was kept on the "Peterhead" diet alone, another group had the "Peterhead" diet supplemented by the addition of milk and of green vegetables. The environment and treatment of both groups was otherwise identical. The rate of growth, the reproductive capacity, and the general health of the rats was followed generation after generation: each animal, when it died from illness or "old age," was examined.

Many of the old rats on the unsupplemented "Peterhead" diet had a condition of the gut which macroscopically closely resembled diverticulosis and diverticulitis in man. Microscopically, however, the condition differed from that in man. Some of the rats on the diets supplemented with milk and green vegetables also suffered but the incidence was not so great. These findings were reported in the *B.M.J.*[2]

We were commendably cautious and refused to jump to conclusions. Unfortunately, the intention to extend and elaborate the experiments on the rats with special reference to the condition of the gut could not be put into effect.—I am, etc.,

R. C. GARRY

Laich Dyke,
Dalginross,
Comrie,
Perthshire

1 Orr, J. B., Thomson, W., and Garry, R. C., *Journal of Hygiene*, 1936, **35**, 476.
2 Lubbock, D. M., Thompson, W., and Garry, R. C., *British Medical Journal*, 1937, **1**, 1252.

Comparison of Drugs for Asthma

SIR,—Dr. K. N. V. Palmer and others (27 March, p. 727) have done a one-dose, nonblind test comparing 300 mg of proxyphylline (a theophylline compound) with two new β_2-receptor stimulant drugs. They found no significant changes after this proxyphylline, and conclude that "proxyphylline . . . cannot be recommended for the relief of asthmatic airway obstruction at least when given by mouth." They seem to be unaware of the fact that such a dose will not give higher serum level than a maximum of 8 μg/ml. Hauge and Gamst[1] found that a single oral dose of 400 mg of proxyphylline produced a maximal serum level of about 8 μg/ml. Recent studies[2] in five healthy volunteers using proxyphylline 600 mg twice daily yielded a mean serum level of 10 μg/ml during 1-3 hours after the first dose of 600 mg. After five days of therapy the serum levels at the corresponding times were around 18 μg/ml.

We have done two double-blind studies comparing proxyphylline in two dosages and placebo in 17 patients with chronic obstructive airway disease. Reversion of bronchoconstriction was evident by a mean increase of FEV_1 of 30% after two inhalations of Isoprenaline Medihaler O. Proxyphylline 150 mg four times daily for 48 hours produced a mean serum level of $8{\cdot}7 \pm 0{\cdot}9$ μg/ml but no significant changes in FEV_1, either after 24 or after 48 hours. With a dose of 300 mg four times daily, however, the serum level averaged $18{\cdot}3 \pm 1{\cdot}1$ μg/ml after 48 hours. FEV_1 increased on the average 8·5% after 24 hours ($p<0{\cdot}05$) and 18·7% after 48 hours ($p<0{\cdot}01$). Placebo therapy produced no significant changes in FEV_1.

These data confirm that proxyphylline, given in a sufficient dose for a time long enough to produce adequate serum levels, produces a statistically significant and a clinically noticeable reversion of airways obstruction, as measured by the FEV_1. Thus, I cannot agree with the statement quoted above.—I am, etc.,

LENNART TIVENIUS

Örebro, Sweden

1 Hauge, H. E., and Gamst, O. N., *Tidskrift for den Norske Laegeforening*, 1967, **87**, 535.
2 Graffner, C., Personal communication.

Analgesics in Terminal Disease

SIR,—A patient of mine has recently returned from a teaching hospital with unhappy memories. She has extensive pelvic carcinomatosis needing narcotic analgesics, but because she was written up four-hourly she often underwent an hour or more of agonising pain before she could be given the next dose.

She is now at home with a liberal supply of analgesics by her bedside and for the moment is more or less relaxed and free of pain. She has made it quite clear however that she will not consider returning, when the need arises, to the same hospital but only to her local cottage hospital. Here, although she knows that she will not receive the same degree of surgical skill, she is assured of relief at any time and that no one, and especially herself, will be watching the clock.

Is it not time that we adopt a more liberal attitude to prescribing and abandon the rigid time schedules?—I am, etc.,

P. B. SCHOFIELD

Walton on Thames

Levodopa and Anticholinergic Drugs in Parkinsonism

SIR,—Dr. R. C. Hughes and others (29 May, p. 487) described the effect of withdrawal of anticholinergic drugs on patients receiving levodopa for Parkinsonism. They found that only 11 out of 34 patients were able to tolerate withdrawal for more than eight weeks. They suggest that "the synergism which seems to exist between anticholinergic remedies and levodopa may be due to inhibition of dopamine inactivation by anticholinergic drugs."

The fact that patients suffering from Parkinsonism require anti-acetylcholine drugs may have a different explanation. We know that dopamine is released in the corpus striatum by impulses originating in the substantia nigra. Thus the nigra-striatal tract is composed of fibres closely resembling sympathetic postganglionic fibres except that the former release dopamine and the latter release noradrenaline. Recently the evidence that sympathetic fibres release noradrenaline through the prior release of acetylcholine has been greatly strengthened.[1] This release of acetylcholine becomes obvious when noradrenaline is removed, as by the injection of reserpine, for then stimulation of sympathetic fibres to the heart,[2] the spleen,[3] and the blood vessels releases acetylcholine which is not used in releasing noradrenaline.

It might therefore be expected that when