CORRESPONDENCE

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Working Hours of Junior Staff

SIR,—We are members of the consultant clinical staff of King's College Hospital. King's is one of the hospitals chosen by the Department of Health and Social Security for a preliminary study of the effect of the reduction in hours of duty of junior hospital staff which is due to come into effect on 1 July. A small team from the Department has been working at King's for some time and recently they put some of their tentative proposals to the physicians and surgeons.

As far as we can see, if duty hours are reduced one of the following must result:

(*a*) Junior doctors continue to work the same duty hours as at present but are paid more—that is, some duty which is now routine becomes "overtime".

(*b*) The efficiency of the junior hospital doctors' work is in some way improved so that they accomplish as much as they do now in a shorter time.

(*c*) More junior doctors are recruited to fill the gaps.

(*d*) Senior hospital doctors work harder than they do at present and cover their own juniors one night and one weekend in three.

(*e*) The standard of hospital service declines.

There are objections to all these points:

(*a*) There is, as far as we can gather, no intention that the Department will make more money available to hospitals to implement these new proposals. If junior doctors claim more overtime pay it would be at the expense of other hospital activities.

(*b*) Junior hospital doctors already claim overtime payment on occasions, which suggests that they cannot always keep up with their work under the present duty periods. From our experience we do not see how more than marginal improvements in the efficiency of their methods of work can be achieved, nor do we know of any proposals to this end.

(*c*) There is already a shortage of junior hospital doctors; the Department, as far as we know, does not propose to provide money to employ more.

(*d*) The D.H.S.S. team at King's suggested that on some of the firms the consultants should act as their own juniors on one night in every three. The team was considering only one hospital, which perhaps explains why they did not think of the consequences of this proposal for the consultant who is on the staff of two or even three hospitals. We are no more willing to act as our own juniors than are the consultants of Cheltenham (9 March, p. 459). Furthermore, this proposal might have an effect on recruitment to the consultant ranks. The alternative appeal of general practice might appear even more attractive than it does at present. The junior doctors who have pressed for a reduction of hours may not have considered all the consequences which success would bring for themselves. After about six or seven years as registrars many of them will have 30 years as consultants.

(*e*) If junior doctors' working hours are reduced we cannot see how a decline of service to patients can be avoided. Not only will there inevitably be discontinuity of clinical care but the quality of that care is likely to suffer. The houseman who is called to a patient in the evening may never have seen him before and know little about his disease or its treatment. For example, the D.H.S.S. team at King's proposed that specialist firms should be on duty for each other; thus the cardiac firm's house physician would be first on call for the liver unit. The management of the patient with bleeding oesophageal varices is not likely to be improved by this arrangement.

We find it difficult to understand how any representatives of hospital authorities could have agreed to the proposed reduction in the working hours of junior staff.—We are, etc.,

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Naproxen (Naprosyn) and Gastrointestinal Haemorrhage

SIR,—One of the most difficult things in assessing the merits and demerits of any new drug, the therapeutic debit/credit balance as it were, is the frequency and severity of the side effects it may cause. The rightful place any new drug will take in current therapeutics is usually not apparent until it has been in general use for several or many months. Whatever the results of the initial clinical and pilot trials may have been, the frequency and severity of side effects become truly apparent only after the drug has been widely prescribed to the general

public, which is, of necessity, the ultimate guinea-pig. It was only after some years of general use of the corticosteroids that cataract formation was recognized as a late complication, and the ocular complications of chloroquine and the haematological hazards of phenylbutazone and oxyphenbutazone were not immediately apparent. The antirheumatic agent ibufenac was withdrawn soon after general release because of hepatic toxicity.

Where anti-inflammatory/analgesic (antirheumatic) compounds are concerned it is the upper gastrointestinal tract that is particularly at risk, for in greater or lesser degree all drugs of this class can cause dyspepsia, mucosal erosion, haemorrhage, deeper ulceration, and even perforation. One of the best tolerated of the newer antirheumatic agents has been ibuprofen (Brufen), and recently a number of other propionic acid substances have been marketed in the hope that they might prove more effective clinically while no more toxic than this agent.[1] One of these newcomers is naproxen (Naprosyn). In many clinical trials[2] this substance proved less toxic to the upper alimentary tract than aspirin and other antirheumatic agents. Hill *et al.*[3] deliberately gave the drug to 27 patients who had suffered major gastrointestinal symptoms on other agents; only three of the 27 had persistent reactions to naproxen. Gastroscopic studies[4] on 12 healthy volunteers showed evidence of gastric irritation in all 12 after taking 4·86 g daily of aspirin for seven days but in only one subject after taking naproxen 500 mg daily for the same period. The very careful and extensive trials on this substance showed it to be in general well tolerated by the upper gastrointestinal tract.

Nevertheless, since it has been generally released I have in the past three months had to discontinue naproxen because of gastrointestinal intolerance in six cases, three having gastrointestinal haemorrhage, necessitating emergency admission to hospital for blood transfusion in two cases.

Case 1—A man aged 64 with rheumatoid arthritis had had melaena on indomethacin four years previously. He remained reasonably well and free from gastrointestinal symptoms on enteric-coated prednisolone 2·5 mg twice daily until he was started on naproxen 250 mg twice daily on 22 December 1973. Two weeks later, on 7 January 1974, abdominal pains commenced and gradually increased. On 17 January he noted that his stools had become black, and he discontinued naproxen on 21 January. When I saw him on the next day he had obvious melaena and was anaemic. He made an uneventful recovery on conservative measures without admission to hospital or blood transfusion.

Case 2—A woman aged 68 with osteoarthrosis had been free from gastrointestinal symptoms while on phenylbutazone 100 mg twice or thrice daily over the previous five months. Ten days after changing to naproxen 250 mg twice daily profuse melaena occurred, necessitating emergency admission to hospital for blood transfusion.

Case 3—A man aged 43 had had intermittent polyarthritis for nine years. In February 1973 he had a severe haematemesis probably due to indomethacin. A barium meal at the time showed no ulcer but doubtful varices at the lower end of the oesophagus. A repeat barium study in November 1973 showed the same appearance, but no varices were seen on oesophagoscopy. D-Penicillamine 250 mg (base) twice daily and naproxen 250 mg twice daily were started. Three weeks later a brisk and heavy haematemesis led to an emergency admission to hospital for blood transfusion. Naproxen was discontinued and D-penicillamine continued 250 mg thrice daily and he improved steadily.

In none of the six cases in which naproxen had to be discontinued was there anything to suggest alcoholic or dietetic indiscretion. Gastrointestinal upsets are notoriously unpredictable, particularly in rheumatoid arthritics, and an antirheumatic drug tolerated for months or even years may suddenly produce gastric complications. In the three cases quoted above naproxen was used because of its reputed non-toxicity. Nevertheless, gastrointestinal intolerance rapidly became evident. These cases are reported only to advise caution in the use of a new antirheumatic agent which is being very widely used.—I am, etc.,

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1 Hart, F. D., *Pharmaceutical Journal*, 1973, 211, 519.
2 *Scandinavian Journal of Rheumatology*, 1973, suppl. 2.
3 Hill, H. F. H., *et al.*, *Annals of the Rheumatic Diseases*, 1974, 33, 12.
4 Halvorsen, L., Dotevall, G., and Sevelius, H., *Scandinavian Journal of Rheumatology*, 1973, suppl. 2. p. 43.

SIR,—It is my practice to check for occult blood in the faeces of patients who are on long-term antirheumatic therapy in view of the known tendency of many antirheumatic agents to cause gastrointestinal bleeding.

Recently I have observed what appears to be an unusually high incidence of positive results in patients treated with naproxen. In order to check whether this may be of any significance I have taken a group of 43 patients, 22 being treated with naproxen and 21 being treated with benorylate. The results of this analysis were as follows: naproxen group of 22 patients—faecal occult blood positive in nine; benorylate group of 21 patients—faecal occult blood positive in four.

I draw no definite conclusions from this work but thought it right to report these results in order to focus attention on the position and to learn other people's experience of the drug, with particular reference to possible gastrointestinal bleeding. —I am, etc.,

S. G. FLAVELL MATTS

Nanpantan, Leics

Aspirin and Myocardial Infarction

SIR,—I write to ask why your leading article (9 March, p. 408) was entitled "Aspirin and Atherosclerosis"? Both the excellent papers you printed in the same issue are concerned with myocardial infarction, though of course I accept that the degree of atherosclerosis is likely to be considerably higher in patients who have had an infarct than in controls. It is also important to distinguish between myocardial infarction and coronary artery thrombosis. Even if at postmortem a thrombus is found in a coronary artery, it is still not established whether it is the cause or the result of the infarct. Clearly these terms are not coterminus.

I entirely agree with Dr. P. C. Elwood and his colleagues (9 March, p. 436) that "platelet aggregation to collagen . . . may not be the most relevant measurement of [? in] the context of coronary artery thrombosis." We know aspirin prolongs the bleeding time—an undoubted in vivo effect—and that it inhibits the release reaction in vitro; but we do not know for sure what contribution the release reaction makes in haemostasis, let alone infarction; nor do we know for certain what initiates platelet deposition in the coronary artery. Thus we do not know which tests are relevant. Nevertheless, I agree with Dr. Elwood that it is essential, in so important a clinical condition and with so safe a drug, to try a higher dose level, even though 2·4 g of aspirin daily had no effect in a small trial in preventing postoperative deep vein thrombosis, which is at least in part a different pathological process.[1] A drug with so many pharmacological effects could well alter other platelet functions, though none are recorded; just as plausibly it could alter other mechanisms involved in myocardial infarction.

While I am writing may I correct a reference? There is no such journal as *Haemorrhage*. Dr. Elwood's reference 7 will be found in *Thrombosis et Diathesis Haemorrhagica*, 1966, **16**, 752.—I am, etc.,

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1 O'Brien, J. R., Tulevski, V., and Etherington, M., *Lancet*, 1971, 1, 399.

SIR,—Your leading article (9 March, p. 408) refers to the potential value which continuous intake of aspirin may have in protection against atherosclerotic heart disease. Your evidence for a negative relation between aspirin and atherosclerosis is partly based on a necropsy study[1] in which a strikingly lower incidence of fatal myocardial infarction in patients with rheumatoid arthritis was found in comparison with control cases. However, is it justified to explain the low incidence of atherosclerosis in patients with rheumatoid arthritis by the intake of aspirin over long periods?

It seems relevant to refer to an investigation[2] in which the ventral costal cartilages were radiographed in 95 patients with rheumatoid arthritis and in 107 patients with osteoarthrosis between the ages of 30 and 80+ years. In both groups of patients a lateral view of the abdominal aorta was also taken. There was a very low incidence of marked calcification of the ventral costal cartilages and of the aorta in the patients with rheumatoid arthritis but a high incidence of marked calcification of these tissues in the patients with osteoarthrosis. Roughly 75% of the patients with rheumatoid arthritis were women. Taking into account that calcification of the ventral costal cartilages starts at an earlier age and is more intense in females than in males[3] and that calcified aortas are more common in women than in men after the age of 65 years[4] the differences were even more striking.

The concept was advanced that in rheumatoid arthritis the osteoporosis and the low incidence of significant calcification of the ventral costal cartilages and of calcified atherosclerosis could be ascribed to a common denominator—lack of affinity of mesenchymatous tissues for calcium. The result of the above-mentioned necropsy study is also in support of the concept of a negative relation between rheumatoid arthritis and atherosclerosis. In view of the inherent antagonism between the two diseases it is suggested that patients with