used in many places; but the occurrence related by my patient was the first example of this use of music about which I have heard.

It would be interesting to hear if others have tried the use of music in this way and, if so, whether it is thought to have given significant satisfaction and relief of anxiety to patients.

JOHN LUCEY

Hockering,
Woking, Surrey

Clinical rheumatology

SIR,—Clinical rheumatology may have advanced, but Dr A St J Dixon (16 April, p 1015), while making some comments about rehabilitation with which many would agree, singularly fails to progress the discussion about what clinical rheumatologists should do. Like Professor W W Buchanan (11 September 1976, p 628) he works in a specialised unit serving a large population and from this intense but restricted experience generalises about what is needed in a district general hospital. As one who works in a district general hospital in an area generously endowed with rheumatologists I find it difficult to recognise the picture he paints.

Discussions about rheumatology are bedevilled by varying estimates of the prevalence of rheumatoid arthritis, most of which are based on criteria^{1,2} which almost certainly overestimate the amount of clinically significant disease—for example, the British League against Rheumatism has two current publications quoting prevalence rates of 1%³ and 1% and 2%.⁴ The confusion is well illustrated by the Sudbury report,⁵ which revealed a prevalence of probable or definite rheumatoid arthritis of 2.5% using the old criteria² and of 0.35% using the much more stringent 1966 New York criteria.⁶

If the Sudbury experience using the New York criteria is projected on to a moderate-sized district general hospital serving a population of 150 000 it suggests that in this group there would be only 500–550 patients with rheumatoid arthritis. At least half of these would have only mild or moderate involvement, and when one considers that all the other generalised rheumatic diseases combined form a smaller problem than rheumatoid arthritis itself it can be seen that at such a hospital there is no major career to be made out of looking after “rheumatoid arthritis and its complications and those diseases that resemble it in some way.”

Mechanical and degenerative disorders of the locomotor system are the commonest rheumatic problems—the field of orthopaedic medicine—and one of the major problems of clinical rheumatology is to decide who should look after these patients in hospital. If orthopaedic surgeons abound one of the solutions proposed by Dr Dixon of a person combining internal medicine and rheumatology who would deal primarily with polyarthritis may be an excellent answer, though as internal medicine will itself always be a demanding discipline there may be a danger of creating a number of consultants for whom rheumatology is only a second interest. If orthopaedic surgeons are scarce then there may be a useful role for the rheumatologist who tackles both orthopaedic medicine and polyarthritis.

There is no clear solution and geographical factors will always play an important role in delineating some appointments. None the less,

using the district general hospital concept we need more discussion about what type of rheumatologist is required there and in particular what the relationship to orthopaedics should be. Without this discussion we know neither how many rheumatologists are needed nor how best to train them.

PETER BLOWER

Greenwich District Hospital,
London SE10

- ¹ Ropes, M W, *et al*, *Bulletin on Rheumatic Diseases*, 1958, 9, 175.
- ² Kellgren, J H, *Bulletin on Rheumatic Diseases*, 1962, 13, 291.
- ³ *The Challenge of Arthritis and Rheumatism*, British League against Rheumatism, 1977.
- ⁴ *Rheumatism, the Price We Pay*, British League against Rheumatism, 1977.
- ⁵ Cathcart, E S, and O'Sullivan, J B, *New England Journal of Medicine*, 1970, 282, 421.
- ⁶ Bennett, P H, and Burch, T A, *Bulletin on Rheumatic Diseases*, 1967, 17, 453.

Cimetidine in alleviation of gastric cancer pain

SIR,—We have noted significant pain relief in two patients with malignant gastric ulcers who have been treated with cimetidine.

The first patient, a man aged 63, was referred with a history of dyspepsia for several years which had become worse in the previous three months. Barium meal confirmed the clinical diagnosis of a 1.5-cm lesser-curve gastric ulcer. Cimetidine 200 mg thrice daily and 400 mg at night was started. When the patient attended for gastroscopy three weeks later he reported complete relief of his pain. At endoscopy the mucosa was growing into the ulcer, which was obviously healing. Biopsy showed adenocarcinoma. Subtotal gastrectomy was subsequently performed.

The second patient, a man aged 66, was referred with severe epigastric pain due to carcinoma of the stomach with liver secondaries. The diagnosis was confirmed histologically by endoscopy. Laparoscopy confirmed the clinical impression that palliative surgery was not feasible. Cimetidine 200 mg thrice daily and 400 mg at night resulted in significant relief of pain after seven days.

We consider that the symptomatic relief of pain in malignant gastric ulcer by cimetidine can be misleading and emphasises the need for endoscopic and histological assessment of gastric ulcers, but that this drug is a useful addition for the clinician in the relief of pain from gastric cancer.

C L WELSH
J L CRAVEN
DAVID HOPTON

York District Hospital,
York

Thyroid function tests and oral contraceptives

SIR,—The paper by Dr W A Burr and others (19 February, p 485) has shown that the derived free thyroxine index (FTI) may give fallacious results in certain groups of patients.

We have been interested in the measurement of thyroid function during the ovulatory cycle in normal women and in women taking oral contraceptives. Serum total thyroxine (T₄) was measured by a radioimmunoassay method based on that of Challand *et al*.¹ Triiodothyronine (T₃) uptake was measured by a commercial kit method (Thyopac-3, Radiochemical Centre, Amersham). The FTI was calculated for each sample. Serum T₄ and serum T₃ uptake were measured on the 5th and 20th days of the ovulatory cycle of 20 women (controls). The FTI (T₄/T₃ uptake)

was calculated from these determinations. Serum T₄ and T₃ uptake were measured on the 20th day of the cycle of 20 women taking Minilyn (lynoestrenol 2.5 mg and ethinyloestradiol 0.05 mg).

There was no significant difference between T₄, T₃ uptake, and FTI between the samples taken on the 5th and 20th days of the ovulatory cycle of the control group. Comparison of the FTI between the control group and those patients receiving Minilyn showed a highly significant increase in the FTI ($P < 0.0001$ using the Mann Whitney test) in the patients on oral contraceptives (see table). However, while there was good correlation ($r = 0.82$) between the T₃ uptake and serum T₄ in the control group on the 20th day of the cycle, in the patients taking Minilyn the correlation between these two values on the 20th day was very poor ($r = 0.15$).

Derived free thyroxine index in controls and patients taking Minilyn

	Controls		Oral contraceptive group (20th day)
	5th day	20th day	
Mean	87.35	83.4	111.55
Standard deviation	8.25	10.22	22.74
Range	76–99	61–98	70–163

This suggests that the use of the calculated FTI using the Thyopac-3 method can give falsely high results in patients taking Minilyn.

O DJAHANBAKHCH

Chelsea Hospital for Women,
London SW3

J BARRACLOUGH
T HARGREAVES

Area Department of Pathology,
Exeter

D W SYKES

Royal Devon and Exeter Hospital,
Heavitree, Devon

¹ Challand, G S, Ratcliffe, W A, and Ratcliffe, J G, *Clinical Chimica Acta*, 1975, 60, 125.

Appendicitis and diabetes

SIR,—Professor J P Corridan and his colleagues report (30 April, p 1135) that they found no association between appendicitis and diabetes in individual patients. They assume that since both of these diseases have been attributed to the refining of carbohydrate foods such an association was to be expected.

Their failure to find an association in no way invalidates the hypothesis that these diseases share common causative factors. Appendicitis has been attributed to the increased viscosity of faeces caused by loss of water from the colon resultant on loss of fibre from the diet. Diabetes on the other hand has been related to increased consumption and increased rate of absorption of starch and sugar stripped of their natural fibre packing.

It is possible to increase sugar intake considerably while consuming a diet with adequate fibre to maintain a large soft faecal volume. A friend of mine with 35 years' experience as a medical missionary in Africa saw diabetes appear in his area after the introduction of sugar and sweetened drinks and over 20 years later saw the cessation of new cases when these commodities became no longer obtainable. He saw no case of appendicitis in an African during this time.

Daily stool weights in this community average over 400 g.

Whereas appendicitis and diabetes have been postulated to result from two different aspects of food refining, diverticular disease and varicose veins have both been attributed to changes in faecal viscosity and volume which not only cause increased intracolonic pressure but also increased intra-abdominal pressure during straining a stool. A positive correlation between these two diseases has been reported in individual patients.¹

Professor Corridan and his colleagues are to be congratulated on their study for it is just such a search for associations between diseases in individuals which can be so informative and which is so neglected. It is hoped that others will follow their commendable example and look for other disease correlations.

DENIS BURKITT

Geographical Pathology Unit,
St Thomas's Hospital,
London SE1

¹ Latto, C, Wilkinson, R W, and Gilmore, O J A. *Lancet*, 1973, 1, 1089-1090.

Raised mean cell volume and *Mycoplasma pneumoniae* infection

SIR,—I am writing to correct the impression given by Dr R R Khaund (7 May, p 1220) that a raised mean cell volume (MCV) together with an abnormally raised mean corpuscular haemoglobin (MCH) is "almost diagnostic of the presence of cold agglutinin." It is usual to find a raised MCH with a raised MCV whatever the cause. A more specific clue sometimes seen with a cold agglutinin is a raised mean corpuscular haemoglobin concentration (MCHC). This is due to a falsely low computation of the packed cell volume (PCV) by the Coulter S counter. The passage of agglutinates of two or more red cells at a time through the counting orifice results in a fall in the measured red cell count. This should be compensated by an equal increase in measured MCV, but loss of linearity at the very high MCV figures presumably diminishes this effect. As Dr Khaund points out, warming the specimen corrects these artefacts.

J S WAINSCOT

Pathology Laboratory,
Stoke Mandeville Hospital,
Aylesbury, Bucks

Arthritis in Flemish paintings

SIR,—Hanging the weightiest of theories on the flimsiest of evidence is a favourite pastime of art historians, but should medical history follow their example? Professor J Dequeker's five cases of rheumatoid arthritis are surely nothing of the sort (7 May, p 1203).

The fingers of the Rombauts Christ are a bit floppy, but then someone has driven a six-inch nail through them; and besides, what would be the motive for such a debasement of the Holy Image? (as Holmes would have asked his medical colleague). Surely painting Christ with rheumatoid arthritis would end the painter's career. The Gossaert hands are, as the author suggests, probably merely a mannerism of style, and the few hurried penstrokes in the Van Eyck are hardly enough to hang a diagnosis on.

The other two pictures do seem to show real pathology, but is it rheumatoid? The girl in

the Jordaens grips so easily with her other hand that it is difficult to believe that she has as bad arthritis as it might seem. I suspect that it was not the painter's intention to make the metacarpophalangeal joints appear swollen but merely to bring out their contour for artistic reasons, to contrast with the expanse of white in the ruff. The Van Gent portrait is so frank that the abnormal joint must be taken at face value and, perhaps more specifically, nose value! The man obviously has congenital syphilis. Could the joint not be explained on this basis?

If these are the only examples of joint disease to be found in Flemish painting, it seems good evidence that rheumatoid arthritis was every bit as rare as we thought.

J C W EDWARDS

St Bartholomew's Hospital,
London EC1

Coronary heart disease and the menopause

SIR,—You confuse three issues in your leading article on this subject (2 April, p 862). These are: the relationship of a premature menopause to premature coronary heart disease (CHD); the relationship of a normal menopause to changes in CHD prevalence postmenopausally; and the effects that hormone replacement therapy given during a normal menopause might have on the incidence of CHD. It is necessary to separate them.

The evidence that a premature menopause can lead to premature CHD is stronger than you imply. Two long-term follow-up studies, one of patients with bilateral ovariectomy versus those with unilateral ovariectomy¹ and the other of women with a spontaneous menopause under the age of 40,² showed a higher than expected incidence of CHD 10-20 years later (see table). The Swedish study of women aged 50-54 showed that 76% of women with myocardial infarction had their menopause before 50 compared with 48% of healthy women.³ Further support has come from the finding that 20% of 145 women who had CHD before the age of 45 were postmenopausal,⁴ and the new Framingham evidence⁵ is also supportive although based on small numbers, as you emphasise. This applies to all these studies, since CHD is still uncommon in young and middle-aged women.

Effect of menopause on incidence of CHD

Groups	Age range (years)	Total Nos	Patients with CHD	
			No	%
Spontaneous premature menopause: ²				
6-10 years later	36-45 (mean 38)	10	0	—
11-15 years later	40-47 (mean 43)	13	2	15
16-20 years later	43-55 (mean 48)	12	3	25
Ovariectomy: ¹				
Bilateral	37-58 (mean 51)	35	9	25
Unilateral without hysterectomy (control group)	42-58 (mean 52)	31	1	3
Edinburgh group				
general practice	35-39	681	7	1.8
(5-year cumulative prevalence)	40-44	444	8	1.0
	45-49	487	14	2.9
	50-54	464	15	3.2
Framingham study ⁵				
(8-year cumulative prevalence)	30-39	1000*	1	0.1
	40-49	1000*	20	2.0
	50-59	1000*	67	6.7

*Standardised incidence per 1000 population.

But, as you say, the evidence that a menopause occurring at a normal age puts women at an immediate greater risk for CHD is not strong. Yet this does not contradict the first premise because the studies relating a premature menopause to CHD showed that there was a 15-20 year delay before an increase in incidence was evident. If the analogy were true for normal women it would be around 65 years that a higher incidence might be expected. At this age there are so many compounding influences that it is probable that the effect of a normal menopause would not be discernible and studies of mortality data would not be contributory.

Then the effect of hormone replacement therapy is an altogether different question. In discussing hormonal protection of women against CHD you comment, "Surely it is most unlikely that the same hormones could both protect from and predispose to the same disease." This is illogical. They are not the same hormones. Premature cessation of ovarian function does not lead solely to withdrawal of oestrogenic and progestational hormones but has sudden and profound effects during reproductive viability on androsterone, adrenal and pituitary function, and on lipid and carbohydrate metabolism. Replacement treatment during a normal menopause in order to maintain the physiological premenopausal hormonal balance has not been tested so far as I am aware. I agree, however, with your assessment that artificial delay of the menopause by oestrogenic compounds is unlikely to prevent CHD and, indeed, they may actually increase the risk in women who are already at risk for other reasons.

M F OLIVER

Department of Cardiology,
Royal Infirmary,
Edinburgh

¹ Oliver, M F, and Boyd, G S, *Lancet*, 1959, 2, 690.

² Szajderman, M, and Oliver, M F, *Lancet*, 1963, 1, 962.

³ Bengtsson, C, *Acta Medica Scandinavica*, 1973, suppl 549.

⁴ Oliver, M F, *British Medical Journal*, 1974, 4, 253.

⁵ Kannel, W B, et al, *Annals of Internal Medicine*, 1976, 85, 447.

⁶ Dawber, T R, et al, *Proceedings of the Royal Society of Medicine*, 1962, 55, 17.

Streptokinase, cerebral vascular disease — and triplets

SIR,—Your leading article on streptokinase (9 April, p 927) states that there are no reports of its use in cerebrovascular disease. May I refer you to a study of cerebral vascular disease in pregnancy¹ which included a case report of a patient with triplets who developed an occlusion of the left middle cerebral artery at 29 weeks. Repeated episodes of paresis and dysphasia over the next four weeks suggested recurrent embolism of the left middle cerebral artery. After considering the options it was decided to treat the lesion by anticoagulation.

Streptokinase was infused directly into the left internal carotid artery in a total dose of 40 000 U over six hours. Treatment was then continued with intravenous heparin. At five hours the patient was speaking clearly and a carotid angiogram was normal. Nine hours after the start of the streptokinase infusion the patient went into labour and one hour later was delivered of male triplets weighing 1.7 kg, 1.5 kg, and 1.7 kg, the first two of whom survived. Total blood loss was 500 ml despite full heparinisation throughout labour and