usually rapidly reversible if the early symptoms are taken as an indication to discontinue the oral contraceptives. Some patients do, however, have difficulty in regaining good health after stopping the pill. We have recently found that oral contraceptives and smoking are the most important contributory factors to patients requiring emergency treatment at an acute migraine clinic.⁵ Both men and women smokers come to the clinic after smoking for an average of 20 years, while women taking oral contraceptives come for treatment after an average of less than three years of exposure to the pill. This suggests to me that oral contraceptive use is a more potent cause of vascular disease than smoking, and this has not been generally realised because of the very high oral contraceptive discontinuation rate.

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¹ Gillman, T, in *Biological Aspects of Occlusive Vascular Disease*, ed D G Chalmers and G A Gresham. London, Cambridge University Press, 1964.

² Osterholzer, H O, et al, *Obstetrics and Gynaecology*, 1977, **49**, 227.

³ Grant, E C G, *British Medical Journal*, 1968, **3**, 402.

⁴ Grant, E C G, *British Medical Journal*, 1969, **4**, 473.

⁵ Grant, E C G, et al, *The Migraine Trust International Symposium*, 1976. in press.

A case of twisted logic

SIR,—In your "Clinics in General Practice" (14 May, p 1260) a patient with a three-week-old injury to the ankle requests an x-ray. In spite of the fact that there is no clinical evidence of a fracture the GP trainee is advised to refer her for x-rays for medicolegal and psychosocial reasons.

There are no medicolegal reasons for taking x-rays. There are, however, medicolegal reasons for making an adequate examination and recording the findings on the notes, including a statement that there is no clinical evidence of a fracture. And, if there is any doubt, there are medicolegal reasons for asking the patient to attend for review. Having fulfilled these obligations, no doctor should order x-rays because of an imagined legal obligation. His obligation is to the patient, not to his lawyer. To order x-rays for psychosocial reasons may indeed cut short a consultation and save time on a particular case, but the general effect will be to perpetuate the myth that x-rays are needed in all injuries, thus increasing the work load for the future. In this case the consultant admitted there was no physical indication for radiography but had no hesitation in consenting to an x-ray in order to put on "an impressive show of strength" and convince the patient that there was no fracture. In effect he was putting on an impressive show of weakness by implying that his clinical diagnosis was not reliable and that the patient was right after all to insist on an x-ray.

We surely have a duty to educate our patients. Even if we fail to persuade them that a fracture of the ankle can be diagnosed clinically we will have little difficulty in persuading them that there are possible harmful effects of taking too many x-rays. And we can remind our patients that the exercise of clinical judgment is recommended by the International Commission on Radiological Protection.¹

It is ironical that we tend to blame the legal profession for our failure to avoid unnecessary x-rays, whereas in fact the legal profession is

dependent on the evidence of medical witnesses to establish negligence. Therefore, until we educate ourselves in the need for balanced judgment in ordering x-rays then there is little hope of improvement.² Balanced judgment depends on our being well informed of the pitfalls in the clinical diagnosis of fractures, and in this connection it is vital that casualty departments should have clear instructions on the principles that should govern discretionary policies in ordering x-rays.³

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¹ ICRP Committee, *Protection of the Patient in X-ray Diagnosis*. Oxford, Pergamon Press, 1970.

² Thomas, D F, *British Medical Journal*, 1971, **2**, 105.

³ Fowler, A W, *British Medical Journal*, 1970, **1**, 362.

Heating human milk

SIR,—Your leading article (28 May, p 1372) comments on the results of our study on pasteurisation of human milk and suggests that differences in technique account for the differences in our findings and those of Ford et al.¹ Our detailed procedure for pasteurisation was as follows: since we wished to simulate a technique which might be used in routine practice, we used whole milk direct from the milk kitchen. This was not processed in any way. Aliquots of 1 ml were transferred to glass tubes (75 mm × 10 mm) in a water bath at 62.5°C for 30 minutes. The samples for protein assays were then frozen at -40°C. Because of the small volumes used, we did not monitor the temperature at the centre of the sample, as suggested in your leader, since we considered that the temperatures would rapidly equilibrate.

The sterilisation procedure appears very similar to that used by the Shinfield group, the principal difference in technique being their use of high-speed centrifugation and filtration to prepare the samples. We consider it unlikely that removal of fat and debris would have a major effect on the heat penetration of such a small sample, and we would suggest that it is most likely that the initial processing may in some way render the lactoferrin and IgA more susceptible to heating.

It is clear from these preliminary studies that, as you suggest, a combined exercise to evaluate the more subtle aspects of the use (and abuse) of human milk is urgently needed.

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¹ Ford, J E, et al, *Journal of Pediatrics*, 1977, **90**, 29.

Amniotic fluid AFP in multiple pregnancy

SIR,—Our observations on alpha-fetoprotein (AFP) values in multiple pregnancies agree with those of Dr Sheila L B Duncan and her colleagues (21 May, p 1354). In 14 cases of normal twins, the amniotic fluid AFP levels were within the normal range for singleton pregnancies. The maternal serum AFP levels were, however, elevated.

These findings agree with the fact that a very high proportion of twins, even those which are monozygotic, are in separate amniotic sacs. Only about 2% have a common amniotic cavity.¹ Thus, as far as the amniotic

fluid is concerned, twins in separate sacs may be considered as singleton pregnancies, with one fetal liver producing AFP per sac. At the placental interface, however, there is the AFP produced by two fetal livers to pass into the maternal compartment; hence the maternal serum AFP is higher than in a singleton pregnancy.

It is the 2% of twins which are monoamniotic which should in theory have an elevated amniotic fluid AFP. A recent case of ours shows that this is so. A patient underwent amniocentesis at 30 weeks because the ultrasound findings were equivocal. The AFP level of the amniotic fluid was 9 µg/ml (normal < 1 µg/ml). The patient was subsequently delivered of twins who were joined side by side. All organs were enclosed and there were no defects of the neural tubes. There were two pancreases, two duodenum, and a single large multilobed liver, which appeared to consist of two fused parenchymas.

Thus in a situation where there was the equivalent of two livers and an indisputably single amniotic sac there was an elevated amniotic fluid AFP level. However, since most multiple pregnancies are not monoamniotic, it is anticipated that almost all amniotic fluid AFP values in multiple pregnancies will not differ from those of singleton pregnancies.

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¹ Morison, J E, *Fetal and Neonatal Pathology*, 3rd edn. London, Butterworth, 1970.

Is this a record?

SIR,—I was recently asked to see a patient with the most extensive bilateral Dupuytren's contractures I have ever seen. He is now aged 70 and apparently was placed on the waiting list for surgical correction of the deformities in 1952. Perhaps the best way to describe the increasing deformity is his progressive inability to play musical instruments. When he first presented 25 years ago he was a keen clarinetist and saxophonist. As his Dupuytren's progressed he lost his ability to play these instruments and turned to the drums. He now has difficulty in holding the drum sticks.

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Insurance companies' attitude to psychiatric illness

SIR,—In his letter referring to recent suggestions that insurance companies may be prejudiced in their loading of minor psychiatric illness (21 May, p 1350) Dr Andrew Sims says that the neuroses carry a slightly increased risk of premature mortality. Such an assertion demands formidable diagnostic severity. A random survey of a hundred patients taken after three consecutive years of admissions to a neurosis unit with which I was associated

revealed four deaths, all from the physical conditions the absence from which of clinical confirmation led to their symptoms being interpreted as neurotic.

A young male ataxic was found post mortem to have a glioma tracking selectively down the mid-brain. A woman with unexplained diarrhoea and weakness had a single small secondary in the liver, no primary discovered post mortem. An anxious man with recurrent invaliding chest pain died of a massive infarct. An intractable asthmatic died in an attack, no more helped by psychological than by physical methods.

However one looks at this dip from the barrel it is a reminder of how all too easy it is to allow universally latent psychopathology to fill a gap. The pragmatic actuary's target may be not the psychoneurotic patient but the tenuous nosology.

H A ANDERSON

Lyme Regis

Local corticosteroids in asthma

SIR,—Dr H M Brown (28 May, p 1408) blames you, to my mind unjustly, for altering your earlier, somewhat overoptimistic view of the advantages of steroid aerosols in asthma. Effective local steroid treatment of asthma started as early as 1955¹ with the insufflation of micronised corticosteroid from a powder blower, but it became popular only much later when given as an aerosol from a metered bottle. Replacing systemic by aerosolised steroids depends (a) on the severity of the case, as documented by the systemic maintenance dose, (b) on the accessibility of the surface of the bronchial mucosa. Most published studies show that patients on small maintenance doses (5.0–7.5 mg/day) can often be maintained on aerosol alone. If they require 10 mg/day or more and were given about 800 µg steroid aerosol in addition,^{2–8} this made it possible to reduce the oral dose by 5–8 mg/day. However, if the bronchial mucosa is covered, as is often the case, by tough mucus, this seems to obstruct the absorption of the aerosol, as might be expected.⁹ Unfortunately many authors have not reported how many of their patients had hypersecretion. Also, the prompt dilator effect of beta stimulants from aerosol bottles used by many asthmatics often creates typical conditional reflexes, which may also work after the steroid aerosol although it has no dilator action.

Three practical conclusions seem to follow: (1) Patients with bronchial hypersecretion (particularly those with persistent cough or regular nocturnal cough) are unlikely to benefit from steroid aerosols. (2) Patients with very mild dry asthma, mostly of allergic origin, who inhale bronchodilators not more often than twice daily, will probably benefit from steroid aerosols about 800 µg per day, or, alternatively, from an oral maintenance dose of 5–7.5 mg/day. If they are reasonably fit without either, neither systemic nor inhaled steroids are indicated. (3) Patients who have life-threatening attacks of asthma or are in danger of becoming invalids through asthma should in no circumstances be given a steroid aerosol but require high doses of a systemic steroid, beginning usually with 30 mg prednisone daily in the morning and tapering very slowly off as outlined elsewhere¹⁰ until the daily maintenance dose is found. Only then, and only if this maintenance dose is higher

than 7.5 mg/day, should gradual partial replacement of the systemic maintenance dose by steroid aerosol be attempted. During this process and afterwards continuous observation is necessary for spontaneous increases of the asthmatic obstruction by intercurrent infection or other factors, best noticeable from the increasing number of bronchodilator inhalations required. In such a case the systemic steroid must be doubled at once regardless of the steroid aerosol and, if necessary, other measures taken. In no case should the decision whether to increase the systemic steroid be left to the patient or, in the case of a child, to the parents. It is here that the danger of sudden death threatens, and it is here that I profoundly disagree with Dr H M Brown. I agree, however, in so far as the family doctor often is too reluctant to give high corticosteroid doses. This reluctance has caused, as some of our most experienced observers have shown,^{11,12} unnecessary deaths from asthma. I and my co-workers have observed during 25 years more than 2000 asthmatics requiring long-term steroids, but have seen surprisingly few deaths during this time, mostly in the aged and through non-asthmatic complications; none in children, of whom we have seen fewer.

In my experience steroid aerosols offer an advantage only to patients with moderately severe asthma who need systemic corticosteroids in a dosage of more than 7.5 mg per day. They will often be able to lower the maintenance dose by adding some steroid aerosol, thus perhaps reducing the risk of adrenal suppression.

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- ¹ Burger, J H, and Shaffer, J H, *Bulletin of the New York Academy of Sciences*, 1955, **61**, 56.
- ² Foulds, W S, et al, *Lancet*, 1955, **1**, 234.
- ³ Godfrey, M P, et al, *Lancet*, 1957, **1**, 767.
- ⁴ Stresemann, E, *Klinische Wochenschrift*, 1961, **39**, 198.
- ⁵ Cameron, S J, et al, *British Medical Journal*, 1973, **4**, 205.
- ⁶ Mygind, N, and Hansen, I B, *Acta Allergologica*, 1973, **28**, 211.
- ⁷ McAllen, M K, et al, *British Medical Journal*, 1974, **1**, 171.
- ⁸ Roscoe, P, et al, *British Journal of Diseases of the Chest*, 1975, **69**, 240.
- ⁹ Herxheimer, H, et al, *British Medical Journal*, 1958, **2**, 762.
- ¹⁰ Herxheimer, H, *A Guide to Bronchial Asthma*. London, New York, San Francisco, Academic Press, 1975.
- ¹¹ Macdonald, J B, et al, *British Medical Journal*, 1976, **1**, 1493.
- ¹² Macdonald, J B, et al, *British Medical Journal*, 1977, **2**, 721.

Reliable measuring of blood pressure

SIR,—We now have several effective anti-hypertensive drugs. We have proof that blood pressure reduction is beneficial in those with moderate or severe hypertension. What is the next goal? It would be interesting to know if drug treatment of mild hypertension is worth while, but I share the doubt expressed in your leading article (4 June, p 1429) about the likely result of the MRC trial. I do not think it will be decisive.

In my view probably the greatest need today is for some simple means of determining an individual's usual blood pressure as distinct from that which develops when he is confronted by a doctor (or nurse or technician) and subjected to the discomfort of sphygmomanometry. There is no doubt that this artificial environment adds a variable, unknown, and often large supplement to the ordinary pressure—a supplement which is considerably increased if the medical examina-

tion is considered to carry a threat of loss of livelihood. Under such circumstances I have recorded a pressure as high as 220/120 mm Hg in an otherwise normotensive subject.

It is frequently argued in favour of casual readings that in spite of their deficiencies they are the best guide we possess to prognosis and thus to the need for treatment. Although this is true in relation to large groups of people, casual readings are a poor guide to prognosis in individuals and inferior to basal readings.¹ Others argue in favour of casual readings that a rise in blood pressure with excitement indicates the existence of a state of "labile hypertension" and that this carries an adverse prognosis. Such an inference is completely unproved.

No, it would be an immense advantage if we could eliminate the artefact caused by sphygmomanometry. Blood pressure can be recorded accurately over long periods without the presence of a doctor or other medical worker, by means of an intra-arterial catheter, but so far no reliable means of doing this non-invasively has been devised. The next major advance in the management of hypertension will be the development of a reliable, inexpensive, non-invasive piece of equipment capable of recording the blood pressure repeatedly over a period of an hour or more with the patient in a state of relaxation. The first manufacturer to produce such equipment could expect to make a fortune.

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¹ Smirk, F H, in *Antihypertensive Therapy: Principles and Practice*, ed F Gorss, p 355. Berlin, Springer, 1966.

Bleeding in renal failure: a possible cause

SIR,—Following the report of Kazatchkine et al¹ that factor VIII—von Willebrand factor activity (VWFR) is decreased in the plasma of patients with chronic renal failure and may partly explain the haemorrhagic tendency in that condition, we have measured factor-VIII-related antigen (VIIIa), factor-VIII-procoagulant activity (VIIIc), and VWFR in a group of 20 patients undergoing long-term haemodialysis.

VIIIc was estimated by a standard two-stage assay,² VIIIa by unidirectional immunoelectrophoresis using the Laurell technique,³ and plasma VWFR by aggregation of formalin-fixed platelets in the presence of ristocetin.⁴

The results are as follows:

	Mean % ₀	Range % ₀	Reference range % ₀
VIIIc	267	122–404	50–200
VIIIa	219	93–400	50–170
VWFR	180	83–291	50–200

We confirmed the elevated levels of VIIIc and VIIIa found by Kazatchkine et al and others, but none of our patients had a depressed VWFR level. Indeed, the mean level was towards the upper limit of normal. VWFR levels did not differ significantly from VIIIa levels, but the difference between mean VIIIc and VWFR is highly significant (using two-way variance analysis, $t=4.48$, $P<0.001$) as is the difference between VIIIc and VIIIa ($t=2.66$, $P<0.05$).

We conclude, therefore, that our patients'