restored by adjustment of sensitivity controls in the normal fashion. Continuous recording of two echoes by time-position is not important because respiratory activity is continuously monitored by chart recorder. The time-position signals are merely used to verify the respiratory origin of the recorded signals.

The recording of fetal chest wall velocity provides additional information which may assist in distinguishing between different types of respiratory movement. We make no claim at this stage that this is clinically useful.

The capacity of the system to record movements of small amplitude is evidenced in fig 2 (a) in our paper, while the duration of recording available from our chart recorder is similar to that obtained from other systems. We have found that consideration of the comfort of our patients usually limits the duration of recording to a maximum of about 90 minutes.

We consider Dr Meire's criteria for a fetal breathing monitoring system to be arbitrary and unsupported by the reference he quotes.

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Raynaud's phenomenon as side effect of beta-blockers

SIR,—We read with interest the paper by Dr A J Marshall and others (19 June, p 1498) concluding that the incidence of Raynaud's phenomenon in patients on three different beta-blockers lay between 59% and 22%. These figures are far higher than the incidence measured by other authors or from our own records of extensive clinical trials. Thus in five papers concerning propranolol (Inderal) where Raynaud's phenomenon or cold extremities was recorded the incidence was 8.2%,¹ 5.8%,² 3.8%,³ 3.3%,⁴ and 2.8%.⁵ With atenolol (Tenormin) out of 547 patients treated in 15 trials where in no instance was the dose higher than 600 mg/day the incidence of cold extremities was 2.6%. Clearly these figures are quite different from the 59% and 35% quoted by Dr Marshall and his colleagues.

We would make certain observations on the survey carried out by this Bristol group which may explain this discrepancy. Firstly, it has long been recognised that if one specifically asks about a side effect then one is very much more likely to receive a positive answer than if an open-ended question is used.⁶ This is presumably due to the background incidence of side effect-like symptoms which are part of everyday life. It is for this reason that a placebo control group and a double-blind design are essential when using direct side effect questionnaires. Unfortunately, no placebo group was included in the present survey, although the methyldopa group acted as a control. We consider that because there is evidence that methyldopa may produce peripheral vasodilatation⁷ the use of this as a control is unsuitable.

Further, although dose ranges for the drugs are given in the paper, the average is not. Reference to table II shows that with propranolol the mean fall in diastolic blood pressure was 32 mm Hg, with methyldopa 31 mm Hg, with atenolol 29 mm Hg, and with

oxprenolol 24 mm Hg. It is interesting to note that the rank order in which the three beta-blockers produced cold extremities in this survey is the same order as the falls they produced in diastolic blood pressure. We wondered therefore if the relationship of cold extremities to the dose of beta-blockers has been adequately allowed for, particularly in the case of oxprenolol.

While there can be very little doubt that cold extremities can be a side effect of beta-blockers and while it is probably true that this incidence varies from drug to drug, possibly related to pharmacological effects discussed by Dr Marshall and his colleagues, we would strongly question the conclusion that the incidence is anything like 20%-50%, as suggested.

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- ¹ Zacharias, F J, et al, *American Heart Journal*, 1968, **83**, 755.
- ² Greenblatt, D J, and Koch-Wesser, J, *Drugs*, 1974, **7**, 118.
- ³ Tarazi, R C, and Dunstan, H P, *American Journal of Cardiology*, 1972, **29**, 633.
- ⁴ Conway, J, in *Modern Trends in Cardiology*, vol 3, ed M F Oliver, p 376. London, Butterworths, 1975.
- ⁵ Prichard, B N C, and Gillam, P M S, *British Medical Journal*, 1969, **1**, 1.
- ⁶ Wheatley, D P, *Journal of International Medical Research*, 1975, suppl 3, p 105.
- ⁷ Onesti, G, et al, *American Heart Journal*, 1964, **67**, 32.

Dangers of tinted glass for driving

SIR,—I feel sure that practically all medical men share the views of Professor R A Weale (15 May, p 1212) and Mr M J Gilkes (3 July, p 42) as to the dangers to drivers of using tinted spectacles and windscreens in conditions of impaired visibility.

It is, however, not generally appreciated that tinted screens are already probably illegal in the UK under section 22 of the Motor Vehicles (Construction and Use) Regulations 1973. I pointed this out in May 1975 in an article on safety for a popular motor magazine and as it drew no editorial comment I presume that it is recognised as being the correct interpretation of this section.

As far as I know there has not been a test case, but I would think that a motorist who had suffered injuries from an accident in fog or heavy rain might well be awarded heavy damages on suing the manufacturers of his car if this was a modern one and was fitted with a tinted screen.

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Other tunnels, other nerves

SIR,—We should like to congratulate you on drawing attention to the problem of peripheral nerve entrapment (3 July, p 3). You mention five sites where pressure commonly occurs. Those with a special interest in these conditions will also recall reports of entrapment at other sites—namely, the exit of the supraclavicular nerve through the clavicle,¹ the emergence of the saphenous nerve from the sub-sartorial canal,^{2,3} and the sural nerve at the lateral margin of the ankle.⁴ In the production

of many of these syndromes minor or repeated trauma plays a part.

We believe that entrapment neuropathy is much more common than is realised, but apart from the obvious motor entrapments such as the carpal tunnel syndrome many surgeons are dubious about the validity of this claim. Preoperative demonstration of a conduction defect appears to be the only objective evidence of compression, but facilities for this test—especially with regard to sensory nerves—are not widespread. We would suggest, therefore, that where possible the suspected patient should be referred for such study, as this will eliminate much of the confusion that surrounds the diagnosis.

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- ¹ Gelberman, R H, Verdeck, W N, and Brodhead, W T, *Journal of Bone and Joint Surgery*, 1975, **57A**, 119.
- ² Kopell, H P, and Thompson, W A L, *Yearbook of Orthopedics and Traumatic Surgery, 1960-1*, p 232.
- ³ Mozes, M, Quaknine, G, and Nathan, H, *Surgery*, 1975, **77**, 299.
- ⁴ Colbert, D S, Mackey, D, and Cunningham, F, *Journal of the Irish Medical Association*, 1975, **68**, 544.

SIR,—It is disappointing to find, in an otherwise excellent leading article (3 July, p 3), no mention of abdominal wall pain due to nerve entrapment in the posterior wall of the rectus sheath. This was first described in 1968¹ and subsequently reported elsewhere.^{2,3} Our clinical experience in over 500 cases has been confirmed by other workers^{4,5} and the syndrome well recognised both in Britain and abroad.⁶

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- ¹ Mehta, M, Annual Meeting, Royal College of Surgeons of England, 1968.
- ² Mehta, M, and Ranger, I, *Anaesthesia*, 1971, **26**, 330.
- ³ Mehta, M, *Intractable Pain*, p 122. London, Saunders, 1973.
- ⁴ Applegate, W V, *Surgery*, 1972, **71**, 118.
- ⁵ Cilento, R, *British Medical Journal*, 1972, **2**, 789.
- ⁶ Raftery, H, and de Valera, E, 1st World Congress on Pain, Florence, 1975.

Erectile impotence

SIR,—The leading article on the treatment of erectile impotence (29 May, p 1298) and the recent letter from Dr G R Freedman (3 July, p 44) emphasise the psychological component at the expense of the physical. Your leading article does mention multifactorial causation but says nothing about the appropriate screening for physical or endocrine abnormalities which might be contributing to or causing the impotence. Furthermore, by accepting aging as a sufficient cause for impotence it implies that investigation of impotence in the elderly is unnecessary.

We have recently completed a clinical trial of gonadotrophin releasing hormone in secondary sexual impotence¹ and have screened 34 consecutive patients for mental and physical pathology who were referred by general practitioners, physicians, and psychiatrists. Diabetics were not referred to us and none of the 34 were found to have abnormal glucose tolerance. Despite this a surprising number, 11 of the 34 referrals, were found to be suffering from a physical illness which could explain their erectile impotence—for example, hepatic