for the carers they would implement the advice given in HN(82)33.

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Fetal monitoring during labour

SIR,—Mr Robert S Sawers's leading article (3 December, p 1649) admirably summarises the perplexities that stem from the advent of commercial fetal heart rate monitors. Mr Sawers makes a plea for new and better methods, even better than fetal pH measurements: do we diagnose adults on heart rate?

There are two older methods of measurement not mentioned; I found both of real value in the final differentiation of real fetal distress from physiological bradycardia.

The first method is the phonocardiograph as developed by Palmrich in Professor Antoine's department in Vienna.^{1,2} This records the timing of the fetal heart sounds and thus a determination of both the systolic and diastolic time intervals. The fetus has well developed blood pressure control in

cardiogram is recorded before or at the start of labour changes brought about by over-frequent uterine contractions or placental insufficiency may be noted. One reason why this technique is not well confirmed and supported is that a polygraph pen writing to 200 hertz at least is necessary to capture the fetal R wave, and it is in the quality and bundle branch defects shown in this wave that the diagnosis rests.³ For these observations the patient does not need continuous monitoring provided that she reports to the recording technician at reasonable intervals, and frequently during the initial stabilising of an oxytocic infusion or if abnormalities begin to arise. The two methods give some insight into fetal cardiac and medullary function.

Regarding future research it seems a pity that the Doppler method does not allow the recording of systolic and diastolic intervals; the device is "overdirectional" and if the fetal position moves, as it does with contractions, the recorded rate may show an apparent but not real change in rate: these are limitations to a valuable technique. Fetal blood sampling by the scalp method involves a tedious technique with the patient in the lithotomy position, a position that in itself affects uterine circulation. An easier method assessable by telemetry seems within research possibilities.

The patient may carry a recorder or meter of contraction rate (drugs control frequency of contractions but not the resultant pressure changes). Also the scalp electrode could measure by telemetry the electrocardiographic wave form and when abnormalities were noted and could not be corrected by postural changes then the phonocardiograph could help the final decision on treatment.

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¹ Palmrich AH. Die Variabilität des physiologischen fetalen Phonokardiogramms und dessen Abgrenzung von pathologischen Herztönen. *Zentralbl Gynakol* 1951;73:1699-707.

² Smyth CN, Farrow JL. Present place in obstetrics for foetal phonocardiography and electrocardiography. *Br Med J* 1958;ii:1005-9.

³ Smyth CN. Experimental electrocardiography of foetus. *Lancet* 1953;ii:1124-6.

SIR,—Dr Robert S Sawers concludes his excellent leading article by suggesting that more attention be given to the analysis of fetal heart rate tracings by computer. He goes on to suggest that the widespread application of the microprocessor might make possible the production of a new generation of more intelligent fetal heart rate monitors. In Hull we have been evaluating just such an instrument, and our preliminary experiences were presented at the British Congress of Obstetrics and Gynaecology earlier this year.

The system was designed by Professor Maeda of Tottori University, Japan, and consists of a small microprocessor (Toitu model MT-140) connected in a single unit to a conventional fetal heart rate monitor (Toitu MT-810). The microprocessor is programmed to analyse baseline fetal heart rate and deceleration indices including amplitude, lag time, duration, and nadir. These indices are used to generate a fetal heart rate score every five minutes and a fetal distress index when adverse features appear.

Our initial impression in 60 patients was that this system gives a useful interpretation of patterns of fetal heart rate. A good correlation between fetal distress index, Apgar score, and cord blood pH was obtained in 80%. Further

studies are in progress to identify the causes of the poor correlation in the other 20%.

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SIR,—I was pleased that Dr Robert S Sawers drew attention in his leading article (3 December, p 1649) to the limitations of the now widely accepted method of intrapartum monitoring of fetal heart rate. There is a high rate of false positive traces during normal labour and a more serious and unacceptable high rate of false negative traces when there is fetal distress. All medical and nursing personnel offering intrapartum care should be aware that what they are observing may be an inadequate guide to fetal wellbeing. An awareness of the limitations is also important because it gives lift to the search for more reliable methods, where as yet there has been slow progress and perhaps complacency among many obstetricians.

Dr Sawers points out that concurrent use of fetal scalp sampling may not only improve the fetal outcome but also reduce the rate of caesarean section. Outside of teaching hospitals, however, fetal scalp sampling is seldom used, and, indeed, many hospitals offer no facilities for fetal blood sampling. Surely when the inadequacies of fetal heart monitoring are so apparent this is an unacceptable state of affairs.

For the present and foreseeable future we must accept that fetal heart traces will be the most widely used method of monitoring fetal wellbeing in labour, and Dr Sawers points to the use of computers to improve the accuracy of their interpretation. Though this may soon come there is also room for better training of medical and nursing staff in their interpretation. In most units in Britain the immediate management of labour falls largely to the junior staff, who, on the whole, receive their practical training in the management of labour from the next most junior member of staff. Thus those who are monitoring labour are all too often inadequately trained to do so. Labour is unquestionably the most dangerous time for the fetus, the neonate, and the mother; in these days when perinatal mortality and morbidity are at the fore of obstetric practice is it not something of a paradox that so little of what is done on the labour ward is done by consultant staff? Surely it is time that this traditional state of affairs changed and priority was given to consultant sessions on the labour ward, if necessary at the expense of other clinical duties.

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Migraine, headache, and survival in women

SIR,—Bradford Hill, on page 212 of his textbook, attaches considerable importance to calculating the limits on an estimate of a survival rate by assuming various possible distributions of the unrecorded cases whenever an appreciable number of cases is lost to follow up.¹ He also, on pages 29 and 261, draws attention to the likely unrepresentativeness of volunteers. Put these two points

Typical recording showing changes in the fetal R wave and ST depression during induced labour. M=maternal wave.

utero and uses heart rate under vagal innervation to compensate for any impediment in the placental-fetal circulation. So long as the impediment (unbalanced intraplacental pressures or cord entanglement) is transient only the diastolic time intervals change.

Secondly, the wave form of the fetal electrocardiogram can be recorded from scalp or abdominal electrodes. These methods were first used at University College Hospital in 1948 and described in 1953.³ The scalp electrode gives a wave pattern not confused by maternal complexes. The more convenient abdominal electrodes do pick up maternal heart signals but, as the heart rates are different, in a 10 second recording there will be sufficient fetal complexes clear of artefacts. Often only the R wave rises above the noise levels, but where ST depressions occur they too can be recognised. When a fetal electro-

together in conjunction with the report by Professor W E Waters and others (12 November, p 1442) and one wonders why these authors are so emphatic about the absence of an increased risk of dying in the women aged 45-64 who had had headache and migraine in 1967.

Their report says that 86% of the intended population cooperated. This is a creditable rate, but it implies that about 477 women did not cooperate. If the proportion aged 45 and over was the same as in the cooperative women shown in table I, about 214 women have been omitted from the comparisons in table II. If none had headache and migraine in 1967 and none had died, they would raise the survival rate of their group from 82.0% to $(301+214)/(367+214)$ —namely, 88.6%. Even if seven of them had died the rate would still be no worse than the average in the affected groups, as $(301+207)/(367+214)=87.4\%$, to be compared with $(292+291+241)/(338+326+279)=87.4\%$. These calculations could be elaborated to take account of age, smoking, and taking analgesics and are likely to upset the conclusions from table III. That is to say, a sufficient number of the uncooperative women may have been better risks than those who did cooperate for there to be more uncertainty in their results than they seem willing to grant. As their only reference to bias concerns less healthy women, one would like their report to have discussed this apparent possibility, as Bradford Hill recommends.

It is regrettable that many textbooks and courses on elementary medical statistics do not go beyond a warning that incomplete data may be biased. Nevertheless, some illustrative computations are given by Bradford Hill. As they require only elementary arithmetic, these kind of analyses could be given priority over significance tests and training in the calculation of such measures as the correlation coefficient, important these formulas may be in suitable contexts. Perhaps computer analysis packages should have a warning attached.

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¹ Bradford Hill A. *A short textbook of medical statistics*. London: Hodder and Stoughton, 1977.

*. *The authors reply below.—ED, *BMJ*.

SIR,—As Mr Tweedie points out, his first reference to Bradford Hill is concerned with losses to follow up.¹ We lost only one woman in the follow up period so this is not an important source of bias in our study.

Mr Tweedie is concerned with the likely unrepresentativeness of volunteers. Our survey was, as we stated, mainly haematological and was based on all women in the age range studied in a defined area of south Wales: over 86% cooperated. It is unlikely, though possible, that there were important differences in the prevalence of headache. Even if there was considerably more, or considerably less, headache in the non-responders, however, this would be important in considering the prognostic significance of headache only if the headaches in the non-responders had a different prognosis from those of the 86% who cooperated in the study. MacMahon and Pugh state that the true relation between "exposure" (here headache) and "outcome" (here death)

will be distorted only if the loss through inability to assign individuals to exposure categories is biased with respect to both exposure and outcome.² If the loss is biased with respect to only exposure—that is, if women without headache did not respond—or outcome—that is, none of the non-responders died—then the true relation between headache and mortality can still be estimated. Any bias with respect to outcome is likely to be other than that suggested by Mr Tweedie, as we pointed out when considering the standardised mortality ratio of the population, in that the less healthy (and thus more likely to die) women are likely to be omitted. If we assume that none of the non-responders had died this would reduce the overall standardised mortality ratio to about 92, and even allowing for seven deaths would bring it up to 94, hardly likely in the population we were studying.

In a short paper it is not possible to go through all possible alternative analysis. Guidelines for deciding whether a sample is representative of a population do not appear to us very suitable for a computer package. (Would it take the form: "If percentage non-responders greater than 10, print 'warning, sample unrepresentative?'") Each study should be considered on its merits. We are fully aware of the importance of the problem of selection. Mr Tweedie refers to Bradford Hill page 261, where one of the examples cited is a study on migraine, published by one of us.¹

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¹ Bradford Hill A. *A short textbook of medical statistics*. London: Hodder and Stoughton, 1977.

² MacMahon B, Pugh T. *Epidemiology. Principles and methods*. Boston: Little, Brown and Company, 1970:218.

SIR,—Professor W E Waters and others (12 November, p 1442) put forward two possible explanations for their finding of lower mortality in women aged 45-64 years with headaches or migraine compared with controls: earlier diagnosis and treatment of hypertension in the group with headaches, and more advanced arterial disease in the control group. We would like to suggest a third possibility.

It has been suggested that migraine is a disorder of platelet function.¹ Women are most affected by migraine during their fertile years, and indeed both migrainous and non-migrainous headaches are generally worse around the time of menstruation.² We have therefore compared ex vivo platelet aggregation with various concentrations of serotonin in the postmenstrual (days 7-10) and premenstrual (days 25-28) phases of the cycle in eight women suffering from menstrual migraine and in a control group of nine women. Preliminary results show that the rate of aggregation is significantly reduced in those with migraine (mean (SEM): $12.5(2.82)$ mm/min) compared with controls (mean (SEM): $21.78(2.95)$ mm/min) in the premenstrual phase (Student's *t* test, $p < 0.05$) using low concentrations of serotonin ($0.5 \mu\text{mol/l}$ ($88 \mu\text{g/l}$)). This difference was not detected with higher concentrations of the aggregating agent ($2, 5, 10$, and $20 \mu\text{mol/l}$ ($352, 881, 1762$, and $3524 \mu\text{g/l}$)), and the postmenstrual results were similar in the two groups.

These results were unexpected in view of reports of increased platelet aggregation in migraine,^{1,3} but strict comparison is difficult for a variety of reasons. Firstly, we were studying menstrual migraine specifically and at well defined times in the cycle; secondly, previous data were based on the aggregation response to $20 \mu\text{mol/l}$ ($3524 \mu\text{g/l}$) of serotonin only; and thirdly, aggregation to serotonin was significantly increased only in classical migraine, no difference being found in common migraine.¹ Anovulatory doses of subcutaneous oestradiol implants are successful in treatment of both menstrual migraine and the headaches commonly associated with the premenstrual syndrome,^{4,5} as they remove any biochemical change that normally occurs between the follicular and luteal phases of the ovarian cycle.

Our results of reduced platelet aggregability in women suffering from migraine raise the possibility that this protects them to some extent against heart disease and strokes. Both conditions are associated with increased spontaneous platelet aggregation,⁶ and platelets play an important part in the pathogenesis of atherosclerosis. We now intend to extend our study to postmenopausal women who complain of headaches and migraine.

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¹ Hanington E, Jones RJ, Amess JAL, Wachowicz B. Migraine: a platelet disorder. *Lancet* 1981;ii:720-3.

² Somerville BW. The influence of progesterone and oestradiol upon migraine. *Headache* 1972;12:93-102.

³ Hilton BP, Cummings JN. 5-Hydroxytryptamine levels and platelet aggregation responses in subjects with acute migraine headaches. *J Neurol Neurosurg Psychiatry* 1972;35:505-9.

⁴ Magos AL, Zilkha KJ, Studd JWW. Treatment of menstrual migraine by oestradiol implants. *J Neurol Neurosurg Psychiatry* (in press).

⁵ Magos AL, Collins WP, Studd JWW. Management of the premenstrual syndrome by subcutaneous implants of oestradiol. *Proceedings of the 3rd International Congress on the Menopause, Antwerp 1983* (in press).

⁶ Wu KK, Hoak JC. A new method for the quantitative detection of platelet aggregates in patients with arterial insufficiency. *Lancet* 1974;iii:924-6.

A new treatment for asthma: promotional expediency versus pharmaceutical responsibility

SIR,—For the past 12 years I have enjoyed a close association with Allen and Hanburys Limited. This arose in the course of studies that I carried out to assess the value of beclomethasone dipropionate aerosol and micronised powder in the treatment of asthma.^{1,2} Having developed a high regard for the company's research division at Ware and also for the responsible and ethical manner in which Becotide was introduced and promoted, I was sad to learn of the recent introduction of Ventide, a compound aerosol containing salbutamol and beclomethasone dipropionate.

Last year I was one of several clinicians with particular experience of the use of beclomethasone dipropionate whose opinions were sought by the company concerning its proposal to develop and market Ventide. In a carefully considered report I strongly advised against it, and I know that at least one of the other clinicians gave a similar opinion. The compelling reasons for disregarding this impartial advice are not difficult to surmise and they have nothing to do with improving the