recommending them the authorities have given the opportunity to psychiatrists and nurses to refuse disturbed offenders with the excuse that their hospital does not provide security or that these patients will disturb others. Many of the patients now refused are not patients likely to be accepted by the secure units when they exist. They are individuals often in the category you describe who should be cared for in psychiatric hospitals now (as they always were in the past) and will still require accommodation in them in the future.

Psychiatric hospitals have a responsibility to provide a comprehensive psychiatric service short of maximum security accommodation and psychiatrists have never agreed a policy with the Department of Health and Social Security which would relieve them from that obligation. Despite this it is a selective policy that has slowly been adopted and apparently accepted (by default) by the Department without any alternative service being proposed by anyone. The DHSS says that in the end it cannot insist that a psychiatrist cares for a particular patient. The doctors say the Department fails to provide alternative care. So that in practice there is an impasse. The Department would also make the point that it has distributed some £5m or more annually in 1976 and 1977 to develop "interim facilities" before regional secure units are established, but figures recently published indicate that most hospital regions find that very little of this money is being taken up and spent as intended.

All this is a serious problem which is unique in not being primarily a financial one. It is to be hoped therefore that a more urgent effort will be made by all concerned to solve it. The prisons, after all, cannot refuse people sentenced by the courts as hospitals can. Perhaps the courts should be more selective in deciding who really requires custody. More remands on bail will help.

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The neonatal electrocardiogram and unexpected death in infancy

SIR,—The excellent studies reported by Dr D P Southall and others (3 September, p 597) and by Dr B R Keeton and others (p 600) of cardiac arrhythmias found on routine screening in infants and the conduction disorders in babies found in a collapsed state call for a short comment in view of their recommendations of clinical screening related to cot deaths.

Unexpected death in infancy is a clinical syndrome only. It has varying aetiologies and different causes of death. However, among these there is both indirect evidence, as from papers such as those cited above, and also direct evidence from necropsy studies of hearts that indicate that a group of these deaths have their chief cause within the heart. The suggestion of neonatal electrocardiogram screening for such lesions is an obvious one and when in 1973 we set up a prospective study in Sheffield with a view to identifying features in babies at increased risk of unexpected death we included a neonatal ECG in the study of all high-risk infants. In a cohort of 11 424 babies, we made a special study of 627 who were thought to be at increased risk of unexpected death. In the four babies of this cohort that subsequently died

no abnormalities were noted in the neonatal ECG. One of these deaths was probably of cardiac origin.

From a present logistic viewpoint, if we assume that one in 10 cot deaths has a cardiac cause, which is my present estimate, we would need to score at least 5000 general births to hit one case and if the figures published by Dr Southall and his colleagues are general we would, in this group, pick up perhaps 100 children with arrhythmias with, at present, no evidence that such ECG changes predispose to unexpected death.

The situation at the present time would appear to call for further, more detailed study and refinement in our tools of assessing the heart in infants rather than widespread screening.

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SI units and acidity

SIR,—One of the by-products of the introduction of SI (Système Internationale) units has been to cast doubt on what are the normal mean values for hydrogen ion concentration of the various biological fluids. For arterial blood the customarily accepted mean of normality has been pH 7.40 or 40 nanomoles of hydrogen ion per litre. This mean has been obtained in the past by averaging observed pH measurements from large numbers of samples.

If a series of pH values are taken which average 7.40 and then the average is taken of the SI equivalents the results must differ—an example is given in the accompanying table. This is because averaging logarithmic numbers is a root function (square root, cube root, N^{th} root, depending on how many values there are). In blood gas homeostasis the differences are of academic rather than clinical interest, as the range of pH values is so narrow. When considering other biological fluids such as gastric secretion or urine acidity significant errors can arise. In the example in the table the real mean hydrogen ion concentration of gastric secretion is 30 times more than that obtained by converting the average of the pH values. The implications are that our older textbooks and published articles which give mean pH values are wrong, since these mean pH values are not the true mean hydrogen ion concentrations even if the latter are expressed in pH notation.

The problem is further compounded where standard deviations are concerned. The growing laboratory practice of indicating the degree of abnormality of a particular bio-

pH values and SI equivalents (mol/l) of blood and gastric juice

pH	SI equivalent
<i>Blood</i>	
7.2	63×10^{-8}
7.4	40×10^{-8}
7.6	25×10^{-8}
Average 7.4	42.6×10^{-8} **
<i>Gastric juice</i>	
7.0	10×10^{-8}
5.0	10×10^{-4}
3.0	10×10^{-2}
Average 5.0	3.3×10^{-4} †

*but 42.6×10^{-8} mol/l = pH 7.37.

†but 3.3×10^{-4} mol/l = pH 3.52.

chemical measurement by the number of standard deviations by which that measurement differs from the normal mean imposes a heavy reliance on the validity of that mean. Further, if the standard deviation has been calculated in pH units and subsequently converted, then any connection between this and the true standard deviation is likely to be coincidental.

Fundamental to this is the underlying rationale of reporting biochemical data. Ideally to the clinician any departure from normality should be related to the extent to which that departure is a threat to health. If this threat is either alinear or asymmetrical about the mean, then reporting in terms of standard deviations either in SI mode or logarithmic (pH) mode is a cumbersome irrelevance. If the distribution of normal values for hydrogen ion concentration does not follow a Gaussian curve, then the simple linear scale proposed by the SI system offers no advantage to the clinician.

Could our biochemical colleagues give us guidance? The question is not so academic as it seems—how is excessive acid production to be identified or how is one to judge whether antacids or H^2 receptor blockers are better at controlling intragastric acidity?

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Miscarriages as a cause of spina bifida and anencephaly

SIR,—The data presented by Professor K M Laurence and Dr C J Roberts (6 August, p 361) confirm much of those of Clarke *et al*¹ and would seem to indicate that the reproductive performance of mothers of anencephaly spina bifida (ASB) fetuses improves progressively if it is accepted that a miscarriage represents a greater disturbance than the survival of an ASB fetus beyond 28 weeks' gestation. The behaviour of the control group is predictable, since except at the extremes of the reproductive life span the maternal reproductive capacity remains relatively stable and at its maximum efficiency.²

However, the data from (ASB) families are, in my opinion, open to an alternative interpretation which, like that of Professor Laurence and Dr Roberts, does not lend support to the hypothesis that early miscarriages are the cause of malformations in subsequent pregnancies.¹ Indeed, the latter concept is extremely difficult to accept on teleological grounds since it would seem very unlikely that the major mechanism by which abnormal conceptuses are eliminated should in itself be the cause of the most common of human major malformations.

Postconceptional subfertility must by definition result from the loss of fertilised ova. The greater part of this loss is due to inherent defects within the conceptus and occurs before the first missed period, a relatively small proportion occurring as clinically manifest spontaneous abortions.³ A very small proportion of these defects result in late abortions and premature births. The evidence that the interval between miscarriage and the next pregnancy in both the ASB and control series is similar indicates that the very early losses must be comparable in both groups. Thus the differences in reproductive behaviour would appear to lie in the number of clinically mani-