three points which we would like to put forward for further consideration.

(1) Our daughter found it of very great help that most of the people who knew she was expecting a baby were told about the death by someone else before she came home and had to meet them. In her case the most useful agents for passing on the information beyond the family circle proved to be the vicar and the local baby-sitting club (which naturally includes most of the local young mothers). Obviously circumstances vary enormously, but most parents will know someone who can help in this way, and our observation of the relief caused by knowing that the ground had been prepared for meeting people without embarrassment does make us feel that the point is worth mentioning in the pamphlet.

(2) It may also be worth mentioning that grandparents, too, can feel bereaved. My wife and I were, I think, surprised at the depth of our own sense of loss. The main point here, of course, is that if this is ignored or repressed it can lead to family resentments such as those mentioned in the draft, but if it is faced and shared it can provide strength to both parents and grandparents and can, as with husband and wife, deepen relationships within the family, in this case the wider family.

(3) The members of our family most closely involved did in fact derive much strength from their Christian faith. We quite realise that it may not seem appropriate to speak in such terms as this in a secular pamphlet, but we would like to suggest that some such phrase as "or other minister of religion" might be inserted after "chaplain" if only to remind people that in many cases an understanding priest or minister whom one knows may prove to be one of the most valuable sources of help.

I hope that these comments based on very recent experience may be of some service.

W J H EARL

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Antenatal education

SIR,—Professor R. Beard and others have suggested an information leaflet for patients who have stillbirths (21 January, p 172).

The Rotunda Hospital, Dublin, has just analysed the value of a similar education leaflet but given to antenatal patients. We endeavoured to provide basic information to women in the lower socioeconomic groups by a simple handout given just before the onset of labour in an effort to improve knowledge and attitudes to the event. Knowledge, pain, and anxiety scores in the group receiving the handout (82 patients) and in a matched control group (110 patients) were assessed by the completion of a postnatal questionnaire. The results are shown in the accompanying table.

It can be seen that the educated group (A) had significantly lower anxiety and higher knowledge scores than the control patients (group B). Dropout and non-attendance rates at formal education classes were similar in both groups, as was the length of labour and

operative delivery rate. Although previous studies have shown that informed patients progress more smoothly to delivery,^{1,2} our patients with the highest anxiety scores did not in fact have an increase in the length of labour, a point observed by Wood *et al*³ in a similar study.

While such a method of delivering knowledge is far from ideal it has been shown that the various methods of antenatal education complement each other.⁴ For patients who will not or who cannot attend formal classes it would appear that information leaflets given before labour and containing clear information about the forthcoming events are of definite value as a teaching and anxiety-alleviating aid.

We can endorse the approach of Professor Beard and his colleagues.

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¹ Van Auker, W B, and Tomkinson, D. *American Journal of Obstetrics and Gynecology*, 1953, **66**, 100.

² Davis, C D. *American Journal of Obstetrics and Gynecology*, 1962, **84**, 1196.

³ Wood, C, *et al*, *Australian and New Zealand Journal of Obstetrics and Gynecology*, 1974, **14**, 210.

⁴ Chamberlain, G, and Chave, S. *Community Health*, 1977, **9**, 11.

"Baby and Child"

SIR,—While I am grateful for the space allocated to a review of my book, *Baby and Child* (28 January, p 228), and grateful for crumbs of comfort like "In general, her advice . . . is difficult to fault," Dr D P Addy has charged me with giving some "inaccurate or potentially dangerous information." Please allow me the right of reply to what is almost an accusation of negligence.

I do not state that "allergy . . . is due to an inability of the body to differentiate between harmless and harmful substances." I point out, as part of a lengthy discussion attempting to elucidate a difficult subject for lay people, that people who are subject to allergic disorders manufacture antibodies against substances which cause no reaction in non-susceptible subjects.

I do not say that asthma "can be treated by using a nasal spray." Sprays, aerosols, and inhalants are mentioned as among the possible routes by which *prescribed* drugs may be administered to children by parents when the doctor does not consider it necessary to attend recurrent attacks personally. Aerosol bronchodilators and disodium cromoglycate by inhalator are obvious examples. I do not specify them as I do not wish the book to encroach on the role of the doctor, who must decide what treatment is suitable for any particular patient.

I do not suggest "treatment of a severe attack of stridor . . . before calling for help." I describe the frightening symptoms of severe croup and, starting with the words "you need a doctor urgently but if you are alone in the house," suggest the optimum way to get a seriously distressed child a little extra oxygen while that doctor, an ambulance, etc, are on

their way. To say that this advice "could be fatal in acute epiglottitis" is absurd. In such a life-threatening emergency what else can a lone parent do but try to keep the child breathing while help comes?

I do say that febrile convulsions "seldom last more than a minute or two"—a manner of speaking, perhaps, but part of the overall message, which is, "Don't leave the convulsing child. As soon as the spasms cease, send for your doctor." How can it be dangerous to omit to mention the minute percentage of such convulsions which last for longer?

I do say, "If a child seems perfectly well and has a normal appetite you can disregard the diarrhoea." The paragraph refers to a single episode of loose stool. Careful discussion of the possible importance of continued diarrhoea has already taken place and the paragraph is immediately followed by a list of associated symptoms which should lead a parent to seek medical advice. There is a further reminder of the hazard of dehydration if vomiting occurs simultaneously.

Finally, Dr Addy refers to the final part of the book as a "medical encyclopaedia" and implies that he knows it was not "checked by a doctor." The book is about child development and care. The section of which he complains is an A-Z of all kinds of information, only part of which is medical. It has been read by several doctors. And I have never done the *Times* crossword puzzle in my life.

PENELOPE LEACH

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Physical deformity of Richard III

SIR,—May a non-medical man add a coda to your contributors' speculations on Richard III's anatomy?

After Professor Philip Rhodes's carefully balanced article (24-31 December, p 1650) it was odd to find Drs B Wolman and Gordon Dale (28 January, p 234) paying so little heed to the fact that Shakespeare was a writer of fiction and, in his *Richard III*, one busily engaged in constructing a melodramatic villain to rival Marlowe's. Not only is there no contemporary evidence that Richard III suffered from any deformity, but even the alleged unevenness of his shoulders is uncertain. When the Windsor Castle portrait was on loan to the National Portrait Gallery for a special exhibition in 1973 an x-ray revealed that the raised shoulder-line was an over-painting by a later hand.

Richard has been reported as "small in stature," and no doubt he didn't make much of a showing beside his brothers Edward and George. But which of us would have? They were both burly six-footers. The journal of Nicolas von Poppelau, a man of great strength but unknown height, tells us, however, that Richard was taller than Nicolas by three fingers, although slighter in build. This suggests not "certainly small" (Dr Dale's phrase) but possibly tall and thin.

Shakespeare's literary genius should surely not be allowed to lull us into forgetting that the deformity of Richard III was a Tudor myth rooted in political necessity. When Richard lay dead at Bosworth there were 29 people still alive with a better claim to the throne than Henry Tudor's. His usurping dynasty could maintain itself only by insisting on absolute submission to the crowned and anointed sovereign lord. How then could it excuse its

Knowledge, pain, and anxiety scores (mean $\pm$ SD)

Group	Knowledge	Pain	Anxiety
A (n=82)	6.7 $\pm$ 1.71	3.80 $\pm$ 1.22	2.38 $\pm$ 1.38
B (control) (n=110)	2.6 $\pm$ 1.47	4.93 $\pm$ 1.26	4.17 $\pm$ 1.45
	P < 0.001		P < 0.01

own treachery and regicide? Only by making the last of the Plantagenets a monster. Thanks to Shakespeare the mud has stuck, but mud is mud, not kyphoscoliosis, Klumpke's paralysis, Erb's palsy, Sprengel's deformity, cerebral palsy, or even coeliac passion.

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* * * This correspondence is now closed.—ED,
BMJ.

Ilio-portal shunt demonstrated during lung scanning

SIR,—The paper by Mr I Taylor and others (19 November, p 1320) describing liver perfusion utilising the umbilical vein, prompts me to report the following case.

A 63-year-old man had presented in 1959 with an extensive retroperitoneal mass. Biopsy at laparotomy showed an anaplastic tumour with lymphatic spread. There was an excellent response to multiple courses of external radiotherapy and he remained well with no clinical evidence of residual disease until 1976. In March 1976 he developed pain in the right loin but no definite diagnosis was established. In August 1976 he had intermittent attacks of chest pain and subsequently developed bilateral oedema of the legs. He was thought on abdominal examination to have a mass in the right iliac fossa. He was referred for lung scanning with a provisional diagnosis of deep vein thrombosis and pulmonary embolism.

In an attempt to demonstrate the site of venous occlusion 2 mCi of ^{99m}Tc -albumin microspheres were injected into a dorsal vein of the right foot, a tourniquet being used to divert the radiopharmaceutical into the deep veins of the leg. The passage of the bolus through the iliofemoral region was imaged by a gamma camera. There was distortion of the lumen of the femoral vein at the level of the inguinal ligament, the external iliac vein did not fill, and activity passed into collateral vessels, including the inferior epigastric vein. The anterior lung scan (see figure), performed subsequently, showed peripheral defects consistent with pulmonary emboli. In addition, however, there was a well-defined area of activity in the region of the left lobe of the liver and a small amount of diffuse activity in the right lobe. A right lateral scan confirmed the anterior position of the area of activity in the left lobe of the liver. This could only have reached the liver through the portal circulation, presumably through the umbilical vein via anastomoses at the umbilicus with the inferior epigastric vein. Demonstration of this collateral

circulation led to a diagnosis of occlusion of the inferior vena cava. Further investigations confirmed that this was due to recurrence of his tumour, which once again responded to radiotherapy.

It is well recognised¹ that collateral circulation via the umbilical vein may lead to a "hot spot" in the left lobe on ^{99m}Tc -sulphur colloid liver scans following antecubital injection in patients with superior vena caval obstruction. The corresponding effect of occlusion of the inferior vena cava does not appear to have been reported previously.

These observations have implications for therapeutic perfusion of the liver via the umbilical vein, where uniform perfusion of both lobes is desirable. Portal venography, as used by Mr Taylor and his colleagues, is not a quantitative technique and there is the possibility that contrast may reach both lobes when injected as a rapid bolus but that the left lobe may be preferentially perfused by slow infusion into normal portal flow. It would seem advisable to confirm uniform perfusion of both lobes by injecting ^{99m}Tc -albumin microspheres into the infusion if this imaginative technique for improving survival in colorectal cancer is to be more widely accepted.

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¹ Holmquest, D L, and Burdine, J A, *Journal of Nuclear Medicine*, 1973, **14**, 348.

Heritability of blood pressure

SIR,—Your leading article (21 January, p 127) provides an excellent review of much current opinion, but may I comment on the distributions said to be expected where polygenic inheritance is involved?

In hypertension the distribution curve for first-degree relatives of patients is skewed towards higher values. Skewness, however, is but the first stage in progressing from a "normal" or Gaussian curve towards complete segregation. In the second stage the bulge beyond the confines of the normal curve increases to become a separate peak and the distribution is described as bimodal. In the third stage the distributions underlying the bimodal peaks begin to separate. In the fourth stage the distributions have segregated. Such a distribution can be compared to that where a single gene is involved and full segregation is the acknowledged rule. The four stages can be demonstrated in the case of height. They depend on the degree of hereditary likeness to be expected. For sibling, parent, or child the probability of similar genetic endowment is one-half, for grandparents or grandchildren it is one-quarter. These proportions apply equally to single gene and polygenic inheritance.¹ The stages of segregation also depend on other factors. One is the distance of the selected category from the population mean. Another is the degree of assortative mating. A third is the accuracy and reliability of measurement. A fourth is the presence or absence of modifying genes.

In a study of the stature of 760 university students¹ the distribution of sons' height was analysed according to their parents' height. Assortative mating of parents was measured by the product moment correlation coefficient ($r=0.28$). This helped to raise the correlation coefficients between father and son (0.47), mother and son (0.49), and midparent and son (0.60) close to the maximum theoretical values of 0.5, 0.5, and 0.71

respectively. When further degrees of parental likeness were considered—for example, where all fathers exceeded 71 in (180 cm) and mothers 66 in (168 cm)—the distribution of sons' height was bimodal, with peaks at 72 and 74 in (183 and 188 cm) and a trough at 73 in (185 cm), which was consistent at all higher levels of cut-off with increasing parental likeness. At father 69 in (175 cm) plus and mother 64 in (163 cm) plus the distribution was trimodal, with the third peak at 70 in (178 cm), which is also the mode of the general spread. Where both parents were shorter—for example, where the father was less than 68 in (173 cm) and mother less than 62 in (157 cm)—similar bimodality was found in the sons' distribution, with peaks at 65 and 68 in (165 and 173 cm) and a consistent trough at 66 in (168 cm), whatever the level of shorter cut-off chosen. Complete segregation of peak distributions was found where fathers were less than 66 in (168 cm) tall married to mothers of less than 61 in (155 cm). The sons' height was quite differently distributed in the 487 heterogamous matings where one parent was taller and the other shorter than average in height. It was unimodal, with the peak at 70 in (178 cm).

The above data were presented to the Society for the Study of Human Biology on 21 April 1961. They illustrate the power of "like-mating" to bring about bimodal and sometimes trimodal distributions of the height of offspring. Other comprehensive characteristics such as hypertension may be expected to be affected in a similar way by the marriage of like parents. It is not the *half* inheritance of first-degree relatives that matters most to an individual: it is his *whole* genetic endowment. The chance of having a hypertensive son is greater where both parents become hypertensive and greatest where all four grandparents were affected.

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¹ Penrose, L S, *The Biology of Mental Defect*, p 105. London, Sidgwick and Jackson, 1949.

² Parnell, R W, *Lancet*, 1963, **1**, 1213.

SIR,—In your leading article on this subject (21 January, p 127) it is stated that "the great contribution of Pickering was to emphasise that arterial blood pressure is a continuously distributed variable with no logical sharp boundary between 'normal' and 'abnormal' values." It is worth recalling where the evidence for this began.

In 1923 Dr W C Alvarez¹ examined the blood pressures of about 6000 men of ages from 16 to 40. He found that the greatest number of men had pressures between 120 and 134 mm Hg. Actually 2950 out of the 6000 fell in this range. But there were 1708 whose pressures were greater than 134 mm Hg and there were 1341 whose pressures were less than 120 mm Hg.

Now this very wide variation was unexpected, since from the lowest to the highest it was 85 to 194 mm Hg, and Alvarez therefore measured the blood pressure of 1215 students of the University of California who were all 18 years of age and found about the same range—namely, from 85 to 184 mm Hg (the average being 130 mm Hg). Among the students of 18 years of age no less than 31% had pressures greater than 134 mm Hg. In one out of every 36 men the pressure was greater than 160 mm Hg.

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¹ Alvarez, W C, *Archives of Internal Medicine*, 1923, **32**, 17.

Anterior rectilinear scan showing localised activity in the left lobe of the liver and slight diffuse activity in right lobe in addition to the expected activity in the lungs.