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*** These letters have been shown to Dr Gardosi and colleagues whose reply follows.—ED. L.

SIR,—Professor Chard and colleagues make an important point about ultrasound dating but there is good evidence from comparative studies that biparietal diameter measurement up to 20 weeks can be used for accurate determination of gestational age, with no demonstrable difference between European and Asian,[1] black,[2] and mixed racial groups.[3] We therefore believe that our routine 18–19 week anomaly scans can confirm or correct gestational age across the whole population.

Dr Chang and colleagues miss our distinction between birthweight and growth charts (see figs 1 and 2 in our paper). Our charts for birthweight have the well-known skewed distribution at preterm gestations (fig 1). That is why we disregard all preterm data for the growth chart, and use only the birthweight range of deliveries at 40 weeks. We are thus left with 6 points as 10th, 50th, and 90th centile markers for males and females. The growth component can be calculated from any published, longitudinally derived formula for intrauterine weight gain, and the computer program fits the curve to these points, being the individually adjusted normal 40 week weight limits in each pregnancy. Therefore the slope is different for each combination of physiological variables, and the curve illustrates the growth velocity with which the fetus should reach its expected birthweight at the end of a normal pregnancy (fig 2). We showed that birthweights can thus be seen in relation to their longitudinal growth centiles rather than cross-sectional birthweight centiles; the distinction is necessary for adequate identification of preterm, small for gestational age (SGA) babies.[4] This point is shown by comparison of the centiles of the 34-week baby in figs 1b and 2b.

Prospective determination of an intrauterine weight curve or "growth potential" for each individual fetus may be an alternative but requires at least two mid-trimester scans, at least 6 weeks apart, and inaccuracy in any of the measurements may be significantly magnified by the required calculations.[5] Serial scanning is indicated in high-risk cases but is hardly cost-effective and practicable for the whole maternity population. Our charts are designed for growth screening by improving the accuracy of assessment of previous birthweights and by providing adjusted limits for fundal height measurements and/or an ultrasound fetal weight check in the third trimester. This should improve identification of those pregnancies which do need serial screening.

Professor Steer gives no information about induction rates in his letter or in the original publication of his data on intrapartum complications. It must be assumed that at least some of the higher number of Indo-Pakistani babies labelled SGA were also identified as such antenatally, and that this diagnosis was acted upon. Induction of labour is a well-known contributor to intrapartum difficulties, and differing rates of this therapeutic intervention would make a comparison of soft outcome measures such as fetal-heart-rate abnormalities, meconium, and 1 min Apgar scores meaningless. Harder evidence, from much larger studies with perinatal mortality as endpoint, makes nonsense of the use of the same birthweight standards across different racial groups.[6,7] Steer uses Altman and Coles' nomograms to calculate centiles but appears to ignore the adjustment tables for maternal height and weight that they were published with and that also appear with the original Aberdeen database. The need to correct for race has also been well documented, and it has been proposed to account for this variable by adding another, race-specific weight adjustment before using the nomograms.[8] Failure to make such correction is likely to be the main reason why Steer's Indo-Pakistani population appears to have a higher rate of SGA babies, and our study discussed the need to avoid such pitfalls. An adjusted centile limit is more specific at any level; the 5th centile may be useful for retrospective analysis but for our chart we prefer the 10th centile cut-off, this being the more sensitive marker for prospective decisions on antenatal management (eg, increased surveillance).

Customised growth charts aim to bring well-proven concepts into the realm of everyday clinical practice. They will help obstetricians to screen for fetal growth retardation, by allowing for physiological variables such as maternal race and stature, and not confusing them with real risk factors such as malnutrition, smoking, and low socioeconomic status.

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Magnetic resonance studies in stroke

SIR,—Dr Donnan (Feb 22, p 473), in his concise discussion of present diagnostic techniques in stroke, includes the prospects for magnetic resonance (MR) methods such as MR-angiography (MRA) and MR-spectroscopy (MRS) (on p 476 of Donnan's article, ^{32}P and ^{32}Na should read ^{31}P and ^{23}Na). Although ^{31}P-MRS has been extensively applied in animal models of cerebral ischaemia[1,2] and yields direct information on mitochondrial energy metabolism, there is now evidence that water-suppressed ^{1}H-MRS will have a more widespread clinical application because the proton nuclei provide higher MR sensitivity and localised ^{1}H-MRS depends on hardware configurations identical to those for MR imaging (MRI).[3] Spatial resolution of MRS is still restricted to volumes of interest (VOI) of 1–27 ml, which may cause partial volume averaging between normal and ischaemic tissues. Until now, the concentrations of metabolites cannot be absolutely quantified from in-vivo spectra. However, only ^{1}H-MRS provides direct and non-invasive observation of lactate accumulation in cerebral ischaemia and, within the same examination the metabolic state of neurons can be estimated from the presence of N-acetyl aspartate (NAA).[4]

We used a combined MRI and localised ^{1}H-MRS protocol to examine 10 patients within the first 8 h after stroke, 15 in the subacute stage of hemispheric infarction, and 15 in the chronic stage. 6 were investigated after hemispheric transient ischaemic attack (TIA). MR images demonstrated cerebral ischaemia as early as 4 h after onset of symptoms. Image-targeted, water-suppressed proton spectra were acquired from 27 ml VOIs by means of a stimulated echo (STE) sequence[3] with a repetition time of 1500 ms

LACTATE AND N-ACETYL-ASPARTATE (NAA) IN LOCALISED ^{1}H-SPECTRA OF ISCHAEMIC STROKE AND TIA IN 45 PATIENTS

—	Lactate			NAA		
	0	+	++	N	−	−−
TIA (n=6)	3	3	..	4	2	..
Acute infarction (<8 h) (10)	..	4	6	..	5	5
Subacute infarction (days 1–30) (15)	..	11	4	..	12	3
Chronic infarction (months to years) (15)	15	..	..	3	11	1

Lactate: 0 not present; + raised; ++ greatly raised.
NAA: N normal; − decreased; −− striking depletion.

and an echo time of 270 ms, to rephase the J-coupled resonance of the lactate methyl protons together with the singulet resonances (table).

Lactate was raised in all acute and subacute infarctions for up to 20 days but was absent in the chronic stage. ^{1}H-MRS distinguished acute and chronic ischaemic changes, but our protocol could not differentiate active anaerobic glycolysis from lactate accumulation in necrosis. Sequential ^{1}H-MRS examinations and correlation to imaging results partly compensated for this shortcoming in our study. The introduction of ^{13}C-labelling techniques will remove this difficulty. In 3 patients, we noted moderate lactate values in the vascular region responsible for a TIA.[5] NAA was reduced in acute and chronic infarctions, which supports previous findings[4] that NAA indicates the metabolic state of neurons in areas of ischaemia.

The unique advantages of ^{1}H-MRS are that it can be done with clinical 1·5T scanners on a routine basis and that morphological and metabolic information can be obtained from a single examination. Since MR angiography can be added to MRI/MRS within 60 min total examination time[6] the method will provide a more complete, fast, and non-invasive and therefore economical diagnostic approach to cerebral ischaemia.

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Glutaraldehyde allergy in hospital workers

SIR,—Dr Calder and colleagues (Feb 15, p 433) provide some interesting evidence that glutaraldehyde may act as an irritant in hospital staff who handle it but none that it produces an allergic response, as the title of their letter suggests. Moreover, we do not know how the nurses in their survey were selected nor how representative they are of all nurses exposed to glutaraldehyde. Our data suggest that Calder et al may be overestimating the effects.

With colleagues, I conducted a survey of all 150 staff who were exposed to glutaraldehyde at St Mary's and Central Middlesex Hospitals; some 18 departments in all took part. All subjects completed a questionnaire and had lung function measured once. The principal signs and symptoms noted are shown in the table in which I have divided the group into those with daily exposure and those whose exposure was less frequent. The prevalence of signs and symptoms is generally less than that reported by Calder et al and by others.[1,2] These differences may indicate variations in exposure, selection, or working practices. In this study, symptoms or signs were not significantly related to age, occupational group, frequency of exposure, or length of exposure (in years from first exposure), and this suggests that glutaraldehyde is having a direct, irritative effect.

PREVALENCE (%) OF SYMPTOMS AND SIGNS IN HOSPITAL STAFF EXPOSED TO GLUTARALDEHYDE BY FREQUENCY OF EXPOSURE

	Exposure:		
—	Daily (n=70)	Less frequently (n=80)	Total
Runny eyes	28·6	32·5	30·7
Skin irritation	22·9	22·5	22·7
Runny nose	14·3	22·5	18·7
Discolouration of skin	21·4	12·5	16·7
Upper respiratory tract irritation	17·1	12·5	14·7
Cough	18·6	10·0	14·0
Unpleasant taste	12·9	10·0	11·3
Wheezy chest	8·6	10·0	9·3
Chronic dermatitis	1·4	2·5	3·3

Although just over 9% of all subjects complained of a wheezy chest when they were exposed to glutaraldehyde, none had abnormal lung function tests, not even those who smoked. Although a single test of lung function has limitations, these data do not provide any evidence for chronic airways disease in this group of hospital workers.

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Blood donors and autoimmunity

SIR,—Two of my patients have been rejected as blood donors, having previously given blood several times. In one the reason was vitiligo and in the other hypothyroidism. I wrote to the regional transfusion centre and was told that the UK National Committee for Donor Selection has "consulted an immunologist on this matter". Because of the autoimmune basis of vitiligo the committee had decided, as a matter of national policy, that people with vitiligo should not be accepted as blood donors; a similar explanation was given for the patient with hypothyroidism. I know of no evidence that vitiligo can be transmitted by blood and such a route seems most unlikely. We are constantly told that there is a shortage of blood for transfusion; such exclusion policies must result in the exclusion of many potential donors.

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Potential dangers of laparoscopic insufflator

SIR,—For laparoscopic surgery carbon dioxide gas insufflators capable of delivering at flow rates of 4 l/min or more have been developed. Some insufflators also include a recirculation pump to remove smoke and laser plume from the peritoneal cavity (fig 1). To avoid having too many tubes the recirculation gas return and the insufflator gas input are united at a T junction so that a single gas supply tube passes to the patient. The pressure flow is controlled by a sensor reading from the insufflator line, the assumption being that this reflects intra-abdominal pressure. This may not be so, however, and the consequence could be dangerous over-insufflation of the peritoneal cavity or frequent inappropriate "high-pressure" alarms.

The three possible configurations of the return tubes, when the T junction is used, are shown in fig 2. In fig 2a intra-abdominal pressure will be underestimated, due to the Venturi effect, and over-insufflation might occur with the risk of carbon dioxide embolus. In fig 2 b and c the high-pressure alarm will sound inappropriately because intra-abdominal pressure is overestimated. To overcome these difficulties the T junction has been modified (fig 2d), with the recirculation gas return limb of the T junction narrowed. However, this fixed constriction cannot compensate for the varying gas flow rates generated by a range of recirculating pump speeds and insufflation rates. We have evaluated two commercial modifications of the T junction on a test circuit and conclude that the only accurate measurement of intraperitoneal pressure is afforded by a dedicated insufflation gas line.

It is now our routine practice—and our recommendation to those who use insufflators like the one illustrated in fig 1—that the recirculating gas and insufflation gas be returned via separate cannulae. Care should be taken that the cannulae for the recirculation gas circuit remain intraperitoneal since there is no

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