Volumetric analysis of aeration in the lungs during general anaesthesia

Sir,—I read with interest the article by Reber and colleagues on volumetric analysis of aeration in the lungs during general anaesthesia.1 This is a study of radiographic density of the lung, using data consisting of picture elements (pixels) sized 0.9 x 0.9 x 4 mm. The density of the tissue in the pixel varies with the amount of air it contains, and the authors interpret these changes in two ways.

First, they use density to assess the degree of expansion of the lung tissue, in terms of aeration. Second, they use changes in density during the breathing cycle to estimate regional ventilation. I would agree that the less dense the lung, the better aerated it is likely to be, because air causes less attenuation than lung tissue (or blood, which here seems to have been assumed to remain a constant fraction of the tissue). Air causes attenuation, in the scale used, of 1000 units, whereas collapsed atelectatic lung causes a much greater attenuation of 100 to +100 units. The authors argue that the distribution of pixel density indicates the distribution of poorly aerated and atelectatic lung. They suggest that the amount of lung tissue in an atelectatic area is four to five times greater than in a normally expanded part of the lung. This implies that by expansion with four times that volume of air, one atelectatic pixel would become five pixels with a normal radiographic density, as the radiographic density of air is very small compared with that of lung tissue. We are left uncertain as to how much air needs to be added to “poorly expanded” lung tissue to convert it into tissue with “reduced aeration” or “normal aeration”, although these quantities would be useful for relating the current measures to previous data obtained with other methods.

This knowledge is of particular importance when it comes to drawing conclusions on “distribution of ventilation”, as the authors have done in their subjects both during spontaneous breathing and mechanical ventilation. Here it is useful to remember how earlier studies of regional ventilation were performed, as this may help to elucidate some of the findings in the study of Reber and co-workers. Regional ventilation, measured with radioactive xenon, is usually expressed as ventilation in proportion to the volume of that region at total lung capacity (TLC),2 this is done because at TLC the alveoli are nearly equal in size throughout the lung, and therefore expression of regional volumes and regional ventilation as a proportion of volume at TLC becomes equivalent to expressing these values in terms of “per alveolus”. This is more useful than expressing ventilation or lung volume in terms of lung tissue at other states of inflation. If inflation of each part is unequal, then the amount of lung tissue present in each unit volume (in the present case) depends on the amount of distension of the lung that is being considered. Indeed, it is intuitively obvious that erroneous conclusions could result if regional ventilation were based on any other lung volume than the volume at TLC. Consider 100 ml of collapsed lung tissue which is distended by 100 ml of air. It would then have a volume of 200 ml, and if its regional ventilation were expressed as a percentage of the original tissue volume, it would have received 100% ventilation. However, this volume of atelectatic tissue can probably be fully expanded to a volume of approximately 1000 ml and hence if regional ventilation is expressed as a proportion of the volume of tissue at TLC, this ventilation would be 10%. There may be an analogous effect, of an inappropriate denominator, when radiographic density is used as a measure of aeration. This may explain some of the results when Reber and co-workers have “assumed a gas distribution in proportion to the attenuation changes” that they observed between inspiration and expiration. There may be a relationship but it is most unlikely to be a direct linear proportion between density change and regional ventilation.

They report that the radiographic density of the gravitationally dependent regions is two times greater than that to the upper regions. However, comparison of the data in subjects breathing spontaneously with those whose lungs were ventilated artificially indicates a further difficulty in the use of radiographic density as an indicator of aeration alone. The tidal volumes of the patients breathing spontaneously are unlikely to have been greater than those of the patients undergoing ventilation with volumes of 8 ml kg−1. What is remarkable is that the change in mean lung density caused by mechanical ventilation (54 units) was much less than the density change spontaneous breathing (131 units), although tidal volume was probably less with spontaneous breathing. A factor that could explain this difference is the change in the volume of blood in the lungs during the 10-s breath-hold at end-inspiration necessary for the scan. This change in blood volume in the tissue may well not be uniform from the top to the bottom of the lung. This underlines the fact that factors other than ventilation can affect radiographic density, and this could affect the comparison of measurements made during spontaneous breathing with those made during mechanical ventilation.

Until these questions and uncertainties regarding the proportional relationship of radiographic density and aeration are addressed, I suggest that the inferences from CT studies should be limited to qualitative assessment of aeration, and that the expression “ventilation distribution” is not used.

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Sir,—In his letter, Dr Drummond discusses some important aspects of the measurement of ventilation distribution. We agree that the calculation of ventilation “per alveolus” offers the advantage of taking into consideration the increasing amount of lung tissue, and thus of alveoli, down the lung.

One approach for depicting ventilation per alveolus is to calculate the gas/tissue ratio of a pixel, assuming that the amount of tissue in a pixel reflects the number, or size, of alveoli. In this approach, it can be seen that the difference in ventilation per alveolus of upper and lower lung regions is smaller than per pixel. Using a somewhat different approach, Dr Drummond has also shown this difference and he also pointed out that a shift of blood, either within the lung or in and out of the lung during the breathing cycle, further complicates the analysis. We thus agree with Dr Drummond on these aspects of ventilation calculations. However, relating ventilation to each alveolus is only one way of presenting the results. Ventilation can also be related to the topography of the lung and, for example, to the distribution of atelectasis. In a ventilation-perfusion scan for assessing pulmonary emboli, ventilation is related to pixels, or volume elements, as is the case in our study. These considerations taken together prompted us to present our data in a manner that has not been manipulated.

The amount of gas within a particular pixel of a CT scan can be estimated from the attenuation value of that pixel. This implies that what is not gas is tissue with a certain attenuation value, therefore the measured attenuation is the weighted mean of gas and tissue. In our study, we used the standard scale of Hounsfield units where air has the value of −1000 Hounsfield units (HU) and water 0 HU. We have considered that lung tissue corresponds to water, an assumption that is used frequently for analysis of a CT scan. The changes in attenuation value can be measured in a particular pixel or a region made up by a number of pixels. Therefore, the change in its gas content can be calculated, which correlates to ventilation. This is what we have done. Our data are consistent with those of Rehder, Sessler and Rodarte who used an isotope technique in awake and subsequently anaesthetized subjects. Even though we agree with Dr Drummond that calculations can be made differently, we consider our conclusion, that ventilation distribution is affected by anaesthesia and mechanical ventilation, with an increase in ventilation to non-dependent regions, is correct.

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**Failed intubation during obstetric anaesthesia**

Sir,—Hawthrone and colleagues reported an increase in the incidence of failed tracheal intubation, during Caesarean section under general anaesthesia in their unit, from 1 in 300 in 1984 to 1 in 250 in 1994. As no national data are available they asked if this reflected a national trend. Since mid-1986, data on obstetric anaesthesia have been collected by hospitals in the South West Thames Region. We too have seen a decrease in the use of general anaesthesia for Caesarean section from 66.1% (range 39.8–81.9%) in 1987 to 22.9% (8.8–37.5%) in 1995. The number of general anaesthetics for Caesarean section decreased from 2198 in 1987 to 1320 in 1995. There has been a progressive increase in the incidence of failed intubation, to 1 in 173 in 1994 and 1 in 165 in 1995. Last year in the six units which performed less than 100 Caesarean sections under general anaesthesia, the incidence of failed intubation was 1 in 130; in the seven units that performed more than 100, the incidence was 1 in 188.

Our experience in South West Thames suggests that it is likely that there has been an increase nationally in the incidence of failed tracheal intubation during Caesarean section under general anaesthesia and that one probable cause is anaesthetists’ decreased exposure to obstetric general anaesthesia. In the recently published Confidential Enquiries into Maternal Deaths in the United Kingdom 1991–1993, all direct deaths associated with anaesthesia for Caesarean section involved the use of general anaesthesia. Any increase in the incidence of failed tracheal intubation during Caesarean section under general anaesthesia should be a cause for great concern.

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**The intensive care unit cost of surviving and dying**

Sir,—The recent editorial by Bion and Strunin introducing the issue on multiple organ failure stated that it costs twice as much to die in an intensive care unit as it does to survive, quoting a reference from 1986. This is true when examining costs of all patients admitted to intensive care. In 1995 our intensive care unit cost was an average of £1997 to manage a patient who survived to leave our hospital, but more than twice this at £4116 to treat a patient who eventually died. The median APACHE II score for these patients was 15 (range 1–48).

When analysis of patient sub-groups is undertaken, however, a different picture emerges. We looked at the costs of treating a group of our most critically ill patients, namely patients referred from the specialties of renal medicine and renal transplant. In this sub-group (median APACHE II score 27 (range 14–48)) the costs, during the same period, of treating those who survived and those who went on to die were similar (£4225 and £4708, respectively).

Thus, the quoted 2:1 ratio in cost of treating survivors and non-survivors may be misleading as the overall cost of a survivor depends on the relatively larger number of less ill patients managed in intensive care who have a good chance of survival.

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Sir,—We are happy that Drs Thompson and Spiers agree that overall it costs at least twice as much to generate a non-survivor as it does a survivor from intensive care. For example, a 14-month study of 3600 ICU patients at Guy’s Hospital showed that 15% of that population died, consuming 37% of the ICU budget in the process. Of course, sub-group analysis generates exceptions to this rule, and in the article which we quoted in our editorial, the authors showed this for patients undergoing elective surgery. These variations are of interest, but they do not alter our main theme, that death in intensive care is doubly wasteful of both lives and resources. It makes sense, therefore, to concentrate on prevention of multiple organ failure which was one of the main themes of the July postgraduate issue of the journal.

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Aortic stent surgery

Sir,—We wish to comment on two points made in the letter of Baranowski and Adiseshiah on aortic stent surgery. They noted, and appeared to accept, a mortality of 10% and put forward as a benefit of the stent procedure the fact that "the patient’s trachea can usually be extubated early...".

Our own published experience of conventional elective abdominal aortic surgery using a combined general-extradural anaesthetic technique is that hospital mortality is well below 5%, with the trachea of virtually all patients being extubated immediately after surgery. We believe that most centres consider a mortality rate of more than 5% unacceptable, and we support the view of Thompson and Greiff that the stent technique should be introduced with great caution. In addition, fully informed consent must be obtained for all new procedures, even if they are offered to patients in whom the standard technique is considered high risk. Until the rate of conversion to an open procedure (current 35%) is reduced significantly, the stent operation should not be offered to patients who are not considered fit for conventional surgery.

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Sir,—We agree fully with the comments of Wildsmith and McClure that a mortality rate of 10% is unacceptable and that is why we felt moved to write our letter on reading the first publication by Greiff, Thompson and Langham. In that original report they did not indicate clearly the risks. We also agree that patients should give fully informed consent. It is for that reason that the consent in our hospital is obtained by consultant staff.

A recent audit of our mortality rates for open elective surgery using general-extradural anaesthetic techniques and early extubation indicated our mortality to be approximately 1%. We feel that it is inappropriate to increase the mortality risk in these patients by subjecting them to a stent procedure with a 35% conversion rate and 10% mortality risk. However, in common with many centres, we receive referrals where the local team has not been prepared to undertake an open operation as the surgical risks have been considered too great. In these patients we feel that there is an advantage to the closed technique with the mortality probably being less than that after the open approach in this specific group. We have had several such patients only spending a night on the high dependency unit as opposed to the predicted several days. A controlled study examining different techniques and risk groups needs to be carried out.

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A complication of intra-arterial shunting during carotid endarterectomy

Sir,—A 68-yr old male ex-smoker presented after one episode of amarous fugax affecting his right eye and lasting a few minutes. Resolution was complete and there was no residual neurological deficit. Doppler studies showed a 75% luminal obstruction in the right internal carotid and 65% in the left, and he was scheduled for right carotid endarterectomy.

In the anaesthetic room, a Fosswich 20-gauge (Ohmeda) arterial cannula was inserted in the right radial artery, after failed cannulation of the left, for continuous monitoring of arterial pressure. A typical arterial pressure trace was displayed on the monitor. General anaesthesia was induced with fentanyl, etomidate and atracurium, and maintained with isoflurane and 70% nitrous oxide in oxygen.

When dissection of the carotid artery was complete, arteriotomy was performed and a Pruitt balloon shunt inserted. Immediately, the arterial pressure trace became attenuated and the reading changed from 130/70 to 75/65 mm Hg. Flushing the cannula had no effect and free aspiration of blood was possible. Withdrawing the shunt restored a normal arterial pressure trace. It was concluded that the caudal end of the shunt was lying at the junction of the innominate and subclavian arteries and causing significant obstruction to flow of the latter (see fig. 1).

In addition to making the arterial pressure reading unreliable, this could have had a significant impact on cerebral blood flow because the right vertebral artery is a branch of the subclavian. During carotid endarterectomy this further reduction in circle of Willis supply may be crucial. On the left, the subclavian and common carotid arteries have separate aortic origins, and vertebral artery blood flow could not be compromised in a similar manner.

We suggest that the right radial pulse or pressure should be monitored when a right carotid shunt is placed, so that subclavian occlusion can be detected.

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Figure 1 Diagrammatic illustration of the obstruction to flow of the subclavian artery as the caudal end of the shunt was lying at the junction of the innominate and subclavian arteries.
Transoesophageal echocardiography in anaesthesia and intensive care

Sir,—The delay in appearance of an editorial on transoesophageal echocardiography (TOE) in British anaesthesia literature reflects the slow uptake of this tool by British anaesthetists compared with our colleagues in the continent or across the Atlantic.

TOE represents a large initial cost forcing consideration of its benefits, especially in our health system where its use does not allow immediate remuneration (as in the USA). The plethora of uses and undefined benefits to outcome has generated an impression of an expensive toy for the boys. It is not clear that its use can be largely subgrouped into a monitoring role or a diagnostic role. As anaesthetists we have concentrated initially on its monitoring role, especially in relation to regional wall motion abnormalities signifying ischaemia. Unfortunately, the “dancing doughnut”, as the short axis monitoring view has been derogatorily termed, has not been specifically linked to other monitors available and certainly in non-cardiac surgery has not justified its expense. It is not known if the addition of colour kinesis will significantly improve its usefulness. Although many variables may be derived from Doppler data, including cardiac output and left atrial pressures, this data is usually too cumbersome to allow routine use. However, for the first time TOE allows us to directly assess the volume status of the left ventricle compared with previous indirect estimates of LV filling from pressure data. In addition, with concomitant pressure data TOE can also provide a load-independent measure of contractility.

In contrast, its diagnostic role is now well established in cardiology. That this can translate to the operating room, intensive care or emergency room has now been well documented. Intensive care and the emergency room, TOE represents a portable easily accessible diagnostic tool. In the operating room we should differentiate between cardiothoracic surgery and other types of surgery. Despite its potential the routine use of TOE in non-cardiothoracic surgery has not been justified except possibly as a volume monitor where extensive blood loss is expected or in the diagnosis of hypotension. In cardiothoracic surgery the diagnostic role of TOE has come into its own. While its monitoring role in routine coronary artery surgery is debatable, its diagnostic role in complex valve, paediatric cardiothoracic surgery and artificial device implantation can contribute significantly to surgical success. In addition, scanning the aorta for atheroma plaques and left ventricle for residual air pockets has proved useful. After operation, its use can quickly differentiate between the main causes of hypotension and is invaluable in assessing postoperative deterioration.

This diagnostic role in the intraoperative period is having a real impact on the work of cardiac anaesthetists. For the first time the results of a surgical technique are being reviewed by anaesthetists and subsequent decisions based on them, including the need to go back on bypass and modify the surgery. Where these decisions are to be made, there is no doubt that the level of competence is hard to maintain for the occasional cardiac anaesthetists, who may lack credibility in their ability to defend themselves only in a cardiac centre. This may drive us towards dedicated full-time cardiac anaesthetists, at least where complex procedures are undertaken. Indeed, some centres in the USA do not appoint cardiac anaesthetists unless they are competent in TOE.

Why then has the uptake of TOE been so slow to date in the UK? First, the enormous initial capital expense has been difficult to access. Second, there remains a fear of technological obsolescence, with many developments in the pipeline, including colour kinesis, three-dimensional reconstruction and real-time, dimensional views. In fact, in the 5 yr since the first editorial quoted by Townend and Hutton, technology has moved from single plane probes to Doppler capability, biplane probes and now multiple plane probes with automatic border detection capabilities. Third, an investment of time and training is required before competence is acquired and quality assurance is acceptable. Fourth, its role has not been defined. To these ends, the Society of Cardiovascular Anesthesiologists and American Society of Anesthesiologists have recently published guidelines on perioperative TOE training and use.

In summary, the impact of intraoperative TOE on non-cardiothoracic surgery is yet to be defined. In cardiothoracic anaesthesia, its use is here to stay.

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Haemodilution induces a hypercoagulable state

Sir,—Ruttman, James and Viljoen have described the effects of haemodilution on in vitro coagulation using the TEG, and among their conclusions suggested that 20% dilution with isotonic saline increases absolute clot strength, while at the same dilution with Haemaccel (3.5% polygeline solution) clot strength is unaltered.

We have recently published similar work, but with a range of dilutions (15, 30, 45, 60 and 75%). We found that both the size (clot weight) and strength (using maximal amplitude on the TEG) of clots formed in vitro were reduced considerably with increasing dilutions of both Haemaccel and Gelofusine (4% succinylated gelatin) compared with the same dilutions with saline or Ringer’s solution. Scanning electron microscopy of these clots...
showed a reduction in cross-linkage with the colloids compared with dilution with saline, or controls from undiluted blood. This could be explained by an effect on fibronectin, as other workers have shown that gelatin binds to fibronectin and that infusion of these fluids decreases plasma concentrations of fibronectin. Although Ruttmann, James and Viljoen emphasized that their study did not examine greater or lesser degrees of dilution, our findings raise concern where large volumes of these fluids are given to patients requiring full haemostatic competence, and we endorse his suggestion that in vivo studies using the TEG should take place in patients receiving different fluid regimens.

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Sir,—Mardel and colleagues reported on a study comparing the effects of various dilutions of blood with crystalloid with those with colloid. They found some inhibition of clot maturation, as measured by clot strength, in the colloid group and attributed this effect to the binding of fibronectin to the gelatin molecule. In this respect their results are in agreement with our findings.

However, we are confused by the assertion that “scanning electron microscopy of these clots showed a reduction in cross-linkage with the colloids compared with dilution with saline, or controls from undiluted blood”. Careful reading of the report of Mardel and colleagues shows that their controls were diluted with saline or Ringer’s lactate. Nowhere do they mention an undiluted control.

It is our contention that haemodilution with crystalloid is not inert but activates some aspects of coagulation in an as yet undefined manner. It is therefore difficult to interpret coagulation studies in which haemodilution with crystalloid is used as a control as these do not necessarily show that gelatin haemodilution per se causes reduced clot strength compared with the native condition.

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ERRATUM

The first author of the article is S. Al Ramadhani and not S. A. L. Ramadhani as published. Also, Dr Ramadhini’s qualification is FFARCSI and not FRCA.

We apologize for this confusion.