Flow cytometry

Sir,—With regard to the article by Heine and colleagues,1 we believe that several points require clarification.

The authors claim that the technique using 123-dihydro-rhodamine technique is its flow cytometry base, allowing the study of a single cell population. However, in the study by Heine and colleagues1 such benefit is negated by separation of individual cell populations. The cell separation technique as presented is unclear. The layering of whole blood onto Ficoll-Hypaque density medium would result in a mononuclear cell layer with all granulocytes sedimenting with erythrocytes. However, the light scatter diagrams show mixed cell populations comprising monocytes, lymphocytes and granulocytes. It is difficult to see how the data relate to the methodology given or the need for cell separation in the first place.

The propofol carrier, 10% Intralipid, inhibited respiratory burst of granulocytes in an identical manner to that of propofol. However, any effect of Intralipid on either light scatter or light quenching in the flow cytometry system is not discussed. In addition, cited literature has been misquoted. Intralipid was not shown to suppress T cell-mediated immunity as stated.2 Other studies have confirmed this finding.4 The effects of lipid emulsions on cell function are dependent on cell type and whether or not the study involves isolated cells in vitro or cells after in vivo lipid exposure.5,6 In addition, the antioxidant effects of propofol have not been discussed.

Finally, the use of isotonic saline to dissolve thiopentone and methohexitone preparations inevitably resulted in hypertonic solutions. This may be inappropriate because hypertonic solutions induce a stress response, affect cell function and may also affect respiratory burst activity.7,8 Pre-exposure of cells to anaesthetic agents before stimulation with PMA4 implies that the results may represent the effect of the drugs on protein kinase C rather than on NADPH oxidase directly. This factor is not discussed. Protein kinase C is also affected by hypertonity.9

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Sir,—We thank Drs Galley and Webster for their interest, however, we are not in accordance with the majority of their comments concerning our article.4 They stated that the only apparent benefit of the dihydro-rhodamine technique appears to be its flow cytometry base. With regard to our study design, the most important difference between the applied technique compared with chemiluminescence is that the dihydrodorodamine technique involves actual measurement of the activity of the respiratory burst oxidase. This enzyme is responsible for intracellular superoxide anion production.2 In our study respiratory burst activity of only viable neutrophils was analysed in a whole blood system. As the number of analysed events in flow cytometry is practically unlimited, data with low variation coefficients are produced and sensitivity can be increased. Sedimentation at 1 g and 22 °C was used as a reduction of erythrocytes in the whole blood samples, as shown in the light scatter diagrams.1 Thus no centrifugation or erythrocyte lysis was required and preactivation or impairment of neutrophil function was avoided. This does not imply that chemiluminescence itself is a less valuable method and we did not state so in our article.

Furthermore, Des Galley and Webster claim that the effect of Intralipid on either light scatter or light quenching in the flow cytometry system had not been discussed in our article. We described in detail the effects of 10-fold anaesthetic doses of propofol and Intralipid on side scatter signals (SSC) in the results and also in figure 5. Figure 5 was given as an example of the decrease in SSC signals of neutrophils which were incubated with the 10-fold anaesthetic doses of propofol. We discussed further if the changes in the cell membrane structure induced by lipid solutions may provide an explanation for the observed decrease in the SSC signals.

We apologize for citing the article of Monson and colleagues who reported stimulation of T cells by lipid solutions under impairment of T cell-mediated immunity.1 Suppression of intracellular enzyme respiratory burst oxidase by i.v. anaesthetics is in the first instance an immune compromising effect. It would have been very speculative to discuss a possible antioxidant effect of propofol only on the basis of our in vitro experiments. PMA is a well known and accepted stimulus for respiratory burst which acts directly via protein kinase C.

Finally, control experiments (data not shown in article) with the studied anaesthetics and no PMA stimulation excluded preactivation of neutrophils. Thus the hypertonity of the solutions did not affect the results of the particular system of the study.

The effect of the three different lipid solutions (concentration 600 μg ml−1) on respiratory burst under different stimuli was investigated in a consecutive study not yet published. We studied long-chain triglycerides (Intralipid 10, Pharmacia), LCT/MCT preparation (Lipofundin MCT 10%, Braun) and omega-3 fatty acid (Omegavenös 10%, Fresenius). Suppression of respiratory burst by all three lipid solutions under PMA stimulation (% mean inhibition: Intralipid, 87.9 (SD 8.5), Lipofundin, 78.8 (17.0), Omegavenös, 84.3 (10.7)) was found to be similar to previously described results.1 Under stimulation with E. coli, respiratory burst was suppressed by Intralipid (8.0 (9.3)) and Omegavenös (9.6 (11.1)) and increased by Lipofundin (−42.7 (21.8)). Therefore, suppression of respiratory burst by lipid
solutions seems to depend both on the stimulus and nature of the lipid solution.

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ASA classification and perioperative variables as predictors of postoperative outcome

Sir,—I read with interest the article of Wolters and colleagues1 and I have a comment on their statistical analysis.

They stated “the Student’s t test was used to quantify the difference in the means of independent perioperative variables between ASA classifications”. This test can be used correctly between no more than two groups and it is not used to quantify the difference but just to verify if a difference between means probably exists.2

The authors did not report the SD of values of operation duration (min), intraoperative blood loss, intensive care stay (days) or postoperative stay (days) in relation to ASA, but considering preoperative status in relation to hours of postoperative ventilation they report the following mean (SD) values: arterial hypertension yes 7.3 (37.3), no 6.5 (29.8), previous myocardial infarction yes 13.7 (52.2), no 6.4 (34.3); severe bronchopulmonary disease yes 10.6 (45.5), no 5.7 (32); smoker yes 6.3 (30.8), no 6.9 (37).

Parametric tests such as the Student’s t test or analysis of variance can be used correctly only if the distribution of the variable is symmetrical, when a variable has only positive values and the SD is greater than the mean, the variable must be considered non-symmetrically distributed,2 and therefore parametric statistics cannot be used to describe or verify a hypothesis on such data.

If the authors had used logistic regression for specific postoperative complications they would have obtained more useful results than to define additional risk factors for indefinite “postoperative complications”.3

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Sir,—We appreciate the comments of Magi on our article. Indeed we used the Student’s t test to explore observed differences between the means of selected variables for each of two defined subgroups in order to quantify the probability that this differences exists by chance sampling only, as stated in the first sentence on p. 219 “…in relation to ASA (table 6) we found an increase in duration of operation between ASA I and ASA II–IV combined (P<0.05) and between ASA II and III (P<0.05)…”.

In the next step we used explorative P values as hints to select some variables as possible predictors for postoperative outcome by modelling the probability of a postoperative complication using a logistic regression model.

We agree with Magi in pointing out some well known general rules of thumb for the correct use of the t test, which should be followed, especially when comparing means for small samples.

Additionally, it should be remarked that the methodological properties of the two-sample t test may be poor for small samples, even if the data are distributed normally, if sample sizes and variances differ greatly. On the other hand, for sample sizes that are large enough (as for the data in our article) well known implications from central limit theory allow the use of the t test (as for the data in our article) distributions. This “robustness” of the t test implies that for “large” samples and without the assumption of normality, the nominal P value is approximately the same as the true significance level whenever the null hypothesis holds.

We apologize for giving SD values only for operation duration, postoperative stay and some other variables for the complete sample (c.f. table 2) but not in relation to ASA subgroups. This omission was designed to prevent table 6 from becoming too large.

We again emphasize that the central aim of the study was an attempt to quantify the importance of ASA classification and well known clinical risk factors in the evaluation of complicated surgical outcome by multivariate analysis of clinical data.

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ASA classification and perioperative variables: graded anaesthesia score?

Sir,—As Wolters and colleagues1 pointed out in discussion, a major drawback of the ASA system is assessment of a patient’s “correct” ASA classification by different anaesthesiologists. This has been shown clearly in previous studies2,3 in which, after consideration of audit, we have concluded that the preoperative status cannot satisfactorily be described by it alone. The situation in obstetric anaesthesia is no better.4 Wolters and colleagues overcame this problem by using two experienced anaesthetists to classify each case, which is far removed from routine UK practice.

We believe that an improved scoring system needs to incorporate both physical status (chronic health evaluation) and operative risk. It must be both reproducible and simple to use. The present situation could be greatly improved by combining a minimally modified ASA physical status classification with an indication of relative “anaesthesia risk”, thereby producing a composite graded anaesthesia score (GAS).

Anaesthesia risk is multifactorial, but attempts to incorporate this into a physical status/chronic health score create difficulties and inconsistencies, particularly in respect of patients suffering

Table 1 Graded anaesthesia score (GAS). Morbidity/mortality related to anaesthesia provision for patients with chronic stable intercurrent medical conditions is not an additional risk. Additional risks include: (a) acute exacerbations/disturbances in chronic disease states; (b) condition for which surgery is required; (c) proposed surgical plan and its urgency; (d) trauma; (e) physiological disturbances; and (f) anaesthesia influencing conditions (for example 2 = dental work, pseudocholinesterase abnormalities, URTI etc; 2.5 = aspiration risk, normal pregnancy, moderate obesity, chronic airway management problem, age <1 or >80 yr, anaesthesia related allergies etc; 3 = malignant hyperthermia susceptibility, moderate physiological disturbances following trauma, sepsis or head injury, pre-ecclampsia, etc; 4 = acute airway obstruction, eclampsia, severe physiological disturbances following trauma, sepsis or head injury, morbid obesity, etc)

<table>
<thead>
<tr>
<th>Chronic health/physical status</th>
<th>Additional anaesthesia risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. A normal healthy patient</td>
<td>1. No additional risk</td>
</tr>
<tr>
<td>2.5. A patient with moderate systemic disease</td>
<td>2.5. Moderate</td>
</tr>
<tr>
<td>3. A patient with severe systemic disease that limits activity but is not incapacitating</td>
<td>3. Major</td>
</tr>
<tr>
<td>4. A patient with an incapacitating systemic disease that is a constant threat to life</td>
<td>4. Life threatening</td>
</tr>
<tr>
<td>5. A moribund patient who is not expected to survive for 24 h with or without an operation</td>
<td>5.</td>
</tr>
</tbody>
</table>
Correspondence

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trauma or severe acute physiological disturbances such as those induced by sepsis. 

Remove concern regarding additional anaesthesia risk by grading it separately, concentrate on intercurrent chronic health concerns and the ASA physical status classification system becomes reliable and reproducible requiring but one small modification in respect of a class between II (mild systemic disease) and III (severe systemic disease) to represent the moderate systemic disease which so frequently presents. ASA V is already effectively a composite score. 

The GAS is shown in table 1. It can be seen that all but ASA V patients move from a single to a double digit score with significantly improved “risk assessment”. This is of value to quality assurance, in assigning the appropriate grade of anaesthetist and to medical audit, producing the consistency which would thereby allow meaningful comparison between units. Thus the normally healthy patient with a dental abscess producing acute airway obstruction is scored GAS 1.4 and the cases listed by Owens, Fleets and Spitznagel2 and Haynes and Lawler4 can also be easily and conveniently graded. 

Having confirmed the simplicity and ease of use of GAS in a pilot evaluation, we believe that the advantages of the score should now be the subject of a large study on a regional or national basis.

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Sir,—We are well aware of the problem of consistency in the use of the ASA physical status classification by different anaesthetists. Therefore, in this study we attempted to eliminate this inaccuracy. In addition, the ASA system is used by all anaesthetists in our clinic. 

In order to mimic a “real-life” scenario, Haynes and Lawler1 did not provide a definition of the ASA grades to respondents. In this case it would have been interesting to know if the respondents who were regular users of the system. However, Haynes and Lawley and also Plumer and Rottman2 did not provide any information on this point.

In our study, ASA classification was used only in its original meaning to describe physical status, not as an estimation of anaesthetic risk. The perioperative variables representing outcome were mainly surgical or general perioperative complications and were not manifestations of specific anaesthetic risk, for example aspiration, airway obstruction or malignant hyperthermia.

Therefore, the aim of our study was different from the proposed graded anaesthesia score (GAS) which is intended to represent anaesthetic risk. We would be very interested to see if the prognostic value of GAS can be confirmed statistically in a large study.

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Effect of Sprotte needle orientation on spinal anaesthesia

Sir,—I wish to comment on the study of James and colleagues on the effect of orientation of a Sprotte needle on spinal anaesthesia using 0.5% plain bupivacaine. They demonstrated that when the needle orifice was pointing cephalad, median onset time of sensory block to T4, measured by loss of cold sensation, was significantly faster compared with caudad orientation (5 (interquartile range 3–5) min vs 8 (5–9) min, respectively). This is a small but clinically useful difference when rapid onset is required. However, the maximum level of sensory block was similar in all four groups which implies that the distribution of local anaesthetic within the cerebrospinal fluid (CSF) was also similar. This is to be expected as the major determinants affecting distribution of local anaesthetics after subarachnoid administration are the physical characteristics of the local anaesthetic and patient characteristics. The technique of injection is less important although it may play a role.

The baricity of 0.5% plain bupivacaine is slightly hypobaric at body temperature. Plain bupivacaine has been shown to displace CSF 80–115 min after injection. If inverting in a cephalad direction delivered a higher dose of local anaesthetic to the trough of the thoracic kyphosis, the drug should then “float” upwards along the cephalad border of the kyphosis to a higher level if the patient is in the supine position. The fact that this did not occur implies that a similar dose of local anaesthetic was distributed towards the trough. Hirabayashi and colleagues have demonstrated that the lowest point of the thoracic hollow is T7–T9, which may account in part for the variable spread seen in clinical practice.

Two reasons may explain the lack of difference in maximum spread of local anaesthetic drug in this study. An injection speed of 5 ml min⁻¹ through a Whitacre needle produced turbulent mixing in an in vitro model. Also, changes in body position affect the volume of the extradural venousplexus which can consequently displace CSF. It would be easy to imagine the effect in a confined CSF compartment of these two factors resulting in the drug being thoroughly and similarly mixed whatever the initial direction of the needle orifice.

Other studies investigating the effects of orifice orientation of needles with a lateral eye have produced mixed results regardless of the baricity of the drug used. Those studies which demonstrated a higher block with the orifice pointing cephalad did so because patients had no or very little body movement after spinal anaesthesia was performed. In the study of James and colleagues, patients were placed from the right lateral to the left lateral position, and in another study were changed from the sitting to the lateral position. It is difficult to establish if baricity or bulk displacement of CSF is the major factor responsible for the effects caused by changes in body position. Until a truly “isobaric” local anaesthetic mixture is available (tetracaine crystals which can be dissolved in CSF are not available in the UK) positional effects will always have a prominent role over orifice orientation in determining drug distribution within the CSF. While “non-isobaric” agents are favourable in some circumstances there are arguments against their use.

Finally, the angle of emergence of solutions injected through a modern 25-gauge Whitacre needle into a fluid medium is approximately 35° to the longitudinal axis. The angle is approximately 30° with a 24-gauge Sprotte because of the larger orifice (although the size of orifice has recently been reduced). The clinical consequences of this small difference are unknown but are unlikely to be important as a similar distribution pattern of injectate was produced by both of these needles in an in vitro model.

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Sir,—We agree that many factors influence the maximum sensory block height achieved after injection of local anaesthetic into the subarachnoid space. Serpell stated that the physical characteristics of the local anaesthetic agent and patient characteristics are more important in determining maximum sensory block than the technique of injection. Our study showed that the technique of injection affected not maximum sensory block but speed of onset of block.

The patients we described were pregnant women. The main finding of our study was that the technique of injection for subarachnoid anaesthesia affected the onset time of a sensory block suitable for Caesarean section in women at term. We orientated the side eye of the Sprotte needle cephalad, caudad, right and left lateral, and demonstrated that a cephalad injection produced a significantly faster onset of block. This is a clinically important difference when rapid onset of sensory block is required for Caesarean section.

Maximum block heights achieved were similar regardless of needle orientation so while the speed of onset of block was affected by injection technique, final distribution of local anaesthetic in the CSF is influenced by the many other factors the author mentioned. Posture is particularly important in the pregnant woman at term.1 We induced subarachnoid anaesthesia in the right lateral position and then turned the women supine with a 20° left lateral tilt and this would enhance the spread of local anaesthetic in the CSF.

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Cardiac arrest after Caesarean section

Sirs,—Having read the case report of Scull and Cartl1 we wish to bring to your readers’ attention a similar incident which occurred in our institution recently. A 31-yr-old woman was scheduled to undergo an elective lower segment Caesarean section (LSCS) under subarachnoid anaesthesia at 38 weeks’ gestation because of a breech presentation. The woman was 163 cm tall and weighed 75 kg. The patient was premedicated with ranitidine 150 mg and metoclopramide 10 mg, 2 h before operation and 30 ml of sodium citrate 0.3 mol litre−1 before induction. After giving 500 ml of colloid i.v., subarachnoid anaesthesia was performed with the patient in the sitting position using 0.5% hyperbaric bupivacaine 2.4 ml injected via a 24-gauge Sprotte needle at L2–3.

The patient lay down on the operating table which was immediately tilted approximately 25° to the left. Arterial pressure before subarachnoid injection was recorded at 129 mm Hg systolic (Dinamap), and after 5 and 10 min was recorded at 114 and 105 mm Hg systolic, respectively. After the second of these readings, ephedrine 6 mg was given i.v. At 10 min block height was T3 bilaterally.

At 15 min as surgery commenced, the patient suddenly complained of severe nausea and became increasingly agitated, talking incoherently and flailing her arms. Arterial pressure had just been recorded at 96 mm Hg systolic, and another dose of ephedrine 6 mg was given i.v. After this the patient continued to flail her arms about, but had stopped talking.

After a few seconds of arm-waving, she collapsed and very rapidly became cyanosed. Her pupils were not dilated at this or any later time. The ECG monitor showed a bradycardia of 15–20 beat min−1, but arterial pressure was not measured at this time. Oxygen saturation was noted to be 70%.

Cricoid pressure was applied and manual ventilation with 100% oxygen started via a face mask. The following drugs were given in rapid sequence: atropine 0.6 mg, ephedrine 18 mg, thiopentone 100 mg and suxamethonium 100 mg, and the patient’s trachea was intubated with a 8.0-cuffed oral tracheal tube. The pulse oximeter showed a rapid increase in saturation from 70% to 100% after intubation, and this was maintained above 97% after intubation. Anaesthesia was maintained with 1% isoflurane and 50% nitrous oxide in oxygen. Arterial pressure was recorded at 170 mm Hg systolic just after intubation, with a heart rate of 110 beat min−1.

The operation continued uneventfully and 1 h 15 min after the subarachnoid injection the patient’s trachea was safely and uneventfully extubated. The patient could feel a dull ache in the area of the incision, although the block was not formally tested.

The patient was subsequently interviewed at length. She remembers being unable to breathe or communicate this problem but being still able to move her arms, which she did in order to attract attention. She remembers the mask being applied to her face and air being blown through her nose.

While these are unusual features, this case probably also represents a reduction in venous return (because of aorto-caval compression) leading to bradycardia, hypotension and loss of consciousness. Subarachnoid block has increasingly become the anaesthetic of choice for Caesarean section, particularly in those patients in whom intubation is predicted to be difficult. What this and other cases of respiratory difficulty after subarachnoid anaesthesia2–6 should remind us is that there is occasionally a requirement for tracheal intubation and any anaesthetic plan should take this into account. Care should also be taken to avoid all causes of a reduced venous return.

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Dose–response curve for anaesthetics based on the Monod–Wyman–Changeux model

Sirs,—Iwai and his colleagues1 have matched the Monod–Wyman–Changeux (MWC) ligand–receptor model to

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sigmoid dose–response curves for anaesthetics. The model represents the way in which the proportion of receptor sites occupied increases with increasing concentration of ligand (anaesthetic). Therefore, it seems logical to match the model to the response of a single cell to an anaesthetic when that response can be measured, as in the case of a Cl− current. Indeed, it is fascinating to see that, when the authors matched the model to the response of induced Cl− currents in single cells to halothane, they obtained plausible values for the variables of the MWC model.

If depth of anaesthesia can be measured in a single, whole organism, it would also be reasonable to extend the approach to matching the response of that organism to an anaesthetic. Indeed, mathematically, the model can be matched to any sigmoid curve, matching the response of that organism to an anaesthetic. Indeed, it would also be reasonable to extend the approach to plausibility of induced Cl− currents in single patients. They obtained a close match to the reported EC50 (MAC) and EC95 values.

However, I question the logic of this extension from the response of a single cell or a single organism to the response of a population. The doses are of a comparable nature; the responses are different: in individuals, percentage of maximum response; in a population, percentage of individuals in whom a particular response is present. For example, MAC is the concentration at which 50% of patients fail to respond to a noxious stimulus, classically surgical incision.

The relevant distinction between single organisms and populations is as follows. The variation in the concentration required to produce different Cl− currents in a single cell, or different depths of anaesthesia in a single patient, arise from the need for different degrees of occupancy of some receptor (or possibly combination of receptors). On the other hand, the variation in concentration required to anaesthetize different patients is presumably as a result of variation in genetic and, perhaps, nutritional and environmental factors.

To take an extreme case, suppose that the dose–response curve for depth of anaesthesia in a single organism has a slope which can be matched with an MWC curve with plausible values for its variables. Suppose then, that 1000 clones of that organism were produced and reared under identical conditions. Those clones would all reach any given depth of anaesthesia (such as lack of response to a noxious stimulus) at the same concentration. Thus at just less than the critical concentration, none of the organisms would respond; at just greater, all would respond. Therefore, the population dose–response curve (for the proportion of organisms responding) would have infinite slope, and yet the dose–response curve (for depth of anaesthesia) for each individual would still have the original slope.

The results of this study are very interesting and will, I hope, stimulate further work in this area. However, I think that the authors are a long way from demonstrating that the variables of reaction kinetics related to depth of anaesthesia can be deduced from population dose–response curves.

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Sir,—Dr Mapleson questioned the logic of our extension of the treatment based on the Monod–Wyman–Changeux (MWC) model from in vitro cases to in vivo cases in our article. He mentioned that the dose–response of a population is different from that of a cell: if clones were reared under the same conditions, the population dose–response curve would have infinite slope, and yet the dose–response curve for each individual would still have the original slope. Dr Mapleson’s argument is incorrect, provided that some additional integrated effects do not take place in an organism. He also suggested that the variation in the concentration required to anaesthetize different patients is caused by genetic and, perhaps, nutritional and environmental factors. It is plausible. However, I think the way to better understand the mechanism of anaesthesia on the basis of molecular scheme and our article is one of such studies. As mentioned in our article, we assumed that the variation in concentration came from the variation in fractions of active receptor molecules occupied by anaesthetics between individual patients from a molecular viewpoint.

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Failed intubation drill

Sir,—We read the article by Hawthorne and colleagues with interest.

We agree that there may be a cultural explanation for the disproportionate representation of Afro/Caribbean women presenting for Caesarean section under general anaesthesia. It is well known in the West Indies that West Indian women are reluctant to accept extradural analgesia; indeed, there is no extradural service in our hospital. Further, there may be a higher Caesarean section rate among Afro/Caribbean women because of the increased angle of inclination which predisposes to malposition.

On the basis of the above, one may expect to find a higher rate of Caesarean sections under general anaesthesia and by inference more frequent failed intubations.

The St James’ Hospital failed intubation drill it is stated that on deciding that intubation has failed, the patient is turned on her side and placed head down. We disagree with turning the patient on her side and with placing her head down before the return of spontaneous respiration for the following reasons:

(a) the key principle in the management of a failed intubation is oxygenation without aspiration. In order to achieve oxygenation, it may be necessary to ventilate the patient’s lungs with a bag and mask until the action of suxamethonium terminates; (b) ventilation would be easier if the patient were kept in the supine position with the wedge in place. In the head down position, splitting of the diaphragm is aggravated with concomitant reduction of lung compliance thus making lung inflation more difficult; (c) junior anaesthetists are far more experienced at holding a mask and ventilating patients’ lungs in the supine position than in the left lateral position; and (d) Rosen cautions against turning the patient on her side before the return of spontaneous respiration because this is often impractical and it may be difficult to ventilate the lungs in the lateral position. Afro/Caribbean women at term are quite heavy (average weight in our hospital is 85 kg). It may be difficult to turn her on her side and manage the airway at the same time, and it is quite possible that in the turning process the patient may fall off the table.

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