Hydroxyethyl starch and Gelofusine on pulmonary function in patients undergoing abdominal aortic aneurysm surgery

Editor—We read with interest the study by Rittoo and colleagues. The authors compared the effects of hydroxyethyl starch (HES) and Gelofusine on pulmonary function in aortic aneurysm surgery.

Microalbuminuria was one of the variables used in this study to measure the degree of vascular permeability. Microalbuminuria is non-specific and is known to occur after anaesthesia and surgery. Did microalbuminuria in these patients correspond to other measures of lung injury? These data are not reported.

Plasma elastase is another variable used to assess lung injury. Although it is specific for neutrophil activity, it can increase after red blood cell and cell saver blood transfusion. In this study, the patients received both red blood cell transfusion and cell saver blood, but the quantities administered in each group are not reported. Hence, it is unclear whether one can attribute the increase in neutrophil elastase to lung injury alone.

The authors have used the chest x-ray and \( P_O_2/F_I_O_2 \) to assess the presence of pulmonary oedema. A history of cardiac disease is present in >70% of patients presenting for aortic aneurysm surgery. The incidence of myocardial infarction and heart failure in the postoperative period in the same group of patients is 3.3%. We did not find any attempt by the investigators to rule out cardiac causes of pulmonary oedema.

Resuscitation using colloids has not been shown to be superior to crystalloids. In many practices (including our own), crystalloid solutions are the main resuscitation fluid. Given that colloid molecules do eventually cross even normal vascular epithelium, entering the interstitium where they continue to exert an oncotic effect, we question the long-term benefit of use of colloid resuscitation in these cases. We look forward to studies including a crystalloid resuscitation arm in the experimental design.

A. G. Djalali
V. Srinivasa
N. Sadovnikoff
Boston, MA, USA

Editor—We read with interest the article by Rittoo and colleagues. One of the variables used in this study was based on a similar study in trauma patients. However, their paper is rendered less convincing by several errors that are evident when reading the paper. The power calculation for this study was based on a similar study in trauma patients. As stated in our paper, we agree that there may be more than one explanation for the lower elastase activities in the HES group. Djalali and colleagues raised an interesting question: is HES cardioprotective and the improved lung function a secondary phenomenon? Unfortunately, this was not one of the aims of the study, which would be underpowered to throw light on this hypothesis.

There is now extensive evidence that HES volume expansion has advantages over albumin, gelatin or crystalloid resuscitation. Space will not allow a full reference list, but we cite a recent comparison of crystalloid vs HES in elderly surgical patients demonstrating the anti-inflammatory effects of HES. It was considered unethical in our hospital to use crystalloids only as fluid resuscitation in patients undergoing aortic aneurysm surgery because of the now well-recognized advantages of HES over crystalloid-only volume therapy.

We thank van Hoogstraten and Jagger for their interest in our paper. Microalbuminuria was one of many variables used in this study. In previous studies, we were unable to show that anaesthesia alone caused microalbuminuria, but only in association with surgery. Although we have shown a strong association between microalbuminuria and the \( P_O_2/F_I_O_2 \) ratio following trauma, the aim of the present study was to compare the different colloids and not investigate further this relationship in patients undergoing aortic aneurysm surgery. Plasma elastase activity had previously been linked to lung injury. Plasma elastase can increase after blood transfusion, and the volume of red cell transfused was not significantly different in the two groups of patients studied (960 ml (95% CI 800–1280 ml) vs 960 ml (95% CI 770–1280 ml) for HES and Gelofusine group, respectively). As stated in our paper, we agree that there may be more than one explanation for the lower elastase activities in the HES group.

Because donor blood transfusion may be an independent cause of acute lung injury, we believe this to be an important omission. With respect to the scoring systems used, we would like some more information, especially as most of the data appear to have been transformed into another set of numerical values before being statistically evaluated. The authors comment that the calculation of the lung injury score was computed by the number of components used (the maximum of which was four), but no mention is made of how many patients were scored on fewer than four components, or how many components each patient was scored on. This appears to us to be important as it might statistically skew the data, invalidating the findings. We are also confused by the method of allocating the postoperative hypoxaemia score. Is there any significance to the ratio of 23.3? Why is this number used instead of 20.0 as the cut-off for severity?

We found this to be potentially interesting data, and it is a pity that it has been obscured by a lack of some important information.

R. van Hoogstraten
S. Jagger
London, UK

Editor—We read with interest the study by Rittoo and colleagues. Plasma elastase activity had previously been linked to lung injury. Plasma elastase can increase after blood transfusion, and the volume of red cell transfused was not significantly different in the two groups of patients studied (960 ml (95% CI 800–1280 ml) vs 960 ml (95% CI 770–1280 ml) for HES and Gelofusine group, respectively). As stated in our paper, we agree that there may be more than one explanation for the lower elastase activities in the HES group. Djalali and colleagues raised an interesting question: is HES cardioprotective and the improved lung function a secondary phenomenon? Unfortunately, this was not one of the aims of the study, which would be underpowered to throw light on this hypothesis.
transfusion of large volumes of blood may cause acute lung injury, the volumes of blood transfused were comparable in both groups and we felt that this was not an important issue in this study. However, we accept that omission of these data may have obfuscated readers.

The lung injury score (LIS) was computed by dividing the aggregate score by the number of components used, the maximum of which was four. In seven patients in both groups of patients, only three components were used to compute the LIS. In the remaining patients all four components were used. The postoperative hypoxaemia score is based on published criteria.

D. Rittoo
P. Gosling
R. Vohra
Birmingham, UK


PFA-100® and regional analgesia

Editor—I was interested to read the correspondence from McGlennan and colleagues. The specific message conveyed to the readers was unclear. Should we use the platelet function analyser (PFA-100®) as the decision making tool before instituting an epidural?

It is well known that treatment with non-steroidal anti-infl ammatory drugs (NSAIDs) is associated with platelet dysfunction, resulting in increased bleeding time and closure time (CT). Many short-acting NSAIDs (e.g. ibuprofen) lose their antiplatelet effects within a few hours. The patient described by McGlennan and colleagues had taken an overdose of ibuprofen 4 days before and was successfully treated and discharged home. Considering the reversible effects of ibuprofen on platelet function and its short half-life, it was unlikely that the prolonged CT recorded by the authors resulted from an ibuprofen overdose. This test was carried out 4 days after the overdose.

The authors did not describe the method of measurement of CT in sufficient detail. I found no logic in declining to insert an epidural on the basis of prolonged CT (which was based on a single measurement) alone.

It is well known that CT has limitations. Despite having 98% negative and 94% positive predictive values, this test is not 100% sensitive to all platelet function defects. False-negative and false-positive results are seen. The PFA-100® is also sensitive to a large number of variables. CT results vary if blood is sampled in the morning or in the afternoon. The CT of blood samples collected in the evening are significantly longer. Haematocrit can affect the CT measurement as well. Two cartridges are used for CT measurements; they are CT/collagen–epinephrine (EPI-CT) and CT/collagen–adenosine diphosphate (ADP-CT). These two cartridges have different sensitivity and specificity for different clinical situations. It is also necessary to duplicate the measurement. Duplicate analysis is associated with a mean coeffi cient of variation of 5.7% (ADP-CT) or 7.1% (EPI-CT). For complete evaluation of platelet function, it is advisable to perform a whole blood electrical aggregometry test, which is the gold standard for detecting platelet function defects or nucleotide release assays.

The authors1 stated that the normal limits of CT are hospital specific. It would have been useful to publish the upper limit of normal CT in the pregnant population in their hospital. It may not be appropriate to compare this patient’s CT with a previously reported CT reference, which was not from their hospital. Thus, the significance of this comparison remained doubtful, especially when the patient did not experience any abnormal bleeding.

Currently, no evidence exists to suggest that regional anaesthesia techniques should be avoided in patients taking aspirin or other NSAIDs in therapeutic doses, even though these drugs can prolong the CT by as much as 63%. Many patients taking these medications may have a CT as high as 300 s, depending on the type of NSAIDs used. There is no evidence that the PFA can estimate the risk of haemorrhage in patients on platelet inhibitors. Neither platelet aggregation nor flow cytometry have been shown to have predictive value with regard to a haemorrhagic risk of antiplatelet agents. Nor are there any reported cases of spinal haematoma attributed to the use of aspirin or other NSAIDs. These drugs have been used for several years in a large number of surgical patients who have undergone epidural/spinal analgesia.

N. G. Mandal
Peterborough, UK

Editor—we thank you for the opportunity to reply to Dr Mandal. We are pleased that we have succeeded in our aim—to stimulate debate in this area.