Factors in epidural haematoma

Editor—Published case reports should serve to either raise awareness of a particular condition and or indicate how similar problems might be dealt with in the future. In the case of an epidural haematoma, as reported by Tam and colleagues, these would come together with the aim of showing how this complication, or its consequences, might be avoided. However, this report raises as many questions as it answers.

First, the summary states that ‘standard guidelines’ were adhered to, but within the report it is revealed that the patient received an inadvertent dose of clopidogrel on the first postoperative day, and while she was also receiving dalteparin. This is certainly not according to guidelines and would be a very major aetiopathological factor. The patient was known to have a degree of chronic renal failure and to have been of fairly small size, yet a standard dose of dalteparin was administered. Again, this does not represent adherence to ‘standard guidelines’.

There is no specific mention of her preoperative analgesia, but it would be very unusual for a patient requiring knee arthroplasty to not require significant analgesia. In many cases this would be an NSAID, which would be another aetiopathological factor, but no information is given.

Finally, what is the risk/benefit balance of using a combined spinal–epidural anaesthetic technique, involving significant instrumentation of the vertebral canal, in a patient undergoing a primary knee replacement? Is not 72 h of continuous epidural analgesia more medicine than the condition warrants? If nothing else, it obscured the development of the early features of the haematoma although there seem to have been significant delays from other causes thereafter.

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Editor—We would like to thank Professor Wildsmith and colleagues for their interesting comments regarding our case report. The intention of publishing our case report was to have been significant delays from other causes thereafter. Whilst the patient received an inadvertent dose of clopidogrel 75 mg the morning after surgery, retrospective analysis of the case notes revealed that the haematoma occurred at the time of insertion of the CSE. Furthermore, previous studies have shown that when clopidogrel is administered at a loading dose of 375–400 mg, maximal inhibition of platelet function occurs at 2–6 h but with clopidogrel 75 mg once daily this level of inhibition is only achieved after 3–7 days of repeat dosing.

The patient was not taking anti-inflammatory analgesics because she had a previous history of haemorrhagic gastritis following ingestion of low dose aspirin.

The case we reported highlighted the significant risk factors that can result in epidural haematoma following neuraxial blockade. Certain factors increase the likelihood of developing an epidural haematoma including patient factors such as age, female gender and bony spinal pathology. These factors should be taken into account when considering a regional anaesthetic technique, particularly in the presence of anticoagulants.

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References

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Propofol and memory

Editor—We were unsure from Dr Veselis’ editorial whether he approves of the possibility of patients being awake during general anaesthesia. Phrases such as ‘It will be a brave new world when we can tell a patient...’ ‘don’t worry—you won’t remember this’ with confidence...’; ‘being aware but having no memory... is not a traumatic event... allows ethical research to be done...’; and ‘...the question is
whether we want a monitor to detect unconsciousness or one that detects amnesia? suggest an openness to discussion at least.

It suits anaesthetists to tell patients that awareness is a rare complication, occurring in 1–2 per thousand. However, this figure relates to awareness with recall. We thought that it might be less reassuring to tell them that they have a 16% chance of being awake during surgery, or even ‘you are sure to be awake for some of the time during surgery’.

We asked 60 anaesthetists in our department three questions:

(i) ‘Would it be acceptable if, during your operation carried out under general anaesthesia, you were awake for a period of time, even though you did not remember afterwards?’

(ii) ‘Would it be acceptable if, during your operation carried out under general anaesthesia when you were completely paralysed, you were awake for a period of time, even though you did not remember afterwards?’

(iii) ‘Would it be acceptable if, during your operation carried out under general anaesthesia when you were completely paralysed, you were awake for a period of time and in pain, even though you did not remember afterwards?’

The answers were ‘no’ in 45 (75%), 56 (93%) and 58 (97%) cases of responses. Three anaesthetists who answered yes to question (i) qualified this, commenting that being awake would be acceptable in planned circumstances or with prior consent.

A significant minority of our anaesthetists would be prepared to be awake if not paralysed and not in pain. However, there is no way to guarantee that the surgeon will not inflict pain at some future time during the operation, and this may be more likely if a light level of anaesthesia is aimed for using a ‘depth of anaesthesia’ monitor. Being awake and anxious during surgery may also lead to long-term psychological sequelae.

We think that if most anaesthetists wish to be unconscious rather than amnesic during general anaesthesia, it will be a long time before it is possible to convince the public that this is acceptable or desirable.

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Editor—I am pleased that my editorial was provocative enough to undertake this interesting survey. The results indicate that these anaesthetists, as probably most do, rely on the clinical metric that has served the profession well from the first use of nitrous oxide and ether. Namely, that if a person is unconscious, they will also be amnesic even for painful stimuli. The blissful sleep of oblivion has served us well, and may be the root of the perception that amnesia without awareness is superior to amnesia for events that we are aware of at the time. As will be recalled, the first media event in the use of nitrous oxide was of a screaming patient in front of a ridiculing audience. This untoward response to anaesthesia rests heavily on our collective consciousness, and is reinforced with every new case of awareness. How can one best assuage the visceral fear of failure?

In large part, the solution to awareness is in understanding the processes of complex systems and making these reliable, as awareness is almost certainly due to the administration of too little anaesthesia, frequently as a result of human error or technical malfunction. Beyond that, I propose that if we understood how anaesthesia, or more accurately, each component of the anaesthetic state works, then we would be less terrified of its failure. Although all of us are excellent empiric purveyors of magic potions, our trade would be more secure with this knowledge in hand. As it is now, our best option is to develop reliable monitors of each anaesthetic component. Such endeavours, of course, will be easier when we understand which brain activities relate to anaesthetic actions on consciousness, memory or pain and how these might be best monitored. This state of affairs is currently exemplified by the use of neuromuscular blocking agents with neuromuscular blockade monitors, where both the physiology and monitoring are largely understood.

Short of this, it is always safer and more reassuring to err on the side of over-medication. This approach is increasingly safe as new medications with fewer side-effects and more error resistant pharmacological profiles are developed. Interestingly, these very considerations, serving as the basis for learned and heated controversies about which agent is superior, started as soon as nitrous oxide and ether were first used.

Such heuristics work well, as is attested to by their endurance from the very beginnings of anaesthesia practice. However, let us not be lulled into a sense of comfort where further knowledge of how anaesthesia works in the brain is no longer pursued just because what we do, we do well enough. The methods of practice that I envision, which cause such psychological discomfort today, will only be possible and acceptable when such knowledge is attained.

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References
Useful ectopies

Editor—The use of trans-oesophageal Doppler cardiac output monitoring (Cardio Q, Deltex Medical, Chichester, UK) for perioperative fluid optimization is well documented. Increasingly the benefit of goal-directed perioperative intravascular filling on postoperative morbidity is being realized.1–3

A simple algorithm using boluses of colloid i.v. fluid based on the Starling principle has been followed and has led to more rapid recovery after major surgery, a reduction in postoperative length of hospital stay and 5.3:1 reduction in the incidence of postoperative gut complications compared with central venous pressure targeted controls.1

The Deltex Cardio Q displays a velocity–time curve for every heartbeat. This gives the user the ability to see the data displayed on which the machine has calculated stroke volume and cardiac output. In following the simple stroke algorithm (Fig. 1), occasionally increases in systemic vascular resistance can affect the stroke volume making it slightly more difficult to interpret the filling requirements. However, if a premature atrial ectopic beat occurs, followed by a compensatory pause, this can provide useful additional information. In Figure 2 for example, the premature beat is smaller than the ‘normal’ beat because of reduced filling time. The beat following the compensatory pause has had increased diastolic filling time. If this later beat is larger (greater area under the curve) than the ‘normal’ beats, it shows there is unused filling capacity within the heart and more colloid can be given safely. However, if this beat is of similar size or indeed smaller than the normal beat, then it is likely that the heart is operating at the top of the Starling curve already—consequently further filling would not be advised. This phenomenon can therefore be used to support the use of the oesophageal Doppler fluid algorithm.

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References


Measure CVP
and stroke volume

250 ml of
colloid i.v. over
2 min

Fall in
stroke volume

YES

NO

NO

YES

Measure CVP
and stroke volume
every 10 min

CVP rise < 3 mm Hg
and increase in
stroke volume

1

Fig 1 Oesophageal Doppler fluid algorithm.1

Fig 2 CardioQ screenshot showing the ‘useful ectopic’ phenomenon.