Should a patient be dissatisfied with your management of his or her case, he or she currently has the options of going elsewhere, complaining to the HPCSA or, alternatively, suing you. One would have thought that that was protection enough but, apparently, this is not so; a further arrow is to be added to the patient’s quiver in the form of the Consumer Protection Act. In fairness, the Act was not introduced to specifically protect patients but, given its broad scope and the fact that healthcare has not been excluded, patients may seek to use the Act against you.

So, what is the Consumer Protection Act, when will it be implemented, and how may it affect you? The Act was signed by the president and published in the Government Gazette in April 2009, and the first part of the Act came into effect on 24 April 2010. This first part primarily provides for the establishment of new consumer protection bodies and the formulation of regulations. The second tranche of provisions, which includes the bulk of consumer rights, comes into force on 24 October 2010, unless a decision is made to defer them.

Provisions from at least five previous acts have been simplified and centralised in the Consumer Protection Act. Given that the Act now entrenches fundamental consumer rights, it has been characterised a “bill of rights for consumers”, aimed primarily at protecting the vulnerable.

An area that may affect anaesthetists directly is discussion regarding fees. The HPCSA expects doctors to make patients aware of the costs that they will incur, and the National Health Act mandates it, but the Consumer Protection Act gives patients the advantage. If it cannot be shown that you have discussed fees appropriately, you may be vulnerable to reimbursing the patient some of the fee paid.

One of the major changes the Act brings about is the introduction of strict liability for goods supplied. The claimant no longer has to prove that he or she suffered harm and subsequent economic loss as a result of somebody’s negligence. He or she merely has to prove that harm was suffered as a result of defective goods. This is a significant change that is to the claimant’s clear advantage.

You could ask: “But, what has this to do with me, the anesthetist?” Unfortunately, a great deal. The Act makes all parties involved, from manufacturer to supplier, jointly and severally liable for the harm and loss resulting from defective goods. In lay terms, the consumer can sue anybody in the supply chain and hold that person liable for all the harm caused and costs incurred. It falls to the defendant to involve the other parties.

Let’s put this in perspective. You give a patient a spinal block but, due to no negligence on your part, the needle tip breaks off and requires surgical removal and results in unpleasant long-term sequelae for the patient. Given there was no negligence on your part prior to the introduction of the Act, the patient could only really sue the manufacturer and would have to prove that the failure of the prosthesis was as a result of negligence. It is a very difficult task to sue a manufacturer, probably located overseas, and highly complex and technical arguments would have to be advanced to ensure success. After the Act’s introduction, things become far easier for the disgruntled patient. All that now has to be shown is that the needle failed inappropriately, and that the patient suffered harm and loss as a result of that failure. The patient does not have to sue a faceless manufacturer overseas, but can hold anybody in the supply chain totally liable. In the vast majority of cases, there is only one person in that supply chain that the patient can easily identify: you!

The claimant does not have to show that you were negligent in any way and, indeed, you cannot claim that you were not negligent and, thus, not liable. If the patient can show that the prosthesis was defective and you were part of the supply chain, then you can be held fully liable for the cost of the damages that follow. Unfortunately, defective
Alternative anaesthetic management of an infant with Wolf-Hirschhorn syndrome during repair of an atrial septal defect using total intravenous anaesthesia

To the Editor: Wolf-Hirschhorn syndrome (WHS) is a rare chromosomal disorder caused by a deletion of the short arm of chromosome 4 with an incidence of 1:50,000 births and a 2:1 female preponderance. Characteristic features of WHS are craniofacial dysmorphisms such as microcephaly, micrognathia, prominent glabella (“Greek helmet facies”), dysplastic ears, preauricular tags, hypertelorism, broad and/or beaked nose, short philtrum, cleft palate, downturned corners of the mouth and dental anomalies.1–3 Further clinical features are low birth weight and developmental delay, mental and motoric retardation, muscular hypotonia, congenital heart defects, feeding, genital and renal anomalies, and seizures. However, many children with this disease require general anaesthesia for diagnostic or therapeutic procedures such as magnetic resonance imaging (MRI), anti-reflux surgery, dental surgery, repair of a cleft palate or urological surgery.

Examination showed a girl in good general condition, with the dysmorphic features of hypermetropia, lagopthalmus and micrognathia. She was suffering from psychomotor retardation, muscular hypotonia, gastrointestinal reflux and seizures since the ninth month of age. Because of nutritional difficulties, a percutaneous gastrostomy was in place. A valproate-induced von Willebrand syndrome was diagnosed before.

On the day of operation, anticonvulsive and proton-pump-inhibitor therapy was continued to maintain sufficient drug levels and medication effects. Premedication with 0.1 mg/kg midazolam intravenously allowed transfer of the then sleeping infant to the prewarmed operation room. Standard monitoring was applied (Siemens SC 9000 XL, Siemens AG, Erlangen, Germany).

Routine monitoring included electrocardiography (ECG), pulse oximetry (SpO₂), and non-invasive blood pressure (Siemens SC 9000 XL, Siemens AG, Erlangen, Germany).

During anaesthesia induction, we had to deal with...
the typical features of the disease. Micrognathia was not pronounced, and we did not assume a difficult airway. Due to gastroesophageal reflux with a high risk of aspiration, modified rapid sequence induction seemed to be the correct treatment. With the upper part of the body in a 30° upright position and after three minutes of preoxygenation, a bolus of fentanyl (2.5 µg/kg) was administered. After an additional two minutes, 4 mg/kg propofol was administered. Subsequently, 0.2 mg/kg vecuronium was injected intravenously.4 Seventy-five seconds later, the patient was intubated endotracheally (4.5 mm ID, Vygon, Encouen, France). Anaesthesia was maintained as a total intravenous anaesthesia (TIVA) with repeated bolus administration of fentanyl and continuous infusion of propofol (8–12 mg/kg/h) adapted to surgical stimuli and haemodynamic response. Pressure-controlled ventilation (40% oxygen in air) was adjusted to normocapnia (PetCO₂ 34–38 mmHg). A central venous line was inserted via the inner jugular vein and an arterial catheter was placed into the femoral artery. Temperature was measured by a bladder thermistor and maintained above 36.0 °C using a warming blanket system (Bairhugger, Arizant, Eden Prairie, USA).

Within a total of 148 min, surgery was completed and remained free of complications. The child was transferred to the paediatric intensive care unit, where she was extubated four hours later, showing sufficient pain control and stable respiratory function. The further postoperative course was uneventful, and the neurological state was unchanged.

Ginsburg and Chen et al reported two cases of malignant hyperthermia in children with WHS, admitted for repair of a cleft palate using volatile anaesthetics.4,5 However, Iacobucci et al and Mohiuddin et al described uneventful anaesthesia in three patients undergoing surgery for open reduction of the left hip, bilateral myringotomy tubes and circumcision respectively.2,3 In most of the cases reported, volatile anaesthetic induction and maintenance was reported.1–4 Nonetheless, a general susceptibility to malignant hyperthermia in WHS patients is not likely.1 However, the application of TIVA avoids a residual risk to trigger malignant hyperthermia and might have additional advantages. The antiemetic properties of propofol and the absence of an epileptogenic potential compared to sevoflurane anaesthesia1 influenced our decision to use TIVA.

We would like to conclude that a TIVA using propofol and fentanyl, preceded by rapid sequence induction with propofol and high-dose vecuronium, could be a safe alternative in the anaesthesiological management of children with Wolf-Hirschhorn syndrome.  

References


ERRATUM

In the previous issue, in the guest editorial, Prof Mike James’s affiliation was printed incorrectly (Coetzee JF, James MF. Anaesthetic gas analysers: potential for confusion and errors if you live and work at moderate altitude. S Afr J Anaesthesiol Analg 2010;16(4):6–8). He is Professor and Head, Department of Anaesthesiology, Faculty of Health Sciences, University of Cape Town.

We apologise for any inconvenience caused by the error.