Procedural sedation and analgesia for out-of-hospital breast surgery: an overview of the procedural sedation and analgesia technique

Louw AJ, MBChB, DABSA, MMed, FCA, Sedation Diploma
Private Practising Specialist Anaesthetist, Bloemfontein
Correspondence to: Cobus Louw, e-mail: ajlouw@vodamail.co.za

Keywords: breast surgery, procedural sedation, analgesia, sedation, out of hospital, PSA technique

Abstract
Out-of-hospital surgical procedures are a rapidly growing market. This has led to surgical procedures that were previously limited to the hospital operating room, with general anaesthesia being performed outside the hospital with sedation and regional anaesthesia. Breast surgery, whether cosmetic, reconstructive or diagnostic, also follows this trend. The aim of this refresher course is to give an overview of the nerve supply to the breast, to explain the type of blocks used and to provide an overview of the sedation technique. The audience should have a better acceptance of performing procedural sedation and analgesia (PSA) for this type of surgery out of hospital after this overview. The author will also provide an overview during the presentation of his own current practice of performing these procedures.

Introduction
Out-of-hospital surgical procedures are a rapidly growing market worldwide, especially in the USA.1 Breast augmentation and breast lifts are the most commonly performed cosmetic surgeries in the USA.2 A large number of these procedures are now being performed outside the operating room under procedural sedation and analgesia (PSA) and regional anaesthesia in South Africa. Specialised anaesthetists are still reluctant to perform sedation out of hospital due to their belief that it is not safe practice.1 Currently, considerable retrospective data support the safe practice of out-of-hospital PSA.3

Surgery type
Generally, out-of-hospital breast surgery is performed by plastic and reconstructive surgeons in the office or dedicated sedation units/ambulatory units. This ranges from breast augmentation, breast lifts, breast reductions, nipple reconstructions and breast reconstruction with tissue expanders or permanent breast implants. Most sedations are performed by the surgeon or a sedation nurse. More anaesthetists are becoming involved with PSA as surgeons appreciate the benefit of having to concentrate on the surgery only, and leaving the sedation technique and monitoring to more specialised care. This has the added benefit of the anaesthetist or sedationist being able to perform more specialised nerve blocks. Usually, local infiltrative anaesthesia is combined with light to deep sedation, but this is changing as specialist anaesthetists now perform these with paravertebral, intercostal or pectoral nerve blocks (PECS), combined with sedation and aided by ultrasound imaging.

Nerve innervation of the breast and related tissue
Breast surgery involves three anatomical structures, namely the breast tissue (with overlying skin, nipple and areola), the anterior thoracic wall muscles and the axilla.

The breast tissue and overlying skin and nipple are innervated by perforators from the anterior and lateral cutaneous branch of the intercostal nerve (T2-T5).4,5 The intercostal nerve follows the subcostal groove, together with the intercostal vein and artery, ordered from superior to inferior; vein, artery, nerve (VAN), and is bordered by the parietal pleura and intercostal intimus muscle innermost, and the internal and external intercostal muscle outermost. The lateral cutaneous branches pierce the intercostal muscles in the midaxillary line to form an anterior and posterior
branch. The anterior branches supply the lateral two thirds of the anterior thoracic wall. The anterior cutaneous branch pierces the intercostal muscles just lateral to the sternal border, and supplies the medial third of the anterior thoracic wall.

Branches of the cervical plexus, the lateral and medial pectoral nerve innervate the overlying muscles of the anterior thoracic wall (pectoralis major and minor). The pectoral medial nerve (C8-T1) has its origin in the medial cord of the brachial plexus, and pierces the pectoralis minor to enter the pectoralis major, and supplies the pectoralis minor and part of the pectoralis major. The lateral pectoral nerve (C5-C7) runs between the pectoralis minor and major, and supplies the remainder of the pectoralis major. These are both very important nerves when placing subpectoral prosthesis.

The costobrachial nerve supplies the axilla which is an extension of the lateral cutaneous branch of the second intercostal nerve.

The long thoracic nerve (C5-C7) that supplies the seratus anterior muscle is another affected nerve and is more relevant for reconstructions with latissimus dorsi flaps.

Regional anaesthetic technique

Local tumescent infiltration

Most surgeons perform a form of infiltration with a solution of local anaesthetic that is very similar to their liposuction infiltration solution. This consists of normal saline, lignocaine and adrenaline, with or without a longer-acting local anaesthetic. The sedationist should calculate the maximum allowed dosage for a particular patient, and keep track of how much is used by the surgeon. It is handy to have this solution on hand, even if blocks are being performed. The surgeon can inject this into areas where blocks may have failed.

Paravertebral block

The intercostal nerve can be blocked at its origin in the paravertebral space. Only one level needs to be blocked, with the resultant spread of the local anaesthetic up and down the space. The thoracic paravertebral space of level T4 or T5 is identified three centimetres lateral to the posterior vertebral process by means of the loss-of-resistance technique to saline or air, with an epidural Touhy needle or with a nerve stimulation needle. A “pop” is usually felt as the needle pierces the costotransverse ligament. The needle is “walked off” either superior or inferior to the transverse process, and advanced no further than one centimetre beyond the contact depth of the transverse process. “Walking off” inferior is considered to be safer as the distance from the process to pleura is further than “walking off” superior. A bolus of 10-ml local anaesthetic solution is injected. Ultrasound-guided paravertebral block is becoming the standard, but a steep learning curve is involved. Complications include pneumothorax, epidural spread, contralateral spread, hypotension (especially with bilateral blocks), and local anaesthetic toxicity.

Intercostal blocks

The intercostal nerve must be blocked proximally before the lateral cutaneous branch pierces the intercostal muscles in the midaxillary line. The author blocks patients in the supine position, with arms 90 degrees abducted on arm boards in the midaxillary line. In the author’s opinion, ultrasound guidance is the safest way to block these nerves. At the very least, a nerve stimulator should be used. Advancing a stimulating needle towards the inferior border of the rib will result in direct local muscle stimulation, and after advancing a millimetre or two further, nerve stimulation will cause muscle stimulation of the intercostal muscles more distally as well. A “pop” is usually felt as the needle advances through the internal intercostal membrane. With ultrasound-guided block, a linear probe is placed in the midaxillary to posterior axillary line. Two ribs are identified. The parietal pleura serves as a hyperechogenic line which connects the posterior aspects of the ribs. The needle is advanced in plane to the probe, towards the inferior rib border, to just above the parietal pleura. Three millilitres of local anaesthetic is bolused. The spread of the local anaesthetic pushing the pleura down can be visualised. No spread indicates intrapleural or intravenous injection. At least T3-T5 should be blocked.

Pectoral nerve block

A traditional PECS block aims to block the lateral and medial pectoral nerves in the plane between the pectoral major and minor muscle. This is needed for subpectoral breast expanders or prostheses. A modified PECS block intends to block these same nerves, but also the intercostobrachial nerve, long thoracic nerve and at least two intercostal nerves in the plane between the pectoral minor and seratus anterior muscle. This is needed for axillary clearance. This misses the anterior branch of the intercostal nerve which supplies the medial third of the anterior chest wall as the lateral cutaneous branches are blocked outside the intercostal groove. Supplementation by subcutaneous infiltration needs to be carried out parasternally if needed.

Combining a PECS block with an intercostal or paravertebral block significantly improves analgesia for breast reductions, breast lifts and breast augmentations.

Complications of regional anaesthesia

Pneumothorax

Pneumothorax is one of the most dreaded complications of paravertebral and intercostal blocks. It is for this reason that
the author does not recommend multiple bilateral intercostal blocks without the use of ultrasound. Pneumothoraces from paravertebral or intercostal blocks are rare, and are mostly caused by the surgeon dissecting into the thorax during reconstruction after radiation therapy, or when augmenting underweight patients. Visualising the entire needle and seeing the spread of the local anaesthetic is the only way to ensure that this is prevented when performing the blocks. Ultrasound provides the benefit of quickly diagnosing a pneumothorax by means of four ultrasound signs. It is 92% sensitive and as sensitive as computed tomography without the radiation risk, as well as being fast and more readily available. Ultrasound is also more sensitive, faster and accessible than a chest X-ray. Ultrasound signs of pneumothorax include the absence of lung sliding and B-lines, and loss of the “seashore sign” in M-mode and the formation of lung point. This will be demonstrated during the presentation.

Local anaesthesia toxicity (LAST)
Toxicity is usually due to unintentional intravenous injection, with the development of symptoms within a minute of the injection, or due to rapid absorption and high peak plasma levels. Toxicity usually develops within 8-12 minutes with intrapleural, intercostal and paravertebral blocks. The absorption of local anaesthesia from the intercostal and paravertebral space is faster than it is from any other area. This leads to very rapid (8-12 minutes) and high-peak plasma concentrations which are potentially toxic. Even with low concentrations of local anaesthetic and by remaining within the toxic dosage limits, patients are still exposed to high plasma levels, but without any signs or symptoms of toxicity. Peak concentrations tend to stay high for up to 40 minutes after the initial peak.

Best practice is to stay within the maximum dosage for the patient’s weight, to use the lowest effective concentration of local anaesthetic and to limit the volume injected. Ropivacaine and levobupivacaine have less cardiotoxicity than racemic mixture bupivacaine. Cardiotoxicity develops at almost twice the plasma levels than that of central nervous toxicity. Using a vasoconstrictor slows absorption and decreases peak levels. Factors which increase susceptibility to toxicity should be kept in mind, such as low body mass, renal insufficiency and antiarrhythmic drug therapy. Most case reports of cardiac arrest with local anaesthetic have occurred in patients with renal insufficiency.

Also, signs of toxicity are not obvious in a sedated patient, and the patient might become irritated or confused before convulsing. The author keeps sedation light and remains in verbal contact with the patient while performing blocks, only increasing sedation once the patient has been surgically drapped.

When toxicity is suspected, it is important to treat the patient with intralipid 20% immediately. This must be readily available to the sedationist. A bolus of 1.5 ml/kg should be infused over 10 minutes as soon as the signs of toxicity become apparent. Convulsions can be treated with intravenous midazolam, and the airway managed to prevent hypoxia, hypercarbia and acidosis, which worsen the outcome. Cardiac arrest needs aggressive and effective cardiac compressions and vasopressors. (The reader is referred to www.lipidrescue.org for details of intralipid infusions).

The sedation technique
Sedation techniques differ vastly from institution to institution, as well as among sedationists. Modern sedations consist of the use of benzodiazepine, opiate and propofol, with or without ketamine. Patient satisfaction depends on the extent of amnesia and discomfort during the procedure, as well as the extent of postoperative analgesia and the absence of nausea and vomiting. This needs to be taken into account when choosing a sedation technique. Drugs with a short half-life must be chosen, and different drugs for specific indications used at lower dosages than when using them alone.

Published data for the use of sedation drugs range from diazepam or midazolam (0.02-mg/kg boluses) combined with alfentanil (1-µg/kg boluses), fentanyl or remifentanil and propofol (bolus 0.2-0.3 mg/kg, then 1-4 mg/kg/hour infused, or a target-controlled infusion of 0.8-1.5 µg/ml effect-site concentration) with or without ketamine. Dexmedetomidine has also been studied and is also useful.

It is the author’s view that the use of remifentanil must be weighed against the small therapeutic window of this drug. The author discourages the use of remifentanil boluses and strongly advises not to exceed the ceiling of 0.05 µg/kg/min continuous infusion. This not an ideal drug for out-of-hospital use owing to the incidence of apnoea, nausea and vomiting.

Low dosages of ketamine are very effective as an analgesic and amnestic, but double the incidence of nausea and vomiting, as well as increasing discharge time. Ketamine also stimulates respiratory drive so that hypventilation and airway obstruction are considerably less when ketamine is combined with drugs. Small boluses of 5-10 mg are very effective.

Ketofol solutions are now widely used. The author uses a 1:4 ratio of ketamine to propofol, and prefers giving this as a target-controlled infusion with a Schnider model of 10 mg/ml at 1-1.5 µg/ml effect-site concentration. This is obtained by mixing 40 ml 1% propofol with 10 ml of 1% ketamine, i.e. a ratio of 2.8 mg/ml ketamine to propofol. Too high a concentration of ketamine increases postoperative...
nausea and vomiting, dissociative dreams and discharge time.\textsuperscript{14}

A propofol target-controlled infusion is superior to the use of boluses and continuous infusion. This is titrated at 0.8-1.5 µg/ml effect-site concentration. A low concentration should be commenced and gradually increased until the desired level of sedation is achieved. A simple sedation scale should be used and the aim should be a level 2-3/5 Wilson scale. This level must be documented.

**Conclusion**

PSA for out-of-hospital breast surgery is feasible, but careful attention to detail is needed. Well performed blocks make a positive difference and ensure that the sedation technique can be performed at not too a deep level. Ultrasound has made previously potentially difficult blocks much easier and safer, but it is still dependent on the skill level of the performer. PSA requires a team effort from the sedationist and surgeon. Good communication is essential, and it is important to have realistic expectations. When performing these procedures out of hospital without general anaesthesia, having a calm, patient surgeon and seditionist is key. A back-up plan is necessary in the event of inadequate analgesia. Patient satisfaction and safety is always the most important aspect when considering out-of-hospital surgery.

**References**