Intraoperative Management of Penile Erection by Using Terbutaline

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Penile tumescence, resulting in partial or total erection during a cystoscopy or other operative procedure of the penis, can occur during regional or general anesthesia.1-5 When this does occur, it is very difficult to perform the desired procedure and may even result in the cancellation of endoscopic or other penile surgical procedures. Although various methods and a number of pharmacologic agents have been used to control penile erection, none have been found to produce consistently good results and some are even unsafe to use in the operating room.1,2 Four patients are described in whom terbutaline was used for treating this troublesome complication.

CASE REPORTS

Case 1. A 62-yr-old man, ASA Class II, was anesthetized for cystoscopy, retrograde pyelography, and prostate biopsy. Anesthesia was induced with thiopental iv and maintained with enflurane (1.5%) and nitrous oxide (60%) with oxygen. While preparations were being made to introduce the cystoscope, the patient developed a solid penile erection which did not disappear with the deepening of anesthesia (enflurane up to an inspired concentration of 3%) and decrease in arterial blood pressure from systolic 128 to 88 mmHg. The patient was given 0.25 mg of iv terbutaline, after which the penis became softer and the heart rate and systolic blood pressure increased from 65 to 80 bpm and 88 to 120 mmHg, respectively. Two minutes later, another iv dose of 0.25 mg terbutaline was given. The patient developed complete detumescence in less than 4 min without any other changes in vital signs. The endoscopic procedure and prostate biopsy were performed successfully.

Case 2. A 78-yr-old man, ASA Class II, was scheduled for a transurethral resection of the prostate for prostatic hypertrophy, under spinal anesthesia. The patient was given hyperbaric spinal anesthesia (L3-4 level) using 8 mg tetracaine and 0.8 ml of 10% dextrose (total volume 1.6 cc), resulting in a spinal blockade at the T8 level. As preparations were being made to introduce the resectoscope, the patient developed an erection, making it impossible to introduce the resectoscope. He was given 0.5 mg terbutaline iv. His heart rate increased from 60 to 80 bpm without any appreciable effect on the arterial blood pressure. Detumescence occurred within 5 min. The resectoscope was easily introduced and the prostate was resected in approximately 1 h.

Case 3. A 24-yr-old ASA Class I man was anesthetized for a bilateral inguinal hernia repair along with a varicocelectomy. Anesthesia was induced with thiopental iv, and his trachea was intubated with the assistance of succinylcholine iv. Two surgeons operated, one on each side. After making the incision, a massive erection developed which proved bothersome to the surgeons. Anesthesia was maintained using midazolam (3 mg), fentanyl (400 µg), nitrous oxide (60%), and atracurium (15 mg) iv. Terbutaline 0.5 mg was given iv and the penis became soft and returned to its usual size within 4 min. The heart rate increased from 70 to 110 bpm and the systolic blood pressure increased from 120 to 160 mmHg. The surgery was completed in 60 min, and the trachea was extubated after reversing the neuromuscular blockade. The patient was taken to the recovery room uneventfully.

Case 4. A 45-yr-old ASA Class III man was scheduled for a transurethral resection of the prostate for benign prostate hypertrophy and carcinoma of the bladder. A spinal anesthetic (L3-4) was given using a 25-g needle and 75 mg of lidocaine with 7.5% dextrose (1.5 cc volume premixed), resulting in an adequate block at the T8 level. Preparations were being made to introduce the cystoscope. At this time, a rigid penile erection developed, making it impossible to continue with the procedure. The turgid state continued for almost 10 min without change. Terbutaline 0.25 mg was given iv and resolution of the erection took place within 4 min. The surgical procedure was completed with no noticeable changes in heart rate or arterial blood pressure.

DISCUSSION

Regardless of the type of anesthesia used, whether spinal or general, penile erection can occur.1-5 Penile erection under spinal and epidural anesthesia is reflexogenic if it is a solid block extending well above the midthoracic level. Terbutaline may be a valuable agent to control penile erection.
level, blocking the sympathetic chain. Otherwise, it is both reflexogenic and psychogenic. Under general anesthesia, the penile erection is probably due to both psychogenic and reflexogenic stimuli. The reflexogenic stimuli arise from washing, touching and instrumentation of the area supplied by the pudendal nerve (S2,3,4), whereas psychogenic stimuli arise from the taste and smell of anesthetic agents, exaggerated auditory sensation during the second stage of anesthesia, or even dreams.6

It is estimated that 1% of male cardiac surgery patients who are anesthetized with high doses of fentanyl develop penile tumescence during attempted Foley catheterization.7 Neither cancelling surgery nor changing the anesthetic technique is the answer; the occurrence of recurrent episodes of penile tumescence while using different anesthetic techniques has been reported in the same patient.1 In one case, penile tumescence resulted in three trips to the operating room with multiple anesthetics. Finally a perineal urethrostomy was performed to accomplish the introduction of the resectoscope to remove the prostate.1,8

Although many methods of treatment have met with partial success, these methods often result in unwanted complications. Treatment with a spinal block is unsound physiologically.5 Spraying ethyl chloride on the penis is no better than ice packs; both are ineffective.7 Induction of hypotension using deep general anesthesia or epidural anesthesia, or by using trimethaphan or sodium nitroprusside, has met with failure. The lowering of arterial blood pressure in elderly TURP patients with coronary artery disease can precipitate a cardiac emergency.1

The use of amyl nitrite, a potent vascular smooth muscle relaxant which produces both arterial and venous relaxation, can result in detumescence.2 It is not advisable in patients with elevated intracranial or intraocular pressure, and the vapors are potentially explosive. These vapors can cause a precipitous decrease in arterial blood pressure resulting in reflex sympathetic tachycardia. This can be dangerous in patients with coronary artery disease.2

Ketamine has been used frequently to help control penile erection.1,4,7,8 Ketamine may take more than 1 h to develop flaccidity, making it impractical for the treatment of penile erection in the operating room. The problem with this drug is that, when used in patients who are under regional block (awake), it can cause unpleasant dreams. Disappointing results have been reported under spinal anesthesia once the erection has occurred.1,4,8,9

Neural control of penile erection does not appear to conform to classic cholinergic or adrenergic concepts.10 Peptidergic innervation is mediated through vasoactive intestinal polypeptide (VIP). The presence of vasodilating VIP in animal and human corpora may explain why there is a cholinergic mediated glandular secretion and atrope resistant vasodilation caused by parasympathetic nerve stimulation. Neither acetylcholine nor alpha and beta ad-

energetic blockade will cause an erection.2,6,11 Phystostigmine (a parasympathomimetic) has been used with ketamine (a sympathomimetic) to treat priapism.12 We are at a loss to explain how these two drugs act to relieve priapism and, again, the complex anatomic histology and neuropharmacology is evident.18,19

The latest method used to treat this problem is direct intracorporeal injection of phenylephrine, epinephrine, metaraminol, and dopamine.13,14 This can result in pain, hematoma, infection, and fibrosis of the penis and accidental iv injection. Two deaths have been reported in France due to acute severe arterial hypertension resulting from intracorporal injection of metaraminol for priapism.15

The mechanism of penile erection is a very complex phenomenon involving blood vessels, blood, polsters, tissue fluid, cavernous tissue, tunica albuginea, and the nervous system and its mediators.6,16 Terbutaline is thought to act by relaxing the entire smooth muscle of the cavernous tissue, and arteries, veins, polsters in these blood vessels, and tunica albuginea and its trabeculae in the penis. The cavernous smooth muscles are not only mechanically stretched due to rapid filling of the blood, but they contract (like a stretched spring) against the accumulated blood giving rigidity to erection.16 We believe that the terbutaline relaxes the stretched corporal smooth muscle (like a stretched spring) losing its tensile force resulting in flaccidity of the entire penis and relaxation of the tunica albuginea. The action of terbutaline on the fibrous-elastic-smooth muscle composed tunica albuginea and its trabeculae also makes the penis flaccid. This results in the release of the constriction and a collapsing effect on draining veins brought on by stretched tunica albuginea and elevated intracorporal pressure. The polsters in the penile veins are relaxed along with the smooth muscles lining the veins. This results in the widening of the diameter of corporeal draining veins and removal of impediment to venous blood flow created by contracting polsters during erection. Thus, the blood from arteries, cavernous sinusoids, and capillaries flows easily to the veins and out of the penis, resulting in its detumescence.

In summary, we have shown terbutaline, a beta-2 adrenergic agonist, to be consistently effective against intraoperative penile erection under any type of anesthesia.

REFERENCES

Numerous authors have recommended insertion of central venous catheters for aspiration of venous air embolism (VAE). Others,\(^1,2,\) have recommended insertion of pulmonary artery (PA) catheters for detection of hemodynamically significant VAE. Unfortunately, the lumens of commercially available PA catheters are small, making them "poorly suited for efficient air aspiration."\(^3\) This leaves the clinician in the awkward position of having a catheter well suited for either detection or treatment of VAE, but not both. Furthermore, since significant amounts of air may enter the right ventricular outflow tract and PA,\(^1,2,4,5\) a means to effectively detect and treat significant VAE in either PA or right atrium-superior vena cava (RA-SVC) is needed.

The author has developed a catheter to address this need (fig. 1, 2). Its balloon tip and 70-cm length permit insertion into the PA. The ECG bushing allows accurate RA-SVC positioning without adapters. Multiple side holes, an end hole, and 7 French size allow rapid removal of embolized air. If used with an introducer with an external sterile sleeve, the catheter can be repositioned during surgery, allowing repeated aspiration of air from multiple sites. The introducer side port will also provide an independent central port for drug injection. The catheter is available on request from Arrow International (PO Box 6306, Hill and George Avenues, Reading, Pennsylvania 19610). Its use is illustrated by the following case.

**REPORT OF A CASE**

A previously healthy 47-kg, 175-cm, 22-yr-old woman with a large midline tumor obstructing the outlet of the fourth ventricle, hydrocephalus, and brain stem compression was brought to the operating room for urgent decompression.

A prototype of the catheter described above was inserted 4 cm into the PA via the left subclavian vein, with the depth of insertion as RA-SVC determined by intravascular ECG monitoring during catheter passage. The introducer and sterile sleeve assembly were left in place to allow later catheter manipulation.

Induction of anesthesia with 200 µg of fentanyl and 800 mg of thiopental was followed by 8 mg of vecuronium and tracheal intubation with a 7.5-mm endotracheal tube. Following intubation, anesthesia was maintained with 70% nitrous oxide and oxygen. The patient was placed in the sitting position.

A burr hole and ventriculostomy were placed in the right parieto-occipital area. A midline suboccipital incision was then made. As this incision was being carried down to the bone, the end-tidal CO\(_2\) decreased rapidly to 14 mmHg, followed rapidly by an increase in PA pressure to 39/24 mmHg and a decrease in arterial pressure to an eventual nadir of 20 mmHg systolic. In rapid sequence, the surgeons were notified of air embolism, 10 mg of ephedrine was given iv, and the catheter was aspirated. Nitrous oxide was discontinued and 5 cm H\(_2\)O PEEP was instituted. After inspecting both wounds, the surgeons located an open vein in the burr hole, which was then packed. A total of 25 ml of air was removed from the PA, then the catheter was withdrawn to the RA-SVC position where an additional 10 ml of air was removed. The arterial pressure rapidly recovered to normal. Ten to fifteen minutes were required for PA pressures and end-tidal CO\(_2\) to return to normal, after which the catheter balloon was inflated and the catheter repositioned in the PA. Anesthesia was then maintained with isoflurane in oxygen. Later, there were two similar, but less severe, episodes. Following the operation, the patient recovered fully without apparent sequelae.