Chapter 7

Anesthesia in Hair Transplantation

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Additional information is available at the end of the chapter

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Abstract

Current hair transplantation techniques require a reliable anesthesia for long periods of time (2 h or more). They demand hemostasis of extended surfaces on wide-awake patients. A combination of anesthetic agents and local vasoconstrictors is needed. We present customary technical characteristics of these procedures as local nerve blocks (supratrochlear nerve, supraorbital nerve, zygomaticotemporal nerve, auriculotemporal nerve, retroauricular nerve, lesser occipital nerve, great occipital nerve) and tumescent field anesthesia. The ordinary drug combinations for premedication and procedure are presented. Special emphasis is done to discuss recommendations to cope with undesirable events that may arise during anesthesia (vasovagal syncope, anesthetic toxicity, anaphylactic and allergic reactions).

Keywords: anesthesia, loco-regional, nerve block, hair, graft, transplantation, implant, follicular unit, patient safety, supratrochlear nerve, supraorbital nerve, zygomaticotemporal nerve, auriculotemporal nerve, retroauricular nerve, lesser occipital nerve, great occipital nerve

1. Introduction

Hair transplantation techniques require a reliable anesthesia for long periods of time. Particular features of these procedures include hemostasis of extended surfaces and wide-awake, conscious patients, and a combination of anesthetic agents and local vasoconstrictors is needed. As a matter of fact, local scalp blocks are common practice in reconstruction of traumatic injuries as well as in neurosurgical interventions that involve intraoperative, functional assessments like deep brain stimulation and epilepsy surgery [1]. Another important feature is the obtainment of local tumescence to allow advantageous firmness of the donor and recipient areas [2, 3].

Lidocaine is the preferred local anesthetic nowadays. It is a short-acting, amide agent and its onset takes place 2 min after injection. The effects are 1–2 h long but the concomitant use of adrenaline stretches them up to 4 h. A typical hair transplantation session may involve 2–3 injections of lidocaine with 2–3 h intervals. *Bupivacaine and ropivacaine* are long-acting, local anesthetics. Their price and cardiotoxicity are greater than those of lidocaine but they remain an interesting option when the anesthetic effect should be maintained long after the procedure is finished and they can be associated with lidocaine in the same injection point [4]. Additionally, some surgeons inject bupivacaine or ropivacaine at the end of procedures that demand long incisions to harvest a donor area.

Ester anesthetics (procaine and chloroprocaine) are known to trigger more allergic reactions though their systemic toxicity is lesser than for lidocaine. Their use is less popular than some 30 years ago. *Opioids* (morphine, fentanyl, and meperidine) are still used in some countries: Handling and storing them requires security measures to avoid abuse and trafficking. Their potential depression of respiratory automatism hampers a wider utilization.

Adrenaline (epinephrine) acts both on alpha and beta receptors but beta effects predominate in the long run. It increases systolic blood pressure, cardiac output volume, heart rate and coronary perfusion. It also increases myocardial consumption of oxygen. It may induce ventricular fibrillation.

The dilution of adrenaline is expressed as a ratio. The 1:1000 concentration means that there is 1 g/l or 1 mg/ml. Standard concentrations of 1:100,000 are equal to 0.01 mg/ml. When using combined 2% lidocaine with 1:100,000 adrenaline, the maximal amount of lidocaine will be achieved long before reaching the maximal dose of adrenaline. The first local effect of adrenaline is vasoconstriction. Systemic effects will only be visible after reaching a dose of 0.015 mg. These include palpitations, sweating, tachycardia and increase in systolic blood pressure up to 70 mmHg over the basal line.

It is worth to bring to mind that most local anesthetic agents act as vasodilators: They dilate local blood vessels which increases their absorption area and diffusion in the circulatory system. Therefore, local vasoconstriction at the areas of injection will result in a decreased risk of toxicity and a longer duration of the effect of the chosen anesthetic agent. Moreover, the reduction of perfusion entails a reduction in blood loss.

2. Contraindications and main risks for local vasoconstriction and anesthesia

Direct vascular traumatism may cause local ecchymosis and should be addressed by effective compression of the area. No large vessels are crossed in the commonest procedures. Needless to say, anti-inflammatory drugs and antiaggregation agents like salicylates should be avoided (when possible) during the days before the procedure.

Intravascular injection of the anesthetic mixture must be avoided through the usual cautions of anatomical knowledge and short "suction before injection" routine.

Intranervous injection and damage are rare and may be prevented by the constant pressure flow of the anesthetic product itself that detaches the tissues. Unexpected movements of the patient and changes in angulation of the needle without a slight withdrawal may also lead to kerbed lacerations.

Overdosing is prevented by the previous estimate of doses according to the weight of the patient (**Table 1**) [5].

Contraindications to the utilization of vasoconstrictors include untreated hypertension, episodes of angor, recent myocardial infarction, severe bradycardia, auriculoventricular block (second or third degree), accelerated idioventricular rhythm, untreatable arrhythmias as well as concurrent treatment with amiodarone, procainamide, flecainide, quinidine or disopyramide. Hyperthyroidism warrants the presence of a trained anesthetist during the intervention. Patients taking tricyclic antidepressants should never receive noradrenaline or levonoradrenaline.

Vagal reaction is a repeated event. Some general measures help to reduce their numbers. Excessive lighting and temperature of the operation room must be avoided at all costs. Some patients may appreciate a first visit before the procedure takes place in order to identify it as a familiar, less hostile environment. Many surgeons make the patients lay flat for the simple nerve blocks and slowly make them sit in the final position. This is simpler when comfortable, hydraulic, bendable stretchers or operation tables are used. A disproportionate needle size may cause bigger pain, so the gauges usually have 25–30 gauge diameters. The smaller the caliber, the lesser the pain, and some teams favor the use of 32G needles. As always, an exaggerated syringe volume may cause higher pressure at the point of injection. A small vibration (by rhythmic tapping or by special devices) is well known to detour attention of the patient and make the injection more bearable. Verbal contact must be maintained all along the procedure. All injectable products should be as close as possible to body temperature: a cold injection may unchain a vagal reaction. Application of eutectic mixture of local anesthetics (EMLA) that usually includes lidocaine and prilocaine as patches or cream on the

	Maximum dose	**Duration of effect**	**Maximum dose with adrenaline**	**Duration of effect with adrenaline**
Amides				
Lidocaine	4 mg/kg	30 min–2 h	7 mg/kg	Around 3 h
Bupivacaine	2 mg/kg	2–4 h	3 mg/kg	3–5 h
Ropivacaine	5 mg/kg	2–6 h		
Mepivacaine	4 mg/kg	1.5–3 h	7 mg/kg	2–4 h
Prilocaine	7 mg/kg	30 min–2 h	8 mg/kg	Around 2 h
Esters				
Procaine	5 mg/kg	20–30 min	7 mg/kg	30 min
Chloroprocaine	11 mg/kg	15–30 min	14 mg/kg	30 min

Table 1. Comparison of maximum doses and duration of effect between common local anaesthetic drugs.

intended injection sites 45–60 min before injection is advisable for all patients, especially when they have declared aichmophobic events in the preoperatory anamnesis. Earphones or even movies may help to distract attention. Premedication (as 5–20 mg of oral diazepam) is more and more common.

Allergic reactions are extremely rare. They involve true hypersensitivity and the intensity of the reaction is dependent on the dose of the reagents. There is a marked susceptibility in asthmatic patients. A particular culprit is sodium metasulfite, a preserving additive for adrenaline.

2.1. Attitude in case of anesthetic toxicity

Early signs of toxicity of ester anesthetics include a metallic taste sensation in the mouth, numbness of tongue, muscle trembling, visual alterations and shivering [6]. Later signs are loss of consciousness, convulsions, coma and respiratory arrest. Metabolic acidosis, hypoxia and hypercarbia may be triggered by convulsions before respiratory arrest appears. In the face of central nervous symptoms of toxicity, the patient must be put in the Trendelenburg position and must receive supplemental oxygen. Hyperventilation may prevent the onset of seizures but once the patient gets unconscious, it seems advisable to intubate to secure an open airway. Seizures are controlled by 5 mg bolus of intravenous diazepam, to be repeated every 5–10 min in case there is no response. In case of severe bradycardia, small doses of intravenous atropine (0.5 mg) should be sequentially administered.

Opioids may induce inappropriate euphoria, nausea, vomits and respiratory depression, bradycardia and bronchospasm. Treatment includes intravenous naloxone titrated in 0.1 mg doses every 2 min until reaching a 10 mg total dose.

Benzodiazepines also may induce respiratory depression. This side effect is potentiated by opioids. This adjuvant phenomenon prevents the two families of drugs from being widely used together. Intravenous flumazenil is the common antidote for benzodiazepines and it should always be available in a practice that applies oral premedication.

2.2. Attitude in case of anaphylactic shock

Anaphylaxis is a life-threatening allergic reaction with cardiorespiratory involvement that sets in minutes. It must be remembered that cutaneous signs are not the main sign of anaphylaxis. Sudden wheezing or coughing, complaints of throat tightness or voice changes should trigger suspicion of respiratory involvement. By diminishing circulation of blood, anaphylactic shock itself averts the full treatment of the anaphylaxis. Fortunately, true anaphylaxis is rare but it is the main reason for establishing a venous line before starting these long procedures. Whenever a venous line is not established, adrenalin injectors must be available. All personnel in the operating facilities, including technicians and secretarial staff, should be trained in basic life support. On a precise spot of the premises, the basic reanimation drugs must be conspicuous and ready. Most times, adrenaline will be used when the situation corresponds to impending shock. The first line of actuation includes 0.2–1 mg of subcutaneous adrenaline (0.2 –1 ml in a standard 1:1000 solution). Most severe reactions prompt an intravenous, slow injection of 0.1 mg of adrenaline over 5 min.

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When precociously detected, a mild, allergic reaction may be jugulated by the combined intravenous use of an antihistaminic (typically 50 mg of diphenhydramine) and a corticoid (typically 100–200 mg of hydrocortisone). These can prevent the mast cells and basophils to degranulate, thus inhibiting the release of chemical mediators as histamine, prostaglandins, leukotrienes and tryptase. Antihistaminic drugs are also useful in the treatment of biphasic and protracted types of anaphylactic reaction. The patient must be kept under observation for a minimum of 4 h. Additional treatment may include intravenous fluids and supplemental oxygen. Other vasopressor drugs and intubation are more specifically delivered in emergency rooms.

2.3. Attitude in case of vasovagal syncope

In vasovagal syncope, the patient undergoes short unconsciousness as a result of abrupt descent of blood pressure. It is more frequent when the patient is in standing or sitting position. Some patients may show short nonperiodic jolts. Common prodromal symptoms include copious sweating, nausea, ear buzz, transient aphasia and vision disturbances (like "seeing white" or "clouded"). Some people with recurrent episodes of vasovagal syncope can somehow anticipate them and increase venous return by muscle contraction of their legs, elevation of both hands over the head or lying in prone position with lower limbs on a cushion.

The main risk of such an event is injury from falling. Once the patient has lost consciousness, full supine position without elevation of the head is enough to regain normal, awakened state. Trying to sit or stand up immediately after such an event may result in a new loss of consciousness, and sweating or nausea may persist for several minutes.

3. Premedication

Premedication is more and more common for these procedures though it entails that the patient must attend the premises 30–45 min before entering the operation room.

Oral diazepam in a dose of 5–20 mg is the usual choice. A (desirable) side effect of diazepam consists of elevating the threshold for convulsive effects of lidocaine. The utilization of benzodiazepine has important legal and safety implications and the patient must be formally instructed to avoid driving, handling of dangerous tools and signing legal contracts for 24 h after administration. Barbiturates are no longer in use for this indication.

4. Other intraoperatory cautions

Verbal contact should be maintained and the patient is always instructed to unambiguously verbalize painful sensations and bizarre feelings before the procedure starts. Recurrent assessment of cardiac frequency, blood pressure and pulse oximetry seem advisable, at least every

30 min [7, 8]. Small, commercial devices for the assessment of cardiac frequency and pulse oximetry are relatively inexpensive.

Though many surgeons may boast of never having unexpected events, an intravenous line should be placed once the premedication has done its effects.

As for the blocks, patches of EMLA are an interesting option in aigmophobic patients 45 min prior entering the operation room. They can be administered at the same time as the oral premedication.

In case the procedure is carried out in a small practice away from hospital premises, an evacuation plan with safe medical transport must be always established. Paramedical crew should find no major obstacles for quick transfer and evacuation.

5. Technique of local anesthesia

Different areas of the scalp correspond to different sensory nerves [9–12]. These include the supraorbital and supratrochlear branches of the ophthalmic nerve (V_1), the zygomaticotemporal branch of the maxillary nerve (V_2), the auriculotemporal branch of the mandibular nerve (V_3), the lesser occipital nerve and the retroauricular nerve from the third (and second) cervical roots as well as the greater occipital nerve from the second cervical root (**Figure 1**).

The first phase of anesthesia involves several peripheral nerve blocks by exclusive anesthetic injection WITHOUT adrenaline. The second phase involves anesthetizing and making tumescent the whole operative field by a mixture of adrenaline and anesthetic agent.

5.1. Supratrochlear nerve block

The supratrochlear nerve emerges medial to the supraorbital notch and it lies in the medial third of the upper orbital rim. It supplies sensory innervation to the medial area of the upper eyelid, the root of the nose and to the frontal scalp and forehead. It must be remembered that surgery on one side of the forehead requires a block of the contralateral supratrochlear nerve due to overlapping territories [13]. For a supratrochlear block, the main landmark lies on top of the angle formed by the eyebrow and the nasal spine. At this point the nerve is in contact with the bone (**Figure 2**). After the injection, firm pressure is applied for better anesthetic spread and prevention of ecchymosis.

Complications of this block (and the following one) are very rare but they include hematoma, intravascular injection and eye globe injuries.

5.2. Supraorbital nerve block

The supraorbital nerve emerges through a foramen on the middle of the orbital rim. The foramen is palpable as a clear step in osseous continuity. The supraorbital nerve exits with its vessels through the *foramen supraorbitalis* and continues superiorly between the *levator palpebrae*

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Figure 1. Surface areas that correspond to the sensitive territories of the lesser occipital and retroauricular nerves (green), the greater occipital nerve (pink) and the three divisions of the trigeminal nerve: ophthalmic (violet), maxillary (orange), and mandibular (red).

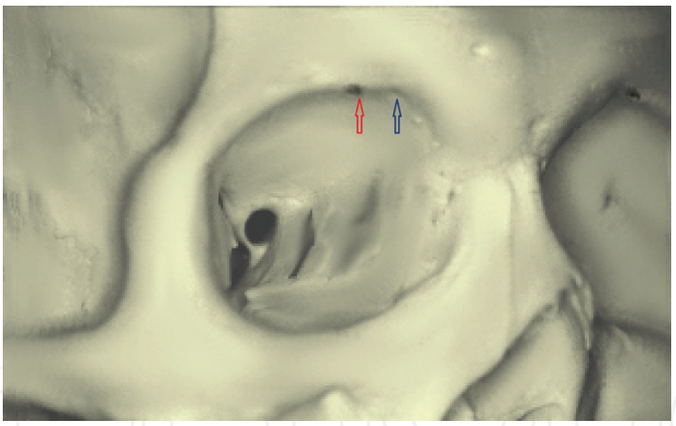

Figure 2. The foramen supraorbitalis (red arrow) lies at the junction of the middle and inner thirds of the superior orbital rim. It marks the emergence of the supraorbital nerve and the supraorbital artery. Mild pressure to the nerve causes some pain. The needle is inserted over the eyebrow level and advanced until touching the periosteum. After injecting 1 ml of lidocaine, the needle is redirected medially and 1 ml of lidocaine is injected along the way. Finally, 1 ml is injected again at the final site that corresponds to the supratrochlear nerve (blue arrow).

superioris and the periosteum. *The foramen supraorbitalis* is easily palpable by following the orbit rim 2 cm from the midline in adults and it loosely corresponds to the same sagittal plane as the pupil when facing the patients head. Previous LeFort fractures may modify reference points. A 25–30 gauge needle is intradermally introduced 0.5 cm under the inferior edge of the eyebrow and is directed medially and cephalad. Once the needle tip is near the supraorbital notch, after

test aspiration, local anesthetic solution (0.5–1 ml) can be injected in an extended subcutaneous wheal. Injection *into* the foramen must be avoided (**Figure 2**).

It is worth noting that the supratrochlear nerve can be blocked immediately following supraorbital nerve block, without removing the needle, by directing the needle about 1 cm toward the midline and injecting an additional 0.5 ml of local anesthetic.

5.3. Zygomaticotemporal nerve block

One of the two branches of the zygomatic nerve (branch of the maxillary division of the trigeminal nerve) is the zygomaticotemporal nerve. It receives sensitive innervation from the temporal area and a small portion of the forehead (**Figure 3**). The nerve lies lateral to the orbital rim in the fossa temporalis [14]. It is usually approached through an injection in the middle third of the upper aspect of the malar branch, targeting the orbital rim and delivering 1 ml of 2% lidocaine while advancing the needle in a 45° angle. The bony rim of the orbit should never be reached to avoid migration inside the orbit. At the final point, 1 ml is carefully injected.

5.4. Auriculotemporal nerve block

The auriculotemporal nerve is a branch of the mandibular division of the trigeminal nerve. It receives sensitive innervation from the posterior area of the temple and the superior two-thirds of the anterior surface of the pinna [15, 16]. It crosses through the parotid gland and continues anterior to the auditory canal escorting the superficial temporal artery. It intersects the zygomatic arch near the surface (**Figure 3**).

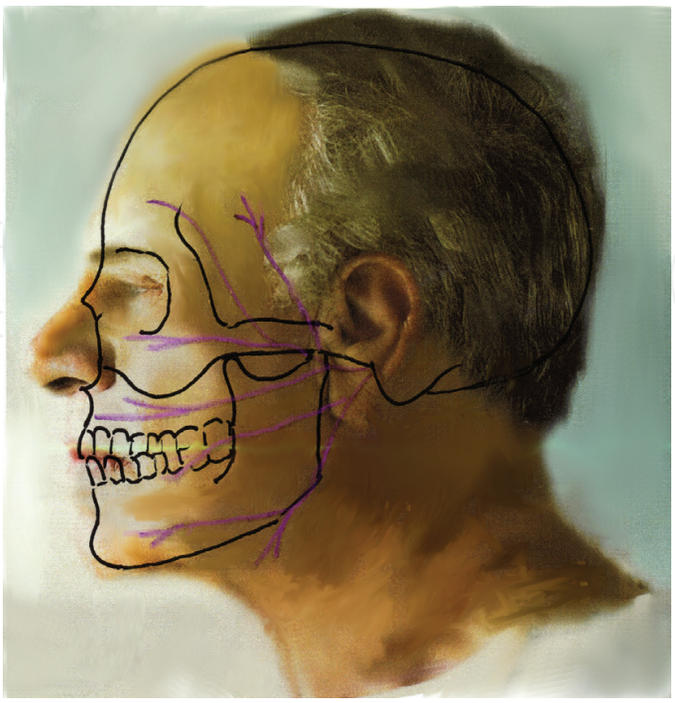

Figure 3. Surface references for the main branches of the trigeminal nerve.

The auriculotemporal nerve is blocked by injecting above the posterior portion of the zygoma, anterior and superior to the tragus and behind the superficial temporal artery that must be avoided. A total of 1–2 ml of 2% lidocaine is delivered after delicate aspiration.

5.5. Lesser occipital nerve and retroauricular nerve block

The lesser occipital nerve is a small branch from the ventral primary rami of the second and third cervical roots. It receives sensitive innervation from the lateral occipital zone and the upper earlobe (**Figure 4**). It runs posteriorly at 45° to the vertical, parallel to the posterior border of the sternocleidomastoid. A subcutaneous injection along the hairline behind the ear is the way to block both nerves [17].

5.6. Greater occipital nerve block

The greater occipital nerve provides cutaneous innervation to the major portion of the posterior scalp from the inion to the vertex. It stems from the second cervical nerve root that comes out between the atlas and the axis. It runs between the *obliquus capitis inferior* and *semispinalis capitis* before piercing the latter muscle. After piercing the aponeurosis of the *trapezius,* it

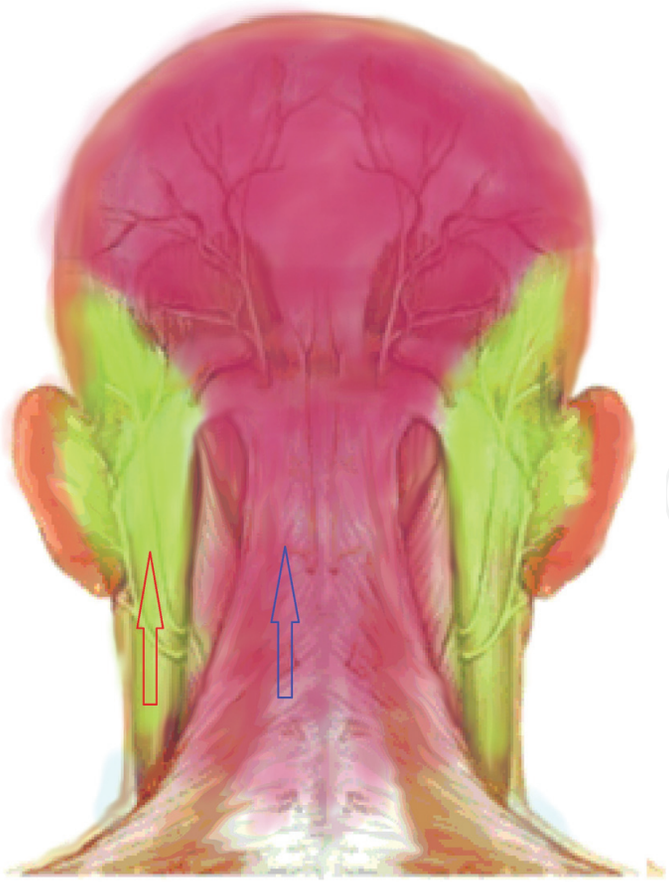

Figure 4. Reference points for the lesser occipital and retroauricular nerves (red arrow) and the great occipital nerve (blue arrow).

becomes subcutaneous distally to the superior nuchal line. Typically, it lies 4 cm lateral to the inion and medial to the occipital artery. The pulsation of the occipital artery is easy to palpate (**Figure 4**). There is considerable variation in its position (1.5–7.5 cm from the inion) [18, 19] and some anesthetists advocate the use of ultrasound probes for safe localization [20].

A 25 G is driven at 90° toward the inion. After aspiration, 1–3 ml of local anesthetic is injected and pressure should be maintained over the site of injection to soak the nerve and to achieve hemostasis once the needle has been withdrawn. Numbness up to the top of the head is a sign of an effective block.

5.7. Field anesthesia

Once the regional blocks have been performed, the surgeon can proceed to the anesthesia of the operative field in a "crown" fashion. Some surgeons advocate only using this kind of anesthesia without nervous blocks (or just the supraorbital and supratrochlear block). Now, a significant, desired outcome is the tumescence of the donor and recipient areas. Adrenaline would prevent hemorrhage and is an integral part of the anesthetic mixture. The injections cause a dissection of the subcutaneous layer providing hemostasis and a firm, stable working plane. However, the onset of the effect is longer than for the blocks and may take about 10–15 min. Anesthesia is achieved by several injection points 1 cm caudally to the hairline or its intended location and each injection point may be used as a departing point for several anesthetic tracts. The number of injection points is highly variable and depends on the preferences and experience of the surgeon. The same applies to the composition of the injected mixture. A common "recipe" employs 50–100 ml of 2% lidocaine and 2–5 ml of adrenaline 1:1000 in 1 l of 0.9% saline (but the whole amount of the mixture is not needed). Some "recipes" include 10 ml of 8.4% sodium bicarbonate to diminish the irritating effects of the low pH of the mixture [2, 3, 21]. Many surgeons add bupivacaine to achieve a lasting effect or even corticoids to reduce the swollen, traumatic appearance of the scalp in the days after the procedure.

5.8. Intravenous sedation

There are remarkable differences in attitudes toward this kind of anesthesia and they vary according to regions and training [21, 22]. The presence of a standby anesthetist to cope with unwanted side effects seems unnegotiable to most surgeons. Nowadays, a combination of midazolam and fentanyl is the preferred option.

5.9. Inhalatory anesthesia

Inhalatory anesthesia is not justified for elective hair transplantation but rare indications include pediatric patients that undergo procedures after burn injuries. Though cumbersome, some clinics use nitrous oxide sedation under supervision of an anesthesiologist or a trained nurse.

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