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Peripheral nerve stimulator vs paresthesia

Anthony R. Brown, MB

“no paresthesiae no anesthesia”

Moore, 1953

“no paresthesiae no anesthesia”

Gentili and Wargnier, 1993

“no paresthesiae no dysasthesiae, but of the failed anesthesia?”

Moore, Mulroy, Thompson, 1994

The above dictums summarize the opposing views regarding the risks and benefits of the paresthesia *versus* non-paresthesia techniques used to perform peripheral nerve blockade. Is there in fact any difference between the two techniques with respect to success and/or complication rate? In particular, does the use of the paresthesia technique increase the risk of nerve damage?

The relationship between peripheral nerve damage and regional anesthesia is controversial. There are many possible causes of nerve damage during the perioperative period, however, in many cases a definitive cause is impossible to establish⁽¹⁻³⁾. In an ASA Closed-Claim Study, Kroll et al. reported that nerve injury was responsible for 15% (227/1,541) of the total number of claims. The majority of these injuries, (61%) occurred under general anesthesia. The ulnar nerve (36%) was the commonest nerve involved, followed by the brachial plexus (23%), lumbosacral nerve roots (16%), and spinal cord (6%). The remaining group was categorized as “other/multiple” nerves (21%). A more recent update, nerve injury accounted for 16% of all claims, with three specific distributions (ulna nerve, brachial plexus, and lumbosacral root) accounting for more than half of all claims for peripheral nerve damage. The author’s conclusion was that most anesthetic-related nerve injuries seem to occur without an identifiable mechanism^(4,5).

The overall incidence of nerve injury is in the region of 0.1% of all surgical procedures^(5,6). Thompson and Liu reported a 0.11% (30/26,167) incidence of postoperative nerve damage. Sixteen of these patients received regional anesthesia, and, after careful evaluation, only 3 cases were suspect regarding the

regional anesthetic. Since most resolve within days to weeks, the problem tends to escape our attention. Although these injuries are rarely related to the regional anesthetic, historically regional anesthesia tends to be the scapegoat⁽⁶⁾.

In a more recent study, Horlocker et al. reported that the frequency of neurological complications in patients undergoing repeated axillary block is similar to that in patients receiving a single regional anesthetic technique. Block techniques included paresthesia, nerve stimulator, transarterial, and combinations of the three. A total of 1,614 blocks were performed on 607 patients. The authors reported 62 neurological complications in 51 patients (i.e. an overall frequency of 8.4% of patients). Only 7/62 (11.3%) of the nerve injuries were related to the anesthetic technique; the remainder (88.7%) were the result of the surgical procedure. In 5/7 cases a paresthesia and/or combination technique was used (paresthesia was elicited in nearly 60% of blocks in the study), in one case a nerve stimulator was used, while in the remaining case the regional technique was unknown⁽⁷⁾.

The causes of nerve damage are legend in number and include pre-existing (often undiagnosed) neurological pathology (e.g. diabetes, uremia, alcoholism, Guillain-Barre syndrome), general anesthesia (mechanisms include direct trauma, pressure, ischemia, plus the potentiating role played by muscle relaxants, hypotension and hypothermia), surgical (malpositioning, prolonged operating time, direct injury to nerves or their blood supply, retractors, tourniquet injuries), and postoperative causes (orthopedic splints and casts, anticoagulation therapy). Regional anesthesia may be associated with nerve damage through a variety of mechanisms. These include excessively high concentrations of local anesthetics, pressure-related effects of high volume injections, the inadvertent injection of a neurotoxic agent, addition of vasoconstrictors to local anesthetics, and direct needle injury. In the latter case, nerve damage may result from direct trauma to the nerve and/or its blood supply, hematoma formation (resulting in nerve compression and/or ischemia), direct intraneural injection, a combination of these factors,

and finally, the use of a paresthesia technique has been implicated.

Numerous authors have suggested that elicitation of paresthesia may result in nerve damage, and that the use of a nerve stimulator and/or a short-bevel needle will reduce the incidence of postblock neuropathies⁽⁸⁻¹³⁾. The counter-argument states that there are no statistically significant clinical data to support the suggestion that nerve damage results from elicitation of paresthesia, and that evidence to support the above arguments are based largely on extrapolation from animal data⁽¹⁴⁾.

Selander et al. drew attention to the role played by the design of the needle tip in nerve injury. They reported a decreased frequency of fascicular nerve damage after needling isolated rabbit sciatic nerve preparations with a 45-degree (short-bevel) needle (8/15) compared with a 14-degree (i.e. regular A- or long-bevel) needle (14/15). The authors concluded that the short-bevel needle produces less injury and should therefore be recommended. In addition, fascicular injury following an intraneural injection occurred in 3/30 nerves with the short-bevel needle compared with 14/30 when a 14-degree bevel needle was used. The authors observed that nerve fascicles have a tendency to slide away from an advancing needle point and that a short-bevel needle significantly reduces the risk of nerve injury when compared with a long-bevel needle. They concluded that paresthesiae should be elicited gently, and that intraneural injections should be avoided⁽⁸⁾.

Rice and McMahon studied the effect of bevel design upon the long-term pathology following intrafascicular. Their findings appeared to contradict those reported by Selander et al. regarding needle design. These authors demonstrated that fine-bore needles inserted into isolated rat sciatic nerves were capable of producing significant and protracted damage. In addition, more severe nerve lesions followed the use of a 27-degree (i.e. short-bevel) needle than a 12-degree (long-bevel) needle⁽¹⁵⁾. It should be pointed out that in Selander et al's study a multifascicular nerve (as would be encountered in clinical practice) was studied rather than a monofascicular nerve as was used in the Rice and McMahon study. Nerve fascicles tend to slide or roll away from the needle point both *in vivo* and *in vitro* especially when a 45-degree short-bevel needle is used as opposed to a long-bevel needle. When a needle comes into contact with a nerve, paresthesia is elicited, and the (awake) patient normally reacts to this. The onset of paresthesia informs the anesthesiologist that the needle is in close contact with the nerve and that, if advanced further, may injure the nerve. The use of a short-bevel needle may press or push the nerve away, thereby giving the anesthesiologist more time to react to a paresthesia before the needle penetrates and possibly injures the nerve. In the Rice and McMahon study the nerve fas-

cicles were deliberately injured with the test needles – a situation unlikely to occur in clinical practice⁽¹⁶⁾. This argument is in fact supported by Rice and McMahon themselves who demonstrated that, during microneurography in awake patients, when paresthesia was elicited, in only a minority of cases was the fascicle impaled (as indicated by neurographic recording)⁽¹⁷⁾. It would therefore appear that the previously described findings of Rice and McMahon would only hold true clinically in the case of inadvertent penetration of a nerve fascicle.

Does the use of paresthesia in fact increase the risk of post-anesthetic neurological sequelae in a clinical setting? In a prospective study, Selander et al. divided patients undergoing hand surgery into a paresthesia and a non-paresthesia (axillary artery pulsation of the needle) group. Mepivacaine 1% with/without epinephrine was the local anesthetic used. Paresthesia was unintentionally elicited in 40% of the non-paresthesia group. Postoperatively 18 (3.4%) of the patients had nerve lesions. In 8 (1.5%) cases the cause was attributed to the effects of surgery, position, or plaster. Postanesthetic nerve lesions occurred in 8/290 (2.8%) patients in the paresthesia group and 2/243 (0.8%) in the non-paresthesia group. In both of the latter cases, unintentional paresthesias were elicited during performance of the block. All of these patients received mepivacaine 1% plus epinephrine. Three patients complained of increased paresthesia on injection, while all complained of a painful paresthesia. Postoperatively, their symptoms varied between light paresthesia lasting a few weeks, to severe paresthesias with aching and paresis lasting more than a year. The question that was raised was whether the needle itself, and/or a direct intraneural injection were responsible for the postoperative neuropathies. The authors concluded that, whenever possible, nerve blocks should be performed without searching for paresthesias, and that, if used, rough and repeated probing for paresthesia, especially with a thick sharp needle can cause nerve damage. They did add, however that “a gentle touch with the needle is probably harmless to the normal nerve”⁽⁹⁾.

The importance of keeping an accurate anesthesia record as an aid to diagnosing a postoperative neuropathy was emphasized⁽⁹⁾. The record should include the type of block, local anesthetic used, its concentration, whether epinephrine was added, the type of needle used, a description of any paresthesia elicited as well as puncture of a blood vessel and/or hematoma formation, tourniquet pressure and time, supplementary blocks and drugs used by the anesthesiologist or the surgeon, and unexpected reactions during the blocking procedure or during the course of the operation.

In an attempt to further define the factors involved in nerve damage, Selander et al compared the effects of an

intrafascicular injection with that of a topical application of bupivacaine on a rabbit sciatic nerve. Topical application, in clinically recommended concentrations, caused no detectable nerve injury, whereas intrafascicular injections caused considerable axonal degeneration and damaged the blood-nerve barrier. The same damage occurred following the injection of physiologic saline solution suggesting that injection trauma rather than injected solution was responsible. Damage to the nerve, however, increased with increasing concentrations of bupivacaine and especially with the addition of epinephrine. Topical application of epinephrine did not cause any nerve damage. The authors commented that epinephrine increases the risk of neurological sequelae once the nerve is damaged (by needle trauma and/or an intraneural injection), and concluded that intraneural injections should be avoided, and plain bupivacaine solutions should be routinely used⁽¹⁸⁾. Concern regarding the addition of epinephrine to local anesthetics has been expressed by others^(19,20). Gentili et al. demonstrated the potential toxicity of local anesthetics in clinical concentrations when injected intraneurally and concur that intraneural injections should be avoided⁽²¹⁾.

In a study by Plevak et al. an axillary brachial plexus block was performed using either a transarterial (i.e. non-paresthesia) or a paresthesia technique. The incidence of persistent paresthesias (defined as longer than 48 hours) occurred in 2/239 (0.8%) of the transarterial group compared with 14/477 (2.9%) in the paresthesia group. The success rate was similar (88% vs 85%)⁽²²⁾. In contrast, Urban and Urquhart reported a similar incidence of paresthesias (9%) on POD 1 following interscalene brachial plexus blockade (22/217 in the paresthesia group, and 3/34 in the nerve stimulator group) with complete resolution in all but one patient by 6 weeks⁽²³⁾.

Operator experience plays an important role in the incidence of complications associated with regional anesthesia. Winchell et al., in a prospective study, reported an incidence of 0.36% (3/854) postblock neuropathies in which paresthesia was elicited by nine experienced anesthesiologists in private practice. Of these blocks, in 835 at least one paresthesia was elicited. Included in the group were 816 axillary, 12 supraclavicular, 12 parascalene, and 14 interscalene brachial plexus blocks. In all cases short-bevel needles were used. As there is unfortunately no control (i.e. non-paresthesia) group, conclusions cannot be reached regarding the comparative safety or efficacy of the two techniques⁽²⁴⁾.

Chambers re-emphasized the fact that needle trauma remains an important factor in nerve damage. The author stated that the incidence of nerve damage may be increased by the paresthesia technique, and that, if this technique is used, it should be used gently and certainly not repeated.

The importance of rapport between the anesthesiologist and the patient was stressed. On the other hand, the possibility of nerve damage is reduced with the use of a peripheral nerve stimulator, particularly if short bevel needles are used⁽²⁵⁾.

The safe and successful use of nerve stimulators is supported by Fanelli et al. who utilized a multiple injection technique to perform 3,996 upper and lower extremity blocks. During the first month after surgery, 69 patients (1.7%) developed neurological dysfunction in the operative limb. Complete recovery required 4-12 weeks in all but one patient, who recovered completely 25 weeks post-block. [Fanelli, 1999 #7405.

Gentili and Wargnier reiterate that a number of studies support the belief that paresthesia increases the risk of nerve trauma. As a result, they prefer the axiom "no paresthesia, no dysaesthesia" to that of "no paresthesia, no anesthesia" as stated by Moore^(26,27).

Moore et al. disagree that the use of a nerve stimulator decreases the incidence of nerve damage. These authors believe that a more accurate statement would be "no paresthesiae no dysaesthesiae, but often failed anesthesia". The nerve stimulator has not been shown to be more reliable than the paresthesia techniques for locating nerves, nor has the nerve stimulator been demonstrated to be clinically safer. In addition, use of the nerve stimulator to locate a nerve, particularly in the unconscious (anesthetized) patient, has not avoided neuropathies. These authors have reviewed six medico-legal cases in which a permanent brachial plexus neuropathy occurred and in which the nerve stimulator was used. None of these cases were reported in medical publications and the authors do not report the details of the cases (for medicolegal reasons). It should be noted, however, that these cases "occurred in semiconscious and unconscious patients, who did not respond when the needle point, in all probability, pierced the nerve's epineurium and the therapeutic dose of local anesthetic was injected". (Moore) The authors conclude by suggesting that there are no statistically significant clinical data to demonstrate that eliciting paresthesia results in nerve damage. Until a prospective blinded major clinical study can provide us with statistically significant information, they believe that authors should not draw conclusions relating to clinical practice that may have significant medicolegal connotations⁽¹⁴⁾.

Barutell et al. provided one of the clearest descriptions of permanent nerve damage following peripheral nerve blockade. During the performance of an interscalene brachial plexus block, the patient suffered a sharp paresthesia on needle insertion. The paresthesia became worse on initiation of the local anesthetic injection. In spite of this, the injection was continued, and was only terminated after the patient lost consciousness and developed respiratory fail-

ure. This report clearly demonstrates that, whichever technique is used, one should never continue injecting local anesthetic if a patient complains of severe pain⁽¹²⁾. This statement is supported by a recent prospective survey of serious complications related to regional anesthesia in France. Of 34 (out of 103,370 regional anesthetics) neurological complications, 21 were associated either with paresthesia during needle insertion ($n = 19$) or with pain during injection ($n = 2$), suggesting nerve trauma or an intraneural injection⁽²⁸⁾. Finally, Kaufman et al. report on seven patients with severe disability established at the time of a peripheral nerve block. In most of the cases, the injection was administered as a routine procedure by an experienced anesthesiologist. All the patients experienced significant discomfort during the block procedure and described their blocks as “unbelievable”, “extremely painful”, the most painful experience of my life”, “more painful than anything I have ever experienced before”, and “the most excruciating pain I have ever experienced”. The patient histories suggest that the condition, which can be resistant to all treatment, in most cases could have been avoided if careful attention had been given to the occurrence of pain during the nerve block. The authors conclude that it is likely that the risk of devastating iatrogenic disability can be minimized if a few basic principles are respected during the administration of peripheral nerve blocks⁽²⁹⁾.

As with nerve damage, the literature does not support a clear-cut advantage of one technique over the other with respect to successful peripheral nerve blockade⁽³⁰⁾. In a retrospective study Horlocker et al. report an increased success rate of the paresthesia technique (90%) over the use of the nerve stimulator (83%) as do McClain et al. (82 vs 75%) and Schroeder et al. (95 vs 88%)^(7,31,32). On the other hand, Smith and Allison reported a higher success rate with the nerve stimulator than with the paresthesia technique during sciatic nerve blockade⁽³³⁾, as does Raj et al⁽³⁴⁾. Extremely high success rates have been reported with the nerve stimulator by Vester-Andersen et al. (98%), Franco et al. (97%), and Tetzlaff et al. (94%)⁽³⁵⁻³⁷⁾. Franco and Vieira report successful subclavian perivascular brachial plexus blocks with a PNS in 97.2% (973/1,001) of patients at a center where this block was originally described by Winnie and Collins⁽³⁸⁾ and traditionally performed using the paresthesias technique⁽³⁹⁾. Kahn and Urquhart however, report a lower success rate (67%) with the nerve stimulator (compared with a transarterial approach) for axillary blocks⁽⁴⁰⁾. Baranowski and Pither concluded that the success rate of axillary blocks was determined by the number of nerves detected rather than whether paresthesia or a nerve stimulator was used⁽⁴¹⁾. The strongest support for the use of a PNS comes from a recent prospective study, by Sia et al. Using a multiple-injection axillary block technique, the authors compared nerve stimulation with

the paresthesias technique. The use of the PNS resulted in a significantly shorter time to perform the block, onset time of the primary block, time to achieve readiness for surgery and total anesthetic time. In addition, the incidence of complete block was greater in the PNS group (91% vs 76%; $p < 0.05$). The frequency of venous puncture was larger in the paresthesias group⁽⁴²⁾.

The issue has become even more confusing following a study by Urmey and Stanton who reported they were unable to consistently elicit a motor response following sensory paresthesia during interscalene block administration. Twenty interscalene block patients were prospectively studied using the paresthesia technique of Winnie with a 22-gauge, 3.8 cm (1½ inch) needle. In 10 patients a short-beveled insulated needle was used (Stimex B-D) and in the following 10 patients a long-beveled non-insulated needle (B-D) was used. Immediately following the report of a paresthesia and prior to local anesthetic injection, the nerve stimulator power was turned on and amperage slowly increased from zero mA to a maximum of 1.0 mA. Presence and location of an upper extremity motor response, if any, were recorded. Following this, 50 ml mepivacaine 1.5% with epinephrine was injected. All 30 patients had easily elicited paresthesias, 22 to the shoulder, 6 to the arm, and 2 to the hand. Only 9 of 30 patients (30%) had visible or reported motor response. All blocks had good evidence of sensory and motor blockade. No patient required general anesthesia. The authors conclude that this study demonstrated that evidence of a sensory response (paresthesia), presumably due to nerve contact, was not associated with the ability to elicit a motor response in 70% of patients, despite stimulation at mAmps that exceed the minimum accepted by most anesthesiologists. Conversely, they conclude that this study provided evidence that, if patients are under general anesthesia (or overly sedated), a lack of motor response does not guarantee that contact with a nerve fascicle has not occurred. Reservations regarding the performance of regional anesthesia in adult patients under general anesthesia have been expressed by others^(14,43,44).

In contrast to the study by Urmey and Stanton, Choyce et al. elicited a motor response after obtaining paresthesia with currents of 0.5 mA in the majority of patients (77%) during axillary brachial plexus blockade⁽⁴⁵⁾. A possible explanation for the discrepant results between the two studies is that the interscalene brachial plexus block may be a poor model to study the relationship of paresthesia and nerve stimulation. Blocking single nerves, such as individual nerves of the lower extremity, would be a more appropriate model than a plexus block. For instance, in a series of 100 patients undergoing sciatic nerve block, Davies and McGlade found that a nerve stimulator elicited a motor re-

sponse in 95%, whereas paresthesia was elicited in only 44% of patients (Table I).

In a multi-institutional study, the authors attempted to determine the relationship of low-current nerve stimulation to paresthesia during interscalene brachial plexus blockade. Low current peripheral nerve stimulation was used to localize the brachial plexus in 64 consecutive patients undergoing shoulder or arm surgery under an interscalene block. Once a motor response was obtained, the patients were queried regarding any sensation or pain localized to the neck, shoulder, or limb. Sensory distribution of the block and motor strength of the arm muscles were tested postoperatively to assess the extent of blockade. Ninety-five percent of the blocks provided successful surgical anesthesia. None of the patients spontaneously reported classical paresthesia during needle advancement

or nerve stimulation. However, upon careful questioning after motor response was obtained, the majority of patients (55%) reported painless sensations traveling down to their hands or fingers. Most patients (71%) spontaneously reported mild paresthesia on injection of local anesthetic. The authors concluded that classical paresthesia is absent or minimal when a low current nerve stimulation technique (≤ 0.6 mA; 100 μ sec, 2 Hz) and slow needle advancement technique are used to localize the brachial plexus during an interscalene block. However, if carefully questioned, more than half of patients may report a nonspecific, painless sensory sensation in the distribution of the brachial plexus. In addition, most patients report a mild radiating paresthesia during the injection of the first few ml of local anesthetic. Finally, low-current intensity nerve localization allows a high success rate of

Table I. Peripheral nerve stimulator vs paresthesia: Pros and cons.

| PNS | Paresthesia |
|--|--|
| Objective endpoint Continuous feedback facilitates and hastens performance of the block Minimal patient cooperation required therefore anxious patients can be appropriately sedated | Subjective "All-or-none" response Patient must be: •awake •cooperative •able to respond reliably Can be an unpleasant experience |
| Minimal discomfort Useful when paresthesia may be unreliable, e.g.: presence of a language barrier, the mentally impaired, extreme anxiety, conditions such as uremia, diabetes | |

Table II. Requirements for the successful use of a Peripheral Nerve Stimulator.

The success rate can be maximized and the complication rate minimized when using the nerve stimulator provided the following requirements are met:

- (a) Short-bevel, insulated needles should be used as well as an appropriate peripheral nerve stimulator (Table III). In an insulated needle the current output is limited to the tip of the needle instead of along the entire needle shaft. This facilitates needle placement⁽⁵¹⁾.
- (b) The low-output negative terminal of the stimulator should be connected to the needle ("negative to needle to nerve") as a significantly greater current is required for stimulation when the needle is connected to the positive terminal⁽⁵²⁾. This increases the risk of unintentional neural contact.
- (c) The initial current strength need never be greater than 1.5 mAmps.
- (d) An appropriate motor response should be sought, e.g. a biceps twitch (musculocutaneous nerve) is not an appropriate response when performing an axillary brachial plexus block).
- (e) The objective is to achieve an appropriate motor response at/below 0.3 mAmps (Table IV).
- (f) Prior to performance of the block, the patient should be sedated to comfort, but never over-sedated.
- (g) Once the correct position of the needle has been confirmed, gentle aspiration should be followed by the injection of 1 ml of local anesthetic, followed again by gentle aspiration. The rest of the dose should be injected slowly, interrupted by frequent (every 3-5 ml) gentle aspirations throughout.

Table III. Peripheral nerve stimulators: desirable characteristics.

| |
|---|
| 1. Constant current output |
| 2. Clear meter reading to 0.1 mAmps (digital display preferable) |
| 3. Variable output control |
| 4. Linear output |
| 5. Clearly marked polarity of stimulator terminals |
| 6. Short pulse width (50 – 1.00 µsec) |
| 7. Pulse of 1 Hz (1 pulse per second) |
| 8. Battery indicator |
| 9. High and low output (neuromuscular block monitor vs peripheral nerve location) |
| 10. High quality alligator-type clips (so that good electrical contact is made with the stimulating needle) |
| The needle should serve as the cathode (i.e. "negative to needle to nerve") |
| Check equipment prior to use (linear and constant response). |

Table IV. Peripheral nerve stimulator - Success rates.

| Current strength | Success rate (%) |
|------------------|------------------|
| (mAmps) | ISB/AxB |
| 0.6-1.0 | 36/25 |
| 0.5 | 82/80 |
| 0.4 | 84/81 |
| 0.1-0.3 | 93/92 |

Bernstein RL, Rosenberg AD. *Orthopedic Anesthesia and Related Pain Syndromes*. 1993:p.174.

the interscalene block without undue discomfort to the patient.

Are there disadvantages to the use of a nerve stimulator? Cost is certainly a factor that should be taken into account. The use of a multifunctional peripheral nerve stimulator (i.e. high output for twitch monitor, low output for nerve blockade) will limit the additional cost of purchasing a nerve stimulator specifically for peripheral nerve blockade (Table II). These stimulators are sturdy and have a prolonged lifespan. While not ideal, short-bevel uninsulated needles may be substituted for insulated needles. It has been suggested that an additional pair of hands is required when using a nerve stimulator technique⁽⁴⁶⁾. This author totally disagrees with this argument. It is in fact easier and faster to perform a block with a nerve stimulator without the aid of an assistant as the constant back and forth communication is eliminated, and the operator can concentrate on the more important task of communicating with the patient⁽⁴⁷⁾.

For those preferring to keep both hands "on the field", a device, in which a foot pedal is used to control the current, has been described⁽⁴⁸⁾. With respect to requiring an extra pair

of hands to inject the local anesthetic, this requirement applies to either technique.

Based on the above, a review of the literature does not support one technique over the other with respect to rate of successful blockade or incidence of postoperative neuropathies. This author, however, supports the use of a peripheral nerve stimulator for the following reasons:

a) The incidence of nerve damage is more closely related to the skill and experience of the individual performing the block than whether a paresthesia or non-paresthesia technique is used.

b) Use of the nerve stimulator does not guarantee that elicitation of paresthesia will be avoided or that nerve damage can be completely avoided. Equipment failure, lack of a "gentle" technique, and other factors may come into play^(49,50).

c) The likelihood of an inadvertent intraneural injection is reduced by performing nerve blocks in (adult) patients who are not anesthetized or heavily sedated.

d) While the success rate and the complication rate might be similar (in experienced hands), performing a peripheral nerve block with a nerve stimulator has advantages other than the possible reduction in nerve damage. These include:

(I) Less patient cooperation is required, i.e. less dependence is placed on the patient's subjective report of a paresthesia compared with the objective endpoint of an appropriate muscle twitch.

(II) Continuous feedback permits adjustments in needle positioning which may hasten performance of the block.

(III) Nerve blocks can be performed in patients in whom communication may be difficult (e.g. language barrier, mentally challenged).

(IV) Patients can be sedated to the point at which they are comfortable but awake enough to report severe paresthesia or pain on injection. This level of sedation may however interfere with the reporting of "clinical" paresthesia.

(V) Paresthesia can be (and frequently is) an unpleasant experience, whereas use of the nerve stimulator very seldom is. Patient discomfort is therefore minimized

(VI) In pediatric patients the risk benefit ratio favors performance of regional anesthesia after induction of general anesthesia.

(e) Scrupulous documentation of all paresthesias elicited (by design or unintentionally during a nerve stimulator technique), as well as the presence or absence of pain during the injection, is essential. This recommendation is strongly supported by Horlocker et al. and Auroy et al. who reported that all cases of persistent paresthesia after spinal, epidural, or peripheral nerve blockade occurred in the same topography as the associated paresthesia^(7,28).

REFERENCES

1. Caplan R, Posner K, Cheney F. Perioperative ulnar neuropathy: are we ready for short cuts? *Anesthesiology* 1994;81:1321-3.
2. Warner M, Warner M, Martin J. Ulnar neuropathy: incidence, outcome and risk factors in sedated or anesthetized patients. *Anesthesiology* 1994;81:1332-40.
3. Stoelting R. Nerve injury during anesthesia. *ASA Newsletter* 1994;58:6-9.
4. Kroll D, Caplan R, Posner K, et al. Nerve injury associated with anesthesia. *Anesthesiology* 1990;73:202-7.
5. Kroll D. What does closed claims review tell us about regional anesthesia. *Am Soc Reg Anesth* 1992:221-3.
6. Thompson G, Liu A. Perioperative nerve injury. In: Benumof J, Saidman L, eds. *Anesthesia and Perioperative Complications* St. Louis: Mosby-Year Book, Inc., 1992:160-72.
7. Horlocker T, Kufner R, Bishop A, et al. The risk of persistent paresthesia is not increased with axillary plexus block. *Anesth Analg* 1999;88:382-7.
8. Selander D, Dhunér K-G, Lundborg G. Peripheral nerve injury due to injection needles used for regional anesthesia: an experimental study of the acute effects of needle point trauma. *Acta Anaesth Scand* 1977;21:182-8.
9. Selander D, Edshage S, Wolff T. Paresthesiae or no paresthesiae? Nerve lesions after axillary blocks. *Acta Anaesth Scand* 1979;23:27-33.
10. Selander D. Axillary plexus block: Paresthetic or perivascular. *Anesthesiology* 1987;66:726-8.
11. Partridge B, Katz J, Benirschke K. Functional anatomy of the brachial plexus sheath: implications for anesthesia. *Anesthesiology* 1987;66:743-7.
12. Barutell C, Vidal F, Raich M, Montero A. A neurological complication following interscalene brachial plexus block. *Anaesthesia* 1980;35:365-7.
13. Passannante A. Spinal anesthesia and permanent neurologic deficit after interscalene block. *Anesth Analg* 1996;82:873-4.
14. Moore D, Mulroy M, Thompson G. Peripheral nerve damage and regional anaesthesia (editorial). *Brit J Anaesth* 1994;73:435-6.
15. Rice A, McMahon S. Peripheral nerve injury caused by injection needles used in regional anesthesia: influence of bevel configuration, studied in a rat model. *Brit J Anaesth* 1992;69:433-8.
16. Selander D. Peripheral nerve injury caused by injection needles (letter). *Anaesthesia* 1993;48:323-4.
17. Rice A, McMahon S. Peripheral nerve damage and regional anaesthesia (letter). *Brit J Anaesth* 1993;70:527.
18. Selander D, Brattsand R, Lundborg G, et al. Local anesthetics: importance of mode of application, concentration and adrenaline for the appearance of nerve lesions. An experimental study of axonal degeneration and barrier damage after intrafascicular injection or topical application of bupivacaine (marcain). *Acta Anaesth Scand* 1979;23:127-36.
19. Myers R, Heckman H. Effects of local anesthesia on nerve blood flow: studies using lidocaine with and without epinephrine. *Anesthesiology* 1989;71:757-62.
20. Partridge B. The effects of local anesthetics and epinephrine on rat sciatic nerve blood flow. *Anesthesiology* 1991;75:243-51.
21. Gentili F, Hudson A, Hunter D, Kline D. Nerve injection injury with local anesthetic agents: a light and electron microscopic, fluorescent microscopic, and horseradish peroxidase study. *Neurosurg* 1980;6:263-71.
22. Plevak D, Linstromberg J, Danielson D. Paresthesia vs nonparesthesia - the axillary block. *Anesthesiology* 1983;59:A216.
23. Urban M, Urquhart B. Evaluation of brachial plexus anesthesia for upper extremity surgery. *Reg Anesth* 1994;19:175-82.
24. Winchell S, Wolfe R. The incidence of neuropathy following upper extremity nerve blocks. *Reg Anesth* 1985;10:12-5.
25. Chambers W. Peripheral nerve damage and regional anesthesia (editorial). *Brit J Anaesth* 1992;69:429-30.
26. Moore D. Supraclavicular approach for block of the brachial plexus. In: Moore D, ed. *Regional Anesthesia* Springfield, Illinois: Charles C. Thomas, 1953:221-42.
27. Gentili M, Wagnier J. Peripheral nerve damage and regional anaesthesia (letter). *Brit J Anaesth* 1993;70:594.
28. Auroy Y, Narchi P, Messiah A, et al. Serious complications related to regional anesthesia: results of a prospective survey in France. *Anesthesiology* 1997;87:479-86.
29. Kaufman BR, Nystrom E, Nath S, et al. Debilitating chronic pain syndromes after presumed intraneural injections. *Pain* 2000;85:283-6.
30. Goldberg M, Gregg C, Larjani G, et al. A comparison of three methods of axillary approach to brachial plexus blockade for upper extremity surgery. *Anesthesiology* 1987;66:814-6.
31. McClain D, Finucane B. Interscalene approach to the brachial plexus: parasthesiae versus nerve stimulation. *Reg Anesth* 1987;12:80-3.
32. Schroeder L, Horlocker T, Schroeder D. The efficacy of axillary block for surgical procedures about the elbow. *Anesth Analg* 1996;83:747-51.
33. Smith B, Allison A. Use of a low-power nerve stimulator during sciatic nerve block. *Anaesthesia* 1987;42:296-8.
34. Raj P, Montgomery S, Nettles D, Jenkins M. Infraclavicular brachial plexus block: a new approach. *Anesth Analg* 1973;52:897-904.
35. Vester-Andersen T, Christiansen C, Hansen A, et al. Interscalene brachial plexus block: area of analgesia, complications and blood concentrations of local anesthetics. *Acta Anaesth Scand* 1981;25:81-4.
36. Franco C, Veira Z, Winnie A. The subclavian perivascular block performed with a nerve stimulator. *Int Mon Reg Anesth* 1998;10:45.
37. Tetzlaff J, Yoon H, Brems J. Interscalene brachial plexus block for shoulder surgery. *Reg Anesth* 1994;19:339-43.
38. Winnie A, Collins V. The subclavian perivascular technique of brachial plexus anesthesia. *Anesthesiology* 1964;25:353-63.
39. Franco CD, Vieira ZE. 1,001 subclavian perivascular brachial plexus blocks: success with a nerve stimulator. *Reg Anesth Pain Med* 2000;25:41-6.
40. Kahn R, Urquhart B. Transarterial technique is superior to nerve stimulator for axillary block (abstract). *Reg Anesth* 1997;22:21.
41. Baranowski A, Pither C. A comparison of three methods of axillary brachial plexus anaesthesia. *Anaesthesia* 1990;45:362-5.
42. Sia S, Bartoli M, Lepri A et al. Multiple-injection axillary brachial plexus block: A comparison of two methods of nerve localization-nerve stimulation versus paresthesia. *Anesth Analg* 2000;91:647-51.
43. Moore D. "No paresthesias-no anesthesia", the nerve stimulator or neither? (letter). *Reg Anesth* 1997;22:388-90.
44. Benumof JL. Permanent loss of cervical spinal cord function associated with interscalene block performed under general anesthesia. *Anesthesiology* 2000;93:1541-4.
45. Choyce A, Chan VW, Middleton WJ, et al. What is the relationship between paresthesia and nerve stimulation for axillary brachial plexus block? *Reg Anesth Pain Med* 2001;26:100-4.
46. Phillips W. An argument against the use of the nerve stimulator for peripheral nerve blocks. *Reg Anesth* 1992;17:309-10.
47. Brown A. Unassisted peripheral nerve blocks (letter). *Reg Anesth* 1993;18:137-8.
48. Hadzic A, Vloka J. Peripheral nerve stimulator for unassisted nerve blockade. *Anesthesiology* 1996;84:1528-9.
49. Urmey W, Stanton J, O'Brien S, et al. Inability to consistently elicit a motor response following sensory paresthesia during interscalene block administration. *Reg Anesth Pain Manag* 1998;23:7.
50. Stark R. Neurologic injury from axillary block anesthesia. *J Hand Surgery* 1996;21A:391-6.
51. Ford D, Pither C, Raj P. Comparison of insulated and uninsulated needles for locating peripheral nerves with a peripheral nerve stimulator. *Anesth Analg* 1984;63:925-8.
52. Tulchinsky A, Weller R, Rosenblum M, Gross J. Nerve stimulator polarity and brachial plexus block. *Anesth Analg* 1993;77:100-3.