Peripheral nerve damage and regional anaesthesia
CORRESPONDENCE

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Sir,—I wish to comment on the recent critical editorial on nerve injury during regional anaesthesia . I agree with the authors that it is probable that only a proportion of neural symptoms after regional anaesthesia occur as a direct result of the anaesthetic technique. However, there is a paucity of satisfactory epidemiological data with regard to the incidence of such symptoms and their aetiology. In their editorial, Moore, Mulroy and Thompson appear to confuse two different issues: the ability of various needle designs to provoke neural injury and the extent to which the provocation of paraesthesiae results in neurological symptoms after regional anaesthesia .

I wish to address the points raised in the editorial which concern needle design studies. I agree that there is a need for clinical data in this area, indeed the most recent article discussed this in detail . However, in the absence of adequate clinical investigations, it is not acceptable for Moore, Mulroy and Thompson to dismiss the evidence from carefully conducted animal studies as not being of relevance, merely because it does not fit their own hypotheses. Furthermore, these two studies differ considerably in their fundamental aims and therefore it is fallacious of Moore, Mulroy and Thompson to dismiss them as a single entity. The first study examined the ability of intraneurally, but extrafascicularly, positioned needles to enter nerve fascicles . In this respect, there is an advantage in the use of short–bevelled needles. In the second study , the needles were deliberately placed intrafascicularly in order to investigate the axonal consequences of accidental fascicular impalement; here there was an advantage in the use of long–bevelled needles and perhaps “pencil–point” needles . Moore, Mulroy and Thompson are clearly confused with respect to this important anatomical difference as they appear to believe that both of these studies investigated the consequences of intrafascicular penetration. Furthermore, the use of the emotive word “harpooning” to describe the techniques of lesioning sciatic nerve used in these two studies is not appropriate to a scientific review. Clinical studies, however challenging, need to be performed to confirm the optimal bevel design of regional anaesthesia needles and clinical recommendations cannot be made until such data are available. Nevertheless, such clinical studies need to be guided by the results of basic science studies .

Furthermore, contrary to the beliefs of Moore, Mulroy and Thompson, it is very simple to differentiate between extra– and intrafascicular needle placement by using a sophisticated nerve stimulator. Much smaller currents are needed to stimulate axons from an intrafascicular, as opposed to extrafascicular, electrode. However, it should be noted that the majority of nerve stimulators sold as being suitable for regional anaesthesia are not capable of reliably operating within the required range.

In addition, the incidence of neuropathy symptoms after intrafascicular injury of axons is known from studies of microneurography (when percutaneous electrodes are deliberately inserted into human nerve fascicles to obtain single axon recordings) . During the experiments the majority of subjects reporting paraesthesiae as nerve fascicles are impaled and 10% of subjects experience persistent paraesthesiae for a few days after the experiment, but serious complications are rare . The electrodes used in microneurography are slightly smaller than regional anaesthesia needles. However, it is known from animal studies that the needles used in regional anaesthesia produce a proportionately greater degree of axonal injury when fascicles are accidentally impaled .

Prospective, randomized, clinical studies investigating both needle design and the role of nerve stimulators in preventing regional anaesthesia–related neuropathies are required to complement existing animal data. However, the implication of the last sentence of Moore, Mulroy and Thompson “that we should refrain from publishing evidence from carefully conducted basic science studies until the clinical data are available, because medicolegal cases may be compromised” is clearly ludicrous.

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Sir,—The editorial on peripheral nerve damage and regional anaesthesia by Moore, Mulroy and Thompson is remarkable for several reasons. One expects a well designed and important message in an article with such a prominent position. Unfortunately, this editorial does not fulfil these expectations; instead it illustrates the authors' rather old–fashioned views and limited respect for basic science.

In short, the editorial contains the following messages:

(1) Animal data are of little value and should not be allowed to guide our clinical practice.

(2) Clinical adverse events whose frequency does not reach statistical significance do not happen and therefore need not be considered in clinical practice.

(3) Adverse events where patients are injured should not be reported in medical publications, as this could prejudice the outcome of court proceedings.

(4) Eliciting paresthesiae while performing neural block is not combined with any risk of neural injury.

(5) The “axiom” of Moore, Mulroy and Thompson: “No paresthesiae, no anaesthesia” is not outmoded.

These remarkable statements stand well for themselves, but some comments are needed.

(1) Animal experiments are often used in investigating mechanisms behind adverse effects of medical treatment. In many instances there are species–specific effects, for example because of enzymatic or immunological differences, which make direct application to humans uncertain. However, traumatic injuries and their consequences are less species–dependent, and nerve lesions caused by trauma by injection needles can be expected to be very similar between species. To disregard experimental results from anaesthetic practice may prove deleterious for both patient and doctor.

(2) The lack of a statistically significant relationship between active paresthesia and nerve lesions in the cited study does not mean that this relationship does not exist. It is more likely to be a consequence of insufficient statistical power. The report by Plevak, Linstromberg and Danielson arrived at the same values as Selander, Edshage and Wolff , but also did not reach statistical significance. In spite of this, the authors stated: “the higher incidence of neurological sequelae demonstrated in the PT (paresthesia technique) group in both studies, allows us to conclude that paresthesia should be avoided during axillary block.” This conclusion is supported by a meta–analysis, based on the results of both studies, in which the difference reached statistical significance ($P < 0.05$, Fisher's exact test).

(3) It is indeed unfortunate that the medicolegal climate in the United States results in such suggestions. It would be unethical not to inform our colleagues about possible risks and complications involved in medical treatment which may cause injury and suffering to our patients. Moore, Mulroy and Thompson, however, ignore their own recommendations when they publicize anecdotal information on six cases of neuropathy following the use of nerve stimulators in regional anaesthesia! .

(4) Paresthesiae can be elicited in various ways. A tap on the “funny bone” that results in an ulnar paresthesia does not normally result in a neuropathy. Bonica wrote in 1954: “While it is true that repeated and rough probing of nerves may cause neurological sequelae, gently touching the nerve with the needle point does not cause any clinically apparent damage” . I see no difficulty in accepting this statement, which agrees with our conclusion: “When performing a nerve block, paresthesiae should be elicited with the greatest care, or if possible avoided, in order to reduce the risk of nerve lesions” . A means of minimizing needle trauma in peripheral nerve blocking is to use a short–bevelled needle which, with its very low risk of penetrating nerve fascicles, offers “safer” paresthesiae . Neuropathies following the use of a nerve stimulator may be the result of misuse of the stimulator (too high energy level), an inability to differ between mechanically and electrically induced paresthesiae, or both.

(5) The “axiom” of Moore, Mulroy and Thompson has never been convincingly proven. Instead, there are several articles which do not show better success rates of axillary blocks performed with a paresthesia technique than without .

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References:

Sir,—Thank you for the opportunity to reply to the preceding correspondence which is relative to peripheral nerve damage and regional anaesthesia. Unfortunately, in attempting to defend their bench research, Rice and Selander have ignored the intent of the editorial. Insinuating that we were attempting to belittle their research and that of others is likewise “ludicrous” (Rice)—nothing could be further from the truth! Our own numerous investigations substantiate this statement.

Evidently, Rice agrees with the purpose of our editorial. He states, “Clinical studies, however challenging, need to be performed to confirm the optimal bevel design of regional anaesthesia needles and clinical

recommendations cannot be made until such data are available," and "it is very simple to differentiate between extra– and intrafascicular needle placement, by using a sophisticated nerve stimulator."

To conclude, Rice indicates he has the "tools" (microneurography) so that a major, clinically statistically significant investigation could be conducted. Therefore, Rice and Selander should collaborate to prove or disapprove their theories as to whether long, short, or pencil–point needles, and/or the nerve stimulator will avoid or reduce to a minimum neuropathy when performing peripheral nerve block. Until then, the point made in the conclusion of the editorial is valid, that is: "until a prospective, blinded, major clinical study provides us with statistically significant clinical information, we believe that authors should not draw conclusions relating to clinical practice and which may have significant medicolegal connotations."

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• PERIPHERAL NERVE INJURY CAUSED BY INJECTION NEEDLES
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Sir,—One reason for the study by Rice and McMahon seems to be to question an earlier experimental study on the acute effects of needle point trauma by Selander, Dhunér and Lundborg . When criticizing that study, Rice and McMahon unfortunately made inappropriate comparisons. They studied the amount and long-term pathology of injuries in the rat monofascicular sciatic nerve after penetrating it with long- (12?) or short- (27?) bevel injection needles, whereas Selander's group aimed primarily at the frequency and acute morphology of nerve fascicle lesions after piercing the rabbit multifascicular sciatic nerve with a long- (14?) or short- (45?) bevel needle.

The experiments by Selander, Dhunér and Lundborg showed that the nerve fascicles easily slid or rolled away from the needle point both in vivo and in vitro, especially when the 45? bevel was used. This led to a significantly smaller frequency of fascicle injury after piercing the nerve with the 45? or short-bevel (SB) compared with the 14? or long-bevel (LB) needle.

When a needle hits a nerve, paresthesiae are elicited and the (awake) patient normally reacts to this. The paresthesia informs the anaesthetist that the needle is in close contact with the nerve and that, if advanced further, the needle may injure the nerve, and it seems that most anaesthetists agree with this view . Selander, Edshage and Wolff and Plevak, Linstromberg and Danielsen found that the use of a paresthesia searching technique for axillary plexus blocks (with long-bevel needles) increased the frequency of post-anaesthetic nerve injuries compared with a non-paresthesia technique. The use of short-bevel needles may press or push the nerve away, thereby giving the patient and the informed anaesthetist more time to react to a paresthesia before the needle penetrates and possibly injures the nerve.

Rice and McMahon used a short-bevel needle with a bevel angle of only 27?. Their short-bevel needle was also 13% thicker than the 12? long-bevelled needle used. To injure the single fascicle of the rat sciatic nerve "the needle was introduced into the exposed nerve at mid thigh level at an angle of 45? and left undisturbed for 10 min". This would not be possible unless the animal or patient was deeply asleep! It apparently implies that each nerve was severely injured by the needle, and under such unconventional conditions it may not be surprising that the 27? needle caused more severe lesions than the 12?. However, as seen in table I of their paper, the acute lesion on day 1 was worst after the nerve was pierced with the transversely orientated long-bevel needle, as in Selander's study.

In the histological study, something termed "intraneural disruption" was assessed and "this was defined as the degree of disruption to the internal elements of the nerve and included such factors as the reaction at the site of injury, the gliosis... scored on a 0–5 scale..." (my italics). Other morphological changes studied were "evidence of axonal degeneration" and "evidence of disorganized regeneration of fibres", both without closer description and scored only on a present or absent basis—that is, 1–100% = yes; 0 = no. In order to create a global assessment of nerve fascicle injury (which had sufficient security to enable statistical analysis...) ..., "observations of epineural disruption" (my italics) were included, also without a word of definition. No microscopic pictures were included to illustrate these changes.

The statistical handling of these histopathological features was puzzling: "... a table of high and low score point allocation was constructed". The summed injury score was "allocated to the low score table if the

epineural or intraneural score was less than 2.5 or when no evidence of axonal degeneration or disorganized fibre regeneration was observed. The converse applied to point allocation to the high score group" (my italics). It seems as if this system makes a variety of interpretations and a high degree of bias possible.

In their Discussion, Rice and McMahon did not discuss the relevance and validity of their results. The authors seem not to understand the fundamental differences in study design and needle penetration techniques between their study and that of Selander, Dhunér and Lundborg. In the latter study, only a quick needle penetration of a multifasciculate nerve was made, to study the frequency of fascicular injury, whereas Rice and McMahon stated: "The rat sciatic nerve used in our study consists of only one fascicle at mid-thigh level and nerve (fascicle) penetration was ensured in all cases". How, then, is it possible to conclude that "The results of these experiments suggest that when nerve fascicles are impaled by commercially available injection needles (of similar diameter), lesions occur less frequently—if they are induced by an LB needle compared with an SB needle" (my italics). Obviously, all nerve fascicles were deliberately injured with the test needles!

The clinical significance of paresthesiae was overlooked also when Rice and McMahon could not confirm the recommendation of Selander, Dhunér and Lundborg to use a 45?-bevel needle in clinical anaesthesia. Instead, they suggested that "there may be grounds for recommending the use of LB needles, especially if the bevel is aligned parallel to the nerve fibres". This is probably true if one deliberately penetrates the nerve with the needle before injecting the local anaesthetic, but such a technique cannot be recommended, as an intraneural injection severely increases the risk of nerve injury .

Fortunately, nerve injuries caused by regional anaesthesia are rare, but even so, they may cause severe and long-lasting suffering for the unfortunate patient and perhaps also for the anaesthetist. It is naturally important that any recommendations concerning methods or techniques for clinical use are based on sufficient clinical experience and basic research.

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Sir,—The aim of our study was not to "question" the much-quoted study by Selander, Dhunér and Lundborg , but to complement it by examining different aspects of an animal model, over a time course more appropriate to the natural history of nerve injuries. As Selander points out, there are fundamental conceptual differences between the two papers. He examined the extent to which injury of the perineural covering of nerve fascicles was influenced by bevel design, over a very short period of time. We described the influence of needle bevel upon the severity and consequences of intrafascicular lesions, should accidental fascicular penetration occur. Selander implies that we were unaware of these differences or chose to ignore them. This is quite clearly not the case, as we were at pains to point out several times in our text. Indeed, the major part of the discussion was devoted to alerting the reader to these differences.

We fully agree with Selander that all possible steps, such as avoidance of paraesthetic techniques, should be taken to avoid fascicular injury. Our study examined the effects of bevel design upon the severity and duration course of intrafascicular lesions, following fascicular penetration. The ability of different bevels to penetrate nerve fascicles (or trunks), or to push them away, in the clinical situation of the non-surgically exposed nerve is, at present, unknown. We shall therefore refrain from making dogmatic recommendations until such evidence is available. However, in answer to Selander's question: we did not measure ease of fascicle penetration, but our impression was that it was, as would be expected, easier to enter the fascicles with the long-bevel needles; these commercially produced needles possess a secondary cutting bevel. We suggest that long- and medium-bevel needles without a cutting bevel (or even "pencil-point" tips) should be investigated as a compromise between the supposed relative difficulty of entry into a nerve fascicle with short-bevel needles and the severe lesions which we have shown to occur should fascicles become impaled with such needles.

The two needles we used were of a similar size and both designs are used commonly in the practice of regional anaesthesia. Our method of impaling the nerves has been used by others investigating mechanical nerve injury and is not dissimilar to that used by Selander in his study. In answer to his point regarding the angle of entry, this was 45? to the nerve. We decided to use this angle having examined the techniques of others investigating mechanical fascicular injury . Why did Selander's group choose 90? ?

We dispute Selander's postulate that fascicular penetration is possible only in deeply anaesthetized animals or humans. We have both undergone many fascicular penetrations of the ulnar nerve during the

course of microneurography recordings with a 30-g recording electrode. Fascicular penetration (as demonstrated by microneurographic recordings) is surprisingly difficult to predict on symptomatic grounds alone . Also, many anaesthetists perform regional anaesthesia in patients under general anaesthesia.

With regards to the comments on our summed nerve injury scores. The whole point of this exercise was to reduce the inevitable bias and observer error of single histological features. We displayed most of the individual histological data in table I, so that the reader might draw his own conclusions. The epineural scores were excluded to avoid repetition, as they were very similar to the intraneural scores; they have been published elsewhere . The sensitivity of the summed nerve injury scores was shown clearly, when the same technique was used to investigate the more minor injuries associated with microneurography .

Several representative photomicrographs were submitted with the original manuscript, as we also felt that the reader should be able to form a visual impression of the type of lesions being discussed. They were omitted from the published version at the suggestion of the editor.

Selander has misunderstood our use of the term “frequency”. Of course, all fascicles were impaled; this was the aim of the study. The term frequency was used to describe the incidence of observed lesions following fascicle injury.

In the discussion we stressed the various differences between the study by Selander, Dhunér and Lundborg and ours. We referred to this well known study because, despite the obvious fundamental difference from our paper, it is the only relevant publication in this field. We did not attempt to make any clinical recommendations from our results, but demonstrated that, should accidental fascicle penetration occur, the lesions observed following the use of short-bevel needles are more severe and take longer to repair than those from long-bevel needles. We stated that a great deal more basic and clinical work needs to be performed before definitive statements can be made regarding the design of needles for use in regional anaesthesia. We most certainly do not advocate the practice of intraneural injection! Clinical practice has already been changed once following a single, limited, animal study which has never been backed up by clinical research. We are reluctant to make further recommendations until a great deal more basic and clinical research has been performed in order to elucidate this issue.

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References:

• PERIPHERAL NERVE DAMAGE AND REGIONAL ANAESTHESIA CORRESPONDENCE

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Sir,—We thank Drs Baranowski and Buist for their interest in our paper and the accompanying editorial. For the record: nerve penetration occurred in vivo and nerves were not “isolated”, in contrast with a previous study ; also the needles were not “fine-bore”, but standard injection needles.

We examined the consequences of accidental penetration of nerve fascicles and demonstrated that, in this respect, long bevels were superior to short. We agree totally that all efforts should be made to prevent fascicle penetration, and hope all anaesthetists exercise “care” when performing regional anaesthesia, whatever their technique. However, as we stated, there are many other factors involved in needle-induced nerve injury—for example, the ability of different nerve needle bevels to penetrate nerve fascicles in clinical practice. As this area is supported at present only by the type of anecdotal evidence which Baranowski and Buist quote, definitive comment is precluded until substantive data are available. However, it is salutary to note that Sjöström and colleagues [personal communication, 1992] have used long-bevelled needles for more than 17 000 supraclavicular blocks, using a paresthesia technique. Eighty percent of their patients were followed up; three cases of serious nerve injury were apparent, but on further investigation they all were found to have resulted from coincidental (surgical) lesions. Dental surgeons have been advised for some years to use long bevelled needles .

We cannot be so certain as Baranowski and Buist of detecting nerve fascicle penetration. During microneurography in awake humans when paraesthesiae were elicited, in only a minority of cases was a fascicle impaled (as indicated by a neurographic recording). To be certain that fascicular penetration had occurred, it was necessary to elicit sensations by electrical stimulation at currents of less than 40 mA. The

nerve stimulators used in regional anaesthesia are not capable of operating reliably in this range, and are able merely to detect proximity to a nerve trunk .

The optimum bevel configuration of needles used in human regional anaesthesia remains to be ascertained.

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