"*I would have everie man write what he knowes and no more.*"—MONTAIGNE

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EDITORIAL

Peripheral nerve damage and regional anaesthesia

Publications in *British Journal of Anaesthesia* in 1992 and 1993 [1–7] observed that the relationship between peripheral nerve damage and regional anaesthesia is still controversial. Some authors have suggested that nerve damage results from eliciting paraesthesia or is dependent on the design of the bevel of the needle used for performing blocks. Others have suggested that neuropathy may be avoided by using a nerve stimulator. However, it is clear that there are no statistically significant *clinical* data to support the suggestion that neuropathy results from any of these factors. The contention that peripheral nerve damage may occur directly from regional block is based, we believe, largely on extrapolation from animal data.

In 1977, Selander published a thesis [8] based on two prospective clinical articles on nerve lesions after axillary blocks [9, 10] and three rabbit investigations [11–13]. In one of the clinical studies of 154 patients [9], the design involved avoidance of paraesthesia. However, "paraesthesiae were accidentally elicited in 54 patients (39%)... but no neurological sequelae were found". In the other clinical study which involved 533 patients, paraesthesiae were sought deliberately and obtained in 290 [10]. In the remaining 243 patients where paraesthesiae were "to be avoided", they occurred unintentionally in 94 (40%) patients [10]. Clinical evidence of nerve damage occurred in eight of the 290 patients (2.8%) and in two of the 243 patients (0.8%), but "no statistical difference was found in the frequency of nerve lesions between the two groups" [10]. Despite this finding, the article concluded "that whenever possible nerve blocks should be performed without searching for paraesthesiae". This conclusion overlooks the obvious fact that in this study they occurred even when not being sought [10]. Furthermore, in ending an editorial in 1987, Selander [14] reviewed only the same non-statistically significant *clinical evidence* obtained between 1977 and 1978 [9, 10] and concluded that one should "use a perivascular technique, preferably a catheter or a short-bevel needle, and try to avoid paraesthesiae". It is not clear if he was still defending his 1978 thesis and thereby perpetuating the views obtained by extrapolation from animal data.

It should be clear to anaesthetists that data obtained from rabbit [11–13, 15] and rat [1] investigations in which neuropathy results from "harpooning" sciatic nerves, may be markedly different from what occurs when eliciting paraesthesia in humans. In these "*in vitro*" and "*in vivo*" animal studies, the sciatic nerves were impaled so that the entire bevel of the needle was buried below the epineurium, that is they were intrafascicular (intraneural) (fig. 1). It is not possible for anaesthetists to verify that such intraneural needle placement occurs when paraesthesia is elicited in humans. Furthermore, in the field of regional anaesthesia, few would disagree with the statement of Scott and colleagues [16] that "animal studies should be accepted only as rough guides as to the situation in humans".

Of the articles published in the 1993 issues of *British Journal of Anaesthesia* [3–7], that of Gentili and Wargnier [5] requires further comment. These authors advocated the use of the nerve stimulator and obviously they prefer the axiom "no paraesthesiae no dysaesthesiae" to the outmoded "no paraesthesiae no anaesthesia" (axiom) of Moore. However, it would have been more accurate had Gentili and Wargnier stated "no paresthesiae no dyasesthesiae, but often failed anaesthesia?". The nerve stimulator has not been proved to be more reliable than techniques of finding the location of peripheral nerves using paraesthesiae and it has not

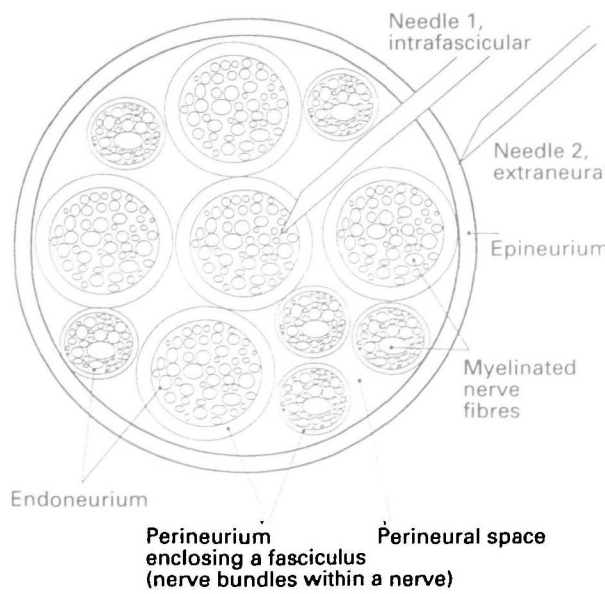

Figure 1 The cerebrospinal nerves contain numerous nerve fibres collected into bundles and enclosed in connective tissue sheaths; a small bundle is termed a *fasciculus*. Each fasciculus is surrounded by a connective tissue sheath, the *perineurium*. Individual nerve fibres are held together and supported by a connective tissue *endoneurium*, continuous with septa from the perineurium. A small nerve may be a single fasciculus, but nerves usually contain several fasciculi held together by connective tissue investment, the *epineurium*. (Reproduced from [17].)

been demonstrated clinically to be safer. We believe that they have exceeded what is scientifically valid in using the term "outmoded", that is, obsolete. It is unfortunate that they and other correspondents [1–4, 6, 7] may have undermined those many anaesthetists who may prefer to, or merely by chance, obtain paraesthesia. Lastly, in defence of the nerve stimulator, they stated "we believe that paraesthesiae increase the risk of this complication (nerve damage)..." [5]. However, it is unfortunately the case that using the nerve stimulator while attempting to locate a nerve, particularly in unconscious patients, has not avoided neuropathy. We have reviewed six medico–legal cases in which permanent brachial plexus neuropathy occurred and in which the nerve stimulator was used. Unfortunately, with this as with many other complications of anaesthetic techniques, lawyers frequently advise their clients not to report such instances in medical publications, in order to avoid prejudicing the outcome of court proceedings.

In conclusion, we suggest that there are no statistically significant clinical data to demonstrate that eliciting paraesthesia results in neuropathy. While all the theoretical studies [1–7, 14] are most interesting, it is doubtful if they influence anaesthetists who have mastered one technique of regional anaesthesia to change to another. We agree with Chambers [2] when he observed "There are immense practical difficulties involved in conducting a major clinical study of the effect of needle type and other factors which may be involved in the aetiology of nerve damage caused during regional anaesthesia". Therefore, 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 medico–legal connotations.

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