Morbidity Associated with Subdural Electrodes

To The Editor: We were interested in reading the paper by Wyler, et al., on the complications of long-term seizure recording using subdural strip electrodes (Wyler AR, Walker G, Somes G: The morbidity of long-term seizure monitoring using subdural strip electrodes. J Neurosurg 74:734–747, May, 1991). We commend the authors on carrying out this needed study and on their very low complication rate. We wish to comment on two aspects of the study, however.

We were surprised to learn of the degree of cerebrospinal fluid (CSF) leakage around the electrodes in this study, a problem which these authors managed with sterile dressing changes. We use these same electrodes in our patients, but control CSF leakage with a simple technique. We tunnel our electrode wires 6 to 8 cm away from the burr-hole incision using a No. 12 styled ventricular needle. While the needle is in place, one or two 2-0 nylon sutures are passed around the needle. After the electrode has been passed and the needle is withdrawn, the sutures are tied down tightly around the electrode, thereby sealing the track. A 3-0 nylon suture is then placed in a purse-string fashion at the exit site to further seal the skin around the electrode. Using this technique, we have had minimal or no CSF leakage around as many as 10 electrodes in a single patient.

We were also surprised at the choice of cefazolin as a prophylactic antibiotic for a neurosurgical procedure as it does not penetrate the central nervous system well. Although the infection rate was extremely low in this report, perhaps it could be reduced even further by using an antibiotic that penetrates the central nervous system.

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Reference


Response: We thank Drs. Ross and Henry for their thoughtful letter concerning our recent article. Their technique of tying down the electrode wires is excellent. Early in our series we used a very similar technique but had an electrode lead break at the suture. This required opening the burr-hole wound to retrieve the electrode. As a result, we have accepted the philosophy that we would rather have the electrode move accidentally than the electrode’s lead break. Their technique is effective in decreasing cerebrospinal fluid (CSF) leakage, but we have not found leakage to be a major problem, and our infection rate seems sufficiently low to support our bias.

With respect to the choice of antibiotics, they are quite right; cefazolin does not penetrate the central nervous system well. It is our supposition that infections are not due to direct contamination of the CSF but to a backward migration of infection from the skin. Therefore, we are more concerned with giving antibiotics that have a high concentration in the soft tissues leading to the intracranial space than we are about a high concentration of antibiotic in the CSF. Nonetheless, they raise good points, and we appreciate their thoughts.

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Thalamotomy Lesions

To The Editor: This is in reference to the recent article by Tomlinson, et al. (Tomlinson FH, Jack CR, Kelly PJ: Sequential magnetic resonance imaging following stereotactic radiofrequency ventralis lateralis thalamotomy. J Neurosurg 74:579–584, April, 1991). Among the clinical recommendations made at the end of the article for creating a single thalamotomy lesion is the statement that the 1.6-mm probe with an exposed tip length of 3 mm was heated to 78°C for 60 seconds following physiological localization. The lesions that this produced resulted in a good clinical response. It is stated in the article that those patients with a troublesome proximal component of an upper-extremity tremor were controlled by withdrawing the probe 3 mm and repeating the procedure, thus extending the lesion superiority. It should be stressed that, prior to any lesioning, neurophysiological testing should be done, including not only microelectrode recording as stated in the article but also microelectrode stimulation (monopolar and/or bipolar). Stimulation is important for localization and also for predicting areas where lesioning could cause potential deficits, such as speech alteration, paralysis, hypesthesia, and visual alterations, among others. It has been our experience that individualization of the target and of the lesion parameters is required for each thalamic stereotactic procedure.

There is no standard technique for localization and lesioning, nor is there a standard size for a lesion because it is not specific enough for tremor of the upper extremity. For example, hand representation is frequently found at about 15 to 16 mm off the midline and is a target for hand tremor. Face representation is usually medial to the hand, and foot representation is between the area of the hand and the internal capsule location. The internal capsule is usually found at 18 mm off the midline. Lesioning of this structure should certainly be avoided. Not infrequently, marked variations of the parameters occur from case to case. The width of the third ventricle and the placement of the burr hole, as well as the frame angles of the stereotactic probe trajectories, also cause variation in the planning.
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of the procedure. The authors state that, in their study, the target area is the ventralis oralis posterior nucleus (VOP). We prefer to do the microrecording and microstimulation in the contralateral ventralis caudalis nucleus (VC) for sensory mapping, followed by exploration and lesioning at the ventralis intermedius nucleus (VIM) which lies immediately in front of the VC nucleus and just behind the VOP nuclei. The lesion can be made 2 to 3 mm anterior to the sensory representation of either the face, hand, and/or foot, which initially is located in the VC.

A standard Zone 2 lesion, as per this article, had a mean diameter of 7.3 mm (range 3.8 to 10 mm). Such variation is likely to include the VIM as a target; with a volume of 10 mm it could include, as well, representation of the face, hand, and possibly even the internal capsule and sensory thalamus. Although it is mentioned in the article that no permanent complications were noted in any of these patients, it would be of interest to know what kind of complications were encountered in light of the size of the lesions made.

In this study, the use of ventriculography is discussed. Years ago, we stopped using ventriculography because of its potential dangers to the patient. Most stereotactic frames are now compatible not only with computerized tomography but also with magnetic resonance imaging. The latter study is used for stereotactic planning as well as for identification and localization of the third ventricle in the anterior commissure (AC) and the posterior commissure (PC) and the AC-PC plane. This decreases the risk of complications to the patient. It should also be stated that radiological resolution at this time is still not precise enough to identify the demarcation of the different nuclei within the thalamus.

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RESPONSE: The purpose of our article was to document the magnetic resonance (MR) imaging appearance and temporal evolution of radiofrequency ventralis lateralis (VL) thalamotomy lesions made in the treatment of movement disorders. It was not intended as an extensive description of the thalamotomy method: a complex subject in itself. Significant individual spatial anatomical variability in the localization of various subcortical targets in relation to radiologically established landmarks necessitates elaborate neurophysiological corroboration in every neuroablative procedure performed for movement disorders. Readers interested in such technical details are referred to a series of prior publications.1-6 Briefly, the homuncular arrangement in the ventralis posterior nucleus (VP) defined by semicroelectrode recording can be used to determine the proper laterality of somatotopically specific lesions in the ventralis oralis posterior/ventralis intermedius nuclei (VOP/VIM). As an aside, considering the marked variation in the width of the third ventricle and axial configuration of the thalamus between individuals, measurements made from the midline as suggested by the author of the above letter are meaningless. However, before becoming too dogmatic on where lesions should be placed in the treatment of movement disorders, we should bear in mind that there are considerable differences of opinion among very experienced neurosurgeons on the "ideal anatomical" lesion site. Lesions made in various positions within VL, medial globus pallidus, and pallidofugal fibers have been reported to abolish tremor in 80% to 93% of patients.7

It is clear that lesions should be of sufficient size to permanently control tremor. Lesions of insufficient size are associated with tremor recurrence, even though tremor may have been abolished at surgery.9 Furthermore, the fact that no permanent complications were encountered in the group of patients reported in our paper indicates that the lesions were not too large, as the author of the letter attempts to imply. Even though the average diameter of the Zone 2 lesion was 7.3 mm in the early phase, late-phase lesions had contracted to an average of 5 mm in Zone 2. This is consistent with inferred lesion diameters resulting from so-called "selective VIM thalamotomy."8 I sincerely doubt that tremor could be permanently controlled by smaller lesions. Even so, I disagree with the comment that a lesion can be made 2 to 3 mm anterior to the region in which somatosensory evoked responses are obtained. In some cases, this can result in contralateral sensory loss and possibly painful dysesthesias; variability in the anterior/posterior position of the neurophysiologically defined anterior border of the VP is also significant.6

The necessity for positive-contrast stereotactic ventriculography is a matter of personal preference. Transient problems with new isotonic positive-contrast agents are rare and the risk of permanent complications negligible. I derive considerable security from precise localization of the anterior and posterior commissures and the intercommissural line, which defines within 1 mm the inferior aspect of the thalamus.3 The height (Z) coordinate of the final lesion is dependent on accurate localization of the inferior aspect of the thalamus and is critical for an effective result.

Computerized tomography (CT) and MR imaging do provide useful information on the axial configuration of the third ventricle and thalamus, and localization of the thalamocapsular boundary. However, one must be aware that field strength distortion can occur with stereotactic MR image localization. This can vary from day to day and differ between various MR imaging units. Field strength distortion at the periphery of the image (where stereotactic fiducial markers are located) can introduce significant errors in calculated target coordinates. Admittedly, no distortion occurs on CT scans, but we have had occasional problems identifying the commissures. In my opinion, positive-contrast ventriculography is the "gold standard" in neuroradiological localization for functional stereotactic surgery, and
I am not yet willing to dispense with the information that this examination provides.

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Intramedullary Schwannoma

To The Editor: I enjoyed the recent case presented by Herregodts, et al. (Herregodts P, Vloemberghs M, Schmeding E, et al: Solitary dorsal intramedullary schwannoma. Case report. J Neurosurg 74:816-820, May, 1991), which describes the second solitary intramedullary schwannoma demonstrated by gadolinium-enhanced magnetic resonance imaging (the first was published by Gorman, et al. in 1989). Unfortunately, the authors’ review of the literature is flawed by multiple errors, both of commission and omission, which should be brought to light since these tend to be perpetuated in the literature.

Although their Table I includes 36 cases, the quoted reports of Penfield (1932), Rasmussen, et al. (1940), and Sloom, et al. (1964), correspond to a single case encountered at the Mayo Clinic and reported by these different authors in a number of publications on various topics. Similarly, the case reported by Guidetti (1967) is the same reported in 1982 by Cantore, et al., as Case 1. Furthermore, the report of Rout, et al. (1983), contains only one case and not two, as presented in the table. The two cases referred to by Lang and Bridge (1959) should not be included either, since the information originally published is insufficient, and the data included in the table are not necessarily accurate. In addition, Van Duijn’s case (1971) is of a 24-year-old woman, not a man as recorded. Finally, the cases reported by Schmitt (1975) and Pardatscher, et al. (1979), are not solitary schwannomas and their intramedullary location is, at best, debatable. Both obviously do not belong to that table.

Errors of omission also are present. The review failed to include the previously reported cases presented in my Table 1.13-5,8-10,11 The cases reported by Prakash, et al.,8 Aryanpur and Long1 and Solomon, et al.,10 are included because, from an anatomical and developmental perspective, their location in the pons and medulla brings them closer to the spinal intramedullary schwannomas than to those intra-axial schwannomas described in the cerebral or cerebellar hemispheres.1 In summary, there are 37 cases of intramedullary schwannoma reported to date. Of these, 18 occurred in males and 19 in females, contrary to the 2:1 ratio reported by Herregodts, et al.

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Table 1

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Location of Lesion</th>
<th>Duration of Symptoms</th>
<th>Degree of Removal</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prakash et al., 1980</td>
<td>14, F</td>
<td>pontomedullary</td>
<td>3 yrs</td>
<td>subtotal</td>
<td>static</td>
<td></td>
</tr>
<tr>
<td>Kang &amp; Song, 1983</td>
<td>47, M</td>
<td>C4-5</td>
<td>1 yr</td>
<td>total</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>Young et al., 1983</td>
<td>33, F</td>
<td>T-1-11-conus</td>
<td>3 yrs</td>
<td>total</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>González et al., 1985</td>
<td>29, F</td>
<td>C2-5</td>
<td>1 yr</td>
<td>total</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>Drapkin et al., 1985</td>
<td>30, F</td>
<td>C3-5</td>
<td>3 yrs</td>
<td>total</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>Solomon et al., 1987</td>
<td>69, M</td>
<td>medulla-C3</td>
<td>4 yrs</td>
<td>total</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Aryanpur &amp; Long, 1988</td>
<td>50, F</td>
<td>medulla</td>
<td>6 wks</td>
<td>total</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>Herrmann et al., 1988</td>
<td>51, M</td>
<td>C2-T1</td>
<td>?</td>
<td>subtotal</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Marchese &amp; McDonald, 1990</td>
<td>72, F</td>
<td>C4-6</td>
<td>6 yrs</td>
<td>subtotal</td>
<td>improved</td>
<td></td>
</tr>
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