Measurement of tissue impedance in conjunction with computed tomography-guided stereotaxic biopsies

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SUMMARY Twenty-two patients undergoing CT-guided stereotaxic biopsies had intraoperative monopolar and bipolar impedance monitoring along the trajectory of the biopsy. Patterns of tissue impedance were retrospectively correlated with CT scans demonstrating decreased impedance generally corresponding to low density regions and increased impedance to the enhancing lesions.

The development of computed tomography-guided stereotaxic biopsy systems has allowed neurosurgeons to obtain tissue samples from deep-seated and crucially situated intracranial lesions. In most systems, the biopsy probe is directed to an intracranial target based upon predeterminated stereotaxic coordinates. With any tumours, more than one biopsy is necessary to obtain representative tissue samples, requiring either movement of the patient and/or additional probe passes with resultant increased risk or inconvenience. Precise knowledge of the structures through which the biopsy probe passes would allow selective tissue samples from different areas within the same tumour, obviating this problem and allowing biopsies to be performed more safely. One potentially useful method is continuous monitoring of tissue impedance. The electrical impedance in tissue is the total opposition to current flow and is measurement of the ratio of the voltage to the resulting current that is generated in the tissue. Ohm’s law for calculating the resistance with an alternating current is Z (impedance) = E (voltage in volts)/I (current in amperes). In a physiological state, this is dependent upon the fluid, myelin, glial and neuronal densities, electrode tip exposure and electrolyte concentrations present in the tissue.

Since 1921, attempts have been made to use impedance recording to locate brain tumours during free hand biopsies. In 1923, Grant was able to demonstrate, in 30 formalin-fixed cadaveric brains using an early impedance monitor, that the impedance in gliomas was approximately ½–½ of that found in normal brain. Investigators have since shown correlation between tissue impedance and anatomic localization in monkey brains, in the human basal ganglia, and in clinically determining cord penetration during percutaneous cordotomies. When used in conjunction with free hand biopsies in an attempt to locate brain tumours, intraoperative impedance recording has not been consistently useful. However, in none of these studies was CT scanning available to compare with the impedance patterns. With CT-guided stereotaxic biopsies, localisation of the tumour has become less of a problem than with prior techniques. Rather, selection of optimal biopsy points, adequate tissue sampling, and safety of probe trajectory are now major factors. Our prospective study was designed to evaluate intraoperative impedance monitoring during CT-guided stereotaxic biopsies in solving these problems. In this paper, we report our experience in 22 patients.

Materials and methods

Stereotaxic procedures were performed on patients with deep, surgically inaccessible lesions, lesions in exquisite areas of the brain, or in patients where a preliminary biopsy diagnosis would influence the final surgical procedure. Fourteen of the patients were males and eight were females. Eleven patients had glioblastoma multiformes (GBM), three anaplastic astrocytomas, two astrocytomas, one a malignant glioma, one an oligodendroglioma, one an infarction, one an ependymal cyst, one an arachnoid cyst, and one a primary CNS lymphoma (table 1).

All patients were biopsied utilising a modified Gildenberg procedure in a Todd-Wells stereotaxic frame or using a Brown-Roberts-Wells frame. Prior to biopsy, a Radionics (Radionics, Inc, Burlington, Mass) thermister electrode was inserted to target. Both the monopolar electrode, with a tip length of 0·0 mm and a tip diameter of 1·6 mm, and the
bipolar electrode, with a tip length of 4-0 mm and a tip diameter of 1-1 mm, were insulated and of stainless steel. A Radionics (RFG-3B) radiofrequency lesion generator system provided impedance monitoring. Using a 50 kHz measuring frequency, the RFG-3B drives a sine wave constant current relative to the unknown impedance at the tissue-electrode interface. The resulting voltage is rectified to produce a measurable DC signal proportional to tissue impedance (personal communication Dr Eric Cosman). This value was recorded at points along the probe’s trajectory from the cortex to target. Tissue impedance was recorded from the cortex to target. Four general areas were specifically recorded: the cortex, the subcortical white matter, the area adjacent to the target, and the target point itself. For all patients, the biopsy was done via a burr hole where the dura could be sharply opened and the cortex exposed for visual inspection. In eight patients, a monopolar electrode, a bipolar electrode, and the biopsy catheter were passed sequentially. In five cases, biopsies were performed both at target and along the trajectory path.

Following accumulation of the impedance patterns, the preoperative CT scans were reviewed to determine if any correlation existed between patterns of impedance and the associated intracranial structures through which the probe passed. CT patterns were generally categorised as demonstrating a ring enhancing lesion (REL), an enhancing lesion with surrounding low density (ELLD), a low density lesion (LDL), or a low density lesion with an enhancing nodule (EN).

Results

The results are summarised in tables 1 and 2.

### Impedance Measurements

**Cortex:** Measurements of monopolar impedance ranged from 150–700 ohms at the cortex. Of the 32 measurements made, the mean value was 400-5 ohms with a standard deviation of 168.8 ohms and a median value 350 ohms. In seven cases, more than one recording was made on the same patient. In each of these, the passage of the electrode was through the same cortical opening and in all cases the impedance reading at the cortex was higher on later measurements.

**White matter** Twenty-eight measurements were made of impedance in the subcortical white matter. The values ranged from 150–800 ohms. The mean value was 440-3 ohms with a standard deviation of 197.2 ohms and a median value of 425 ohms. In 13/20 cases, the impedance in the white matter was higher than in the cortex on the first catheter passage. In seven cases, more than one catheter passage occurred. In 2/7, the impedance on the first passage was greater than that on the second passage, while in 4/7 the impedance reading was higher on the second passage.

### Table 1  Computed tomography monopolar impedances (ohms)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Location</th>
<th>Pathology</th>
<th>Lesion</th>
<th>Biopsy site</th>
<th>Cortex</th>
<th>White matter</th>
<th>Adjacent to target</th>
<th>Target</th>
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<tbody>
<tr>
<td>1</td>
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<td>GBM</td>
<td>LDL with EN</td>
<td>EN</td>
<td>450</td>
<td>600</td>
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<td>400</td>
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<tr>
<td>2</td>
<td>Basal ganglia</td>
<td>GBM</td>
<td>Multiple REL</td>
<td>RE</td>
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<td>500</td>
<td>600</td>
<td>500</td>
</tr>
<tr>
<td>3</td>
<td>Parietal</td>
<td>GBM</td>
<td>REL</td>
<td>RE</td>
<td>250</td>
<td>275</td>
<td>250</td>
<td>250</td>
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<tr>
<td>4</td>
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<td>Lymphoma</td>
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<td>200</td>
<td>150</td>
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<tr>
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<td>250</td>
<td>350</td>
<td>300</td>
</tr>
<tr>
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<td>Parietal</td>
<td>Astrocytoma</td>
<td>LDL with EN</td>
<td>LD</td>
<td>200</td>
<td>300</td>
<td>175-250</td>
<td>200</td>
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<tr>
<td>7</td>
<td>Basal ganglia</td>
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<td>ELLD</td>
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<td>200</td>
<td>100</td>
<td>150</td>
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<td>225</td>
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<td>300</td>
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<td>Malignant glioma</td>
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<td>RE</td>
<td>350</td>
<td>200</td>
<td>—</td>
<td>100</td>
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<td>10</td>
<td>Frontal</td>
<td>GBM</td>
<td>REL</td>
<td>RE</td>
<td>425</td>
<td>—</td>
<td>—</td>
<td>300</td>
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<td>Astrocytoma</td>
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<td>LD</td>
<td>200</td>
<td>—</td>
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<td>100</td>
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<td>—</td>
<td>—</td>
<td>300</td>
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<td>500</td>
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<tr>
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<td>700</td>
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<tr>
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<td>475</td>
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<td>475</td>
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<td>LDL with calcium glioma</td>
<td>CA +</td>
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<td>600</td>
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<tr>
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<td>Quadrigeminal cistern</td>
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<td>LDL</td>
<td>LD</td>
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<td>725</td>
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</table>
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In one case, the readings were the same.

**Area adjacent to target** There were 26 impedance recordings. In general, a change in impedance was noted 1–2 cm prior to reaching the target. The mean value for these measurements was 399.0 ohms with a standard deviation of 207.9 ohms and a median value of 350 ohms. In 17/25 recordings, there was a decrease in impedance from the subcortical white matter to the region adjacent to biopsy. In all of these cases, a low density region in white matter was seen adjacent to the target on CT scan. In the eight cases where the impedance rose, five examples were in the basal ganglia or thalamus. In these cases, it was hypothesised that the trajectory of the probe would pass through the dense fibre tracts in their trajectory toward target, with this causing the resulting rise in impedance. In one case, the lesion biopsied had virtually no surrounding low density and the change in impedance was felt to correspond to the entrance into the lesion itself. In two cases, the changes in impedance were only 25 and 50 ohms, respectively.

In many patients, a more complex pattern of change emerged with multiple changes occurring as the target point was approached. This was especially true of ring enhancing lesions and lesions in the basal ganglia.

**Target** There were 31 recordings at the predetermined target site. These ranged from 100–800 ohms. The mean value was 380.5 ohms with a standard deviation of 183.9 ohms and a median value of 350 ohms. Compared to subcortical white matter, 17 biopsy targets had lower, three had equivalent, and eight had higher impedance levels. In comparison to the area adjacent to the target, 11 measurements showed a decrease, 11 showed an increase, and four were unchanged. Of these last four, two were cysts, one an infarct, and one was white matter adjacent to tumour.

**Comparison with CT pattern**

Certain patterns emerged from this study when impedance results were compared with the CT scan. As mentioned above, a general pattern of decreased impedance was seen in oedematous white matter with an increase noted at the approximate point of an enhancing rim or nodular area of enhancement. If a central low density area was entered, the impedance level would then again fall. Of six ring enhancing lesions, four had a pattern of decreased impedance in the surrounding low density region, increased impedance in the enhancing rim, and decreased impedance in the central low density. In one case of a ring enhancing lesion in the basal ganglia, the reverse was seen and in one patient (patient 17) where multiple biopsies were performed, the pattern varied according to the site biopsied. In six patients with ELLD on CT scan, five had a decreased impedance corresponding to the low density region followed by an increase in impedance at the enhancing target. In one case (patient 4) of a lymphoma in the basal ganglia, the reverse was seen. There were six low density lesions. Two of these had enhancing nodules and one had an area of calcification. In the two with nodules, one had a decreased impedance in the low density area with an increase at the nodule, while the second had a decrease in the low density region with a further decrease at the nodule. In this last case (patient 1), the enhancing portion had a significant amount of necrosis. In the low density lesion with calcification (patient 21), the impedance in the low density lesion was decreased in comparison to surrounding white matter, while the impedance at the point of calcification was further decreased. In three low density lesions, an infarct and two cysts, the area adjacent to target and at target was essentially the same.

**Comparison of Impedance values with histologic pattern**

In four cases of ring enhancing lesions, biopsies were performed both at the predetermined target and at an additional area along the trajectory of the needle. In all cases, there was a pattern of increased impedance 1–2 cm before target and decreased impedance at target. In all cases, the target point with the decreased impedance represented predominantly necrotic tissue, while the area of increased impedance represented viable tumour. The respective values were 300–310, 100–200, 150–200, and 115–300 ohms. In one patient (patient 17), the decreased impedance noted once oedematous white matter was entered was maintained until target where only white matter was seen. On a subsequent biopsy, a pattern of increased impedance followed by decreased impedance was seen adjacent to target with viable tumour and necrosis seen respectively. In one patient with an ELLD lesion, biopsy at the target point was an anaplastic astrocytoma, while biopsy 1 cm away had an impedance of 125 ohms and proved to be oedematous white matter.

**Comparison of monopolar and bipolar measurements**

In eight patients, both monopolar and bipolar measurements were made (table 2). In terms of both absolute values and patterns of impedance, a poor correlation was seen between the two systems. When initial attempts were made to correlate patterns of impedance and CT scans, it was subjectively felt that monopolar measurements were more beneficial.

**Illustrative cases**

**Patient 6** A 68 year old, Caucasian male developed adult onset seizures. These were characterised by
speech arrest, focal twitching of the right upper extremity followed by generalised movement and loss of consciousness. A CT scan demonstrated a mixed density lesion in the left parietal region (fig 1). At the time of surgery, the left parietal lesion was approached through a left frontal burr hole. In approaching the first biopsy site, which represented the enhancing portion at the edge of the low density lesion (labelled A), the monopolar impedance was 200 ohms at the cortex and 300 in the white matter. There was a rapid change to 175 ohms 2 cm from target which abruptly increased to 250 ohms 1·5 cm from target, then dropped to 200 ohms at target. The bipolar electrode was then passed and had a pattern of 350 ohms at the cortex, 325 ohms in the white matter with a gradual decrease to 300 ohms until 2 cm from target when there was an abrupt decrease to 250 ohms which increased to 325 ohms at target. The biopsy catheter was then inserted to target and four quadrant samples taken which was felt to be a low grade astrocytoma on frozen section. The biopsy catheter was then withdrawn 2 cm where the fluctuating impedance had first been noted. A second biopsy was performed with the return of bright red blood. The catheter was rapidly withdrawn. The patient was observed for 20 minutes with no decline in neurologic status. In transit to computed tomography, however, he acutely declined and a CT scan was performed, demonstrating a large haemorrhage. The patient was then taken to surgery where the bleeding site was found to correspond to a sulcal vessel.

Patient 7 A 58 year old male presented with progressive hemiparesis. On CT scan, an enhancing lesion with a small central low density area and surrounding low density were seen in the right basal ganglion (fig 2). The impedance at the cortex was 350, at the white matter it became 300 with gradual drift down to the 175–150 range until 1 cm from target when there was an abrupt increase to 225. Biopsy at that point was consistent with a GBM.

Patient 16 A 57 year old female had a subacute history of memory loss and episodic confusion. A CT scan was performed demonstrating a low density lesion in the left frontal area (fig 3A). Subsequently, these episodes became more frequent and a second CT scan was performed which demonstrated a pre-

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**Table 2 Comparison of monopolar and bipolar impedance parameters**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Computed lesion</th>
<th>Tomography target 1</th>
<th>Cortex</th>
<th>White matter</th>
<th>Adjacent to target</th>
<th>Target</th>
</tr>
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<tbody>
<tr>
<td>3 REL</td>
<td>RE</td>
<td>MP 250</td>
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<td>250</td>
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<tr>
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<td>200/225</td>
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<td>5</td>
<td></td>
<td>BP 250</td>
<td>300</td>
<td>275</td>
<td>300</td>
<td>275</td>
</tr>
<tr>
<td>6 LD with EN</td>
<td>LD</td>
<td>MP 200</td>
<td>300</td>
<td>175–250</td>
<td>400</td>
<td>300</td>
</tr>
<tr>
<td>7 ELLD</td>
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<td>BP 250</td>
<td>200</td>
<td>100</td>
<td>175</td>
<td>175</td>
</tr>
<tr>
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<td>RE</td>
<td>BP 350</td>
<td>325–300</td>
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</tr>
<tr>
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<td>BP 425</td>
<td>300</td>
<td>150–175</td>
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<td>225</td>
</tr>
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<td>11 LDL</td>
<td>LD</td>
<td>BP 200</td>
<td>200</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Fig 1 Patient 6. An enhanced CT scan demonstrating a mixed density lesion in the left parietal region.
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Fig 2  Patient 7. An enhanced CT scan demonstrating an enhancing lesion with a small central low density area and surrounding low density.

Previously unvisualised smaller lesion in the ipsilateral sylvian fissure region. A MR scan was then performed showing a well circumscribed left frontal lesion separate from the ventricle (fig 3B). At the time of biopsy, the monopolar electrode impedances were 500 ohms at the cortex, 550 in the white matter with an abrupt change to 450 ohms 2 cm from target, maintaining the same reading until target. Initial aspiration returned 3 cc of clear fluid suggestive of CSF. Following this, no further fluid was returned and four quadrant biopsies were performed. On touch preparation, columnar epithelium suggestive of ependyma was seen. Initial frozen sections, however, revealed only normal white matter. A second target spot 5 mm lateral to the first but within the central low density of the lesion was selected. The impedance reading at the cortex was 600 ohms which increased to 675 in the white matter. Approximately 2 cm from target, offscale infinite impedance suddenly developed which was maintained until target was reached. This was felt to represent entrance into an air-filled cystic cavity. An unenhanced CT scan performed postoperatively demonstrated a cystic cavity separate from the ventricle with an air-fluid level present (fig 3C). The final pathology was consistent with an ependymal cyst.

Patient 17  A 68 year old Caucasian male with a history of prior dementia secondary to multiple vascular infarctions and subsequent progressive hemiparesis. A CT scan was performed demonstrating a large ring enhancing lesion in the right parietooccipital area (Figure 4). The first biopsy was at the anteriormost edge of the ring enhancing lesion (Target B). Impedance monitoring was 600 ohms at the cortex with an increase to 700 in the white matter. This decreased to 675 ohms within 2 cm of the cortex and maintained this level until target. A biopsy was performed and demonstrated only normal white matter. Target C, an

Fig 3  Patient 16. (A) An enhanced CT scan demonstrating a low density lesion in the left frontal area. (B) A magnetic resonance scan showing a well circumscribed left frontal lesion separate from the ventricle. (C) A postbiopsy unenhanced CT scan demonstrating an air-fluid filled cystic cavity.
Patient 17. A CT scan demonstrating a large ring enhancing lesion in the right parietooccipital area. (A) The first target biopsied. (B) Second point biopsied. The impedance levels are outlined in the text.

Anteromedial portion of the enhancing rim of the mass, was biopsied next. The monopolar impedances were 700 ohms at the cortex, 800 ohms in the white matter, 850 ohms 1 cm from target, and 750 ohms at target. Necrotic material was seen on frozen section. The catheter was withdrawn 1 cm from target at the level of highest impedance. Tissue was consistent with an anaplastic astrocytoma.

Patient 21 A 34 year old Caucasian male developed focal seizures at 30 years of age. A CT scan demonstrated a low density lesion in the left frontal region with central calcification. The patient was followed clinically on anticonvulsants and was stable until 2 months prior to biopsy when he developed increasing seizure frequency despite the addition of a second and third anticonvulsant. A repeat CT scan was unchanged (fig 5). The first biopsy was of the anterior low density portion of the lesion. The monopolar cortical impedance was 350 ohms, at the white matter it was 475 ohms, becoming 400 ohms 1 cm from target and 350 ohms at target. A biopsy was suggestive but not diagnostic of an oligodendroglioma. A second target point corresponding to the central calcification was selected. The monopolar impedance was 390 ohms at the cortex, 520 ohms at the white matter, 600 ohms 2 cm from target with an abrupt drop to 420 ohms at target. Frozen section demonstrated calcification and cellular change diagnostic of an oligodendroglioma.

Patient 22 A 61 year old Caucasian male with history of severe depression admitted for ECT. Two years previously, he had been shunted at another hospital for supposed normal pressure hydrocephalus. On CT scan, a large quadrigeminal region low density lesion was noted in addition to markedly enlarged lateral and third ventricles. Metrizamide was injected by
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radiograph demonstrated the electrode to be at target. A fenestrated ventricular catheter with a 20 spinal needle as stylette was inserted to the point of resistance. The spinal needle was then inserted forward with a return of clear CSF and the catheter advanced over the tip. Metrizamide was injected through the needle, demonstrating a biloculated cystic structure (fig 6B).

Discussion

The purpose of this study was to determine if intraoperative impedance monitoring would be of benefit in selecting target points for intraoperative biopsy and in allowing the surgeon a better idea of the exact location of his biopsy probe at any time during the procedure. From our limited data, monopolar impedance monitoring is useful for both purposes. However, the value of the impedance pattern is more important than the absolute values obtained. Although this study is retrospective and has utilised crude reconstruction of the pathway of the probe, the patterns of impedance seen with various intracranial lesions appear to be reasonably consistent. In order to interpret the impedance reading intraoperatively, it is crucial that a clear idea of the potential structures through which the electrode may pass be carefully kept in mind. In this way, the patterns of impedance appear to be useful in interpreting when an enhancing lesion, an area of low density, a cystic structure, a calcified lesion or the ventricle is entered. Of equal importance, a lack of change in the impedance can be suggestive that a particular target has not been reached. This is especially true in mixed density or ring enhancing lesions where the enhancing rim generally represents viable tumour, and the central low density is necrosis. Selection of an appropriate biopsy site in these lesions is crucial.

In general, oedematous white matter will demonstrate a decrease in impedance when compared to subcortical white matter. As a specific lesion is then entered, the impedance will either further decrease, as is seen with cystic and many low density lesions, or will increase, as is seen in different types of enhancing lesions. With ring enhancing lesions, where adequate tissue sampling may require biopsies to demonstrate both necrosis and viable tumour, impedance monitoring allows a single needle pass to selectively sample different portions of the lesion. Usually, if the pattern of decreased impedance in the peritumour oedematous white matter followed by an increase at the point of entrance into the enhancing rim is not seen, as in patient 17, this suggests that the enhancing edge has been tangentially missed. If the low density centre of a ring enhancing lesion is the selected target, or if it is inadvertently entered during biopsy, the gen-
eral pattern has been for a terminal decrease in the impedance level. Additionally, ongoing impedance monitoring appears to be useful in calcified and other mixed density lesions and with patient 22, continuous impedance monitoring may allow the precise course of a catheter to be known and guided, maintaining the desired trajectory through a structure such as the ventricle. It cannot be overemphasised, however, that interpretation of the impedance pattern is based upon a clearly defined idea of the potential structures through which the probe may be passing. Misinterpretation of impedance patterns can result in serious errors. In retrospect, an awareness of the significance of the rapid changes in impedance seen with the passage of the probe through a sulcus in patient 6, might have avoided the intraoperative haemorrhage that occurred.

In general, impedance readings appeared to correspond to density changes noted on CT scans. Organ et al evaluated the impedance levels in 14 patients with intracranial tumours. In 13/14 patients, the tumours could be localised by a change in impedance. In nine of the patients, the tumour was noted to have an abnormally low impedance, while in four there was noted to be an abnormally high impedance. The authors noted a close correlation between the physical characteristics of the tissue and the impedance levels. The nine tumours with low impedances were produced by physically soft tumours, while the four tumours with high impedances were all meningiomas. They also noted transient high impedance levels in the capsules and pseudocapsules of the soft tumours. These initial observations were supported by Becker et al who recorded the impedance with both bipolar and monopolar electrodes in five patients with intracranial tumours, noting reduced impedance in three primary astrocytic tumours and in a soft meningothelial meningioma. In three patients with intracerebral haematomas, normal impedance levels were seen. Subsequent work by Broggi and Frauzini has also noted impedance changes in relation to apparent differences in tissue type.

Organ et al suggested that decreased impedance was related in certain instances to enlarged interstitial spaces. From our data, it appears that decreased impedance is associated with decreased density as seen on CT scan. Examples of this are the decreased impedance of oedematous white matter compared to normal white matter and the more obvious increase then decrease in impedance seen as the probe goes from an enhancing rim into a low density area. The sensitivity of these changes and the exact mechanism are not known. The nature of our data which consist of a retrospective interpretation of impedance patterns based upon the patterns of density seen on CT scan preclude a more precise analysis. We are currently attempting to plan prospectively our trajectories in such a way that angled coronal and sagittal CT reconstructions can be directly compared with impedance patterns intraoperatively.

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