A practical technique for monitoring extradural pressure

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The authors discuss the technical problems involved in the continuous measurement of intracranial pressure, and describe a simple, inexpensive extradural strain gauge system that can be manufactured by most research units. The system has the merits of coplanar construction, a ready zero reference, and simplicity in design and use. The theoretical limits of the extradural method are presented and discussed in relation to transducer design.

Key Words • intracranial pressure • continuous monitoring • extradural pressure • coplanar construction

This paper describes the construction and use of a simple, cheap, balanced strain-gauge system implanted extradurally. We have used this system in the experimental laboratory for 7 years and in the clinic for 6 years in the assessment of cases of communicating hydrocephalus.

Material and Method

Transducer Construction

The transducer used in our patients was developed from the original used in intracranial pressure (ICP) measurement in cats by Corne and Stephens,9 modified for human use by Corne, et al.,4 and later by Dorsch, et al.7 Its present form is that of a small, cylindrical block of epoxy resin with a diameter of 12 mm, which fits easily into a standard 12 mm burr hole. The height of 13 mm is a few millimeters more than the thickness of most skulls. Projecting from one end is a small loop of stainless steel, which serves as a handle to facilitate introduction and removal (Fig. 1).

The dural face of the transducer is hollowed out into a small, air-filled chamber, which communicates with the outside atmosphere by a narrow polyethylene tube. Covering this chamber is a flexible membrane of thin latex rubber, which closes the sensing face of the transducer. The chamber contains a flat metal sensing arm, with one end embedded in a side wall of the chamber and the other end free approximately in the center. This arm bears two double metal foil strain gauges, one on each face (Fig. 2). Electrical connections between the gauges are made within the epoxy body, forming a full resistive bridge configuration; four insulated wires emerge to the outside with the polyethylene tube. Pressure applied to the sensing face of the transducer is actually
measured by slight deformation of the free end of this sensing arm, causing changes in the resistance of the gauges.

An essential feature of the construction is that the tip of the sensing arm be made exactly coplanar with the outer edge of the epoxy body, neither projecting beyond nor recessed inwards. This construction will eliminate the effect of tension in the dura mater.

Electrical and other characteristics of the transducer, summarized in Table 1, are perfectly adequate for clinical or experimental use, especially with the availability of a method of baseline check.

Use of the Transducer

A standard 12 mm precoronal burr hole is made on the dominant side; the other side is thus available for ventricular puncture for calibration or treatment. The hole should be carefully made at right angles to the plane of the skull, so that the transducer when inserted can be truly coplanar with the inner surface of

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**Fig. 1.** Photograph of two completed transducers.

**Fig. 2.** A diagram of sensing face (upper) and vertical section (lower) of a transducer for use in adults.
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TABLE 1

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Measurement</th>
</tr>
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<tbody>
<tr>
<td>transducer size</td>
<td>diam. 12 mm, height 13 mm (human), height 7 mm (animal)</td>
</tr>
<tr>
<td>gauge resistance</td>
<td>120 ohms</td>
</tr>
<tr>
<td>gauge factor</td>
<td>approximately 2.05</td>
</tr>
<tr>
<td>electrical configuration</td>
<td>four active arm bridge</td>
</tr>
<tr>
<td>recommended excitation</td>
<td>1 to 2 V</td>
</tr>
<tr>
<td>sensitivity at 1 V input</td>
<td>3.5 to 5 μV output per mm Hg applied pressure</td>
</tr>
</tbody>
</table>

Tested full-scale deflection (FSD) at applied pressure of 200 mm Hg:
- overload capability: > 500 mm Hg
  - in vitro: < 1 mm Hg/day
  - in vivo: <10 mm Hg/day
- baseline drift: with temperature drift <0.2 mm Hg/°C
- sensitivity drift: spontaneous <0.75%; with temperature <0.3%/°C
- linearity: ±0.75% FSD
- hysteresis: ±0.75% FSD
- frequency response: flat to 60 Hz
- resonant frequency: > 80 Hz
- membrane strength to external inflation: > 150 mm Hg above applied pressure

the bone. Extradural hemostasis is made perfect, and the depth of bone measured. The transducer is then implanted to the same depth and cemented in place with acrylic resin. The four wires and the polyethylene zeroing tube are brought out through the skin incision, which is sutured in two layers over the transducer in the usual way. The polyethylene tube, free of kinks, is left in the head bandage with its end open and unimpeded. The four wires are connected to a recorder and to the energizing supply, which should be 1 to 2 V.

To check the baseline or zero position of the recording, a small volume of air is injected along the polyethylene tube; usually less than 0.25 ml is needed. The recorder trace is observed during this maneuver, and injection is stopped as soon as pulsations cease on the trace. At this point the transducer membrane and dura mater and intracranial contents have been lifted away from the tip of the sensing arm, which is thus lying free within the chamber surrounded by air at the same pressure, and therefore registering zero pressure. Zero checks can be made as often as necessary. However, the recording usually becomes stable within 1 day, and thereafter the check is only necessary once or twice daily.

Removal of the device at the end of the period of study requires a second small operation, which can be done under either local or general anesthetic.

The transducer has been used a total of 28 times on 27 patients, without serious complication. One patient who had an implant lasting 42 days developed mild scalp cellulitis after removal of the device, but it cleared quickly with antibiotic treatment.

The gauge used in animal experiments is basically the same, modified for the thinner skull. It is 8 mm in height, with no projecting handle, and as such would be suitable for use in infants and small children. It has been used extensively in our laboratory work with good results; accuracy was maintained in one animal during a 9-month period of implantation. Implantation and operation are exactly the same as in the device for humans.

Discussion

Episodic measurement of CSF pressure has been used in neurological and neurosurgical practice since the introduction of lumbar puncture by Corning in 1855. In recent years, however, the value of direct and continuous measurement of intracranial pressure has been recognized in a variety of clinical neurosurgical situations. It has been used during the induction of anesthesia in tumor cases, in the early postoperative period as a monitor of potential hemorrhage or spreading edema, and in the management of severe head injuries. There have been suggestions that intracranial pressure rises may give warning of hemorrhage in cases of intracranial aneurysm, and we ourselves have reported its usefulness in the differentiation between communicating hydrocephalus and cerebral atrophy. Further experiences with this last problem are reported in the accompanying paper. In the animal laboratory, also, measurements of intracranial pressure have become important in the assessment of blood flow changes following cerebral infarction or following the establishment of focal expanding intracranial lesions.

There are advantages and disadvantages in every available technique for measurement of intracranial pressure. Fluid-filled manom-
eters connected directly to cerebrospinal fluid spaces have certain disadvantages, including the possibility of infection, damping in a fluid-filled column, and the difficulty of maintaining patent connections; all of these problems are increased in demented, semiconscious, or restless patients.

The simplicity of our system allows for substitution of a mechanical or electrical manometer placed externally and connected by a fluid system to the CSF spaces. The risks of infection are less, but they still remain. Also, although this is the system that has been used with great success by the pioneers Guillaume and Janny and Lundberg, the disadvantages of blockage of the small catheter and of possible fluid leakage remain. In addition, the external manometer's position needs to be changed to maintain some arbitrarily chosen baseline relationship to the changing position of the patient.

Transducers implanted in the skull have the obvious advantages of direct measurement of the pressure, so that changes in the patient's position have much less effect; a variety of devices has been designed for either extradural or subdural implantation. The extradural device is particularly attractive because the intact dura mater forms an excellent barrier against potential infection and also prevents leakage of CSF, which tends to get into the mechanism of subdural transducers however firmly they may be en-cased. We believe that extradural pressure measurement can be perfectly accurate if proper precautions in construction and insertion are observed.

Objections to the use of extradural methods of measuring ICP are based on the interposition of the tough, inelastic dura mater between a measuring device and the source of pressure and its supposed adverse effect on pressure measurement due to reduced sensitivity and errors related to changes in dural tension with changes in ICP. It can be shown that this need not be the case, either in theory or practice.

A vector quantity, such as a force, is known to have a zero component at right angles to its line of action (Fig. 3). A force of magnitude \( x \) acting in direction \( OX \) will have a component along a line \( OR \) at an angle \( \theta \) to that line equal to \( x \cos \theta \). At right angles to this line, along \( OY \) or \( OY' \), \( \cos \theta \) and thus the component of \( x \) is equal to zero. This principle is used in the construction of our pressure transducer, in which the tip of the sensing arm is made exactly coplanar with the surface of the sensing face of the device. Similarly, during insertion of the transducer, care is taken to keep this face coplanar with the inner surface of the skull bone.

During use, then, the plane of the dura and the transducer membrane is at right angles to the line of action of the ICP, which acts directly outwards against the tip of the sensing arm. Tension in the dura mater may change with changes in intracranial pressure, but forces due to such change will have zero component in the direction of force due to the ICP. Figure 4 demonstrates the effect of coplanar construction. Where \( D \) is the force due to dural tension and \( P \) is that due to intracavitary pressure, \( D \) will have no component at right angles to the dura. Where the gauge is indenting the dura inward, the angle of \( D \) will be altered so it will have a resulting force along the line of action of the force \( P \).
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With coplanar construction the two forces are at right angles, and the resulting component is zero.

It is true in theory that the outward deformation of the sensing arm caused by the intracranial pressure being measured will permit outward expansion of the dura mater and transducer membrane so that it is no longer coplanar. However, the actual deflection of the sensing arm tip has been calculated to be very small, approximately 30 μm with a rise in applied pressure of 100 mm Hg, and thus any loss of coplanarity is negligible. In one of our earlier transducers the sensing arm projected beyond the face of the transducer. The effect of not using coplanar construction appears in the resulting recording (Fig. 5). Even at zero CSF pressure, the device still registers pressure due to residual sensing arm deformation by the dura, with an error of 5 mm Hg or more. In fact, in some cases, the graph of deflection against pressure was not linear, with an apparently increasing sensitivity at higher pressure due to the increase in dural tension as ICP rose. By contrast, with coplanar construction and insertion, the graph of extradural pressure against cisternal pressure was perfectly linear and had a zero intercept (Fig. 6). The “coplanar principle” in construction has been used for measurement of pressure from outside the organ wall, with intraocular, intrauterine, and intraarterial pressures, and was first adapted for measurement of intracranial pressure by Schettini, et al.

When intracranial pressures have been measured simultaneously by the extradural method and by means of CSF pressure, the results have often differed. In nearly all of these reports, extradural pressure was higher than CSF pressure, and usually the divergence became greater at higher pressure levels. Differences of 30 mm Hg in humans, and 20 mm Hg in animals, have been reported. Differences have been quite variable even within the same series. Thus, in one of seven cases reported by Jørgensen and Riisved, the two pressures were essentially the same over a range of 20 to 115 mm Hg, while in another case a difference of 15 mm Hg was noted at a ventricular pressure of 125 mm Hg.

One explanation for these differences is a lack of coplanarity in construction or insertion of the extradural device, as in the use of fluid-filled bags that naturally indent the dura. Similar objections apply to the use of perforated metal capsules with a fluid connection as well as to other devices which are slipped under the edge of a burr hole into the extradural space. Even a properly constructed coplanar transducer may have a similar effect if it is inserted at an angle to the plane of the bone, or projects excessively beyond the inner table.

Another explanation concerns possible damage to the brain during the making of the burr hole or insertion of the transducer. The resulting local edema would soon cause a rise in the local brain tissue pressure; this pressure may be transmitted to the transducer im-

Fig. 5. Graph showing calibration of extradural pressure against cisternal CSF pressure in a baboon (see text).

Fig. 6. Graph showing comparison of extradural (Y) against cisternal (X) CSF pressure in a baboon (see text). Comparison shows 95% confidence limits.
planted immediately over this area but not involve a large enough volume of brain to affect the mean ICP as measured by CSF pressure.

As discussed extensively by Majors, et al.,28 an extradural device inserted only far enough to flatten the dura over its face will not necessarily obliterate the subarachnoid space underneath. The pressure then measured will be that of the CSF in that area, and with free communication it will equal the general CSF pressure. On the other hand, when the insertion is far enough to flatten the underlying brain surface past the "pial breakpoint," the tissue pressure in the underlying brain will be the factor measured; this will be raised, at least temporarily, by the act of flattening the brain surface, even in the absence of local trauma or edema.

The level at which the CSF pressure transducer is placed is also important. For valid comparison, it must be placed at the same level as the extradural transducer and not at one of the normal reference levels, such as the foramen of Monro. Any difference in transducer level introduces a constant discrepancy between the pressures recorded in the two techniques (Fig. 7).

Apart from the question of trauma during insertion, in human patients natural pathology such as tumor or postoperative edema, may be present in brain near the site where the transducer is inserted. Under these circumstances, with the local subarachnoid space obliterated by swollen brain, the pressure can be transmitted to the general cranial cavity for equalization only via the brain substance; since the brain is a partly elastic, partly plastic material,11 a definite period of time will be needed for this to occur, often with shifts of brain between compartments. With progressive or continuing pathology, as with increasing edema, equalization might never occur.

In the animal experiments referred to above and in our own animal studies, pressure has normally been raised by means of induced hypercapnia. In such a situation CSF pressure rises almost entirely secondarily to increased brain tissue pressure caused by increased cerebral blood volume. When brain swelling is produced in this way, the subarachnoid space beneath the transducer may be obliterated. The transducer will then no longer sense purely diffuse intracranial pressure, but will sense local brain tissue pressure beyond the pial breakpoint. The tendency of brain to bulge when the pia is surgically incised provides a realistic illustration of the tensile strength of this membrane.

As shown by Schettini, et al.,38 and seen in some of our animal experiments (Fig. 8), the extradurally measured pressure, apart from rising higher in hypercapnia, may also remain higher for 20 minutes or more afterward. This may be due to true cerebral "swelling," in this instance resulting from increased interstitial...
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Fluid volume caused by outpouring from the dilated capillaries under raised intravascular pressure. In some of our animals, however, even quite high pressures were reached in hypercapnia with no differential between the two records (Fig. 9). Two possible explanations for this are that the animals did not have any fluid loss into the interstitial tissues, or that the transducers in these cases did not make actual contact with brain surface, so that a free subarachnoid communication was maintained and CSF pressure was being measured by both transducers.

Our studies in human subjects have so far been confined to patients with suspected communicating hydrocephalus, reported in the accompanying article. In a number of these, the extradural transducer recording has been calibrated both directly, with pressure applied to the transducer immediately before or after the study, and in comparison with ventricular

**In vitro calibration**

36 mm deflection = 50 mm Hg

\[ \therefore \text{1 mm deflection} = 1.39 \text{ mm Hg} \]

**Calculation of Slope**

Pen deflection 1 mm = 0.8 mm Hg

\[ \therefore 11 \text{ mm} = 14.6 \text{ mm Hg} \]

\[ \therefore 10 \text{ mm deflection} = 13.8 \text{ mm Hg} \]

\[ \therefore 1 \text{ mm deflection} = 1.38 \text{ mm Hg} \]

**In vivo calibration**

**Ventricular Pressure**

\[ 
\begin{array}{c|c}
\text{Ventricular Fluid Pressure} & \text{mm Hg} \\
\hline
\text{-20} & \text{mm deflection} = 15 \\
\text{-15} & \text{mm deflection} = 15 \\
\text{-10} & \text{mm deflection} = 15 \\
\text{-5} & \text{mm deflection} = 15 \\
\text{-0} & \text{mm deflection} = 15 \\
\end{array}
\]

**Valsalva**

**Jugular compression**

**Fig. 10.** Graphs showing comparison of extradural and ventricular fluid pressures in a patient over a range of pressures, increased by raising intrathoracic pressure or by jugular venous compression.
fluid pressure as measured by a brain cannula connected to an external transducer at the same level as the EDP transducer. In none has any discrepancy been noted between the two pressures (Fig. 10). The explanation for this, we believe, lies in the cause of the raised intracranial pressure in these patients, and in the method used for raising the pressure in calibration.

High ICP in communicating hydrocephalus is primarily caused by imbalance between rates of production and absorption of CSF, and not raised pressure within the brain itself. Thus, any difference between CSF and the brain pressures would tend to be in the direction of a higher CSF pressure. Furthermore, large differences between the two are more common at higher pressures; the pressures in most of our patients, although raised, were not as high as those often found in patients with other pathological conditions.

In our patients, the calibration of extradural against ventricular pressure was carried out either by compression of the jugular veins in the neck, or by an increase in the intrathoracic pressure as manipulated by the anesthetist. The rise in venous pressure with these maneuvers will of course raise cerebral tissue pressure, but will also have the same effect on CSF pressure by the distension of veins in the subarachnoid and the spinal extradural spaces. This is opposed to the effect of hypercapnia, which affects primarily brain tissue pressure, and only secondarily CSF pressure.

In conclusion, the extradural method of monitoring intracranial pressure is satisfactorily accurate, provided proper care is taken in construction and insertion of the pressure transducer. Our transducer fulfils adequate constructional criteria, is of acceptable safety, and is one of the most accurate and stable among devices currently in use. Its capacity for zero checking allows effective elimination of any residual inaccuracy.

References
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This study was supported by the Medical Research Council of Great Britain.

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