Long-term intracranial pressure recording in the management of pseudotumor cerebri

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Long-term monitoring of intracranial pressure (ICP) was used as an aid for the management of four patients with pseudotumor cerebri. After the implantation of a small experimental pressure sensor in the skull, most of the subsequent ICP measurements were made noninvasively by an external interrogator. During the initial study of the patient, baseline ICP recordings were made in the hospital before treatment. Pressure recordings on a 24-hour basis were continued during treatment, which, depending on the case, was with Diamox (acetazolamide), steroids, or thecoperitoneal shunting. After discharge these patients returned weekly over 10 to 22 months for ICP measurement and for ophthalmological examination. Intracranial pressure before treatment showed irregular variations ranging from 100 to 500 mm H2O over a 24-hour period. The efficacy of treatments could be assessed in a few hours by the degree of ICP stabilization. Shunt malfunction was detected by a slow but continuous rise in pressure before full clinical signs were evident.

KEY WORDS — pseudotumor cerebri — continuous ICP monitoring — steroid — Diamox — thecoperitoneal shunt

PSEUDOTUMOR cerebri occasionally is difficult to control and may eventually lead to blindness. At present, measurement of cerebrospinal fluid (CSF) pressure by lumbar punctures, assessment of optic disc swelling, and serial visual field examinations are used to follow and evaluate the course of pseudotumor treatment.

A device is now available that makes possible continuous intracranial pressure (ICP) monitoring over long periods of time. By this device, changes in ICP can be related to patients' symptoms and ophthalmological evaluation. More importantly, it allows one to regulate medications, such as steroids and Diamox (acetazolamide), until ICP decreases. If patients require shunting to reduce excess pressure, then the device serves to evaluate the patency of the shunt.

This paper describes our experience with long-term ICP recording in four patients with pseudotumor cerebri. All patients had several days of continuous ICP recordings before treatment, medical treatment was then instituted, and the ICP monitored. Three patients eventually required thecoperitoneal shunting. All patients were examined weekly after discharge from the hospital.
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pressures were intermittently confirmed with lumbar puncture, because of the experimental nature of the sensor.

**Technique**

**Implantation of the Sensor**

Informed consent is first obtained from all the patients. A small pressure-sensing capsule is implanted, under local anesthesia, in a burr hole in the skull (Fig. 1). Located outside the body but in close proximity to the implanted sensor is an interrogator, which radiates a VHF radio signal that interrogates the implanted sensor. The information about the ICP detected by the interrogator is then recorded on a chart by the bedside recording equipment. The procedure can be performed easily on either in- or outpatients.

The pressure sensors have been functional in patients for over 2 years. This is possible because the sensor contains no batteries or transistors, but only a passive electric circuit consisting of an inductance (L) and a capacitance (C).

**Action of the Sensor**

The ICP sensor is a short cylinder 15 mm in diameter and 6 mm in height. It is essentially a miniature barometer containing a small metal bellows in which a quantity of nitrogen gas is sealed. As such, the sensor measures in terms of absolute pressure which includes barometric pressure as well as ICP. Movement of the bellows from pressure variations will change the value of capacitance (C) in an LC-tuned radio-frequency (RF) circuit contained in the sensor. Hence, the resonant frequency of the tuned circuit in the sensor is a function of the pressure acting on the bellows. The external detector radiates a frequency-modulated radio wave which, through the inductance (L), couples RF energy into the sensor's LC circuit. As the frequency of the RF from the external detector changes, it reaches the resonant frequency of the implanted sensor. When it does, the RF energy absorbed increases sharply and the external monitor measures the frequency at which this large energy loss occurs. From previous calibration, the relationship between the RF resonant frequency of the sensor and pressure on the sensor is established.

The outer case of the sensor is made of a high-strength plastic. When in place in the burr hole, the thin plastic diaphragm on the inferior surface of the sensor comes in contact with the dura but does not obstruct the underlying subarachnoid space. The plastic case is filled with silicone oil which transmits pressure from the plastic diaphragm to the bellows. The overall action of the sensor is described as follows. With increased ICP, the diaphragm of the sensor is deflected inward. This increases the pressure in the silicone oil which compresses the gas-filled bellows. The bellows' movement decreases the capacitance of the LC circuit in the sensor and raises its RF resonant frequency. The higher resonant frequency is detected externally, and is

![Diagram of the intracranial pressure monitoring system](image)

FIG. 1. Diagram of the intracranial pressure monitoring system.
recorded as increased ICP. The resonant or absorption frequency of the sensor LC circuit can be detected through 2 to 3 cm of tissue. In the clinical usage of the ICP sensor, the readings require correction for barometric pressure and for temperature (18 mm Hg/°C) if the patient's temperature is abnormal.

The ICP sensor has been tested in the laboratory and in animals for 5 years. Gross and histological studies of the tissue at the site of the sensor implantation showed the presence of fibrosis but no signs of tissue irritation. The sensor operates over a range of -200 to +1800 mm H2O, with an accuracy of ±50 mm H2O. Laboratory and animal tests show that the version of the ICP sensor used in this study has an upward baseline drift of 1 to 2 mm H2O/day.

**Case 1**

This 24-year-old woman was first evaluated for papilledema in 1973. She was diagnosed as having pseudotumor cerebri and was treated with steroids. Her symptoms promptly remitted. However, in 1976 her headaches and papilledema recurred, after delivery of a baby. She had gained 50 lbs with her pregnancy. She was re-evaluated with computerized tomographic (CT) scan, electroencephalogram (EEG), and brain scan, all of which were normal. Lumbar puncture showed increased pressure of 300 mm H2O. Corticosteroids were again given, with relief of symptoms. However, after steroid therapy was withdrawn her symptoms recurred and she was referred to the Department of Neurosurgery for consideration of theco-peritoneal shunting.

On October 27, 1976, she had a sensor implanted in the right parietal area under local anesthesia. Intracranial pressure was monitored while the steroids were withdrawn (Fig. 2), and she was discharged. She was readmitted on December 18, 1976, because of recurrence of symptoms. Figure 3 shows fundus photographs and a 24-hour recording of ICP before shunting. On December 27, 1976, the patient had a theco-peritoneal shunt placed. During shunting the sensor calibration was verified by comparing lumbar puncture.
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ture and sensor pressure reading. Figure 4 shows one 24-hour pressure recording after shunting; note the low ICP.

Case 2

This 51-year-old woman began to have severe headaches and blurred vision in 1974, at which time an ophthalmologist noted papilledema. She had a CT scan, brain scan, arteriogram, and pneumoencephalogram, all of which were normal. She was treated with prednisone. However, it was not possible to taper off the prednisone therapy without her symptoms recurring. She was referred to our hospital for a thecoperitoneal shunt. On February 23, 1977, she had a sensor implanted under local anesthesia. Pressure was monitored until March 3, 1977, with intermittent lumbar punctures to confirm the pressure recorded by the sensor. Intracranial pressure recording for a 24-hour period before shunting is shown in Fig. 5; note the large fluctuations in pressure and the papilledema. She was discharged home on March 8, 1977, and followed weekly. Her pressures progressively increased until May 1, 1977, when her symptoms recurred.

With the use of the ICP sensor, detailed measurements were made of the ICP during the course of lumbar puncture. Figure 8 shows the tracing from the ICP pressure monitor from which the heart rate of 84/min and respiration rate of 12/min can be determined. The lumbar puncture opening pressure was 240 mm H2O, the ICP sensor read 430 mm H2O (or 310 mm H2O when referred to the lumbar puncture site, which was 12 cm higher than the implanted sensor). The sensor was reading 70 mm H2O too high 68 days after implantation. This was a result of the 1-mm H2O/day baseline drift of this particular sensor. The mm H2O as a result of 20 cc of CSF being withdrawn. At the reduced ICP, the cardiac pulsatile pressure decreased from peaks of 150 mm H2O to peaks of 75 mm H2O, as can be seen in the tracings.

The patient later underwent exploration of the shunt and was found to have loculations around the peritoneal end of the shunt. Revision of the shunt brought her pressures down; however, the patient again had slow but progressive increase of pressure from May 1, 1977, to May 15, 1977, when the shunt had to be removed because of infection (Fig. 6). She has since been followed with Diamox as the only treatment and her pressure has stabilized at 200 mm H2O.

Case 3

This 17-year-old boy noted frequent headaches in 1973. In 1975, he sustained a skull fracture and was admitted to a hospital for 1 week. He complained of persistent headaches and was re-evaluated at another hospital in 1976, where papilledema was

![Fig. 4. Case 1. Left: Intracranial pressure recorded by the sensor from 1 a.m. to 5 p.m. The patient had undergone thecoperitoneal shunting. Pressure readings were brought down to +50 to -50 mm H2O. The sensor sensitivity is ±50 mm H2O hence her pressures were close to 0. Right: Fundus photographs 2 weeks after shunting.](image-url)
FIG. 5. Case 2. Left: Intracranial pressure recorded by the sensor from 1 a.m. to 10 p.m. The patient was not under treatment and had visual difficulty and headaches. Pressures ranged from 150 to 525 mm H₂O. Right: Eye grounds showing severe changes.

noted. He was transferred to our hospital, where EEG, brain scan, CT scan, arteriogram, and pneumoencephalogram evaluation showed no evidence of mass lesion. Therefore, diagnosis of pseudotumor cerebri was made. He was treated with steroids, which improved his headaches. He was then switched to Diamox and discharged; however, as the Diamox was withdrawn, his symptoms recurred and the patient returned for re-evaluation.

On February 15, 1977, a sensor was implanted and the patient was given Diamox, with improvement of his symptoms. The Diamox was withdrawn gradually without the ICP increasing. On November 16, 1977, the patient was admitted after mild head trauma.

FIG. 6. Case 2. Long-term intracranial pressure (ICP) plotted daily from February 25 to November 18, 1977. From February 25 to 28 the patient had ICP reaching 400 mm H₂O, which was relieved by the peritoneal shunt. Postshunt pressures were in the range of 200 mm H₂O. She was then discharged on March 10. There was a progressive rise in ICP from April 24 to May 2. The patient underwent the peritoneal shunt revision after lumbar puncture confirmed increased pressure, and the abdominal end of the shunt was found to be caught in loculations in the abdomen. Pressures increased again from May 3 to 15, when the shunt was again found to be nonfunctioning because of infection and it was removed. She has since been maintained on 4 gm Diamox/day.
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and recurrence of headaches, nausea, and vomiting. Pressure sensor showed elevation to 400 mm H₂O. He was continuously monitored as Diamox was reinstituted. Figure 7 shows the decrease in pressure over 12 hours to 200 mm H₂O brought about with 4 gm of Diamox per day. The patient was monitored into the next day until the pressure stabilized at 200 mm H₂O (Fig. 8).

Case 4

This 28-year-old woman was referred to our neurology service with papilledema, headache, and blurred vision. She had been evaluated 2 months before admission at another hospital because of papilledema and found to have normal brain scan, CT scan, skull films, and arteriograms. Her lumbar subarachnoid pressures were reported to be high. She was treated with prednisone, followed by Diamox, with no improvement of her symptoms.

She had a sensor implanted on December 7, 1977. Two days of baseline recordings of ICP were made, and she then underwent a thecoperitoneal shunt on December 9, 1977. Samples of the pre- and postoperative ICP recordings are shown in Fig. 9; note the decrease in ICP as a result of the treatment.

Discussion

The syndrome of intracranial hypertension and papilledema with no focal neurological signs, together with the clinical findings of normal CSF composition and normal or small ventricles has been recognized as a distinct disease entity for nearly a century. Most writers attribute to Quincke⁹ the first clear description of the disease which he named "serous meningitis."

Symonds¹²,¹³ noted the appearance of the symptoms of intracranial hypertension accompanied by visual problems in children as a complication of otitis media. He renamed the disease "otic hydrocephalus," which he believed was caused by excessive CSF secretion or defective absorption through the arachnoid villi from inflammatory occlusion. Gardner,⁴ who made the first air contrast studies, agreed with Symonds except that he believed thrombosis in the lateral sinus with engorgement of the intracranial veins was sufficient to induce intracranial hypertension. He called the disease "sinus thrombosis causing intracranial hypertension," a disease that he and Symonds agreed was relieved in time with recanalization in the thrombosed veins and the development of collateral channels.
FIG. 9. Case 4. Intracranial pressure (ICP) recordings before (left) and after (right) the installation of a thecoperitoneal shunt. Preoperatively, high ICP was accompanied by plateau waves and by large pulsation from the heart beat. Postoperatively, ICP was normal except for negative ICP occurring when the patient was seated.

Thrombosis of the dural sinuses was suggested as a cause of pseudotumor cerebri by the studies of Ray and Dunbar and Zuidema and Cohen.

Dandy, however, suspected that the increased ICP came about because of excessive blood volume. The rapid changes in the bulges at the temporal decompression sites (2 to 3 minutes) suggested variations in the intracranial vascular bed by vasomotor control. He called the condition "intracranial pressure without brain tumor."

McCullagh was the first to report on a patient with pseudotumor cerebri that could be attributed to menstrual edema. Wagener concluded most cases of pseudotumor cerebri are caused by cerebral edema. To lend support to the argument of edema as the cause, Sahs and Joynt showed intracellular and extracellular edema in biopsy material from 10 patients with pseudotumor cerebri. The recent work on pseudotumor cerebri shows an acceptance of the idea that, in this disease, the same symptoms may arise from a large diversity of underlying causes.

In the past 2 years we have been evaluating an ICP sensor for use in neurosurgical patients. The most valuable use of the ICP monitor has been in the management of patients with a chronically increased ICP, such as in pseudotumor cerebri. Our continuous recordings of ICP in pseudotumor cerebri patients show high and rapidly changing pressure levels (Figs. 3 and 5), a characteristic of this condition previously noted by Dandy. Davidoff and Dyke remarked that the temporal fossa bulges could be used for determining when the patient requires drainage by lumbar puncture or drugs for dehydration. Gardner found the size of the swelling at the decompression site was a good indicator for determining when lumbar puncture was required to relieve pressure. The ICP monitor, however, gives us quantitative records of the complicated temporal behavior of the ICP in these patients (Fig. 6).

Lundberg was the first to use continuous ICP monitoring for neurosurgical patients. He identified three distinct pressure wave phenomena, A-, B-, and C-waves. Johnston and Paterson studied 20 pseudotumor cerebri patients with continuous monitoring of ICP, using Lundberg's method, that is, recording ICP via an intraventricular catheter. They found A-waves (plateau waves) and B-waves in their pseudotumor cerebri patients as we did in ours (Fig. 9). They noted that headache did not seem to be associated with the presence of a plateau wave. This lack of association was attributed to a reduced level of perfusion pressure.

Langfitt, et al., investigated cerebral blood flow in the presence of increased ICP. They showed that an acute rise in ICP decreased cerebral blood flow whereas a gradual increase in ICP will not affect blood flow even at 680 mm HgO. This suggests that in pseudotumor cerebri, the damage to vision may be brought about by acute rises in pressure rather than by chronically high ICP.

All four cases of pseudotumor cerebri studied in this report showed high fluctuation in ICP during 24 hours with pressures occasionally dropping to normal levels (Figs. 3 and 5). This explains the occasional normal pressures observed in pseudotumor cerebri patients. The ICP did not appear to vary in a rhythmic fashion in these patients.

In two of the four patients, Diamox effectively lowered ICP (Figs. 6 and 7). In one patient (Fig. 7), pressures decreased within 12 hours of Diamox administration. This is postulated to be secondary to reduced secretion of CSF. Steroids were effective in lowering pressures in all four patients (Fig. 2). In the three patients who required thecoperitoneal shunt, three shunt malfunctions oc-
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curred. All three malfunctions were detected by the slow but progressive increase in pressure observed on the ICP monitor (see Fig. 6). In the long-term use of this particular ICP sensor we must account for a steady upward drift in baseline pressure of 1 to 2 mm H₂O/day. Thus an occasional lumbar puncture is required in outpatients to correct the calibration.

The sensor implanted into patients with chronically increased ICP allows us quickly to obtain pressure readings on the wards or in the clinic. The evaluation of the optic disc is basically a qualitative method of obtaining evidence of increased ICP. The sensor allows quantitative ICP readings to be obtained quickly. At present the method is encumbered by having to directly read the tuning frequency and correct it for temperature and barometric pressure; however, these corrections will be performed automatically by electronic circuits in the near future.

The ICP sensor implanted in patients provides early warning of acute rises in ICP, and allows the monitoring of chronic pressure elevations. The purpose of the implanted pressure sensor is to detect ICP rises before the onset of papilledema.

The ability to make chart recordings of the ICP of patients in the hospital has greatly improved evaluation of treatment by giving us the ability to observe the changes with treatment. We have been able to incorporate the interrogator within the head dressing of patients for continuous recordings. Patients can disconnect themselves from the monitor and be fully ambulatory.

In summary, we have found that the ICP sensor allowed us to follow treatment and regulate ICP pressure in pseudotumor cerebri patients with very few lumbar punctures. The method was helpful in clinical management of patients by alerting us to the occurrence of acute episodes of high ICP before papilledema developed. The method was more advantageous than daily lumbar punctures and ophthalmoscopic fundus examinations.

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References


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