Intracranial venous sinus hypertension: cause or consequence of hydrocephalus in infants?

CHRISTIAN SAINTE-ROSE, M.D., JACQUES LACOMBE, M.D., PH.D., ALAIN PIERRE-KAHN, M.D., DOMINIQUE RENIER, M.D., AND JEAN-FRANÇOIS HIRSCH, M.D.

Department of Pediatric Neurosurgery, Hospital for Sick Children, Paris, France

From a previous study of achondroplasia as well as from the observation of patients with hydrocephalus associated with craniostenosis, the authors have concluded that an increased superior sagittal sinus venous pressure (SSVP) could be the cause of the enlarged ventricles. However, other workers have demonstrated that an increased SSVP could be the consequence of increased intracranial pressure (ICP). Therefore, the authors undertook a study to determine if there was a physiological test that could distinguish between rare instances of increased SSVP caused by structural and irreversible narrowing of the sinus and those caused by increased ICP.

In 20 hydrocephalic infants and children, pressure was simultaneously measured in the lateral ventricle, the superior sagittal sinus, and the jugular vein. Stable baseline pressures were recorded, as well as the variations observed after the withdrawal of an amount of cerebrospinal fluid (CSF) sufficient to lower ICP to zero. Similar recordings were taken after reinjection of an equal quantity of CSF. In all of the patients, SSVP was increased, but not as much as the ICP. In the cases of hydrocephalus without any associated cranial malformation, and therefore without any likely anatomical interruption of the sinus, CSF withdrawal induced a simultaneous decrease of ICP and SSVP. However, whereas ICP could be lowered to zero, SSVP never fell below the jugular venous pressure, which remained stable (around 5 mm Hg) throughout the recording session. Results were different when sinography demonstrated an anatomical interruption of the sinus, as in cases of hydrocephalus associated with achondroplasia or craniostenosis. In these cases, although ICP was normally lowered by CSF withdrawal, SSVP remained nearly unchanged, usually greater than the jugular venous pressure.

The present study demonstrated that SSVP recording during ICP variations induced by CSF withdrawal permits differentiation between a reversible collapse of the sigmoid sinus due to increased ICP and a fixed obstructive lesion of the sinuses. Based upon this test and the results of sinography, the authors inserted a venous bypass between the lateral sinus and a jugular vein in three patients.

KEY WORDS • hydrocephalus • pseudotumor cerebri • achondroplasia • craniostenosis • intracranial pressure • venous sinus • venography

Various degrees of hydrocephalus are known to be associated with craniostenosis in some patients and with most cases of achondroplasia. In a previous article, we have shown that several clinical, radiological, and surgical arguments favor the hypothesis that, in achondroplasia, hydrocephalus is caused by an impairment of the venous outflow from the brain. In these cases, the computerized tomography (CT) scans show an enlargement of the ventricles and of the cerebral sulci, and angiography and sagittal sinus venography demonstrate a narrowing or an interruption of the sigmoid sinuses and a venous collateral circulation through emissary veins and spinal venous plexuses. However, variations in the venous anatomy occur frequently. Moreover, it is impossible to conclude from nondynamic pictures whether the compression of the sigmoid sinus is the cause or the consequence of hydrocephalus.

To overcome these difficulties, we decided to simultaneously record the cerebrospinal fluid (CSF) pressure and sagittal sinus venous pressure (SSVP) in 31 infants with various conditions. Our presumption was that, in cases of venous outflow impairment, the CSF pressure would not be greater than the SSVP, but equal or nearly equal to it. Actually, we found that, in infantile hydrocephalus, even without any fixed anatomical obstruc-
tion in venous outflow, CSF pressure and SSVP were very similar, the differences being within the limits of errors of the method.

We then studied the simultaneously induced variations of CSF pressure, SSVP, and jugular venous pressure, to determine if the venous outflow impairment was the consequence or the cause of the high intracranial pressure (ICP). If it were the consequence, lowering the CSF pressure should lower the SSVP by removing the outside pressure exerted on the walls of the sinuses. However, if impaired venous outflow were the cause of hydrocephalus, SSVP should not change after a fall of CSF pressure, since that fall would not change the fixed obstruction of the venous outflow. The results of these different recordings in 31 infants are reported here.

Clinical Material and Methods

Thirty-one infants, ranging in age from 1 to 23 months, were studied. The patients suffered from communicating hydrocephalus (six cases), aqueductal stenosis (one case), hydrocephalus accompanying myelomeningocele (six cases), subdural hematoma (one case), cranio-nystenosis (14 cases), or achondroplasia (three cases).

The CSF pressure was measured through a needle placed in the right frontal horn. A second needle was inserted in the other lateral ventricle to remove or reinject CSF. The superior sagittal sinus was cannulated at the site of the anterior fontanel, in the direction of the blood flow. Patency of the cannula was maintained by intermittent irrigation with a saline solution. Jugular venous pressure was recorded near the base of the skull by means of a catheter inserted in a retrograde direction in the jugular vein to a distance of 4 cm. All these catheters were placed so that their tips were at the same horizontal level. They were all connected to a pressure transducer* that was calibrated to zero pressure at the estimated level of the right atrium. After amplification, data were displayed on a computer from which tracings could be obtained on an x-y table.† During the recording session, the child received barbiturate sedation (intra-rectal Nembutal (sodium pentobarbital), 5 mg/kg). General anesthesia was considered unnecessary in all cases.

The first part of this study was devoted to the simultaneous recording of CSF pressure and SSVP (11 cases) to determine a baseline pressure. In the second part (20 cases), in order to study the simultaneous variations of CSF pressure and SSVP, the CSF pressure was lowered to zero by a rapid (1- to 3-minute) withdrawal of CSF, and was later restored to its initial value or more by reinjection of an equal amount of CSF. Four of the 20 patients were suspected of having a fixed obstruction of cerebral venous outflow.

Results

Recordings of Baseline Pressure

In all 11 patients who underwent simultaneous recordings of CSF pressure and SSVP to determine baseline values (Table 1), the CSF pressure was higher than SSVP in seven patients, equal in three patients, and

---

* CSFP = cerebrospinal fluid pressure; SSVP = sagittal sinus venous pressure.

---

TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (mos)</th>
<th>Diagnosis</th>
<th>Baseline Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>CSFP</td>
</tr>
<tr>
<td>1</td>
<td>3</td>
<td>cranio-nystenosis</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>cranio-nystenosis</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>cranio-nystenosis</td>
<td>13</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
<td>cranio-nystenosis</td>
<td>13</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>cranio-nystenosis</td>
<td>22</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
<td>cranio-nystenosis</td>
<td>17</td>
</tr>
<tr>
<td>7</td>
<td>5</td>
<td>cranio-nystenosis</td>
<td>10</td>
</tr>
<tr>
<td>8</td>
<td>18</td>
<td>achondroplasia</td>
<td>25</td>
</tr>
<tr>
<td>9</td>
<td>4</td>
<td>hydrocephalus</td>
<td>23</td>
</tr>
<tr>
<td>10</td>
<td>1½</td>
<td>hydrocephalus</td>
<td>23</td>
</tr>
<tr>
<td>11</td>
<td>14</td>
<td>hydrocephalus &amp; myelomeningocele</td>
<td>25</td>
</tr>
</tbody>
</table>

---

TABLE 2

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (mos)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>5½</td>
<td>hydrocephalus &amp; aqueductal stenosis</td>
</tr>
<tr>
<td>13</td>
<td>6</td>
<td>hydrocephalus &amp; Arnold-Chiari malformation</td>
</tr>
<tr>
<td>14</td>
<td>2½</td>
<td>hydrocephalus &amp; Arnold-Chiari malformation</td>
</tr>
<tr>
<td>15</td>
<td>1</td>
<td>hydrocephalus &amp; Arnold-Chiari malformation</td>
</tr>
<tr>
<td>16</td>
<td>1</td>
<td>hydrocephalus &amp; Arnold-Chiari malformation</td>
</tr>
<tr>
<td>17</td>
<td>½</td>
<td>hydrocephalus &amp; Arnold-Chiari malformation</td>
</tr>
<tr>
<td>18</td>
<td>½</td>
<td>communicating hydrocephalus</td>
</tr>
<tr>
<td>19</td>
<td>10</td>
<td>communicating hydrocephalus</td>
</tr>
<tr>
<td>20</td>
<td>4</td>
<td>communicating hydrocephalus</td>
</tr>
<tr>
<td>21</td>
<td>6</td>
<td>communicating hydrocephalus</td>
</tr>
<tr>
<td>22</td>
<td>19</td>
<td>subdural hematoma</td>
</tr>
<tr>
<td>23</td>
<td>3</td>
<td>cranio-nystenosis</td>
</tr>
<tr>
<td>24</td>
<td>1</td>
<td>cranio-nystenosis</td>
</tr>
<tr>
<td>25</td>
<td>8</td>
<td>cranio-nystenosis</td>
</tr>
<tr>
<td>26</td>
<td>9</td>
<td>cranio-nystenosis</td>
</tr>
<tr>
<td>27</td>
<td>13</td>
<td>cranio-nystenosis</td>
</tr>
<tr>
<td>28</td>
<td>9</td>
<td>cranio-nystenosis &amp; hydrocephalus</td>
</tr>
<tr>
<td>29</td>
<td>14</td>
<td>cranio-nystenosis &amp; hydrocephalus</td>
</tr>
<tr>
<td>30</td>
<td>7</td>
<td>achondroplasia &amp; hydrocephalus</td>
</tr>
<tr>
<td>31</td>
<td>8</td>
<td>achondroplasia &amp; hydrocephalus</td>
</tr>
</tbody>
</table>

---

* CSF = cerebrospinal fluid; CSFP = CSF pressure; SSVP = sagittal sinus venous pressure; JVP = jugular venous pressure.
lower in one patient. In all cases, the difference between the two pressures was small (approximately 3 mm Hg). In the eight patients who showed a clearly elevated CSF pressure (between 15 and 25 mm Hg), the SSVP was increased in the same proportion so that the difference between the two pressures remained small. This elevation of SSVP was observed regardless of the cause of the increased CSF pressure: craniostenosis (Cases 1 to 7), hydrocephalus with (Case 11) or without (Cases 9 and 10) myelomeningocele, or achondroplasia (Case 8). Therefore, the raised SSVP did not seem to be the cause or the mechanism of intracranial hypertension. It should be noted that all patients were infants between 1 and 23 months. A similar study in adults would likely give different results.

Recordings of Pressure Variations

Simultaneous recordings of CSF pressure, SSVP, and jugular venous pressure were made after CSF removal in 20 infants (Table 2). Two different types of recordings were found.

CSF Pressure and SSVP Decrease. In 16 of the 20 infants, there was a simultaneous decrease of CSF pressure and SSVP after CSF removal (Fig. 1). Before CSF removal, CSF pressure and SSVP were elevated, the former usually being slightly higher than the latter. After withdrawal of enough CSF to lower the pressure to zero, SSVP was greater than CSF pressure because, although SSVP had decreased simultaneously with CSF pressure, it did not fall lower than the jugular venous pressure, which remained stable during the whole recording session (around 5 mm Hg) (Fig. 2).

After reinjection of a quantity of CSF equal to that withdrawn, the jugular venous pressure remained stable, but the CSF pressure was usually restored to a level slightly higher than initially. In some cases, SSVP followed this overresponse (Fig. 1), but in others SSVP did not exceed its initial value (Fig. 2).
This simultaneous decrease in both pressures was found in five cases of craniostenosis (Cases 23 to 27), one of subdural hematoma (Case 22), four of communicating hydrocephalus (Cases 18 to 21), five of hydrocephalus with Arnold-Chiari malformation (Cases 13 to 17), and one of aqueductal stenosis (Case 12). In these cases, angiography or sagittal sinus venography did not show any narrowing of the sigmoid sinuses.

These results demonstrate that, in infants, CSF pressure is transmitted through the wall of the dural sinuses so that SSVP variations follow changes in CSF pressure. The SSVP was slightly lower than the CSF pressure, but was never lower than the jugular venous pressure, which was about 5 mm Hg near the superior jugular bulb. When CSF pressure was increased, graphs of CSF pressure and SSVP were parallel at first, but later they diverged when the sinus could no longer be compressed, so that SSVP did not increase further. In these 16 infants, the elevated SSVP was due to a reversible compression of the sinuses by the intracranial hypertension.

**No SSVP Decrease.** In the other four infants, who had hydrocephalus associated with craniostenosis (Cases 28 and 29) or achondroplasia (Cases 30 and 31), CSF removal induced a fall in CSF pressure, but SSVP did not change and remained elevated even when CSF pressure was lowered to zero (Figs. 3 and 4). The SSVP also remained higher than the jugular venous pressure, which, as usual, did not change with variations in CSF pressure. In these patients, it was clear that the elevated SSVP was due to a fixed obstruction of the lateral sinuses. Indeed, this obstruction was demonstrated by sagittal sinus venography and angiography (Fig. 5).

The two types of recordings are clearly shown in Fig. 6, which depicts the variations of the difference between

---

**FIG. 3.** Simultaneous recordings of cerebrospinal fluid pressure (CSFP), sagittal sinus venous pressure (SSVP), and jugular venous pressure (JVP) during removal (−30 cc) and reinjection (+30 cc) of CSF in an infant with achondroplasia and a fixed partial obstruction of the lateral sinuses. P = pressure. **Left:** Slow-speed recording. **Right:** Enlargement of recording at points A and B at left, at a faster speed.

**FIG. 4.** Simultaneous preoperative recordings of cerebrospinal fluid pressure (CSFP), sagittal sinus venous pressure (SSVP), and jugular venous pressure (JVP) during removal (−60 cc) and reinjection (+60 cc) of CSF in an infant with Crouzon’s disease and a fixed partial obstruction of the lateral sinuses. **Left:** Slow-speed recording. **Right:** Enlargement of recording at points A, B, and C at left, at a faster speed. P = pressure; Cal = calibration, and indicates the point where automatic calibration of the recorder occurred.
CSF pressure and SSVP after CSF withdrawal. In the 16 cases (Cases 12 to 27) with a reversible compression of the sinus wall by the CSF pressure, the CSF pressure and SSVP showed parallel variations, so that their difference remained small (−5, −1 mm Hg) after CSF removal. On the other hand, in infants with a fixed obstruction of the sinus (Cases 28 to 31), the difference between CSF pressure and SSVP increased after CSF removal.

**Recordings in an Infant with Venous Bypass**

Based upon these recordings, we decided to insert a bypass graft between the transverse sinus and the jugular vein in an infant with craniostenosis associated with hydrocephalus. In this patient, sagittal sinus venography had shown an obstruction of the sigmoid sinuses (Fig. 5). However, the reduction in CSF pressure after withdrawal of fluid was not accompanied by a decrease in SSVP (Fig. 4). This infant was operated on first for his craniostenosis. Ten days later, a reverse saphenous venous graft was inserted between the left lateral sinus and the external jugular vein according to a technique used by Sindou, et al., and later used by Hitchcock and Cowie. At the level of the transverse sinus, an end-to-side anastomosis was made with interrupted sutures. The suturing was carried out in two steps: 1) between the soft internal layer of the sinus and the tunica interna of the graft; and 2) between the dural wall and the adventitia of the saphenous vein. No drugs were given, and no special management was needed in the postoperative period.

It is important to note that the diameter of the graft, taken in an 8-month-old infant, is about 1 mm and therefore might have a low efficiency. However, in this case, it became progressively larger, as shown 6 months after surgery by sagittal sinus venography (Fig. 7). A comparison between the ICP recordings made before surgery and 6 months afterward (Figs. 8 and 9) shows an improvement postoperatively, especially in the shape and amplitude of the rapid eye movement (REM) sleep waves associated with increased ICP before treatment. Figure 10 demonstrates this decrease in CSF pressure and SSVP. However, CSF withdrawal did not induce any important SSVP decrease. To understand this unexpected result, it should be remembered that the graft...
is located extracranially and cannot therefore be collapsed by CSF pressure. Simultaneously with this reduction in ICP, the size of the ventricles became progressively reduced, as shown in Fig. 9. This ICP reduction is, moreover, proven by the decrease in the ratio of ventricular area/brain area measured on pre- and postoperative orbitomeatal CT scans taken at the same level 18 mm above the foramen of Monro. This ratio was 0.30:1 before surgery, 0.27:1 6 months later, and 0.22:1 a year after the operation.

Two other infants (13 and 5 months old) with hydrocephalus, craniostenosis, and a venous outflow impairment with a huge collateral circulation in the scalp (Fig. 11A) were operated on using the same technique. In these two cases, the venous graft was also very small (Fig. 11B). Preoperatively, ICP was very high and remained so immediately after surgery, due to inefficiency of the small nondilated venous graft. Because of the risk to vision, it was decided to not wait any longer for dilatation of the graft, and a ventriculoperitoneal (VP) shunt was inserted. In these two patients, the venous graft remained patent and increased in size within 6 months (Fig. 11C). Remodeling of the skull, which could not have been done without an interruption of the venous collateral channels in the scalp, became possible and was performed without any complication. In these two cases, the venous bypass graft was thus useful. However, these two reports cannot be used to show that a relationship does not exist between venous outflow impairment and hydrocephalus, since the graft was too small when it became imperative to insert a VP shunt.

Discussion

Two questions have often been debated in experimental and clinical studies: Is SSVP dependent upon CSF pressure? Can an increased SSVP be the cause of hydrocephalus?
Venous sinus hypertension and hydrocephalus

FIG. 9. Same patient as in Figs. 4, 5, 7, and 8. Computerized tomography scans taken at the same level, 18 mm above the foramen of Monro, preoperatively (left), at 6 months (center), and at 1 year (right).

FIG. 10. Same patient as in Figs. 4, 5, 7, 8, and 9. Simultaneous recordings of cerebrospinal fluid pressure (CSFP), sagittal sinus venous pressure (SSVP), and jugular venous pressure (JVP) 6 months postoperatively, during removal (−25 cc) and reinjection (+25 cc) of CSF. Left: Slow-speed recordings. Right: Recordings at points A, B, and C at left, at a faster speed. P = pressure.

FIG. 11. Sinography in a patient with craniosynostosis and hydrocephalus. A: Before surgery, there is stenosis of the left lateral sinus (arrows S) and a huge venous collateral circulation of the scalp. B: One month after the operation, a narrow but patent venous graft (arrows G) is seen. C: Six months postoperatively, the venous graft is patent and dilated (arrows G).
Under normal conditions, CSF pressure is generally higher than SSVP, this pressure gradient being necessary to CSF flow. However, the relationship between CSF pressure and SSVP has been interpreted variously by different workers. Weed and Flexner thought that CSF pressure variations did not influence SSVP, which remained stable in their investigations. On the contrary, Bedford showed, in a study of dogs, that an increased CSF pressure correlated with a decreased SSVP. In Wright's study, intracranial hypertension caused a limited and transitory elevation of SSVP. Several authors have shown that SSVP was elevated when CSF pressure was increased, whereas Martins, et al., demonstrated that, in the adult, the consequence of intracranial hypertension on SSVP was unpredictable. Norrell, et al., thought that SSVP and CSF pressure variations would be the same in cases of hydrocephalus accompanying myelomeningocele because of the Arnold-Chiari malformation and the abnormal lateral and sigmoid sinuses. Shulman and Ransohoff, however, showed similar variations of CSF pressure and SSVP in all cases of infantile hydrocephalus.

It should be noted that the different conclusions of these studies may be due to variations in the clinical or experimental conditions: the torcular of the dog is intrasosseous; the lateral sinuses are in an abnormally low position in myelomeningoceles; the sinuses of the adult are certainly less easily collapsed than those of the infant; and the compression of a sinus probably depends upon the chronicity of the intracranial hypertension. Our data agree with the findings of Shulman and Ransohoff. In infantile hydrocephalus, with or without Arnold-Chiari malformation, CSF pressure and SSVP are elevated, but SSVP is usually slightly lower than CSF pressure. The SSVP is determined by the cerebral blood flow and by the vascular resistance. In our study, ICP variations were too small to modify the cerebral blood flow. Moreover, a modification of the cerebral blood flow should have resulted in a change in jugular venous pressure. Therefore, it can be concluded that the increased SSVP is due to a reversible collapse of the sinus caused by the intracranial hypertension.

Langfitt, et al., showed that intracranial hypertension due to an inflated supratentorial balloon would collapse the sagittal sinus in the monkey. In man, it is very likely that the collapse takes place in the posterior fossa, at the level of the sigmoid sinuses. Several clinical studies have demonstrated such collapse. Another argument also favors this hypothesis: the round shape of the sigmoid sinuses, which allows a partial collapse.

Our physiological data allow for distinction between reversible collapse of the sinus and fixed obstruction. Moreover, the venography and angiography performed in our patients with fixed obstruction confirmed the narrowing of the sigmoid sinuses and showed the development of a collateral circulation. The varying functional efficiency of these collateral channels might explain the large range of the SSVP values measured in these cases. The differentiation between reversible and fixed obstruction is of utmost importance since, in the former instance, in which the collapse is the consequence of the increased CSF pressure, it would not be appropriate to insert a venous bypass. In cases of fixed obstruction, however, further studies will be necessary before we can know if such a bypass on the venous outflow is efficient enough. In craniostenosis, with impairment of the venous outflow, the present study does not demonstrate whether the fixed obstruction is the irreversible consequence of chronic high pressure in the posterior fossa or of primary meningeal or osseous maldevelopment. In achondroplasia, other investigations have shown that the obstruction to the venous outflow was due to a bone narrowing at the jugular bulb.

Our study suggests that an increased SSVP may be the cause of hydrocephalus, since the four cases of fixed obstruction were characterized by dilated ventricles. Such a relationship is still being discussed. In experimental investigations, Bering and Salibi were able to produce hydrocephalus in dogs by occluding all the major drainage routes in the neck, whereas Guthrie, et al., by blocking the torcular Herophili, produced intracranial hypertension with normal-sized ventricles. Several authors have performed clinical studies and concluded that an increased SSVP would not result in hydrocephalus but in pseudotumor cerebri. In a series of 79 cases of benign intracranial hypertension in infancy and childhood, Grant identified nine patients who showed mild ventricular dilatation. Other authors have conducted studies restricted to infants and showed high venous outflow pressure would result in ventricular dilatation. Moreover, some reports have demonstrated that the lowering of the venous outflow pressure results in a decrease of the ventricular size, as occurred in our first patient. Wu and Swaiman reported a case in which communicating hydrocephalus was inadvertently induced in a neonate by bilateral jugular vein catheterization; the removal of one catheter resulted in a return to normal ventricular size within 14 days.

It is clear that, at least in infants, an increased SSVP produces hydrocephalus and enlargement of the subarachnoid spaces. One explanation for the mechanism for the enlargement of the subarachnoid spaces of the convexity was proposed by Young, who suggested that, in infants, the elastic resistance to deformation of the skull sutures is probably less than that of the peri-ventricular brain tissue.

A distensible skull appears a prerequisite if elevated venous pressure is to cause ventricular and subarachnoid enlargement. In this study, the three infants with craniostenosis associated with hydrocephalus had a large anterior fontanel and at least two sutures were not involved. We agree with Rosman and Shands, who suggested that the difference in outcome from
Venous sinus hypertension and hydrocephalus

raised pressure (hydrocephalus or pseudotumor) is age-related, and, more specifically, that it depends on whether the cranial sutures are patent or closed. When the cranial sutures are patent and the skull distensible, an increase in venous outflow pressure results in macrocrania and hydrocephalus; when they are closed, the equilibrium of the pressures within the skull does not allow any ventricular dilatation, but results in a high CSF pressure.

This study has identified two groups of patients with infantile hydrocephalus. In one group, which is the most common, hydrocephalus results in a reversible collapse of the sigmoid sinus and in an elevated SSVP, a consequence of the increased CSF pressure. In the other group (achondroplasia and some rare craniosenoses), the consequence of a fixed obstruction of the venous outflow is an elevated SSVP, an increased CSF pressure and, when the skull is distensible, a dilatation of the ventricles and of the subarachnoid spaces. Our study demonstrates that SSVP recording during CSF pressure variations allows good discrimination between these two groups of patients.

Acknowledgments

We wish to acknowledge Drs. M. Mayer and J. M. Servant for their help in this work, and Miss J. Bercet for her fine secretarial assistance.

References


Manuscript received February 23, 1983. Accepted in final form November 28, 1983.
Address reprint requests to: Jean-François Hirsch, M.D., Department of Neurosurgery, Hôpital Necker, 149 Rue de Sèvres, 76016 Paris, France.