EFFECT OF CUTTING THE TENTORIUM CEREBELLI ON THE RESPONSE TO CRANIOCEREBRAL TRAUMA

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The tentorium cerebelli, in its situation as a virtually unyielding membrane, can contribute to injury of the midbrain or pons produced by edema or increased pressure from the temporal lobes or cerebellar hemispheres.

The experiments reported here were done to determine whether or not the absence of a taut tentorium would protect the brain stem from the effects of direct cranial trauma. It was also possible to obtain an idea of the effect of tentorium vs. "no tentorium" on the production of cerebellar pressure cone.

MATERIAL AND METHODS

1. Experimental Animals. Twenty-six dogs were used. They were selected from similar breed lines and were all within the age range of from 18–24 months. They weighed from 14 to 20 lbs.

The tentorium of the dog consists of a sheet-like extension of dura mater between the cerebellum and cerebrum. It is attached posteriorly to the internal protuberances of the occipital bone, and anterolaterally to the "crista petrosa" of the petrous bones, which is a shelf-like projection, probably the canine analogue to the bony tentorium of the cat.1 The anatomical relationships of the tentorium to the skull may be seen in Figs. 1, 2 and 3.

2. Operative Procedure. Sterile technic was used. Animals were anesthetized with intravenous Nembutal Sodium (0.5 mg./lb.). Ether was not used because of its transient hypertensive effect which may contribute to edema of the brain. In the prone position, a T-shaped occipital incision was made. Four burr holes were made (Fig. 4) and a quadrilateral segment of occipital bone was removed. The dura mater was entered and the occipital lobe on each side was retracted upward. The tentorium overlying each cerebellar hemisphere was incised from lateral mesiad, at an angle of about 45° with the sagittal midplane. Care was taken to completely divide the free tentorial edge at its "notch," thus releasing all tension of the membrane. The occipital dura mater was then closed and the bone flap was replaced, following which a layered closure of muscle and skin was performed. There were no infections. Animals in the control group underwent the same procedure, except that their tentorii were not cut.

The second stage was carried out in the experimental group only, and consisted in applying blunt trauma to the top of the skull. This was done from 3–4 weeks after tentorial division, under Nembutal anesthesia.

A carotid artery was cannulated and blood pressure was recorded by means of a

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Fig. 1. Lateral aspect of skull of dog. Dotted lines represent sutures. Outline topography of cerebrum, cerebellum, and plane of tentorium.

Fig. 2. Sagittal section through skull showing plane of tentorium and its bony attachments.

Fig. 3. Basal view of skull showing coronal plane of basal tentorial attachments.
mercury-calibrated strain gauge, bridge amplifier and ink-writer (Sanborn). A tracheal cannula of Tygon tubing was inserted.

A diagram of the apparatus used to apply trauma is shown in Fig. 5. An upright member 12 inches high stands at right angles to a grooved baseplate. The groove is fitted with a sliding tampon that holds the dog's head securely against the upright. A lubricated slot in the upright contains the male member of a 5-lb. metal weight. The upright forms one limb of a solenoid circuit. When current is applied, the weight descends. The measured force of impact is 18.12 kg./cm.² Solenoid acceleration is used instead of springloading, because with a spring the initial velocity (except for gravity) is greatest at the onset of motion, whereas with solenoid activation the initial velocity is greatest when the weight strikes. The head is adjusted so that the weight falls on the sagittal plane of the skull at the vertex. The inferior surface of the skull rests evenly on the mandibular angles and both auditory bullae. After trauma the animals were carefully treated, especially as regards fluid balance, and all were sacrificed at 6 weeks by intracardiac Nembutal.
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RESULTS

Animals that had their tentorii severed did better statistically (Chi Square) than the controls (Tables 1 and 2). Survivors were classified as those animals that lived for 4 days with minimal or regressing signs of concussion, other neurological deficit, or cardiovascular decompensation. Those that appeared moribund early, but later improved, were classified as survivors if they lived 5 days. Independent evaluation of the animals' condition was made by observers who did not know the experimental animals from the controls. Mean survival time was greater for the experimental group. Although indices of vital signs are hard to compare when there are many parameters involved, it is a definite impression that those experimental animals that died had a more benign course until the acute onset of their demise. This can be gathered from the blood-pressure data and clinical course. Animals that survived 5 days did not die during the 6-week period. Animals that died had predominant cerebral edema. The brain was pushed down into the tentorial opening, which was plugged so that fluid could not pass from the basal cisterns to the upper subarachnoid space. The accumulation of fluid at the base of the brain increased medullary pressure.

The control survivors at sacrifice had hydrocephalus caused by pressure of the brain stem containing the narrow aqueduct of Sylvius against the hard tentorial edge. None of the experimental survivors (cut tentorii) had hydrocephalus. More information regarding this point might have been obtained if survivors had been allowed to live another month, although the controls with hydrocephalus had clear-cut ventricular dilation, and there is little reason to doubt that the same condition had had enough time to develop in the experimental animals. Mean basal cisternal manometric pressures at autopsy were above normal in the controls and within normal range in the experimental group.

Pressures in the basal cisterns upon opening the skull (stage II) were above normal in 3 experimental animals, and in 8 controls. In the experimental animals that died one subdural hemorrhage was found, one temporal lobe laceration and one case of pontine and midbrain hemorrhage caused by impact of the brain stem against the basi-occiput. These occurred despite the cut tentorii. Concussion states could be determined by the time required for recovery from "anesthesia," which was about 30 min. in normals after maneuvering of the head in the trauma apparatus, etc. Eleven animals in the experimental group reacted from anesthesia. Reaction time in these varied from 45 min. to 4 hrs. with 3 requiring over 2 hrs. Eight controls recovered consciousness, their reaction times varying from 40 min. to 24 hrs., with 7 requiring over 2 hrs.

Early increase in blood pressure was a poor prognostic sign, except in cases in which early acute shock developed. There was poor correlation between deterioration and the development of a high pressure and low
<table>
<thead>
<tr>
<th>No.</th>
<th>Survival (days)</th>
<th>R.P. Post-Trauma*</th>
<th>Consciousness Time of Reaction</th>
<th>Pupils</th>
<th>Deep Tendon Reflexes</th>
<th>Resp.</th>
<th>Basal Cistern Pressure;</th>
<th>Autopsy Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>↓, slow ↓</td>
<td>1st day, Reacted at 21 hrs.</td>
<td>Lt. dilated, fixed</td>
<td>↑ rt.</td>
<td>Reg. 100</td>
<td>Rt. uncal herniation, brain stem to lt., compressing III N.</td>
<td>Minimal cerebellar pressure cone</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>↓</td>
<td>Died 18 min.</td>
<td>Fixed bilat.</td>
<td>0</td>
<td>Irreg. 112</td>
<td>Lt. hippocampal herniation.</td>
<td>Laceration of midbrain, tentorial edge</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>Acute shock</td>
<td>Died 2 hrs.</td>
<td>Early constriction, then dilated, fixed bilat.</td>
<td>Incr. bilat.</td>
<td>De-cerebrate rigidity</td>
<td>Reg. 95</td>
<td>Bil, uncal herniation, stretched post. cerebri with pressure III N. bilat. Com pression of peduncles</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>Normal, then ↓, Pulse ↓</td>
<td>Dilated bilat., not fixed to light</td>
<td>Normal</td>
<td>Irreg. 188</td>
<td>Rt. subdural hematoma.</td>
<td>Uncal herniation with brain stem to lt., slightly buckled dorsally</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>Progressive ↓</td>
<td>Reacted 8 hrs. Progressive ↓</td>
<td>Progressive dilatation rt., fixed to light</td>
<td>↑ lt.</td>
<td>Rate ↑ 151</td>
<td>Brain edema, lacerated uncal processes bilat. Compression of midbrain; stretched rt. VI N. &amp; post. cerebral; pressure cone</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>↓, then ↓</td>
<td>Died 12 hrs.</td>
<td>Constriction, progressive dilatation</td>
<td>Flaccid</td>
<td>Progressive ↓ rate 146</td>
<td>Brain edema, compression both III N., cerebellar tonsillar herniation</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>4</td>
<td>↓, then ↓, then stable</td>
<td>Reacted 4 hrs. Unconscious last 8 hrs.</td>
<td>Progressive dilatation, fixation rt.</td>
<td>↑ lt. Spastic hemiparesis Reg. 160</td>
<td>Hydrocephalus; compression of aqueduct from sides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>5</td>
<td>↓, then normal</td>
<td>Reacted 3 hrs. Neg. Neg. Neg.</td>
<td>Neg. 68</td>
<td>Cerebral edema, hydrocephalus, compression of aqueduct</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>5</td>
<td>No change</td>
<td>Reacted 40 min. Neg. Neg.</td>
<td>Slight ↑ rate 74</td>
<td>Slight cerebral edema, minimal dilatation of ventricles. Tentorial edges snug at midbrain; no herniation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>4</td>
<td>Normal, then ↓</td>
<td>Reacted 3 hrs. Rt. dilated 2nd day</td>
<td>Rt. ↓ 2nd day Rate ↓ 2nd day 122</td>
<td>Small lt. subdural hematomas, lt. uncal herniation, midbrain to rt., cerebellar pressure cone. Compression vein of Galen against splenium</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total 5 day survivals = 4

4 day survivals = 2
3 day survivals = 1
2 day survivals = 1
1 day survivals = 1
0 day survivals = 4

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* Normal = 150/90 mm. Hg
↑ Normal = 50-80/min.
↑↑ Normal = 50-110 mm. H2O
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#### TABLE 2

**Experimental group**

<table>
<thead>
<tr>
<th>No.</th>
<th>Survival (days)</th>
<th>B.P. Post-Trauma*</th>
<th>Consciousness Time of Reaction</th>
<th>Pupils</th>
<th>Deep Tendon Reflexes</th>
<th>Resp.†</th>
<th>Basal Cistern Pressure ‡</th>
<th>Autopsy Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>Slight ↓ then normal</td>
<td>Reacted 80 min.</td>
<td>Constricted, then normal</td>
<td>Slight ↑ rt.</td>
<td>Reg.</td>
<td>92</td>
<td>Minimal cerebral edema</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>Wide pulse pressure (systolic elevation)</td>
<td>Reacted 60 min.</td>
<td>Slightly constricted</td>
<td>Neg.</td>
<td>Reg.</td>
<td>55</td>
<td>Bilateral uncal swelling; no compression of structures in tentorial notch</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>Slight ↓ then ↑</td>
<td>Reacted 4 hrs., late drowsiness</td>
<td>Slight ↓ dilation, lt.</td>
<td>Slight ↓ rt.</td>
<td>Slight rate</td>
<td>95</td>
<td>Generalized cerebral edema. No pressure cone</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>↑ then stable</td>
<td>Reacted 7 hrs., progressive coma</td>
<td>Bilat. dilation (rt. later)</td>
<td>Slight bilat. ↑ (rt. later)</td>
<td>Irreg rate ↓</td>
<td>142</td>
<td>Edema, uncal swelling, compression of peduncles &amp; III N. bilat. more on rt.</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>Acute shock</td>
<td>Died 7 hrs.</td>
<td>No reaction from anesthesia; urinary incontinence</td>
<td>Lt. dilated, fixed</td>
<td>Decrebrate rigidity</td>
<td>Rapid, shallow</td>
<td>92</td>
</tr>
<tr>
<td>6</td>
<td>4</td>
<td>Slight ↓ then ↑</td>
<td>Reacted 1.5 hrs., progressive loss of consciousness</td>
<td>Normal early, bilat. dilation late, not fixed</td>
<td>Regular, late ↑ shallow</td>
<td>114</td>
<td>Cerebral edema; no deformities noted</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>5</td>
<td>Slight ↓ then normal</td>
<td>Reacted 50 min.</td>
<td>Neg.</td>
<td>Neg.</td>
<td>Neg.</td>
<td>87</td>
<td>No abnormalities</td>
</tr>
<tr>
<td>8</td>
<td>4</td>
<td>↑ progressive ↓</td>
<td>Reacted 68 min.</td>
<td>Progressive bilat. dilation</td>
<td>Progressive ↓ greater on rt.</td>
<td>Progressive ↓</td>
<td>150</td>
<td>Large lt. subdural hematoma. Uncal swelling with pressure on lt. peduncle; midbrain pushed to rt. Both III Ns. slightly stretched lt. more than rt., but rt. bent over post. cerebral artery</td>
</tr>
<tr>
<td>9</td>
<td>5</td>
<td>Neg.</td>
<td>Reacted 90 min., normal thereafter</td>
<td>Neg.</td>
<td>Neg.</td>
<td>Early ↓, returning to normal in 3 days</td>
<td>78</td>
<td>Minimal cerebral edema. (Absence of tentorium must allow greater vertical mobility of cerebrum, thereby reducing superficial cerebral damage at site of trauma)</td>
</tr>
<tr>
<td>10</td>
<td>5</td>
<td>Early ↓ then ↑</td>
<td>Reacted 165 min.</td>
<td>Slight dilation throughout course</td>
<td>Slight ↓ lt.</td>
<td>Neg.</td>
<td>91</td>
<td>Microscopic edema only. (Question of early irritation of III Ns. which should produce constriction of pupils unless permanent damage ensued)</td>
</tr>
<tr>
<td>11</td>
<td>4</td>
<td>↑</td>
<td>No reaction</td>
<td>Both constricted</td>
<td>Slight ↑ lt.</td>
<td>Irreg. Normal rate until late, then ↑</td>
<td>110</td>
<td>Severe laceration rt. temp. lobe; clot compressing rt. peduncle; uncal herniation toward midbrain on rt., pushing it to lt. despite tentorial cutting</td>
</tr>
<tr>
<td>12</td>
<td>5</td>
<td>Neg.</td>
<td>Reacted 43 min.</td>
<td>Neg.</td>
<td>Early facial paralysis recovered</td>
<td>Neg.</td>
<td>54</td>
<td>Negative</td>
</tr>
<tr>
<td>13</td>
<td>5</td>
<td>Slight ↑</td>
<td>Reacted 70 min.</td>
<td>Neg.</td>
<td>Neg.</td>
<td>↑ early, then normal</td>
<td>66</td>
<td>Negative</td>
</tr>
</tbody>
</table>

**Totals**

- 5 day survivors = 8
- 4 day survivors = 2
- 3 day survivors = 1
- 2 day survivors = 1
- 1 day survivors = 0
- 0 day survivors = 1

- 13

* Normal = 150/50 mm. Hg
† Normal = 30-38/min.
‡ Normal = 50-110 mm. HgO
pulse rate, which occurred in only one control. Representative blood pressure responses are presented in Figs. 6 and 7. Neurological signs other than blood-pressure changes and alterations in the level of consciousness included pupillary constriction or dilation, and alterations in muscle tone and deep tendon reflexes varying from flaccidity (usually associated with profound shock) to spastic hemiparesis or decerebrate rigidity.

Microscopic examination was made (eosin-methylene blue stain) of representative cortical sections in animals who presented “negative findings” at autopsy.

Structures in the tentorial notch that were most affected by uncal herniation were the midbrain (pushed to one side or buckled dorsally), the 3rd cranial nerves, posterior cerebral arteries, and the cerebral peduncles. In one case the vein of Galen was occluded. When cerebellar “tonsillar” herniation was present the medulla was also crowded.

![Fig. 6. Blood pressure during trauma. Arrows indicate application of trauma in all figures.](image)

(A) Control dog No. 7. Note darkening at top of record which on increasing speed of paper is seen to represent prolongation of systolic ejection phase. Died 4 days.

(B) Experimental dog No. 4. Oscillations are seen in some animals during light Nembutal anesthesia. The cause is unknown. They are not related to respiration. Died 3 days.

(C) Experimental dog No. 8. Decreased amplitude of systolic ejection phase after trauma. Died 4 days.

(D) Control dog No. 6. Died 12 hours after trauma.
DISCUSSION

Sunderland\textsuperscript{10} has recently published a beautiful series of anatomico-clinical studies in humans, relating pressure to the neurological signs. These include changes (as found in animals) in consciousness (mesencephalic compression), pupillary constriction or dilation (III N. irritation or com-
pression), contralateral and/or ipsilateral corticospinal signs (cerebral peduncles), visual disturbances (posterior cerebral artery), and less common syndromes caused by involvement of the trochlear and abducesens nerves, and the vein of Galen. The tentorium is covered by the cisterna ambiens and basalis and filling of these with brain is caused not by local swelling but by displacement, primarily mechanical. The degree of displacement permitted by the falx, the infundibulum, the basal attachments of the brain, the fiber tracts and blood vessels, have been described by Žertag. Zülch reviewed the gross and microscopic differences between brain edema and swelling. Following trauma, edema of the brain stem is often more prominent than brain swelling. Brain swelling produces an increase in volume which can lead to a shift in intracranial structures and symptoms of herniation. Subsequent circulatory changes may be hyperemic or infarctive. In cases of trauma brain swelling may occur in slight degree in “primary” form, or may be secondary to edema.

Displacement probably begins when the brain yields locally to increasing pressure. Adjacent portions of the ventricles are deformed and the reserve space of neighboring cisterns and overlying sulci is filled with brain. The shift often goes beyond the midline of the cisterns and indents the corresponding portion of the opposite hemisphere. The local increase in pressure is not confined to the homolateral hemisphere but apparently extends into the opposite hemisphere by displacement of tissue between the edge of the falx and the base. Finally the pressure is transferred axially from structures above the tentorium to those beneath it, leading to herniation of the cerebellar tonsils into the foramen magnum. When pressure is increased first in the posterior fossa, upward displacement through the incisura tentorii is possible. A moderate amount of sagittal displacement is possible along the axis of the brain stem from the medulla to the region of the quadrigeminal plate. This is particularly important in the symptomatology of axial displacement of the brain stem in dogs. Anatomical relationships between the intact tentorium and the cisternal spaces give rise to different forms of internal herniation. Displacements in the interhemispheric cisterns occur mainly in the anterior portion. Posteriorly, to reach significant size they must follow dorsal injuries which have displaced the corpus callosum downward. If there is equal increase in volume of the two hemispheres, lateral displacement will be absent, but the corpus callosum may be pressed upward against and even transected by the lower edge of the falx. Herniation at the incisura tentorii and the foramen magnum produces midbrain compression (temporal pressure cone) or constriction of the medulla oblongata (tonsillar pressure cone). Occlusion of veins draining the midbrain and pons results in venous stasis or infarction by compression. The vascular changes may be of greater importance than the presence of swollen brain tissue per se against the brain stem. In herniation into the cisterna ambiens, the opposite border of the midbrain may be
pressed against the tentorial edge, becoming indented and subsequently softened hemorrhagically (Kernohan’s tentorial notch: ipsilateral pyramidal syndrome).

In occlusion of the aqueduct, the suprapineal recess can expand and push its way under the tentorium against the superior cerebellar vermis. The brain substance in dogs in this situation may perforate at various places, most frequently on the medial wall of the trigonum of the lateral ventricle, and the cerebrospinal fluid escapes through the opening, forming a cyst of the subarachnoid space. This may extend into the cisterna ambiens in the direction of the cerebellum. It does not connect with the ventricles or subarachnoid space. In humans this is accomplished surgically by ventriculocisternostomy.\textsuperscript{13} In the cisterna ambiens temporal lobe gyri may pass through the incisura tentorii, displacing the midbrain to the opposite side. In dogs hemorrhagic infarcts in the medial aspects of the occipital lobes may follow this. In some cases uncal grooving by the edge of the tentorial notch resulted in a linear laceration of the temporal lobe and complete separation of tissue medial to the groove. In humans, the lateral displacement of the midbrain may be greatest at the tentorial level.\textsuperscript{4,5}

The acute quadrigeminal syndrome, consisting in attacks of decerebrate rigidity, usually follows increased pressure from below (posterior fossa injuries). This can also occur in downward displacement with a large tentorial pressure cone.\textsuperscript{9} The cerebellar pressure cone is a herniation into the cisterna magna which produces grooving or definite displacement of the cerebellar tonsils, or transforaminal displacement of an elongated tonsillar cone, most marked in young people because of pliability of the bony canal. Parts of the hippocampus and uncus can be pressed into the basal cisterns, forcing the peduncle toward the other side and compressing the third nerve. Infarct-like lesions in dogs occurred in all these herniations depending on contributory factors, for example, impairment of circulation at the calcarine region following herniation into the cisterna ambiens.

"Intracisternal brain swellings" are “indicators” of shifting processes,\textsuperscript{2,3,9,11} where the advancing portions of the brain suffer distortion as they are caught against dural folds. The cisterns partially protect the brain from sharp dural and bony edges by their fluid cushion, allowing a certain degree of mobility of parts of the brain in their vicinity.

SUMMARY

1. Twenty-six dogs, half of whom had had their tentorii cerebelli previously cut bilaterally, were subjected to a controlled cranial trauma.

2. The immediate response, clinical course, survival time and autopsy findings were significantly more favorable in animals whose tentorii were cut. The procedure does not lower significantly the incidence of cerebellar pressure cone, but apparently the pressure cone can be distributed in a way that is less lethal than with an intact tentorium.
3. The dynamics of mechanical displacements of the brain with respect to the tentorium are discussed, as well as correlations between structures related to the tentorial opening, and clinical signs.

The valuable advice of Drs. John Halloran and Peter Brandt, and the technical assistance of Mr. Michael Halloran, are greatly appreciated.

REFERENCES