INTRODUCTION

In recent years there has been an increasing prejudice against lumbar puncture, from both the profession and the laity; neurosurgeons, especially, have protested against this procedure in cases showing evidence of increased cerebral pressure and in expansive posterior fossa lesions, and have asserted that there is danger of herniation of posterior fossa contents into the foramen magnum with medullary pressure and serious or fatal outcome. To quote Cushing 1 in this connection as far back as 1909: 'The withdrawal of cerebrospinal fluid from the spinal meninges by a lumbar puncture is often hazardous, as it may tend to a sudden wedging of the bulb in the opening, with anemia and paralysis of the vital centres.' This statement has not been proved by factual evidence; it by no means follows that the presence of constricted cerebellar tonsils in the foramen magnum, found either operatively or at necropsy following lumbar puncture, is directly due to lumbar puncture, and much less follows that such a condition is due to a sudden wedging of the posterior fossa contents. Moreover, this cerebellar annulation is found in cases not punctured. It is rather here contended that such anatomical findings attributed to puncture are due to brain œdema, and when present take place slowly and not suddenly. No experimental evidence of movement of posterior fossa contents by fluid pressure variations between communicating ventricles, cisterna, and subarachnoid spinal spaces is reported, even in distortions of the posterior fossa. The diagnostic importance of cerebrospinal fluid analysis is unquestioned and often of deciding influence when operative intervention is considered. It is the purpose of this paper to bring forth evidence that a spinal tap performed according to a strictly defined technique is a reasonably safe and justifiable procedure in cases of intracranial hypertension. The alternative of ventricular puncture, a major operative procedure, entailing as it does skull trephine and brain puncture, is not to be lightly considered; moreover, the mortality of ventricular puncture is admittedly higher than any statistics following lumbar puncture. I should like to

* From the Division of Neuropsychiatry, Stanford Medical School. Read before the Neuropsychiatry Section of the California Medical Association at its Sixty-second Annual Session, Del Monte, April 24—27, 1933.
emphasize that a neurological complication following puncture is not necessarily due to puncture, and shall quote examples in confirmation thereof.

In the early part of 1932 I proposed to Dr. Jules H. Masserman, then Senior Resident in Neuropsychiatry at the Stanford Medical School, that he should undertake experiments directed, firstly, to the determination of the presence or absence of posterior fossa movement in fluid pressure variations, and secondly, the explanation of the so-called herniation of the cerebellar tonsils in the foramen magnum, in order to clear up conflicting experience and controversial points. These detailed experiments, largely due to the original ideas and painstaking labour of Dr. Masserman, have formed the subject of two papers recently submitted jointly by us to the Archives of Neurology and Psychiatry and soon to be published. Briefly, these experiments were as follows.

**EXPERIMENTAL DATA**

I. **ANATOMIC INVESTIGATIONS.**—These were made—

(a) To obtain roentgenograms of the human ventriculo-subarachnoid system under various degrees of fluid pressure.

(b) To investigate the effects of rapid fluid drainage of one portion of the system on the manometric pressures in other portions.

(c) To study the effects of artificially produced distortions of the brain on intracranial hydrodynamics.

Fresh cadavers were prepared as follows: The cranial circulation was ligated, and its pressure hydrostatically maintained. Needles were inserted into the lateral ventricles, basal cistern, and lumbar spine, and each needle was connected to a manometer. The cerebrospinal fluid was then replaced by a radiopaque solution, and control roentgenograms of the skull taken to show the outlines of the ventriculo-subarachnoid system under various degrees of fluid pressure. At each pressure level lumbar and cisternal drainages were performed, and possible movements of the intracranial contents studied by comparing subsequent encephalograms with the control. During each drainage, also, the manometric readings in other portions of the ventriculo-subarachnoid system were recorded, and the descending pressure curves plotted. Various parts of the brain were then distorted in situ by the injection of melted parowax, and the effects of such distortions on intracranial hydrodynamics studied by the above methods. The conclusions were as follows:

1. The lateral ventricles and the cisternal and lumbar subarachnoid spaces normally remained in free communication under all experimental pressure variations.

2. Rapid cisternal or lumbar release of high intracranial pressures did not produce movements of the tentorium, cerebellum, or medulla.

3. Distortion of a cerebellar lobe forced it down against the medulla and caused a partial hydrodynamic block between the lateral ventricles and the lumbar sac.
II. The Influence of Rapid Reduction of Intracranial Pressure on the Occurrence of Brain Oedema.—This investigation is an attempt to evaluate the possible pathophysiological effects of rapid fluid decompression of the ventriculo-subarachnoid spaces.

A. Pairs of dogs were similarly anesthetized and their intracranial pressures slowly raised by hydrostatic means from normal to 750 mm. of water. The intracranial tension of dog (A) was kept at this level for four hours, whereas that of dog (B) was rapidly reduced after the first half-hour. At the end of the four-hour period sections were taken from each brain for study. The brains were then removed and desiccated. The series of (B) brains showed a more pronounced microscopic tissue oedema and a greater percentage of water loss by desiccation than did the corresponding series of (A) brains.

B. In nearly all cases the spinal fluid pressure returns to its original level within from three-quarters to three hours following lumbar drainage of 10–40 c.c. of fluid. The rise may then continue to give a cranial hypertension lasting a variable length of time.

If more than 35 c.c. of spinal fluid is rapidly removed and the pressure is then allowed to restore itself, a second drainage of the same amount of fluid will produce a greater drop of spinal fluid pressure than did the initial drainage. This may be interpreted to indicate an encroachment on the volume of the subarachnoid spaces, either by vascular engorgement or brain oedema.

CLINICAL DATA

One hundred and three unselected cases of lumbar puncture in cases of intracranial pressure or in cases of suspected intracranial pressure are here reported, including those showing some degree of papilledema. These latter, 12 in number, may or may not be relevant to the series, but have been included because of the considerable importance of this symptom in the cranial conditions under discussion. The following tabulations from the detailed charted records of these cases will present in concise form the pathological states and sequelae:

**LUMBAR PUNCTURES**

<table>
<thead>
<tr>
<th>Total Cases</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verified tumours</td>
<td>58</td>
<td>56</td>
</tr>
<tr>
<td>Diagnosed tumours, unverified</td>
<td>28</td>
<td>27</td>
</tr>
<tr>
<td>Brain abscesses, verified</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Papilledema</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td><strong>Total Cases</strong></td>
<td><strong>103</strong></td>
<td><strong>100</strong></td>
</tr>
<tr>
<td>Verified Tumours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supratentorial</td>
<td>39</td>
<td>67</td>
</tr>
<tr>
<td>Midbrain</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Subtentorial</td>
<td>17</td>
<td>29</td>
</tr>
<tr>
<td>Cerebellopontile angle</td>
<td>6</td>
<td>10</td>
</tr>
</tbody>
</table>
THE PROPRIETY OF DIAGNOSTIC LUMBAR PUNCTURE

VERIFIED TUMOURS—Continued

<table>
<thead>
<tr>
<th>Condition</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choked disc, measured</td>
<td>16</td>
<td>28</td>
</tr>
<tr>
<td>Papilloedema</td>
<td>37</td>
<td>64</td>
</tr>
<tr>
<td>Spinal fluid pressure increased</td>
<td>23</td>
<td>40</td>
</tr>
<tr>
<td>Pressure normal</td>
<td>18</td>
<td>31</td>
</tr>
<tr>
<td>Pressure not noted</td>
<td>16</td>
<td>29</td>
</tr>
</tbody>
</table>

SEQUELÆ

| Incidence of symptoms (all)         | 11     | 11         |
| Severe headache                     | 4      | 4          |
| Nausea and emesis                   | 2      | 2          |
| Convulsions                         | 1      | 1          |
| Death                               | 4      | 4          |
| None                                | 92     | 89         |

DEATHS FOLLOWING LUMBAR PUNCTURE

Case I.—425, female child, age 4½ years. Fundi showed papilloedema. Spinal fluid pressure not noted; 4 c.c. withdrawn. No initial symptomatology or shock. Death in 13½ hours, preceded for half an hour by convulsions and bulbar failure. Necropsy.—Rupture of highly vascular sarcoma in left anterior thalamus. 'Cerebellum, pons, and medulla normal.'

Case II.—392, female child, age 8 years. Fundi showed choked discs. Spinal fluid pressure greatly increased, 2 c.c. withdrawn. Condition before puncture grave, cyanosis and cardiac complications, headache. Death in six hours. Necropsy.—Small orange-sized cystic tumour mass, right parietal region; base of brain normal.

Case III.—88,593, male, age 36 years. Diagnosis: Tuberculous meningitis. No mention of fundus examination. No definite neurological findings. Maniacal. Lumbar puncture done, 10 c.c. withdrawn, 40 mm. pressure. Became more maniacal. Second puncture two days later, 30 c.c. withdrawn, pressure not noted. Died 17 hours later. Necropsy.—Syphiloma of brain, floor of third ventricle. No mention of posterior fossa herniation.

Case IV.—69,649, male, age 50. Diagnosis: Cerebrospinal syphilis; one positive spinal fluid. Fundi normal. First lumbar puncture half an hour after 0·6 gm. arsphenamine intravenously; pressure 17·5 cm., 25 c.c. withdrawn. Second puncture 5½ hours later, pressure 17·5 cm., amount not noted. Third puncture half an hour after 0·6 gm. arsphenamine 16 days later, pressure 8·5 cm., 20 c.c. withdrawn. Fourth puncture 5½ hours later, pressure 10·5 cm., 20 c.c. withdrawn, 'considerable reaction.' Fifth puncture five days later, pressure not noted, 30 c.c. fluid withdrawn, replaced by mercurialized serum. Sixth puncture four hours later, pressure 18 cm., 18 c.c. withdrawn. 'Marked systemic reaction.' Three hours later complained of severe headache, soon had to be given artificial respiration. Died 40 hours after puncture. Necropsy.—Cystic glioma, right hemisphere of cerebellum, 5 cm.
in diameter. 'On lower surface of the cerebellum there are cone-shaped projections on each side of the medulla, the one on the right is longer and measures about 2½ cm. in length and projects about 2 cm. from cerebellum. Corresponding process on the left measures 1½ by 1 cm.'

DEATHS IN CASES WHERE PUNCTURE WAS POSTPONED

Case I.—584, single male adult. Symptoms: dystonia, occipital pain and rigidity. Impression: tuberculous meningitis. Course in hospital: died the morning after day of entry. Necrospy.—Tumour $5 \times 3$ cm. in right cerebellar hemisphere.

Case II.—3,123, single female, age 14 years. Symptoms: sudden onset of occipital headache, left-sided weakness, right third nerve palsy. Course in hospital: became comatose, died $5\frac{1}{2}$ hours after examination. Necrospy.—Hæmorrhage into right lateral ventricle.

DISCUSSION

Of the four deaths following lumbar puncture, only one occurred in a posterior fossa lesion, a pathological locus supposed to be particularly favourable to puncture accidents. This death occurred in one of the two cases which fulfilled the foregoing experimental postulates of improper lumbar decompression and drainage producing vasomotor shock and brain œdema; therefore these two cases cannot be reasonably included in a series of technically proper lumbar punctures and have been retained in the series because of the inclusion of every case of suspected expanding lesion. The remaining two cases of deaths both showed supratentorial lesions and the puncture may not have influenced the fatalities. That this point of view is not unreasonable is indicated by the two cases reported which died before a contemplated puncture; had a puncture been done in either of these cases it would doubtless have been incriminated with the fatality, as were the two punctured cases on the 'post hoc' premise. In none of the fatal punctured cases was death sudden, as would be expected by sudden wedging of posterior fossa contents into the foramen magnum with paralysis; the shortest time interval was six hours (P 392).

Acusticus tumour is considered to favour untoward sequelæ from puncture by dislocation of posterior fossa contents. Six such tumours were punctured in the series with no alarming after-effects or fatalities.

A number of the cases showed remarkably high intracranial pressures ($P$ 1,229, 90·0 cm. (of $H_2O$); $P$ 2,467, 85·0 cm.; $P$ 4,266, 72·0 cm.; 58,145, 60·0 cm.), indicating communication between the cerebral ventricular-subarachnoid and spinal spaces, and the absence of a foraminal block, as has been imagined in the presence of such degrees of pressure.

The literature on the subject shows a surprisingly small incidence of immediate death following lumbar puncture as compared with the total
THE PROPRIETY OF DIAGNOSTIC LUMBAR PUNCTURE

fatalities, and indicates that if lumbar puncture plays a rôle it is very doubt-
fully concerned with sudden wedging of the bulb, which would produce
alarming or fatal results. Thus, in a 20-year period, 1895–1915, Schönbeck in a comprehensive article practically covering the lumbar puncture period up to the time of his publication reported 71 fatalities, amongst which 87 were in tumours (11 infratentorial) and 13 due to hæmorrhage. In 10 cases death occurred after such an interval after puncture as to render the relationship questionable; in seven cases of immediate death the majority were associated with hæmorrhage. In one internal case (Curschmann) a tuberculoma was found pressing on the floor of the fourth ventricle. In only three cases was wedging of the cerebellum reported in the posterior fossa.

An important article by Masson reports punctures in 94 cases of verified intracranial tumours (82 infratentorial) without a fatality, and but one death (22-hour interval) in an unverified tumour series of 106 cases.

The fatalities of Schönbeck in the series here reported reveal a surprising predominance of supratentorial over infratentorial fatalities. In 40 punctured cases of verified brain tumour reported by Comfort the only fatality possibly related to puncture occurred in rupture and hæmorrhage of a frontal tumour. Fremont-Smith and Putnam recently discussed the present status of lumbar puncture as performed in the Boston City Hospital and stressed the small number of reported fatalities in recent years. They call attention to the value of very refined methods of diagnosis in early cases. In the discussion Horrax agreed that the danger was very slight with a low grade of choking. Puusepp reports 18 infratentorial tumours punctured without accident.

The considerable difference in the fatal statistics of the series of Schönbeck and of Masson represents in the Schönbeck series many cases punctured without the precautions and refinements of diagnostic technique that have been gained by experience and also many cases of actual decompression, and in the Masson series a purely diagnostic procedure carefully and intelligently done. This leads to the consideration of the technique of a properly performed lumbar puncture.

The patient should lie in a horizontal side position and the needle should not be of larger bore than 19 gauge. A mechanical manometer, as that of Claude, is preferable to a calibrated tube because of better fluid manometric control. At the first pressure reading jugular compression should be made to determine the patency of the craniospinal pathways; should the fluid pressure not vary by compression or release, indicating a craniospinal block, fluid should not be withdrawn; also, particular care should be observed when the fluid is xanthochromic, which may indicate a soft and vascular ventricular or surface tumour and danger of hæmorrhage. If the fluid pressure responds to jugular compression this test should be repeated after 2·5 c.c. are withdrawn, and the normal pressure, 15·0 cm., should not be reduced to lower than 10·0 cm. and an abnormally high pressure by not more than 50 per cent. No more than 5 c.c. of fluid should be withdrawn in tumour suspects, and less if any
untoward symptoms develop, such as pain, respiratory, or pulse variations. *Fluid withdrawal should be done slowly, drop by drop.* After the puncture the patient’s body should be inclined so that the head depends and kept so for twenty-four hours.

A satisfactory fluid analysis may be made with 5 c.c. of fluid. To quote Dr. H. A. Wyckoff, Director of the Stanford Clinical Laboratory:

- The minimum quantity of cerebrospinal fluid which will suffice for a fairly complete diagnostic examination is 4·8 c.c., used as follows:
  - Wassermann—2 c.c. (qualitative).
  - Colloidal gold—0·1 c.c.
  - Protein, total—1·0 c.c. (quantitative).
  - Globulin—0·7 c.c. (Nonne and Noguchi).
  - Sugar—1·0 c.c. (quantitative), making a total of 4·8 c.c.

This amount requires the utmost care in pipetting and does not allow for repeating any of the tests, which is sometimes desirable. There are still other reasons why it is very undesirable to have such small quantities of fluid with which to work. I earnestly advise that at least twice this amount of cerebrospinal fluid (say 10 c.c.) be sent whenever the nature of the case permits.

**SUMMARY AND CONCLUSIONS**

1. Clinical and experimental evidence is here presented to support the contention that there is no proof of sudden wedging or movement of the posterior fossa contents into the foramen magnum following spinal tap in open craniospinal pathways, even in the presence of increased intracranial pressure. There is, however, reason to believe that so-called herniation is an expression of general brain swelling and oedema and may be due to changes in intracranial pressure and vasomotor paralysis and shock from various causes. Sudden and excessive fluid withdrawal by spinal tap may produce such oedema, as well as similar withdrawal of fluid from other parts of the cerebrospinal fluid system.

2. Of the four deaths in the series two can be excluded as due to properly performed diagnostic punctures; two may or may not have been determined by diagnostic punctures.

3. The incidence of post-puncture accidents is greater in supratentorial than infratentorial tumours and the incidence of sudden death is greatest in vascular lesions.

4. Admitting that even a properly performed lumbar puncture may in exceptional cases hasten the course of a prognostically serious and often hopeless pathological condition found in intracranial hypertension (as in Case no. 425 of this series), this is not a just criterion by which to designate the procedure as unsafe; rather, the accumulated experience of late years would justify the opinion that when properly done the procedure is reasonably safe, because of the low mortality—only two reported cases (Putnam) in
recent years—and justifiably indicated because of the valuable diagnostic information it affords.

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The Propriety of Diagnostic Lumbar Puncture in Intracranial Hypertension

Walter F. Schaller

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