How Idiopathic is Idiopathic Intracranial Hypertension?

Dr Grant Bateman MD
Director MRI
John Hunter Hospital
Newcastle Australia
Definition of IIH

- **Idiopathic intracranial hypertension**: (Pseudotumour cerebri, BIH)
  - overweight middle aged females
  - headache / papilloedema / visual obscuration
  - CSF pressure (>25cm h$_2$o) with normal composition

- Normal CT / MRI or the suggestion of slit like ventricles

- Summary; An elevated CSF pressure of undisclosed cause.
Standard MRI findings

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They all have elevated sagittal sinus pressure

Elevated intracranial venous pressure as a universal mechanism in pseudotumor cerebri of varying etiologies Dean G. Karahalios, MD, Harold L. Rekate, MD, Mazen H. Khayata, MD and Paul J. Apostolides, Md

Upstream pressure depends on the flow, resistance and downstream pressure

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MRV findings

- Thrombosis 10%
- Stenosis (fixed or dynamic) 75%
- Patent sinuses 15%
1. Thrombosis of Sinuses

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2. Stenosis of Sinuses: fixed stenoses
Stenosis of sinuses: Dynamic stenoses

Symptomatic IIH

Asymptomatic after lumbar puncture.

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How significant are Dynamic stenoses?

37 mmHg

No gradient

21 mmHg
Mixed disease?

Department of Neurology, Royal Prince Alfred Hospital, Sydney, Australia.

RESULTS:
Before stent placement, the mean superior sagittal sinus pressure was 34 mm Hg (462 mm H(2)O) with a mean transverse sinus stenosis gradient of 20 mm Hg. The mean lumbar CSF pressure before stent placement was 322 mm H(2)O. In all 52 patients, stent placement immediately eliminated the TSS pressure gradient, rapidly improved IIH symptoms, and abolished papilledema. In 6 patients, symptom relapse (headache) was associated with increased venous pressure and recurrent stenosis adjacent to the previous stent. In these cases, placement of another stent again removed the transverse sinus stenosis pressure gradient and improved symptoms. Of the 52 patients, 49 have been cured of all IIH symptoms.

The mean jugular bulb pressure was 14 mmHg!

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3. Patent Sinuses
Patent sinuses: 2 populations?

Resolution of pseudotumor cerebri after bariatric surgery for related obesity

Case report

**TRIMURTI NADKARNI, M.D., HAROLD L. REKATE, M.D., AND DONNA WALLACE, R.N., M.S., C.P.N.P.**

Division of Neurological Surgery, Barrow Neurological Institute, St. Joseph’s Hospital and Medical Center, Phoenix, Arizona

**TABLE 1**

Dural venous and right atrial pressures in two obese patients with pseudotumor cerebri*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Weight (lb)</th>
<th>SSS†</th>
<th>TS (rt/lt)</th>
<th>SS (rt/lt)</th>
<th>JB (rt/lt)</th>
<th>IJV (rt/lt)</th>
<th>INJ</th>
<th>SVC</th>
<th>RA</th>
<th>IVC</th>
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<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td>13/17</td>
<td>12/16</td>
<td>12/16</td>
<td>9/15</td>
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<td>16</td>
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<td></td>
<td>preop</td>
<td>227</td>
<td>17</td>
<td>13/17</td>
<td>12/16</td>
<td>12/16</td>
<td>9/15</td>
<td>16</td>
<td>16</td>
<td>16</td>
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<tr>
<td></td>
<td>postop</td>
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<td>9/8</td>
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<td>8/8</td>
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<td>6</td>
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<td>300</td>
<td>28</td>
<td>21/21</td>
<td>15/19</td>
<td>15/18</td>
<td>13/16</td>
<td>14</td>
<td>12</td>
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</tr>
<tr>
<td></td>
<td>preop</td>
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</tr>
<tr>
<td></td>
<td>postop</td>
<td>170</td>
<td>16</td>
<td>14/13</td>
<td>8/12</td>
<td>8/10</td>
<td>8/10</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>

* All values are presented in millimeters of mercury. In the transverse and sigmoid sinuses and in the jugular bulb and internal jugular vein, pressures were measured on the right side (rt) and the left side (lt). Abbreviations: IJV = internal jugular vein; INV = innominate vein; IVC = inferior vena cava; JB = jugular bulb; RA = right atrium; SS = sigmoid sinus; SVC = superior vena cava; TS = transverse sinus.

† The SSS pressure is a mean of pressures measured at the anterior, middle, and posterior SSS.

Pressure = flow x Resistance

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**Blood Flow Results**

<table>
<thead>
<tr>
<th></th>
<th>Total flow mLs/ min</th>
<th>SSS flow mLs/ min</th>
<th>SSS flow as a % of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal n=14</td>
<td>880</td>
<td>415</td>
<td>48</td>
</tr>
<tr>
<td>Thrombosis n=12</td>
<td>900</td>
<td>240</td>
<td>27</td>
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<tr>
<td>Stenosis n=32</td>
<td>1020</td>
<td>360</td>
<td>35</td>
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<td>Patent n=9</td>
<td>1360</td>
<td>440</td>
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</tbody>
</table>

Pressure = Flow x Resistance

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Does elevated venous pressure alter cerebral blood flow?


Jugular ligation does not increase intracranial pressure but does increase bihemispheric cerebral blood flow and metabolism.

Chai PJ, Skaryak LA, Ungerleider RM, Greeley WJ, Kern FH, Schulman SR, Hansell DR, Auten RL, Mahaffey SF, Meliones JN.

There was a significant increase in right-side (44.7 +/- 2.0 vs. 38.8 +/- 2.4 mL/kg/min; p < .05) and left-side (42.9 +/- 2.3 vs. 38.7 +/- 1.9 mL/kg/min; p < .05) cerebral blood flow 5 mins after venovenous ligation when compared with baseline values.

Venous occlusion increases CBF by 20% above normal due to altered metabolism with more lactate

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Controversy Hyperemia

32 yrs old male
CSF pressure 35 cm H2O
CSF biochemistry and cytology normal.
Conclusions

- Idiopathic intracranial hypertension is caused by elevated venous pressure in all cases.
- Elevated venous pressure is due to thrombosis, stenosis, hyperemia, elevated central venous pressure or combinations.
- Is it still idiopathic?