Extrajugular pathways of human cerebral venous blood drainage assessed by duplex ultrasound

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THE INTERNAL JUGULAR VEINS (IJVs) are thought to represent the main outflow pathway for cerebral venous blood in humans. However, the clinical observation that bilateral resection of the IJV is usually well tolerated suggests the presence of alternative, nonjugular pathways (9). Such a route exists in the form of the anatomically complex vertebral venous system (1, 7, 8, 16). Part of this system are the vertebral veins (VVs), which have been shown to serve as venous collaterals in cases of jugular flow obstruction (5, 9). A recent ultrasound study of healthy volunteers demonstrated that the pattern of cerebral venous drainage changes, even under physiological conditions, depending on the body position (23). Whereas flow in the IJVs dominated in the supine position, a marked jugular flow reduction and a concomitant flow rise in the VVs were seen when the subjects changed into the erect body position. However, the magnitude of flow increase in the VVs did not match the decrease measured in the IJVs. The authors, therefore, posed the hypothesis that alternative pathways, like the spinal epidural veins as a part of the internal vertebral venous system, might have been the reason for the observed difference (23). In addition, the deep cervical veins and other ultrasound inaccessible parts of the external vertebral venous system have to be considered (1, 4). To study the potential role of the deep neck veins, venous blood volume flow (VBVF) was measured by duplex ultrasound in both VVs during bilateral IJV and during circular neck compression.

MATERIALS AND METHODS

The study was conducted in 12 volunteers [3 women, 9 men, age 29 ± 6 (SD) years], free of cardiovascular disease. All subjects gave informed consent. Color-coded duplex sonography was performed by using a 3- to 11-MHz linear transducer (HP Sonos 5500, Hewlett Packard). Insonation was carried out in a head straight and supine body position at a midcervical level during normal breathing of the probands. VBVF was calculated as described previously (13, 14, 20, 23) by multiplication of vessel area and time-averaged velocity (mean of all frequencies over two to three heart cycles) by using the HP Sonos 5500 software (Fig. 1). In case of marked respiratory variations of time-averaged velocity and vessel cross-sectional area (CSA), measurements were performed during brief apnea after a normal expiration. The CSA of the IJV was measured in the horizontal plane by using the B-mode image, whereas VV-CSA was calculated from VV diameters obtained in the sagittal plane, assuming a circular vessel shape. VBVF at rest was assessed in both IJVs and VVs. Additional VBVF measurements were then carried out in the VVs during a transient complete blood flow obstruction of both IJV, lasting ~1–2 min. Flow cessation, confirmed by duplex ultrasound, was achieved by applying a constant manual pressure on both IJVs at the submandibular level. A third VV flow analysis was performed during circular neck compression at the same level by using an elastic band. This procedure also led to IJV flow cessation. For statistical analysis, nonparametric repeated-measures ANOVA (Friedman test) and Dunn’s multiple-comparison posttest were performed. A P value of <0.05 was considered significant.

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Two volunteers (subjects 6 and 7) were additionally studied by venous magnetic resonance angiography (Magnetom Vision, 1.5 T, Siemens, Germany) during manual jugular vein compression to analyze whether flow changes in the nonjugular venous system could be visualized. For data acquisition, a saturated FLASH two-dimensional sequence was used (repetition time 33 ms, echo time 9 ms, one signal acquired, 3-mm slice thickness, 260-mm field of view, 192 × 256 pixel matrix).

RESULTS

All vessels were successfully insonated at rest and during the two modes of venous compression (Fig. 1). Compression tests were well tolerated, with subjects mainly noticing the applied neck pressure and some degree of facial swelling but no other side effects like headaches or visual disturbances. IJV flow cessation during circular neck compression was achieved by a mean neck circumference reduction of 4.5 cm (range 3–7 cm). Mean total venous outflow, calculated as the sum of VV and IJV volume flow at rest, was 766 ± 226 ml/min, ranging from 390 to 1,130 ml/min. The IJVs contributed 94% (720 ± 232 ml/min) and the VVs 6% (47 ± 33 ml/min) of total VBVF (Table 1). The following compression tests led to a significant VV flow increase (P < 0.0001). Bilateral manual IJV compression resulted in a VV flow of 128 ± 64 ml/min (P < 0.05), comprising now 17% of the total VBVF. The circular compression led to a further nonsignificant mean flow rise (186 ± 70 ml/min, P > 0.05, 24% of total VBVF). This increase, however, was only seen in 7 of the 12 subjects.

DISCUSSION

Although the extracranial portion of the arterial cerebral inflow has been investigated in detail by different modalities, only little attention has been paid to the venous drainage of the brain. The IJVs have been considered to present the most important pathway for venous blood returning from the brain. This assumption was based on angiographic studies and cerebral blood flow analyses with nitrous oxide, labeled erythrocytes, and thermodilution techniques (12, 21, 15, 25), which were all performed in a supine body position. However, anatomic investigations as well as clinical observations in patients after bilateral radical neck dissection suggest coexisting IJV-independent alternative venous drainage pathways (9).

Venous anatomy. The main jugular blood drainage pathway leads from the superior sagittal and the transverse sinuses via the sigmoid sinuses into the IJVs, which meet the superior cava vein via the brachiocephalic vein. Competent valves usually impede retrograde flow in the IJV. In contrast, the vertebral venous system forms a freely communicating, valveless “network” of longitudinal and transverse venous vessels. It consists of an internal part, the intraspinal epidural venous plexus, and an external paravertebral part, both continuing throughout the entire length of the spinal column. The system communicates with deep thoracic and lumbar veins, intercostal veins, azygos and hemiazygos veins, as well as with the inferior venous cavities.

Table 1. Individual IJV and VV blood volume flow measurements

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, yr</th>
<th>Gender</th>
<th>Total IJV at Rest, ml/min</th>
<th>Total VV at Rest, ml/min</th>
<th>Total VV comp 1, ml/min</th>
<th>Total VV comp 2, ml/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>F</td>
<td>450</td>
<td>110</td>
<td>180</td>
<td>320</td>
</tr>
<tr>
<td>2</td>
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<td>M</td>
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<td>50</td>
<td>100</td>
<td>100</td>
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<tr>
<td>3</td>
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<td>F</td>
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<td>30</td>
<td>M</td>
<td>440</td>
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<td>110</td>
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<td>5</td>
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<td>M</td>
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<td>20</td>
<td>160</td>
<td>240</td>
</tr>
<tr>
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<td>M</td>
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</tr>
<tr>
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<td>M</td>
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<tr>
<td>8</td>
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<td>M</td>
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<td>20</td>
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<td>130</td>
</tr>
<tr>
<td>9</td>
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<td>30</td>
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<td>40</td>
<td>140</td>
</tr>
<tr>
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<td>23</td>
<td>F</td>
<td>640</td>
<td>10</td>
<td>120</td>
<td>120</td>
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<tr>
<td>12</td>
<td>23</td>
<td>M</td>
<td>880</td>
<td>60</td>
<td>100</td>
<td>170</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>29 ± 6</td>
<td></td>
<td>720 ± 232</td>
<td>47 ± 33</td>
<td>128 ± 64</td>
<td>186 ± 70</td>
</tr>
</tbody>
</table>

IJV, internal jugular vein; VV, vertebral vein; M, male; F, female. Total IJV flow is shown at rest. Total VV flow is shown at rest and during bilateral manual IJV compression (comp 1) and circular neck compression (comp 2).
VVs and the deep cervical veins, which are located in the longitudinal part of the external vertebral venous system. VVs represent the main longitu-
dinal component of the external vertebral venous system. VVs, deep cervical veins, and the external jugular vein finally join the brachiocephalic vein. The CSA of the internal vertebral venous system at skull base level has been estimated to reach up to one-fourth of the combined jugular CSA, whereas the total CSA of the vertebral venous system might even surpass that of both IJVs (3, 4).

Ultrasound assessment of VBVF. In the midcervical region, IJVs and VVs are easily accessible by duplex ultrasound (11, 13, 14, 23). The presented VBVF measurements are in good agreement with previously obtained ultrasound data. Different research groups found a combined mean IJV flow of 740 ± 209 and 700 ± 270 ml/min (14, 23) and a mean VV flow of 40 ± 20 ml/min (23) in the supine body position. The accessible total mean venous outflow of 766 ± 226 ml/min in our group of healthy volunteers corresponds well with ultrasound assessed global arterial inflow, which has been reported within the range of ~500–950 ml/min (18–20) and with a mean value of 727 ± 102 ml/min for subjects aged 20–39 yr (17). Therefore, the IJVs have to be considered the main outflow pathways in the supine position. This, however, was shown to change completely when the subjects turned into the upright position, as IJV flow fell from 700 ± 270 to 70 ± 100 ml/min in a group of volunteers, whereas VV rose from 40 ± 20 to 210 ± 120 ml/min, leaving an unexplained remaining mean difference of ~450 ml/min (23). In our study, a complete IJV flow cessation was ensured by bilateral manual compression. This indeed caused the expected VV increase (mean 82 ± 57 ml/min). Interestingly, individual values varied greatly from 100 to 450 ml/min (subject 6) to one subject without a detectable flow rise (subject 7). Venous magnetic resonance angiography in subject 6 showed bilateral prominent IJVs at rest and a noticeable signal increase in both VVs as well as the deep cervical veins during IJV compression (Fig. 2). In contrast, subject 7 demonstrated multiple cervical veins at rest that showed a pronounced signal increase during compression. These veins probably explain why no ultrasonographic VV flow increase could be detected during the bilateral IJV compression. However, circular neck compression in this subject did result in a VV flow increase of 100 ml/min, confirming the predominating nonjugular pathway via the deep cervical veins. In contrast, nonjugular drainage patterns in subject 6 seem mainly to follow the VVs as fewer deep cervical veins could be visualized. Combined with a deeper location of the cervical veins, predominantly surrounding the splenius capitis muscles, circular neck compression probably did not result in any significant flow obstruction, explaining the absent VV flow rise. Over-

all, the additional cervical vein obstruction led to a further VV flow increase of 58 ± 53 ml/min (range: −10 to 140 ml/min), yielding VV flow values compensating 19% (one-fifth) of the jugular drainage capacity. A similar VV capacity of 26% (one-fourth of jugular venous flow) was seen within the upright body position (23).

For interpretation of these data, a number of technical limitations as well as anatomic-specific characteristics of the insonated vessels have to be considered. An unintentional concomitant carotid artery compression could, for instance, lead to a reduced arterial inflow and subsequent overestimation of the diverted venous flow. However, this seems very unlikely as the applied jugular pressure was low, and B-mode insonation of the carotid CSA during compression did not show vessel deformation. In our VBVF measurements, a circular-shaped VV was postulated for flow calculations, whereas real vessel CSA at rest is probably noncircular in most cases, changing into a more circular shape due to an intraluminal pressure rise during the compression tests (Fig. 2). This might have led to an under- or overestimation of VV flow, especially at
rest. Furthermore, known venous anatomic variations, like the doubling of VVs, might have been overlooked or missed in our approach. However, even considering these potential systematic errors, >50% of jugular blood flow seems to follow other routes so that the VVs are unlikely to be the main nonjugular drainage pathway. Our results suggest that the deep cervical veins represent one of the relevant additional alternative pathways, particularly as they turned out to be the most prominent veins besides the VVs on our MRI scans, all together probably extending the CSA of the VV. The only moderate further VV flow increase during circular neck compression (8% of total jugular flow) is probably due to a limited compression effect on deep cervical veins caused by their anatomic location (Fig. 2). This hypothesis is further supported by case reports of patients in whom intraspinal pressure studies were performed by lumbar puncture after bilateral radical neck dissection (22). Under application of bilateral manual pressure to the cervical muscle bed, the registered pressure in two patients rose from 22 and 25 cmH₂O to 65 and 60 cmH₂O, respectively (22).

Finally, the intraspinal epidural system, already demonstrating a flow increase during IJV compression on MRI, has to be considered. The distribution between these two compartments remains unclear, as no validated technique exists to directly assess blood flow within these regions.

In conclusion, we have shown in a group of young healthy volunteers that powerful additional venous pathways besides the IJVs exist and may compensate for IJV flow cessation. The VVs as important parts of the vertebral venous system are probably not the main nonjugular drainage pathway. Instead, the variably developed deep cervical veins as well as the intraspinal epidural venous system have to be considered. Knowledge of the individual venous drainage patterns might be of clinical relevance in patients undergoing IJV resection or patients suffering from raised intracranial pressure.

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REFERENCES


