REVIEW ARTICLE

Jugular Venous Reflux

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Cerebral venous outflow impairment is known to produce cerebral dysfunction in many clinical and animal studies. However, knowledge and understanding of the cerebral venous system is far less than that of the arterial system. The internal jugular vein, which is easily observed by color-coded Doppler sonography, is one of the main tracts for cerebral venous drainage. Recently, internal jugular venous reflux has been found to be related to several neurologic disorders. These associations suggest that the mechanism of these disorders is related to cerebral venous outflow impairment. In this article, we will briefly introduce the cerebral venous system and extracranial venous drainage pathway, then specifically review the characteristics of the internal jugular vein, its branches, and the hemodynamic factors involved in internal jugular venous reflux.

KEY WORDS — cerebral venous outflow impairment, cerebral venous system, jugular venous reflux, venous valve incompetence

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Cerebral Venous System (CVS)

Cerebral circulation encompasses both the arterial and venous system (CVS). The venous system contains approximately 70% of the blood volume, with approximately three-quarters contained within small veins and venules [1,2]. An alteration in cerebral venous volume would influence cerebral hydrokinetics and intracranial pressure [2,3]. In addition to cerebral venous drainage impairment, an alteration in cerebral venous volume would produce brain edema and/or decreased cerebral perfusion, leading to cerebral dysfunction [3–8]. Despite its significance in cerebral circulation, in contrast to the cerebral arterial system, the CVS is far less described and studied.

Jean-Martin Charcot (1825–1893), a great teacher and physician, created the foundations for neurology as an independent discipline and has had a phenomenal influence on contemporary neurology [9–11]. Charcot proposed a topographic distribution of brain diseases according to the distribution of cerebral arteries, but paid little attention to the CVS. In the first half of the 20th century, his students, also very celebrated neurologists, established the principle of neuropathology. Since then, extensive studies and observations have been carried out on the cerebral arterial system compared with those on the CVS. Insufficient description makes for a poor understanding of the physiology and pathology of the CVS, which might lead to the underestimation of cerebral venous disorders [3].

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Clearly, further basic science and clinical studies are needed to increase our knowledge and understanding of the CVS.

Cerebral Venous Blood Drainage

Venous drainage from the cerebral hemispheres consists of two systems, the superficial and the deep venous system [12–15]. The superficial system drains blood from the cortex and the superficial white matter via the cortical veins, and is collected by the dural sinuses. Among several collecting dural sinuses, there are two important sinuses: the superior sagittal sinus (SSS) draining dorsolaterally, and the cavernous sinus which drains anteroventrally. The SSS starts at the foramen cecum and courses along the gentle curvature of the inner table of the skull, within the leaves of the dura mater, to reach the confluence of sinuses, the torcular herophili. Along the way, the sinus receives several superficial cortical veins. The transverse sinus then drains the SSS, equally on both sides in only 20% of cases, and asymmetrically in more than 50%, depending on the configuration of the torcular herophili [12,15]. In 20% of cases, one transverse sinus drains the SSS totally (most often on the right side) and the other drains the straight sinus which belongs to the deep venous system [15]. The cavernous sinus extends from the superior orbital fissure to the petrous apex. It receives orbital venous and middle cranial fossa drainage. From the cavernous sinus, blood drains posterolaterally along the superior petrosal sinus into the transverse sinus and inferolaterally along the inferior petrosal sinus into the sigmoid sinus.

The deep cerebral venous system drains the deep white matter and regions surrounding the lateral and third ventricles or the basal cistern [12–14,16]. Three veins unite just behind the interventricular foramen of Monro to form the internal cerebral vein. These include the choroid vein, septal vein and thalamostriate vein. The choroid vein runs from the choroid plexus of the lateral ventricle. The septal vein runs from the region of the septum pellucidum in the anterior horn of the lateral ventricle and the thalamostriate vein runs anteriorly in the floor of the lateral ventricle in the thalamostriate groove between the thalamus and lentiform nucleus. The internal cerebral veins of each side run posteriorly in the roof of the third ventricle and unite beneath the splenium of the corpus callosum to form the vein of Galen. The vein of Galen is a short (1–2 cm long), thick vein which passes posterosuperiorly behind the splenium of corpus callosum in the quadrigeminal cistern. The internal cerebral vein and the vein of Galen receive the basal veins of Rosenthal and the posterior fossa veins and drains to the anterior end of the straight sinus where it unites with the inferior sagittal sinus. The basal vein of Rosenthal begins at the anterior perforated substance by the union of the anterior cerebral vein, middle cerebral vein and the striate vein. The basal vein of Rosenthal on each side passes around the midbrain to join the vein of Galen [17,18]. The main collecting vein for the deep venous system is the straight sinus, which receives the venous blood from the vein of Galen and flows into the transverse sinus (most often into the left side) [15]. The basal vein of Rosenthal is an important collateral pathway for the internal cerebral vein and the vein of Galen and connects with the superficial sylvian vein via the deep sylvian vein [16].

There are many medullary veins in cerebral white matter, which can be subdivided into the superficial and the deep medullary veins [12]. The superficial medullary veins drain 1–2 cm of white matter and are connected to the superficial cerebral veins. The deep medullary veins drain the deep white matter and flow into the subependymal veins of the lateral ventricle walls.

Venous drainage of the posterior fossa mainly depends on the galenic system and the petrosal system, and to a lesser extent, the tentorial veins and the transverse sinuses [12–14].

Extracranial Venous Drainage Pathway

Most of the cerebral venous drainage is via the extracranial venous pathway in the neck. Other

extracranial venous outflow pathways, such as the emissary veins of the middle cranial fossa draining the superficial and the deep middle cerebral vein contribute to a lesser extent [12]. The main cerebral venous outflow tract in the neck consists of the internal jugular vein (IJV), vertebral venous system, and the deep cervical veins (cervical soft tissue veins) [19–23]. These three venous pathways show multiple anastomoses between them in the neck, especially in the region of the craniocervical junction [19,21]. Among them, IJV and the vertebral vein can be detected easily by color Duplex ultrasound.

IJV is the largest vein in the neck and is considered to be the most important cerebral venous outflow pathway. Venous flow from the superficial and deep venous system is toward the transverse sinus then the sigmoid sinus, which drains into the IJV. The IJV is joined by the subclavian vein to form the brachiocephalic vein. The confluence of the bilateral brachiocephalic vein is the superior vena cava (SVC), which ultimately drains venous blood into the heart.

The vertebral venous system consists of two components, one is the vertebral venous plexus and the other is the vertebral vein (e.g. the vertebral artery venous plexus) [9,21,24,25]. The vertebral venous plexus can be subdivided into the internal (posterior and anterior internal vertebral plexus) and the external (posterior and anterior external vertebral plexus) vertebral plexus [9,21,24,25]. Complex connections of the cerebral venous outflow with the vertebral venous system over the craniocervical junction have been displayed in several human cadavers and angiographic studies [19,21,26–28]. The anterior condylar confluent (ACC) is the most important connection between the intracranial cerebral venous circulation and the vertebral venous system. The ACC appears as an anatomic constant whose tributaries are the anterior and lateral condylar veins, inferior petrosal sinus, and the IJV. Numerous anastomosis of the ACC make it a crossroad between the cavernous sinus, dural sinuses of the posterior fossa, and the posterior cervical outflow tract (vertebral venous system and deep cervical veins). The posterior and lateral condylar veins allow for connections with the external vertebral venous plexus, whereas the anterior condylar veins are related with the internal vertebral venous plexus. There are anastomosis between the anterior internal vertebral venous plexus, vertebral vein, and deep cervical veins within the craniocervical junction region.

The pterygoid plexus and facial veins are the other important extracranial venous collateral pathways. The pterygoid plexus communicates with the cavernous sinus and ultimately drains into the external jugular vein [9]. The facial vein can receive venous flow from the reversed superior ophthalmic vein blood flow when the venous pressure in the cavernous sinus is high [29,30].

Extracranial venous drainage is position-dependent [12,20,22,25,31]. In the supine position, the IJV is the main route for cerebral venous drainage. One ultrasonographic study in the supine position showed that a total jugular flow volume of more than two-thirds of the global cerebral arterial inflow volume was present in 72%, and less than one-third of the global cerebral arterial inflow volume was found in only 6% of normal individuals [23]. The existence of an extra-jugular route for cerebral venous outflow was evidenced by the fact that a unilateral or bilateral radical neck dissection was well tolerated in affected patients [32–34]. Extra-jugular venous pathways for cerebral venous drainage include the vertebral venous system and deep cervical veins [19–23]. These extra-jugular venous pathways are responsible for cerebral venous outflow in the upright position when the IJV is collapsed because of both increased external pressure [12] and decreased IJV venous pressure [35] when upright. This phenomenon is thought to contribute to an evoked venous resistance at the outlet of the cerebral venous system in the upright position [12,31,36].

Venous return is altered by the pressure gradients of blood flow into the thorax resulting from respiratory variations in intrathoracic pressures [12,37,38]. Inspiration is known to augment venous return to the heart by the generation of negative intrathoracic pressure. An increased distance

between the sternum and the thoracic outlet arteries at deep inspiration improves venous drainage of the left IJV into the left brachiocephalic vein [39,40]. In contrast, breath-holding results in increased abdominal and thoracic pressure, which produces a decrease in venous outflow via the cervical veins [41].

IJV and IJV Valve

The IJV is the main extracranial route for cerebral venous outflow, especially in the supine position. On duplex sonography of the extracranial arteries, the cervical veins were not routinely examined, because clinically relevant symptoms were not evident. In the past, duplex sonography has been used to diagnose pathologic conditions of the IJV, including dural arterio-jugular venous fistula and jugular or central venous occlusion caused by thrombosis or external compression. Recently, several neurologic disorders have been found to be associated with abnormal flow patterns in the IJV, which were detected by duplex sonography [39,40,42–48].

The examination procedure for the IJV is similar to duplex sonographic examination of the extracranial carotid arteries [49]. In the longitudinal plane, the IJV is identified lateral to the common and internal carotid arteries (Fig. 1). Using color coding, the IJV can easily be distinguished from the arteries by their normally opposite direction of flow. The sonographic characteristics of the cervical veins are compressibility which occurs with the transducer when slight pressure is applied, change in the vessel lumen caused by breathing, and the typical Doppler venous signal [49]. Great care should be taken to avoid compression of the cervical veins during sonographic examination, and usually a significant amount of ultrasound gel is required. During exhalation, the IJV lumen expands, whereas it is narrowest at the end of inhalation because of decreased intrathoracic pressure [49]. During deep inhalation, the IJV might collapse [49]. A venous Doppler signal is characterized by a continuous spectral waveform of low pulsatility, modulated by respiratory activity and the cardiac cycle (Fig. 1) [49,50]. Furthermore, a Valsalva maneuver (VM) is helpful in confirming the venous flow. Normally, the increase in intrathoracic pressure caused by the VM leads to a decrease in venous flow velocity [49,51]. The IJV flow velocity normally decreases on expiration and accelerates on inspiration [49].

The existence of the IJV valve (IJVV) was initially described by Harvey [52]. The IJVV is located about 0.5 cm above the union of the subclavian vein and the IJV at the lower limit of the jugular bulb (Fig. 2) [50,52–55]. IJVVs are seen in 96.8% of the general population [53,55]. In the majority (93–99.1%) of subjects, the IJVV is bicuspid [53,55]. The valve closes once during each cardiac cycle [54]. The closure of the valve occurs during diastole when the

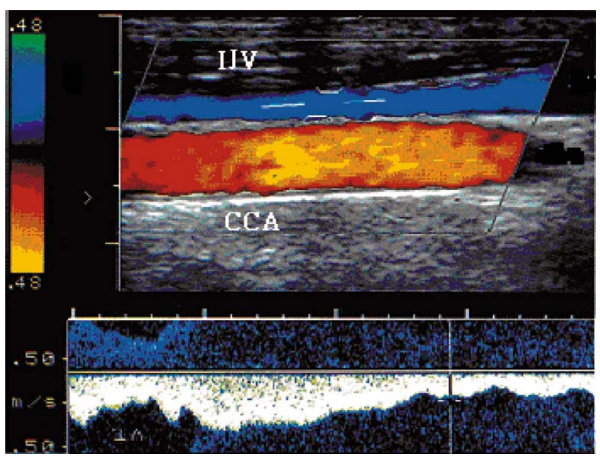

Fig. 1. *In the longitudinal plane of color-coded duplex sonography, internal jugular vein (IJV; blue) is identified lateral to the common carotid arteries (CCAs; red).*

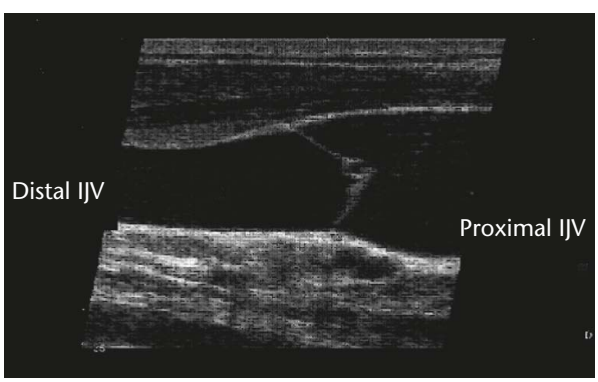

Fig. 2. *Internal jugular vein (IJV) valve is located about 0.5 cm above the union of the subclavian vein and the IJV at the lower limit of the jugular bulb.*

atrium transmits backward pressure from the right atrium into the SVC and then into the IJV [54]. The valve is then open in mid-systole [54]. The morphology and motion of the IJVV can be shown by high-resolution ultrasonography [50,54,55]. The IJVV serves an important role in preventing backflow of venous blood and backward venous pressure into the cerebral venous system during conditions of increased venous pressure or intrathoracic pressure [52–55]. Without a competent IJVV, a sustained or prolonged retrograde-transmitted venous pressure via the IJV might lead to cerebral venous hypertension or congestion [52–55].

The competence of the IJVV can be easily assessed using color duplex sonography. A retrograde flow detected by color duplex or in the Doppler spectrum spontaneously or during VM is considered IJVV incompetence (IJVVI) (Fig. 3) [49,53,54]. A retrograde flow in the IJV might result from disturbed venous flow caused by pulsation of nearby arteries or valve closure-related mechanical oscillation. Nedelmann et al [56] found a cutoff value for the duration of the detected retrograde flow during VM, to differentiate between a physiologic jugular venous reflux and a true IJVVI. The duration of the retrograde flow, which equals or is greater than 0.88 seconds, used as the criteria for IJVVI yields 100% sensitivity and 100% specificity. In our research, we have used 0.5 seconds as the cutoff value to define IJVVI for a convenient comparison between studies [46,57,58].

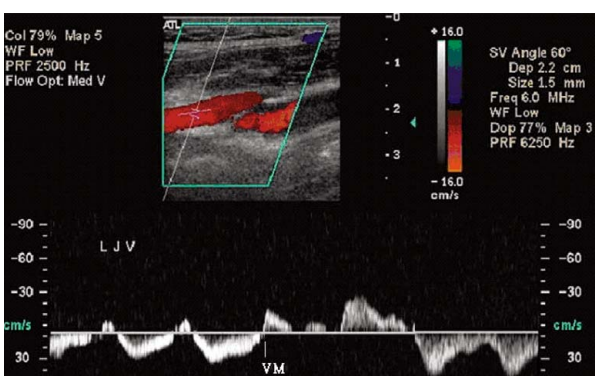

Fig. 3. *Retrograde flow detected by color duplex or in the Doppler spectrum during Valsalva maneuver (VM) is considered internal jugular vein valve incompetence.*

IJVVI is frequently seen in patients who have an elevated central venous pressure, such as congestive heart disease [59,60], tricuspid valve regurgitation [59,60], primary pulmonary hypertension [61], and chronic obstructive pulmonary disease [61]. These findings support the hypothesis that venous valve incompetence is acquired and linked to venous hypertension [62,63]. IJV wall injury after central venous cannulation is another presumed etiology of IJVVI [64]. In several studies, IJVVI was found in 20–40% of normal individuals, depending on the imaging method and the study population [44,53,55]. In a study with a large sample of healthy individuals ($n=121$), IJVVI was found more frequently in older subjects and in men [55].

Recently, several neurologic disorders have been found to be related to IJVVI. These disorders are transient global amnesia (TGA) [39,40,42–45], transient monocular blindness [46], cough headache [47], and primary exertional headache [48]. This association suggests that cerebral venous hypertension or congestion might play a major role in the mechanism of these disorders.

Branches of Internal Jugular Vein (JVBs)

Several tributaries drain into the IJV in the neck. From rostral to caudal, those IJV branches (JVBs) are the facial, lingual, superior thyroid and middle thyroid veins [30,65]. The facial, lingual and superior thyroid veins may join each other to form the thyrolinguofacialis, linguofacialis or thyrofacialis veins, respectively, before joining with the IJV into which they drain [30,65]. Bilateral JVBs interconnect with each other at the midline to form anastomosing plexi, which might serve as collateral channels to maintain adequate venous drainage [30,66].

Venous valves in the IJV tributaries have been described in a cadaver study [65]. The frequency of venous valves (number of venous valves/cm) was 0.24 ± 0.16 in the facial vein, 0.07 ± 0.15 in the lingual vein, 0.05 ± 0.10 in the superior thyroid vein, and 0.22 ± 0.40 in the middle thyroid vein [65].

In veins with valves, retrograde venous hypertension could be prevented by competent valves [53].

JVB and its flow pattern can be detected using color-Doppler sonography [58]. In our previous study, we isolated the IJV using a cross-sectional view from the neck base to the submandibular level to detect any JVB in the neck (Fig. 4) [58]. Once a branch was detected, we then moved the probe to show the longitudinal view of this branch to reveal the flow direction in the branch using color Doppler, to see if it flowed into or away from the IJV (Fig. 5). The Doppler spectrum measurement was then used to confirm the flow direction in the branches. Venous reflux in JVB can be provoked by a VM if the JVB venous valve is incompetent.

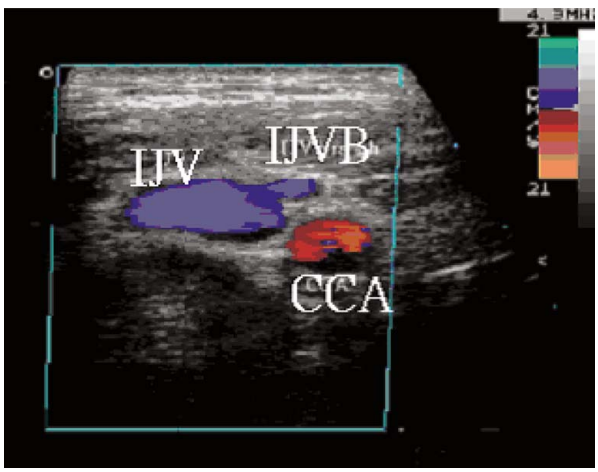

Fig. 4. *Internal jugular vein (IJV) is isolated by a cross-sectional view from the neck base to the submandibular level to detect any internal jugular vein branches (IJVBs) in the neck. CCA = common carotid artery.*

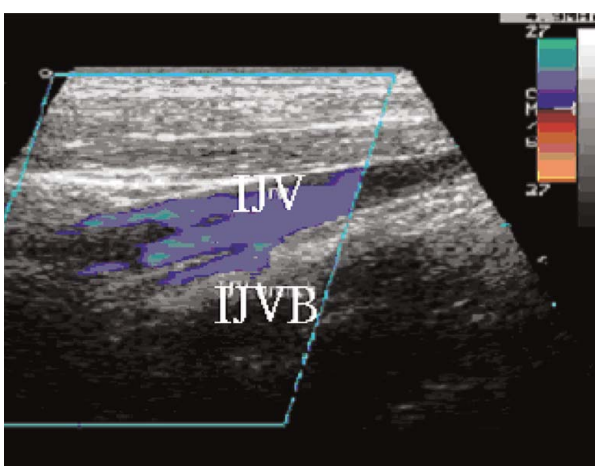

Fig. 5. *Once a branch is detected, the probe is then moved to show a longitudinal view of this branch to reveal the flow direction in the branch by color duplex, to see if it flows into or away from the internal jugular vein (IJV). IJVB = internal jugular vein branch.*

JVB venous reflux is significantly associated with IJVVI and greater IJV blood-flow volume, respectively, both of which may reflect higher IJV pressure transmission during VM [58]. Therefore, venous reflux in JVB might indicate an increased IJV pressure, which could weaken the valve competence, even in cases without IJV venous reflux [58]. This sonographic finding can be applied to the further study of diseases, which are thought to be related to an elevated IJV pressure.

Internal Jugular Venous Reflux (JVR)

A pressure gradient determines the flow direction of veins [12]. In venous segments with existing venous valves (e.g. the proximal IJV), venous reflux requires a reversed pressure gradient and an incompetent venous valve connecting both poles of the pressure gradient [67]. However, in venous segments without existing venous valves (e.g. the distal IJV and intracerebral veins), just a reversed pressure gradient could produce venous reflux [39,40,68,69]. Therefore, the existence of an incompetent venous valve is not always necessary for venous reflux. In JVR, there is an abnormal (reversed) pressure gradient resulting from increased venous pressure proximally with or without an incompetent venous valve. JVR indicates an increased proximal venous pressure, which might impede cerebral venous outflow and induce neurologic dysfunction [39,40,42–45].

In physiologic situations, most frequently encountered reversed pressure gradients result from many Valsalva-like activities which increase intrathoracic pressure. These activities include coughing, straining to defecate, sexual intercourse, and heavy lifting, etc. During these activities, JVR will occur if the IJVV is incompetent. This transient, episodic venous reflux might impede cerebral venous outflow or transmit back venous pressure into the cerebral venous system [39,40,42–45]. Long-term

repeatedly increased pump back pressure by these Valsalva-like activities may damage the venous valves, leading to valve degeneration and eventually incompetence [58,62,63]. This explains the high frequency of IJVVI seen in the elderly [55]. Furthermore, this episodic venous hypertension in the IJV following Valsalva-like activities might be exaggerated by conditions which chronically increase the central venous pressure, such as congestive heart disease [59,60], tricuspid valve regurgitation [59,60], primary pulmonary hypertension [61], and chronic obstructive pulmonary disease [61]. Therefore, there is a higher frequency of IJVVI in these conditions.

If there is a persistent and high (above the pressure threshold of IJVV competence) reversed pressure gradient, such as in central venous obstruction, JVR will be continuous (Fig. 6) [39,70–76]. The level of JVR depends on the extent of the pressure gradient and the sufficiency of venous collaterals [40]. In our previous studies, venous reflux could be as high as that of the cerebral basilar plexus (Fig. 7) [39,40]. This continuous JVR has been mostly reported on the left side because of the anatomic characteristics of the left brachiocephalic vein. The left brachiocephalic vein has a more obtuse angle and a longer length before joining the SVC than the right brachiocephalic vein. Besides, the left brachiocephalic vein goes through the narrow space between the sternum and the thoracic outlet arteries before entering the SVC. This narrow space might compress the left brachiocephalic vein to stenosis or even occlusion, resulting in JVR (Fig. 8). This phenomenon has been reported in normal individuals with a frequency of 0.2–0.4% [71,72]. A higher frequency in the elderly was observed, which might be due to the more frequently engorged thoracic outlet arteries in elderly people [75]. In our previous studies, left brachiocephalic occlusion caused

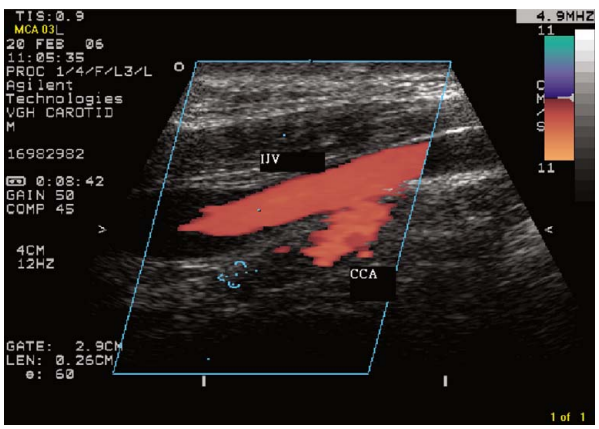

Fig. 6. *Continuous jugular venous reflux at rest. CCA = common carotid artery; IJV = internal jugular vein.*

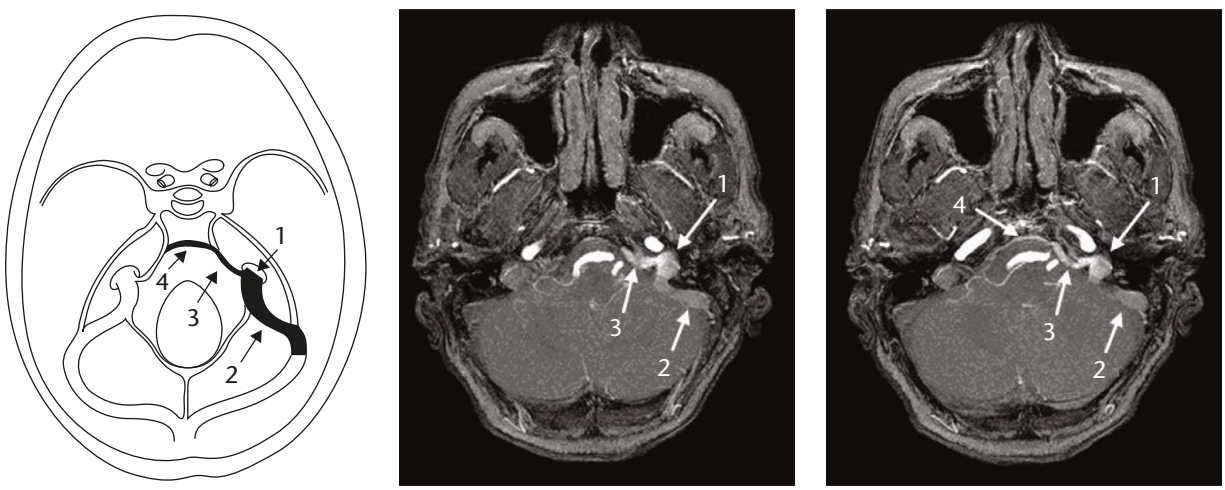

Fig. 7. *Venous reflux was shown from the left internal jugular vein (IJV) to the sigmoid sinus and through the inferior petrosal sinus to the basilar plexus. Veins with abnormal bright signals (indicate venous reflux) on three-dimensional time-of-flight magnetic resonance angiogram are solid black in color in the diagrams. Arrow 1 indicates the left IJV; arrow 2, left sigmoid sinus; arrow 3, left inferior petrosal sinus; and arrow 4, basilar plexus.*

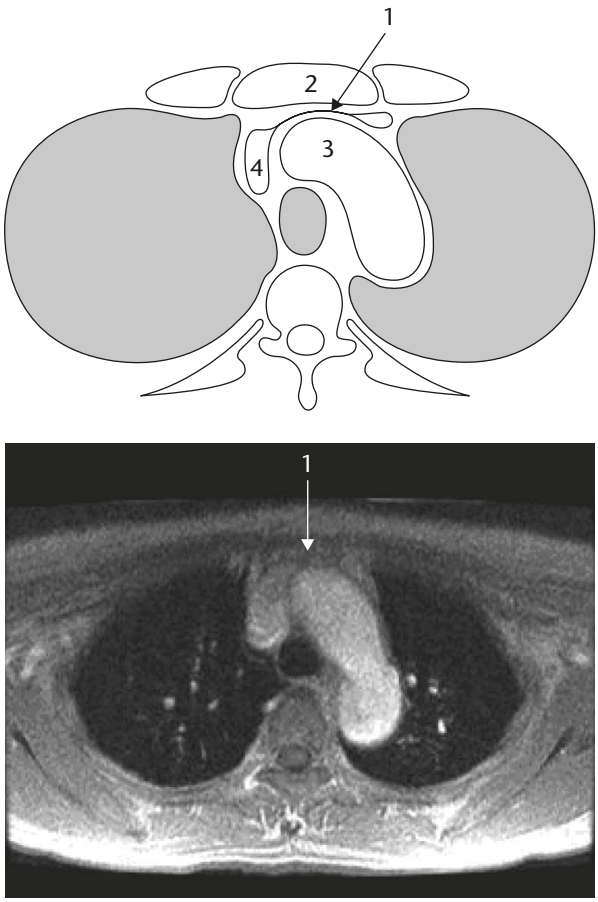

Fig. 8. *Left brachiocephalic vein (1) is compressed to occlusion between the aortic inlet arteries (3) and the sternum (2). "4" indicates the superior vena cava.*

by compression in the space between the sternum and the thoracic outlet arteries was significantly more frequent in patients with TGA [39,40]. This suggests that cerebral venous outflow impairment might play a major role in the pathophysiology of TGA.

The other causes of central venous obstruction producing continuous JVR are mediastinal goiter [77], mediastinal masses [78], aortic aneurysm [79], venous thrombosis (SVC syndrome) [80], and severe congestive heart failure [81]. To determine whether there are other sources (e.g. decreased proximal venous distensibility, increased venous flow volume, specific respiratory patterns, or abnormal blood components), which produce increased proximal venous pressure resulting in JVR, requires further investigation.

The Detection of JVR

In the initial study of IJVV competence, patients were examined using invasive venography [60]. The functions of both IJVVs were studied during cardiac catheterization with increased thoracic pressure caused by coughing. Later, the competence of the IJVV was assessed extensively using color duplex sonography owing to its noninvasive and practical characteristics [39,40,43–48,53,54]. A retrograde flow detected by color duplex or in the Doppler spectrum spontaneously or during VM was the diagnostic criterion for IJVVI. Some studies then used air contrast ultrasonography to detect IJVVI [55,82]. Air contrast ultrasonographic venography was performed by monitoring the IJV before and after VM with B-mode real-time ultrasonography following intravenous injection of agitated air and saline (1 mL of air and 9 mL of normal saline manually agitated). The injection was randomly performed on the right or left antecubital vein in those studies. IJVVI was determined when air bubbles were seen distal to the valves. There has been no correlation studies carried out to compare the examination results from invasive venography, conventional color duplex sonography, and air contrast ultrasonography. However, there are some technical issues which should be addressed when using air contrast ultrasonography to detect IJVVI, especially when left brachiocephalic vein compression is the reason for JVR [83]. The assumption that the side of injection does not influence detection of IJVVI is based on limited clinical experience [55]. When air contrast is injected into the right antecubital vein, the air bubbles are not transmitted to the left IJV even if the left IJVVs are incompetent because of compression of the left brachiocephalic vein. Moreover, we have found that there is a venous valve in the left brachiocephalic vein, which can prevent transmission of the air bubbles to the left IJV [83]. These factors might result in an underestimation of the frequency of left IJVVI. The occurrence of right IJVVI may be overestimated, because the air bubbles might appear in the right IJV without IJVVI via the abundant venous collaterals in the

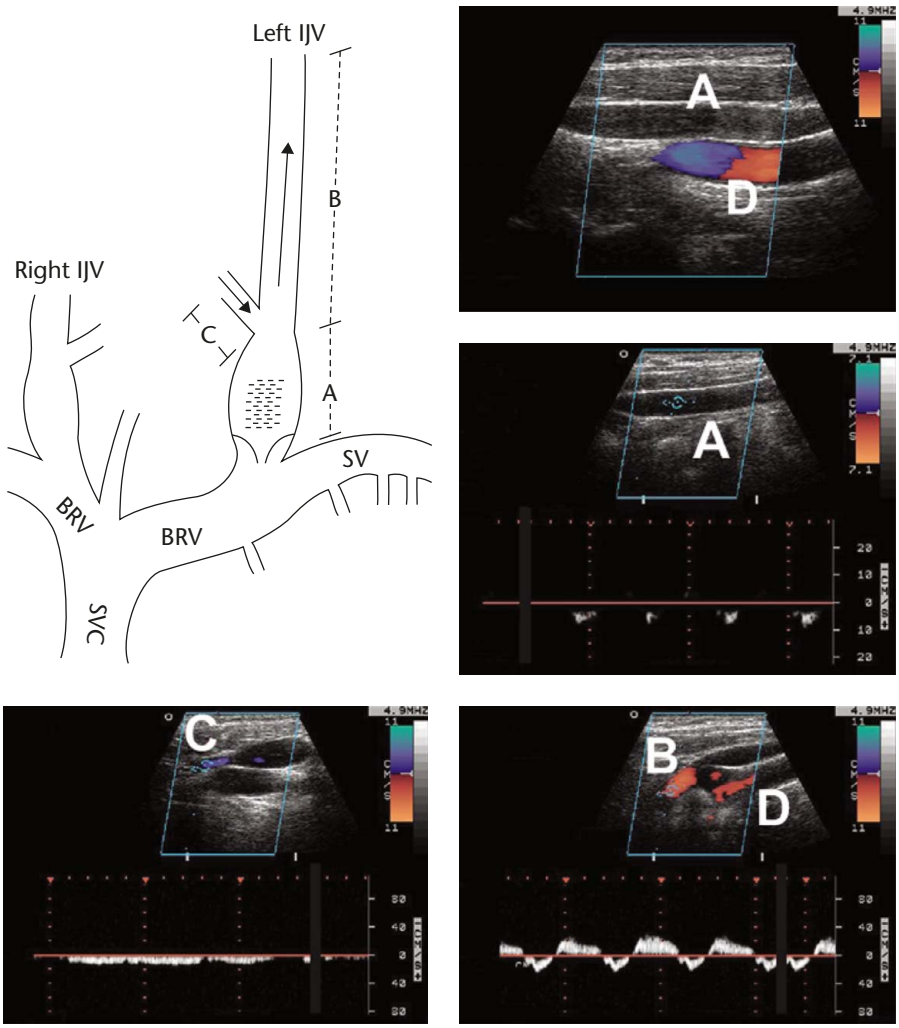

Fig. 9. *Diagrams and sonographic studies showing segmental reversed flow in the left distal internal jugular vein (IJV) in a transient global amnesia patient. The proximal segment of the left IJV (A) shows spontaneous echo contrast with trickle flow. The distal segment of the left IJV (B) shows reversed flow. The flow direction of the left IJV branch (JVB) (C) is into the left IJV. The flow patterns in the right IJV and JVB are normal. "D" indicates the common carotid artery. BRV = brachiocephalic vein. SV = subclavian vein; SVC = superior vena cava.*

case of left IJVVI when air contrast is injected into the left antecubital vein. The venous collaterals have been demonstrated by upper extremity digital subtractive venography in patents with left brachiocephalic compression [39].

As previously mentioned, just a reversed pressure gradient is needed to produce a venous reflux in a venous segment without a valve, such as in the distal IJV. We have found a segmental reversed flow in the left distal IJV and in JVB, respectively, with no venous reflux found in the proximal IJV at baseline in some patients with TGA (Fig. 9 and 10) [40]. These conditions might be omitted if only the proximal IJV is examined. Furthermore, JVR detected at rest might return to normal antegrade flow during VM, especially in cases of left brachiocephalic vein compression caused by the narrow space between the sternum and the thoracic inlet arteries (Fig. 11). The VM usually lasts for 10–30 seconds. Therefore, to maintain a breath-hold for such a time, patients are usually asked to or spontaneously perform a deep inspiration before the VM. This deep inspiration releases the increased IJV pressure and improves the venous outflow via the IJV by increasing the distance between the aorta and the sternum, and might compensate for the VM effect of

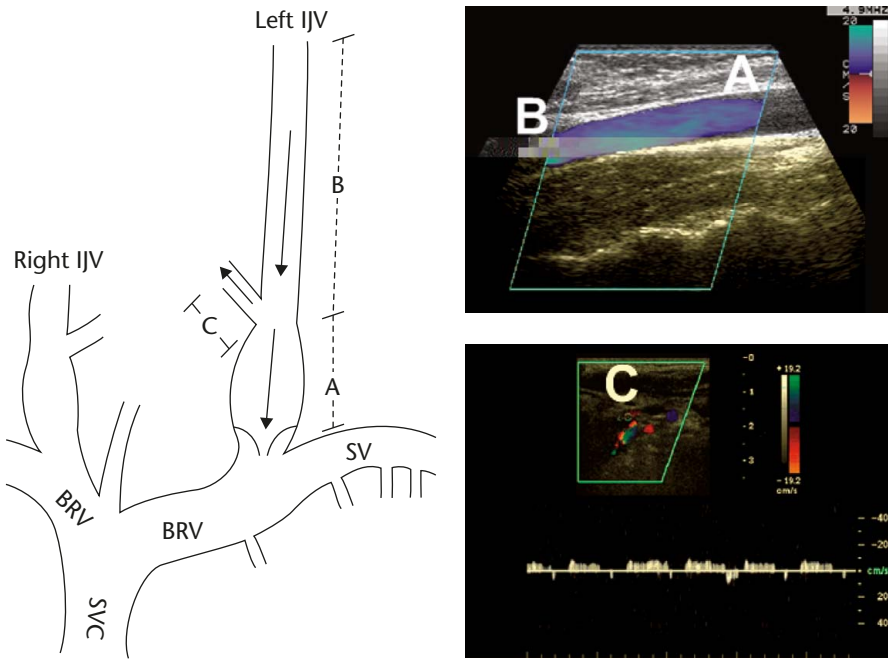

Fig. 10. *Diagrams and sonographic studies showing isolated reversed flow in the left internal jugular vein branch (JVB) in a transient global amnesia patient. The proximal (A) and distal (B) segments of the left internal jugular vein (IJV) show normal anterograde flow. However, the direction of flow in the left JVB (C) is away from the IJV, i.e. reversed flow. The flow patterns in the right IJV and JVB are normal. BRV = brachiocephalic vein; SV = subclavian vein; SVC = superior vena cava.*

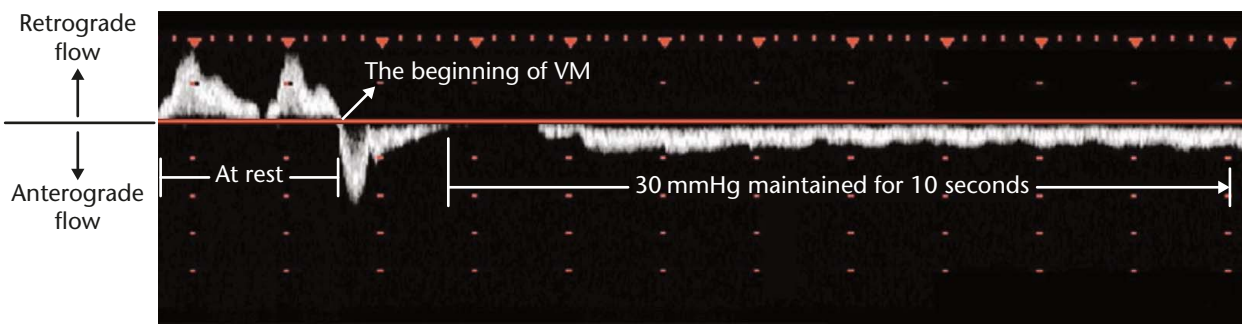

Fig. 11. *The Doppler spectral study of the left internal jugular vein (IJV) during the Valsalva maneuver (VM) in a transient global amnesia patient with a continuous reversed flow in the left IJV. The retrograde flow at rest changed to anterograde flow because of a spontaneous deep inspiration at the beginning of the VM. During the VM with the intrathoracic pressure maintained at 30 mmHg, the flow in the left IJV shows a decreased but still anterograde flow.*

increased IJV pressure. Thus, the increased IJV pressure caused by VM might not be high enough to produce a pressure gradient for retrograde flow in the IJV though incompetent IJVVs. The abnormal retrograde flow in these patients at rest would not be seen during VM. Therefore, in studying JVR, we suggest that sonographic studies should include the whole length of the IJV and JVB in the neck. The examination should be done first at rest, and then during the VM. This thorough study could avoid any underestimation of the frequency of JVR in normal individuals and in patients with neurologic disorders, which are thought to be caused by cerebral venous outflow impairment.

Conclusion

JVR indicates an abnormal reversed pressure gradient, which might impede cerebral venous outflow

and/or transmit venous back pressure into the cerebral venous system. The magnitude of the pressure gradient, the sufficiency of venous collaterals, and the competence or existence of venous valves will determine the extent of venous reflux. More and more neurologic disorders are thought to be related to JVR. We have developed a sonographic examination to investigate the hemodynamics of the IJV and its branches. This thorough examination avoids underestimation of the frequency of JVR in normal individuals, and in patients with neurologic disorders which are thought to be caused by cerebral venous outflow impairment. The definite hemodynamic, histologic and molecular effects of JVR on the brain requires further investigation.

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