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Hemodynamics of the Internal Jugular Vein: An Ultrasonographic Study

*Shinn-Kuang Lin[1,2], Yeu-Jhy Chang[3,4], Fu-Yi Yang[1]**

[1]*Stroke Center and Department of Neurology, Buddhist Tzu Chi General Hospital, Taipei Branch, Taipei, Taiwan*
[2]*School of Medicine, Tzu Chi University, Hualien, Taiwan*
[3]*Stroke Center and Department of Neurology, Chang Gung Memorial Hospital, Taoyuan, Taiwan*
[4]*College of Medicine, Chang Gung University, Taoyuan, Taiwan*

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Abstract

Objective: To establish a simple classification of internal jugular venous flow using ultrasonography.

Materials and Methods: We retrospectively analyzed the sonographic findings of 1600 consecutive patients who were referred to the neurosonographic laboratory. Abnormal jugular venous flow was classified into five groups: (A) markedly decreased flow velocity; (B) stasis and/or thrombus formation; (C) reversed flow; (D) increased turbulent flow; and (E) pulsatile turbulent flow. Groups A to C were categorized as slow flow groups and groups D and E as high flow groups.

Results: Abnormal jugular venous flow was found in 182 (11%) patients. A slow flow pattern (73%) comprised the majority of abnormalities. Eighty-nine percent of patients in the slow flow groups had abnormalities in the left internal jugular vein (IJV) and 92% of those in the high flow groups had abnormalities in the bilateral IJV. Incompetence of the jugular valve was the most common cause of slow flow in the IJV. Most of the increased turbulent flow was found in patients with hyperthyroidism and in pregnant women. Arteriovenous malformation and carotid-cavernous fistula were the causes of pulsatile turbulent jugular venous flow. Associated clinical symptoms occurred in only two (1.5%) patients (with a concomitant left IJV and left subclavian vein thrombosis) in the slow flow groups, and in 34 (68%) patients in the high flow groups.

Conclusion: Most of the abnormally decreased jugular venous flow occurred on the left side. Decreased venous flow or even thrombosis of one side of the IJV was usually asymptomatic. Increased jugular flow was associated with a physiological hyperemic state in cerebral hemodynamics. Increased awareness and angiographic studies are indicated in patients with pulsatile turbulent jugular venous flow because of a high correlation with intracranial arteriovenous shunting. (*Tzu Chi Med J* 2009;21(4):317–322)

*Corresponding author. Department of Neurology, Buddhist Tzu Chi General Hospital, 289, Jian-Guo Road, Xindian, Taipei, Taiwan.
E-mail address: fuyi127@tzuchi.com.tw

1. Introduction

Traditionally, the internal jugular vein (IJV) has served as an indicator of right side cardiac function via measurement of central venous pressure and changes in the velocity of jugular venous flow [1–3]. Ranganathan et al suggested that the right atrium behaves as a conduit rather than a capacitance chamber [3]. Recent studies have focused on either the position of the IJV to facilitate cannulation with a central venous catheter, or thrombosis of the IJV [4,5]. Central venous catheter insertion is reported to be the most common cause of IJV or subclavian vein thrombosis [6–9]. Unlike the carotid artery, the vascular wall of the IJV is much more flexible with a variable lumen diameter. It is difficult to perform a quantification study of IJV flow. A jugular venous Doppler signal on ultrasound is represented by a continuous spectral waveform of low pulsatility, modulated by respiratory activity and the cardiac cycle without arterial pulsation (Fig. 1F). Many factors influence IJV flow, including postural change, respiration, cardiac function, and local pressure from the transducer of ultrasound equipment. Different patterns of IJV flow have been previously described; however, a Doppler classification of IJV flow has not been carried out. In this study, we attempted to establish a simple classification of IJV flow according to B-mode and Doppler findings on ultrasonography.

2. Materials and methods

Since 1994, more than 10,000 patients have been referred to our neurosonographic laboratory for color-coded carotid duplex sonography including that of the IJV. We conducted a retrospective analysis of the IJV of the first 1600 consecutive patients, including 40 patients with hyperthyroidism. Carotid duplex sonography was performed with an ATL Ultramark 9 or ATL 2000 HDI system (ATL Ultrasound Inc., Bothell, WA, USA), which contains a 5–10 MHz transducer for high-resolution real-time B-mode imaging. The patients were examined in the supine position with the neck slightly rotated away from the side of examination. Measurement was performed at maximal venous extension during a normal respiratory cycle at a level 1.5 cm inferior to the palpable anterior surface of the cricoid cartilage for the IJV. The sonographic examination began with transverse scans to identify the jugular veins and carotid arteries. Subsequently, the IJVs were scanned in the sagittal (longitudinal) plane to assess the magnitude and morphology of intraluminal echoes. Physiological features of the IJV were also assessed sonographically. Distensibility was defined as the increase in area measured in the transverse plane during a Valsalva maneuver. Normal compressibility was recorded as the change of venous shape on transducer pressure. Beating venous valves are typically located caudally at a level 3 cm inferior to the palpable anterior surface of cricoid cartilage in the IJV. Pulsed-wave Doppler was applied to detect the IJV flow. Normal IJV flow does not show regular arterial pulsations corresponding to heart beats. Instead, respiratory pulsation due to an increase and decrease in intrathoracic pressure upon respiration can be observed (Fig. 1F).

Abnormal jugular venous flow patterns were classified into five groups (Table 1). Group A had markedly decreased flow velocity with the peak velocity below 10 cm/s (Fig. 1A). Group B had stasis and/or thrombus formation with spontaneous echo contrast, with a homoechogenic or heteroechogenic lesion scanned in the lumen by B-mode imaging studies and very low or no flow detected on Doppler study (Fig. 1B). Group C had reversed flow on Doppler study (Fig. 1C). Group D had increased turbulent flow with markedly increased flow velocity and vibratory waveforms on Doppler study (Fig. 1D). Group E had pulsatile turbulent flow related to arterial pulsation on Doppler study (Fig. 1E). Groups A, B and C were categorized as slow flow groups while groups D and E were categorized as high flow groups.

3. Results

Abnormal jugular venous flow was found in 182 (11%) patients. Abnormal IJV flow patterns in these patients are summarized in Table 1. A slow flow pattern comprised the majority of abnormalities. Eighty-nine percent of patients in the slow flow groups had abnormalities in the left IJV and 92% of those in the high flow groups had abnormalities in the bilateral IJV. Incompetence of the jugular valve was the most common cause of slow flow in the IJV. Table 2 summarizes the etiology of high flow in the IJV. Hyperthyroidism (40%) was the most frequent cause of increased turbulent flow in the bilateral IJV in group D. All pulsatile turbulent IJV flow in group E resulted from intracranial arteriovenous fistula. Twenty percent of patients with abnormal IJV flow had associated clinical symptoms. In the slow flow groups, only two (1.5%) patients with a concomitant left IJV and subclavian vein thrombosis had associated clinical symptoms. In the high flow groups, 68% patients had associated clinical symptoms (Table 3).

4. Discussion

The IJV is the most important cerebral venous outflow pathway in the neck. Anatomical variation partly accounts for the inability to cannulate the IJV in certain patients. Ultrasound examination has been reported to be useful in quickly establishing the position of

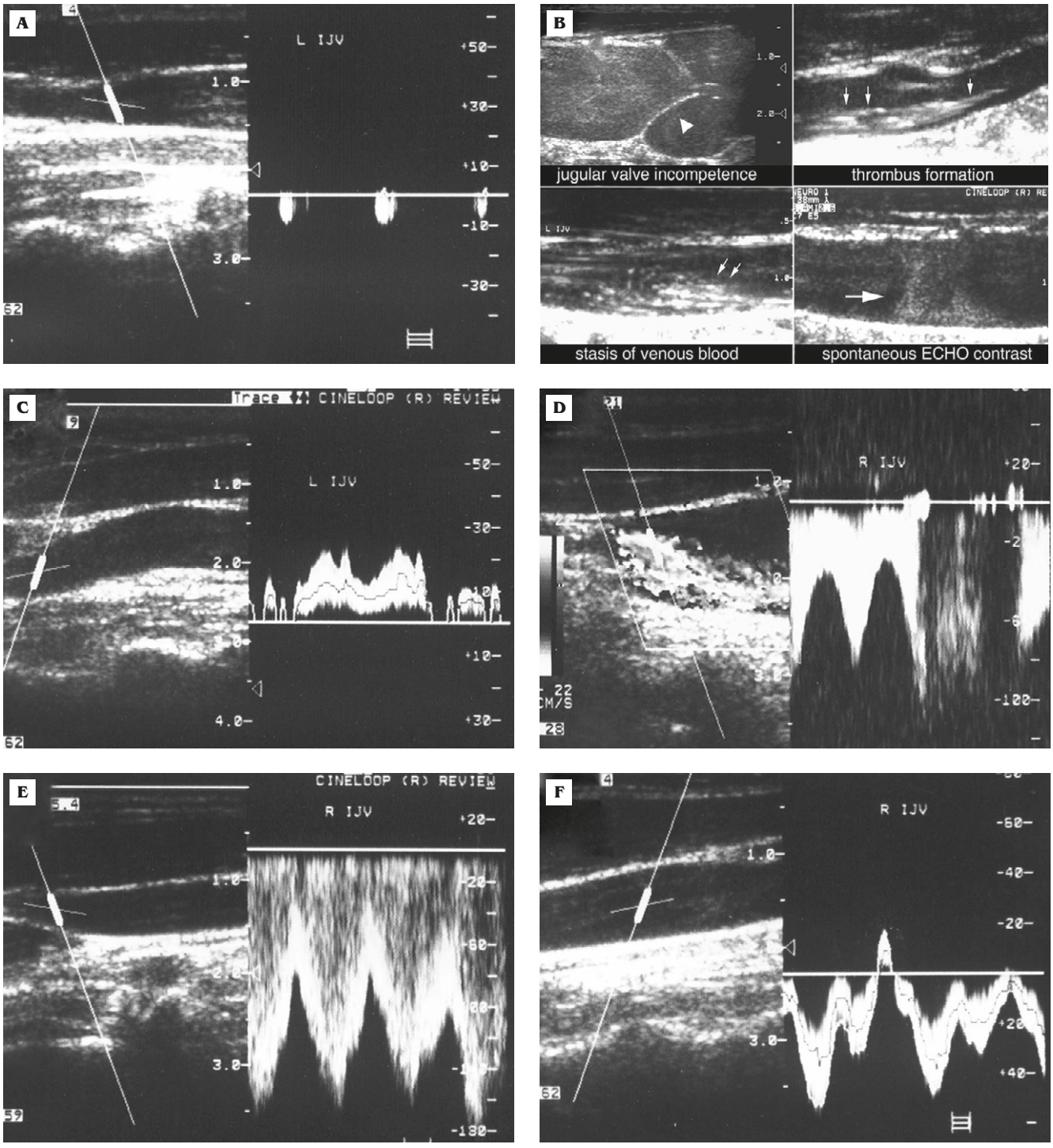

Fig. 1 — Abnormal flow in the internal jugular vein. (A) Group A: markedly decreased flow. (B) Group B: stasis and/or thrombus formation with spontaneous echo contrast in the lumen. (C) Group C: reversed jugular venous flow. (D) Group D: turbulent flow with markedly increased flow velocity and vibratory waveforms. (E) Group E: pulsatile turbulent flow. (F) Normal flow in the internal jugular vein.

the IJV [10]. Using ultrasound, Clenaghan et al found that a Trendelenburg tilt increases the IJV diameter and hence improves the chances of successful cannulation [11]. The IJV is an uncommon site of spontaneous venous thrombosis. Reported causes include intravenous drug abuse, jugular vein catheterization, neck dissection, a hypercoagulable state associated with malignancy (Trousseau's syndrome), neck injury and ovarian overstimulation syndrome [12]. Symptoms of IJV thrombosis vary. Suppurative jugular

Table 1 — Summary of abnormal internal jugular venous flow by ultrasound study

Classification of abnormal internal jugular venous flow	Lesion side involved			Total (n=182)
	Right	Left	Bilateral	
Slow flow groups (Groups A, B, C)				132 (73%)
A. Markedly decreased flow velocity	3	43	4	50
B. Stasis and/or thrombus formation	5	55	1	61
C. Reversed flow	1	20	0	21
High flow groups (Groups D and E)				50 (27%)
D. Increased turbulent flow	2	1	36	39
E. Pulsatile turbulent flow	1	0	10	11

Table 2 — Causes of increased internal jugular venous flows in patients in the high flow groups

Cause	Lesion side involved			Total (n=50)
	Right	Left	Bilateral	
Increased turbulent flow in the IJV	2	1	36	39
1. Hyperthyroidism	0	0	20	
2. Pregnancy	0	0	9	
3. Spontaneous intracranial hypotension	0	0	1	
4. Intracranial AVM	0	0	2	
5. Venous hums	0	0	2	
6. Unknown cause	2	1	2	
Pulsatile turbulent flow in the IJV	1	0	10	11
1. Intracranial AVM	1	0	6	
2. Carotid-cavernous fistula	0	0	4	
Total	3	1	46	50

IJV=internal jugular vein; AVM=arteriovenous malformation.

Table 3 — Observed associated diseases in patients with abnormal internal jugular venous flows

Abnormal internal jugular flows	Associated diseases, n (%)
Slow flow groups (Groups A, B, C; n=132)	2 (1.5)
IJV and SCV thrombosis	2
High flow groups (Groups D and E; n=50)	34 (68)
Hyperthyroidism	20
Arteriovenous malformation	9
Carotid-cavernous fistula	4
Intracranial hypotension	1
Total (n=182)	36 (20)

IJV=internal jugular vein; SCV=subclavian vein.

thrombophlebitis (also known as Lemierre syndrome) with thrombosis of the IJV is a serious condition that requires emergency treatment. Diagnosis of IJV thrombosis or venous distention can be easily carried out with ultrasound, even in patients who do not have evidence of jugular venous distention on physical examination (13).

Normally, most of the superficial cerebral venous flow drains into the superior sagittal sinus and then into the transverse sinus, mainly on the right side. The deep venous flow drains into the straight sinus and then into the transverse sinus, mostly on the left side. It is not surprising that the volume and velocity of cerebral venous flow drains into the heart unevenly through the bilateral IJVs with a predominance on the right side. According to our findings, abnormalities in the IJV on real-time duplex sonography are not uncommon (11%). Most abnormalities were found in the slow flow groups (73%) and 89% of abnormal slow flow was detected in the left IJV. In this study, insonation of the jugular valve was not achieved successfully in every patient owing to the deep location of the IJV. Possible causes of slow venous flow in the IJV include asymmetry of flow from the anatomical variations described above, incompetence of the jugular valve, increased central venous pressure from congestive heart disease or tricuspid valve regurgitation, and increased intrathoracic pressure from pulmonary hypertension or obstructive pulmonary disease. The jugular valves prevent backflow of venous blood and pressure into the cerebral venous system. Retrograde flow in the IJV with or without a Valsalva maneuver during sonographic study is considered as jugular valve incompetence. Jugular valve incompetence is found in 20–40% of healthy individuals, depending on the methodology and definition (14,15). It is thought to be the major cause of retrograde venous flow in the IJV. Theoretically, once venous return is blocked, subsequent blood stasis occurs in the IJV, leading to local thrombus formation. However, venous return is affected by postural changes. An upright position increases the pressure gradient and hence speeds the venous return. It also results in a partial collapse of the IJV and enhances an extrajugular venous pathway. In a healthy subject, blood stasis in the IJV might spontaneously disappear during daily activity. When activity is restricted or there is severe incompetence of the IJV, venous stasis with subsequent thrombus formation can occur.

Most of our patients with slow jugular venous flow did not have associated discomfort, which was also the case with those with bilateral slow jugular flow. Only two of 132 patients (1.5%) with a concomitant IJV and subclavian vein thrombosis had clinical symptoms of deep vein thrombosis. Since this was not a prospective and postural-related dynamic study of the jugular vein, we did not use a questionnaire on symptoms. However, unilateral jugular reflux or jugular thrombus has been found to be related to several neurological disorders (16,17) such as transient global amnesia, transient monocular blindness (18), cough headache (19), and primary exertional headache (20). For a better understanding of the IJV and to improve the diagnosis of problems in the IJV, underestimation of jugular reflux and insufficient correlation with clinical features are two major issues that must be addressed.

The high flow groups comprised 27% of IJV abnormalities. In group D, increased jugular flow was observed in patients with hyperthyroidism, pregnancy, spontaneous intracranial hypotension, intracranial arteriovenous malformation, venous hum, and unknown causes. Causes of pulsatile jugular venous flow in group E included intracranial arteriovenous malformation and carotid-cavernous fistula. Unlike the slow flow groups, increased jugular venous flow was detected on both sides in 92% patients in the high flow groups. Bilateral pulsatile jugular venous flow has also been previously described in patients with carotid-cavernous fistula (21). As intracranial arteriovenous shunting flow becomes more severe, venous drainage increases through the bilateral IJVs into the heart. A physiological hyperemic state such as hyperthyroidism or pregnancy can induce an increase in jugular venous return as well as an elevation of jugular flow velocity. Further information from an associated history, physical examination, and thyroid function tests is helpful in distinguishing the possible etiology. Chen et al found that the mean maximum flow velocity in the ophthalmic vein was significantly higher in patients with intracranial hypotension than in healthy controls (22). We also found increased flow velocity in the IJV of a patient with intracranial hypotension. The actual mechanism has not been well clarified.

In patients with abnormal intracranial vascular lesions such as arteriovenous malformation, dural fistula, or carotid-cavernous sinus fistula, increased shunting flow from the arteries to the veins results in abnormally elevated venous flow in the IJVs. Pulsatile jugular venous flow is a subsequent effect of venous arteriolization (21). Complications of untreated abnormal intracranial arteriovenous shunting vary according to the hemodynamic changes and include intracranial hemorrhage, subarachnoid hemorrhage, venous sinus thrombosis, intracranial hypertension, increased intraorbital pressure, seizures, and headaches (23). Thus, an increased pulsatile IJV flow usually denotes a serious intracranial vascular lesion. Further angiographic studies, such as magnetic resonance angiography or digital subtracted angiography, are usually necessary to verify the cause.

There were limitations in the Doppler classification of the IJV in this study. Operator-dependent ultrasonographic examination as well as the nonconstant hemodynamic status of jugular venous flow may have caused a variety of results in Doppler waveforms, especially in the slow wave groups. Some adjuvant methods such as the Valsalva maneuver or air-bubble contrast for jugular reflux detection ameliorate problems in the evaluation of the IJV. Another limitation is that this was a retrospective study that did not include a detailed survey of clinical features or a chart review. Bias might have been present in the comparison of clinical symptoms. Hemodynamic effects of the IJV require a prospective study with long-term follow-up of both the clinical features and ultrasound findings.

Carotid duplex sonography is a noninvasive, reproducible tool available at the bedside to evaluate the cerebral arteries, including the jugular veins. Routine carotid duplex sonography usually does not include the jugular veins in most hospitals. Therefore, the importance and frequency of abnormal jugular venous flow could be underestimated. We recommend a quick screening of the IJV during carotid duplex sonography, as it carries no technical difficulty and is not time-consuming. Patients with pulsatile turbulent jugular flow warrant further angiographic studies to investigate the underlying pathological vascular disease.

5. Conclusion

Most abnormally decreased jugular venous flow occurs on the left side. Decreased venous flow or even thrombosis of one side of the IJV is usually asymptomatic. Increased jugular flow is associated with a physiological hyperemic state in cerebral hemodynamics. Increased awareness and angiographic studies are indicated in patients with pulsatile turbulent jugular venous flow because of a high correlation with intracranial arteriovenous shunting.

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