Spontaneous intracranial hypotension

To the Editor: With much interest we read the article by Franzini et al. (Franzini A, Messina G, Nazzi V, et al.: Spontaneous intracranial hypotension syndrome: a novel speculative physiopathological hypothesis and a novel patch method in a series of 28 consecutive patients. Clinical article. J Neurosurg 112:300–306, February, 2010), in which they speculate about the role of the spinal epidural veins and the vena cava system, both in the etiology and the treatment of spontaneous intracranial hypotension (SIH). The authors postulate a hypothesis about the pathophysiology of SIH “based on the current knowledge of the anatomy and the physiology of the epidural veins as well as the observations made in a consistent series of patients with SIH.” They state, “Venous drainage of the spinal epidural space is served by 2 main anatomical complexes. The upper thoracic plexiform venous network drains into the superior vena cava system via the radicular veins. Below L-2, the epidural venous network drains into the inferior vena cava system via a network of large radicular veins. These 2 systems communicate at the thoracolumbar junction,” and the authors continue with a paragraph about the influences of respiration, posture, physical activity (walking, standing), and the cardiac cycle on the venous return from the spinal epidural space to the heart. The authors believe that SIH is caused by the abnormal/elevated loss of CSF, either because of the presence of a leak at the level of a radicular dural sheath or, in the absence of a leak, via the radicular arachnoid villi. They conclude that negative pressure in the inferior vena cava (especially in the erect position and during walking) causes a pressure gradient toward the lower epidural veins and results in “sucking” of CSF, which causes intracranial hypotension. The authors assert that blocking of the (posterior?) L1–2 epidural space with a patch disconnects the upper thoracic plexiform venous network from the lower epidural venous network (below L-1) and stops the presumed “sucking” mechanism.

Dr. Franzini and colleagues described a very rare and intriguing clinical entity, whose etiology is poorly understood. The majority of patients with SIH have a CSF leak at the radicular nerve sheath in the cervical or thoracic area. Nevertheless, the application of a posterior lumbar epidural blood patch has been considered an accurate treatment in most of the patients. We wish to commend Dr. Franzini and colleagues on their attempt to explain both the etiology and the treatment mechanism in this rare disorder. However, we think that there are a number of inaccuracies in the authors’ interpretation of both the anatomical and the pathophysiological characteristics of the vertebral venous system (VVS) and the related venous networks.

The spinal epidural veins (the correct anatomical de-
is situated in the posterior part of the spinal canal, remote from the anterior IVVP. It is evident that no blocking of the IVVP will be achieved with the blood/glue patch constructed by the authors. On the other hand, even if the anterior and posterior IVVPs were blocked at a certain level within the spinal canal, the venous tracts via the basivertebral veins, the intervertebral veins, and the EVVP would physiologically serve as an alternative route for the venous blood toward the vena cava system.

Other points of serious concern are, for one, the per-

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**Fig. 1.** Schematized representation of the connections of the VVS with the vena cava system showing the vertebral vein (a), subclavian vein (b), segmental thoracic (intercostal) and lumbar veins (c), intervertebral vein (d), hemiazygos vein (e), internal thoracic vein (f), superior vena cava (g), inferior vena cava (h), azygos vein (i), ascending lumbar vein (j), sacral venous plexus (k), and the renal vein (l).

ception by the authors that the venous system has only one function: to provide the return of blood to the heart, and also, for another, that blood flow between the VVS and the inferior vena cava is unidirectional (from the epidural veins toward the vena cava). The authors completely ignore that, in humans, the connections between the VVS and the vena cava—and the azygos—system are valveless. In the absence of valves, blood can flow in either direction, depending on changes of the intrathoracic and intraabdominal pressure (respiration, coughing, straining, and so on) and hydrostatic forces (changes of posture, gravity forces). Besides, the authors do not take into account the fact that the volume of the VVS is 20 times larger than that of the contributing arteries. According to Herlihy, the excess of venous channels in relation to the arterial supply of the related spinal structures has to be considered as a provision of nature to equalize venous pressure, to redistribute blood, and, in pathological conditions of the vena cava, to serve as an alternative path for the continuation of the circulation. Others have stressed the role of the IVVP as a safety cushion and in CSF reabsorption. In a recent publication by Watanabe et al. about MR imaging findings in a series of 18 patients with SIH, 14 patients (78%) had distended epidural veins. Apparently, in patients with SIH (lying horizontally in the MR imager) the cervical anterior IVVP is distended. This is abnormal and does raise questions about the patency of the jugular veins in those cases.

In our opinion, current knowledge of the anatomy and the pathophysiology of the spinal epidural veins and the MR imaging findings of Watanabe et al. nullify the hypotheses by Franzini et al. We have to admit that we also are unable to explain how the application of a blood (or glue) patch in the posterior lumbar epidural space, followed by a short period of bed rest, improves symptoms in the majority of patients whose characteristics fit the diagnosis of SIH. It is evident that the VVS plays an important role in the regulation of volume and pressure of the spinal epidural space. Due to its morphological complexity, studies of the pathophysiology of this venous network are very difficult to perform. Franzini et al. already pointed out that pressure measurement within the vena cava of patients with SIH would provide important information. However, in the absence of baseline data, such information will be difficult to interpret. Much research is needed, both on the anatomy and the pathophysiology of the VVS. Hopefully this will provide a rational for the treatment of disorders that are related to this system, such as SIH.

References

9. Schievink WI, Meyer FB, Atkinson JL, Mokri B: Spontaneous

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**Fig. 3.** Schematized representation of the VVS in the lumbar region showing the anterior IVVP (a), posterior IVVP (b), basivertebral veins (c), posterior EVVP (d), anterior EVVP (e), intervertebral vein (f), radicular vein (g), and the ascending lumbar vein (h).
response: We carefully and with great interest read the letter by Groen and coworkers discussing issues raised by the authors in relation to our previous manuscript. We think that, obviously, scientific debate is imperative for improving and refining knowledge, and this is especially true with regard to conditions whose pathogenesis remains obscure. Theoretical arguments are important tools to discovering a pathway toward a more precise definition of the real and general picture in these cases.

Review of the anatomy of the VVS and its functionally related basivertebral and external vertebral systems is explanatory and illustrative; however, the considerations of the authors do not seem to us convincing enough to totally “nullify” out hypothesis:

1) The sentence about our “perception” of the unidirectionality of the blood flow from the VVS to the inferior cava system, but, above all, about our “perception” that the venous system has only one function (that is to say, to provide return of blood to the heart), is quite hazardous; it is obvious that, in the absence of a possible explanatory mechanism about such a complex disease, we intended to track a “baseline” concept to clarify subsequent logical steps that could lead to our hypothesis.

2) It also appears obvious that the posterior spinal epidural space is used by us (and by other groups) to inject the fibrin-blood compound, and it is used because it is actually the safest route to reach the entire epidural compartment. It seems clear that the compound also reaches the anterior IVVP and the anterior epidural space; the presence of the contrast medium in both compartments is demonstrated in Fig. 2 of our previous manuscript. So, even the most important anterior IVVP is involved in such treatment modality.

3) Groen and coworkers state that “even if the anterior and posterior IVVP were blocked at a certain level within the spinal canal, the venous tracts via the basivertebral veins, the intervertebral veins, and the EVVP would physiologically serve as an alternative route for the venous blood toward the vena cava system.” The serious concern about this characterization is that, even if the basivertebral veins, the intervertebral veins, and the EVVP served as an alternative route, the venous plexus, which is actually able to drain the CSF, is the internal plexus, the others being downstream; if, theoretically, the IVVP is blocked, CSF cannot be drained directly into the other venous plexuses.

4) Considerations following the report of Watanabe and colleagues (about the cause-effect relationships between epidural venous distension and the collapse of the dural sac) seem, to us, misleading, in that Groen and coworkers simply write that “after treatment with a blood patch, the dural sac re-expanded and the previously distended anterior IVVP returned to its normal proportions.” However, this actually does not mean that the real cause of the dural sac’s collapse is the venous distension (which should be the reverse of what was hypothesized by our group)—such a hypothesis could also be interesting, but Groen does not attempt to demonstrate it.

We hope our discussion of points, as well as the issues raised by Groen and coworkers, will contribute to a continuation of the debate about such mysterious and debilitating pathological conditions.

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Reference


Please include this information when citing this paper: published online July 2, 2010; DOI: 10.3171/2010.4.JNS10480.