Lamina terminalis fenestration

To the Editor: We read with great interest the recent paper by Chohan et al. (Chohan MO, Carlson AP, Hart BL, et al: Lack of functional patency of the lamina terminalis after fenestration following clipping of anterior circulation aneurysms. Clinical article. J Neurosurg 119:629–633, September 2013). In their study, the authors injected, on postoperative Day 1 following clipping of anterior circulation aneurysms, an iodine-based contrast agent intraventricularly to assess, with CT imaging, the flow into the basal cisterns through a fenestrated lamina terminalis. They concluded that fenestration of the lamina terminalis (FLT) did not result in functional patency of the lamina terminalis when performed as part of surgical clipping for ruptured aneurysms.

We have some remarks and criticisms regarding this article, which leads to clear-cut conclusions. The findings of CT ventriculocisternography were impressive, but somewhat surprising. We do not find a convincing pathophysiological explanation to support the assertion that FLT per se is useless. Furthermore, endoscopic transventricular FLT has been shown to be feasible, effective, and a good alternative to standard endoscopic third ventriculostomy (ETV) in selected cases of hydrocephalus.

The explanation provided by the authors that the frontal lobe, in its normal anatomical position after clipping, seals off the lamina terminalis fenestration may appear rational, but is not fully convincing. An alternative interpretation of the results of Chohan el et al. is the possibility that the fenestration may not be fully functional in the very early postoperative period (Day 1) in patients without acute post–subarachnoid hemorrhage (SAH) ventricular dilation. In this regard, the comparison with the hydrocephalus case treated by ETV could be misleading. Clearly, the patient with chronic hydrocephalus, included as a control in this study, had wide cisternal space and increased intraventricular pressure, whereas patients who have undergone surgery for SAH within the preceding 24 hours and are being treated with external ventricular drainage (EVD) may have a reduced intraventricular pressure and very tight cisternal spaces.

Fenestration of the lamina terminalis is, objectively, a ventriculocisternostomy. There are 2 fundamental requirements for its functioning: 1) the ventricular opening should be sufficiently wide; 2) the cisternal space must be sufficient.

Concerning the first point, maintenance of a large opening of the lamina terminalis should be assured through fine bipolar coagulation of its borders according to the technique originally described by Yaşargil et al. According to Yaşargil, FLT and clot evacuation in the basal and sylvian cisterns in selected cases reduced the shunt placement rate to 8.6%. In 1983, he modified his strategy by performing LTF in all patients and observed a reduction of the need for CSF shunting to 3% of 650 cases. In 1995, Yaşargil further modified his technique by sharply opening the lamina terminalis and briefly coagulating the opened rim to avoid subsequent closure due to adhesions.

The second point is equally important; FLT must be associated with generous subarachnoid dissection, clot removal, and opening of the Liliequist membrane to be functional. Beyond the above-mentioned considerations of Yaşargil about cisternal blood clearance, it has been demonstrated that it is important for functionality of the fenestration. Akyuz and Tuncer, in a randomized study comparing FLT alone and FLT combined with opening of the Liliequist membrane, suggested that this latter strategy reduced the incidence of shunt-dependent hydrocephalus (although the reduction was not statistically significant) and that this positive effect was particularly noticeable in patients in whom a cisternal “overflow” was observed at surgery on opening of the membrane. This corresponded to cases with clots within the fourth ventricle and ventricular dilatation.

Other studies have been focused on the potential advantage of combining FLT with cisternal clot clearance in reducing the incidence of shunt-dependent hydrocephalus.

It is generally believed that hydrocephalus after SAH is of the communicating type. Nevertheless, chronic hydrocephalus after SAH may also be of the noncommunicating type. The type of hydrocephalus that develops after SAH may depend on the location of the ruptured aneurysm and severity of SAH. It is possible that severe SAH (Fisher Grade III and IV) resulting from posterior circulation aneurysms more commonly causes a noncommunicating hydrocephalus by blocking the foramina of Luschka and Magendie, whereas Fisher Grade I or II SAH resulting from anterior circulation aneurysms typically results in a communicating hydrocephalus by obstructing the arachnoid granulations over the cerebral convexities. In the former situation, FLT would potentially be beneficial by providing an alternate route of egress for ventricular CSF.

Finally, our unpublished data demonstrate through MRI techniques, including cine phase contrast MRI, the anatomical patency (Fig. 1) and functional CSF flow through the subarachnoid space in patients who have undergone FLT.

In 1999, we published the first clinical series (52 cases) on the role of FLT in preventing shunt-dependent hydrocephalus. Since then, we have treated more than 400 patients combining FLT, evacuation of blood from the basal cisterns, and opening of the Liliequist membrane.
This resulted in an incidence of shunt-dependent hydrocephalus of less than 4% (unpublished data) compared to reported values of up to 30%. The procedure has been demonstrated to be safe. Nevertheless, we recognize that this issue is still debated and there is no consensus on the efficacy of this procedure. We, and others, are still convinced about the necessity to organize a multicenter trial focused on the identification of specific factors that may influence the incidence of chronic hydrocephalus as well as vasospasm after SAH. This would be important information in the continuing debate between clipping versus endovascular treatment of ruptured aneurysms.

Disclosure
The authors report no conflict of interest.

References

RESPONSE: We thank Tomasello et al. for their commentary on our paper. We agree with their overall assessment in that 1) our data only relate to functional (and not anatomical) patency of fenestrated lamina terminalis, 2) this functional patency was only assessed on Day 1 after FLT, and 3) it may or may not have any bearing on the long-term patency (functional or anatomical) of the fenestrated lamina terminalis. Our choice of early ventriculocisternography was based on the assumption that the likelihood of a fenestrated lamina terminalis remaining patent would be the highest closest in time to the surgery. Moreover, since this procedure has been proposed by some as instrumental in preventing delayed ischemic neurological deficit (DIND), its patency is only relevant (at least for vasospasm) during the first few days after SAH, the time when patients are most susceptible to DIND.

Our technique of performing FLT is very similar to what Tomasello and colleagues describe in their commentary. After meticulous lavage of cisternal spaces and clearance of all visible blood, the external ventricular drain is closed. The lamina terminalis is then opened sharply and widely with an arachnoid knife. The opening is further expanded with microforceps to achieve as wide an opening as possible. In addition, as recommend—ed by Professor Yasargil, the Liliequist membrane is also opened, and blood within each compartment is carefully removed. We, too, believe that FLT is a safe procedure when performed by experienced surgeons.

The reported incidence of hydrocephalus after an-
eurysmal SAH has a wide range (6%–67%). Generally speaking, chronic hydrocephalus occurs in more than 20% of patients with aneurysmal SAH and accounts for nearly 35% of all shunts placed. In a recent meta-analysis, the rate of shunt-dependent hydrocephalus in all patients (treated with clip ligation or endovascular coil embolization) was reported to be 18.4%. There was no statistically significant difference between the surgical and endovascular groups (17.5% vs 19.7%, respectively) in that study.

A number of studies suggest significant reduction in the development of shunt-dependent hydrocephalus due to microsurgical FLT. For example, Komotar et al. reported an 80% reduction in shunt-dependent hydrocephalus in their FLT cohort. Shunt placement rates as low as 4.25%, 6.9%, and 9% have been reported in patients who have undergone FLT. Despite a number of studies supporting microsurgical FLT, a recent systematic review by the same author who initially reported nearly 35% of all shunts placed. In a recent meta-analysis, Komotar reported an 80% reduction in shunt-dependent hydrocephalus due to FLT. A number of studies supporting microsurgical FLT, a recent systematic review by the same author who initially reported an 80% reduction in the incidence of shunt-dependent hydrocephalus due to FLT showed no significant association between FLT and the incidence of shunt-dependent hydrocephalus.

In our own series (unpublished) of nearly 200 patients treated for ruptured anterior circulation aneurysms, the incidence of shunt-dependent hydrocephalus was 7.7%. These patients nonrandomly underwent FLT or did not undergo FLT based on the surgeon’s preference. We found no statistically significant difference in the incidence of shunt-dependent hydrocephalus between the 2 cohorts. Our incidence of shunt-dependent hydrocephalus was much lower than that reported in literature. We believe, like many others, that meticulous intraoperative cisternal lavage and clearance of cisternal blood followed by prolonged and aggressive CSF drainage during the postoperative course, using EVD, lumbar drains, and lumbar punctures (for 4–11 days), promoted clearance of subarachnoid blood, further reducing arachnoid fibrosis and vascular inflammation and improving CSF dynamics.

Finally, our choice of including a patient with aqueductal stenosis and chronic hydrocephalus was merely to validate our technique of ventriculocisternography. We agree that no comparisons can be made between the chronic situation of hydrocephalus and acutely disturbed ventricular and cisternal CSF dynamics of aneurysmal SAH.

In conclusion, we generally agree with Tomasello and colleagues that our study is limited to functional patency of the fenestrated lamina terminalis during the very early postoperative period and does not exclude the possibility that the lamina terminalis remains anatomically open and could be functionally relevant at a later time. We still believe that aggressive clot removal and CSF drainage through EVD and the use of lumbar drains is instrumental in promoting CSF dynamics, at least during the time frame when patients are most susceptible to DIND. It is likely that FLT, along with the above-mentioned maneuvers, plays an important role in reducing the incidence of shunt-dependent hydrocephalus, an idea that is rational, but not settled in the literature.

References


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Internal carotid artery anterior wall aneurysms

To The Editor: The article by Indo and colleagues on the pathogenesis of anterior wall aneurysms (Indo M, Oya S, Tanaka M, et al: High incidence of ICA anterior wall aneurysms in patients with an anomalous origin of the ophthalmic artery: possible relevance to the pathogenesis of aneurysm formation. Clinical article. J Neurosurg 120:93–98, January 2014) left us with ambiguous reflections. Of the two main conclusions drawn by the authors, one is brilliant, while the second is objectionable.

Although the mechanism was hypothesized in the past, Indo and colleagues are the first to prove that aneurysm formation at the anterior wall of the internal carotid artery (ICA) is triggered by arterial wall weakness due to an anomalous origin of the ophthalmic artery (OA). One-fourth of patients with an OA originating...
from somewhere other than the supraclinoid segment of the ICA and only less than 1% of those with normal OA branching presented with an anterior wall aneurysm (p < 0.0001). Given the large cohort (over 1600 angiograms, 1.9% of which depicted the OA branching defect) and the approximate statistical power at the level of 99.9%, the significance of the evidence no longer seems doubtful. The contribution of developmental OA failure in the creation of an aneurysmal bulge at the ICA anterior wall may be regarded as ground-breaking information, although the results are limited to the whole and nonspecific group of aneurysms that consists of the clinically different saccular, blood blister–like, or dissecting entities.\(^5\)

However, the second conclusion made by the authors and pertaining to the association between an anomalous OA origin and the saccular shape of the ICA anterior wall is strongly objectionable for several reasons. The data appear insufficient to substantiate this thesis, as the statistics covered a very small sample; 16 patients harbored a dorsal wall aneurysm, which was confirmed in 10 intraoperatively. Although those results have statistical significance (p < 0.041), our calculations reveal their statistical power as insufficient (52.9%). Secondly, the ICA anterior wall aneurysms, by definition, constitute a subset of either dissecting (including blood blister–like aneurysms [BBAs]) or saccular aneurysms.\(^5\) Furthermore, Indo and colleagues have not included any saccular aneurysms or BBAs other than those that were anteriorly projecting and originating from the nonbranching site of the supraclinoid portion of the ICA. Therefore, several BBAs could have been omitted. It has been reported that only 5 of 40 BBAs protruded from the anterior wall, whereas saccular ones did so occasionally.\(^4\) The preliminary results of our ongoing systematic review of all reported cases of ruptured BBAs also confirmed that these aneurysms are not limited to an anterior projection (Fig. 1).\(^7\) Moreover, the configurational change from blister to saccular shape has been described.\(^2\) A different therapeutic approach is attributed to either a dissecting or a saccular aneurysm; therefore, Indo and colleagues made a valuable step in clinical management by exploring the preoperative factors differentiating these aneurysms. On the other hand, the best treatment option for either the dissecting aneurysm or the BBA has still not been clearly established.\(^1,2,4,7\)

It is also worth adding that Indo and colleagues did not mention multiple aneurysms. More than one cerebral aneurysm is reported in up to one-third of unselected patients, including those with ICA anterior wall aneurysms. Multiple aneurysms in patients with ruptured BBAs were identified in 4.8% of collected cases in the early systematic review.\(^7\) A BBA of the ICA coexisted with another saccular aneurysm arising from the same artery in 4 of 16 patients with multiple aneurysms. To emphasize that issue, we mention that an angiogram from our own series revealed a saccular OA aneurysm and a concurrent dissecting BBA, both arising from the same ICA (Fig. 2). Unlike the theory introduced by Indo and colleagues, the anterior wall aneurysm could be more plausibly attributed to the previously reported mechanisms, such as arteriosclerosis, hemodynamic effects, or an abrupt bend of the ICA.\(^4–6\)

In conclusion, regardless of its drawbacks, the study by Dr. Indo and colleagues\(^2\) contributed a great deal to the understanding of the pathogenesis of the ICA anterior

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**Fig. 1.** Possible projections of BBAs and their distribution. After the anteromedial projection, anterior is the second most frequent projection among ruptured BBAs. The data have been extracted from the database of the international prospective register of systematic reviews (PROSPERO). ACA = anterior cerebral artery; MCA = middle cerebral artery; PCoA = posterior communicating artery.
plaining an anterior wall/BBA pathogenesis: 1) arteriosclerosis; 6) and their cases in the following years. Subjectively selected papers abrupt bending, sheer stress; 5) and 5) anomalous origin of OA. 2)

ulceration causes focal wall defect; 1) arterial hypertension; 4) ICA angiogram (D) and does not appear in the 3D reconstruction with standard threshold settings (red arrow and question mark). The coexistence of a sacular aneurysm and a dissecting BBA on one curve of the artery was noted from clinical and pathological perspectives. Moreover, the significance of such a delicate classification would be extremely limited from clinical and pathological perspectives.

The authors report no conflict of interest.

Disclosure

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Fig. 2. Two different aneurysms of the same ICA. The right ICA angiogram (A, arterial phase; B, capillary phase; and C, venous phase) obtained because of SAH reveals a saccular OA aneurysm (blue arrowheads) and anterior wall BBA unrelated to any ICA division (red arrows). Of note, the BBA appears up to a late venous phase of the angiogram and does not appear in the 3D reconstruction (D) with standard threshold settings (red arrow and question mark). The coexistence of a saccular aneurysm and a dissecting BBA on one curve of the artery was not covered in the paper by Dr. Indo and colleagues. However, it can be explained by the possible increased wall shear stress on the outer curvature of the ICA.

Fig. 3. Graph depicting the increasing number of BBA publications and their cases in the following years. Subjectively selected papers explaining an anterior wall/BBA pathogenesis: 1) arteriosclerosis; 2) ulceration causes focal wall defect; 3) arterial hypertension; 4) ICA abrupt bending, shear stress; and 5) anomalous origin of OA.

References


RESPONSE: We appreciate the comments from Szmuda et al. based on their meticulous analysis of our paper and their experience with ICA anterior wall aneurysms. Below, we summarize their concerns and respond to each.

Szmuda et al. mentioned that our results on the relationship between the abnormal OA origin and aneurysm shape according to angiograms were based on insufficient data with low statistical power. In our study, only 16 ICA anterior wall aneurysms were identified among the 1643 ICAs that were examined (that is, 0.97%). The resulting low statistical power is attributable to this small sample size, which we unhesitatingly admit to as a limitation of our study. Therefore, we cautiously argued that our findings needed to be assessed in combination with other radiological examinations when determining the condition of the aneurysm wall. As Szmuda et al. stated, the configurational change in ICA anterior wall aneurysms over time must also be taken into consideration.

Szmuda et al. observed that some aneurysms could have been excluded from our study, including saccular aneurysms or BBAs not projecting anteriorly and originating from the nonbranching site of the supraclinoid portion of the ICA. If we follow their classification (see Fig. 1 in their letter), aneurysms in our study would simply correspond to a group consisting of anterior-, anterolateral-, and anteromedial-projecting subsets. Our study focused on ICA anterior wall aneurysms, and we believe that our inclusion criteria appropriately defined them in a manner that most neurosurgeons would accept. In addition, given our experience, we assume that it would be extremely difficult to precisely classify the ICA aneurysm projections into 8 categories. Moreover, the significance of such a delicate classification would be extremely limited from clinical and pathological perspectives.
We did not refer to the existence of multiple aneurysms at the ipsilateral ICA in our article. Among 16 ICA anterior wall aneurysms in our study, there was a single coexisting small aneurysm at the junction of the ICA and OA. In addition, we experienced 3 cases of ICA anterior wall aneurysms after enrollment for this study was completed. Of these cases, one showed two coexisting saccular aneurysms arising at the same ICA. Therefore, the incidence of multiple aneurysms in cases with ICA anterior wall aneurysms was 10.5% (2 of 19 vessels). Although the page mentioned by Szmuda et al. did not allow us to access the actual data of their study, our rate does not appear significantly different from the incidence in their report. Szmuda et al. mentioned that such high coexisting rates of saccular aneurysms and BBAs at the same ICA could be more plausibly explained by preexisting theories. However, we continue to believe that neither arteriosclerosis nor hemodynamic stress can completely account for the reason why BBAs are observed with such high frequency in the anterior wall of the ICA, because those factors would also affect other intracranial arteries. We believe that our embryological hypothesis more reasonably explains why the risk of the formation of ICA anterior wall aneurysms is approximately 50 times higher in ICAs with an anomalous OA origin than in those with a normal OA origin.

References
