Posterior circulation in moyamoya

To the Editor: We read with great interest the article by Hishikawa et al.2 (Hishikawa T, Tokunaga K, Sugiu K, et al: Assessment of the difference in posterior circulation involvement between pediatric and adult patients with moyamoya disease. Clinical article. J Neurosurg 119:961–965, October 2013), which clearly shows the clinical significance of steno-occlusive posterior cerebral artery (PCA) lesions responsible for infarction development in both pediatric and adult patients. In moyamoya disease, the leptomeningeal collaterals from the PCA are actually the only effective collateral suppliers of blood to the anterior circulation because bilateral steno-occlusive involvement in the terminal internal carotid artery (ICA) and proximal anterior and middle cerebral arteries prevents the development of leptomeningeal collaterals from each other and collaterals through the circle of Willis. Therefore, the steno-occlusive changes to the PCA give rise to the development of infarction in this disease process.

We completely agree with the authors’ finding that even less advanced steno-occlusive lesions in the ICA are associated with ipsilateral steno-occlusive PCA lesions in pediatric patients, while the severity of the steno-occlusive lesions in the ICA and the prevalence of ipsilateral PCA lesions correlated positively in both pediatric and adult groups. We believe this finding indicates that the vascular changes in this disease affect the PCA earlier in children than in adults, although they initially affect the ICA, and then the PCA in both children and adults. In pediatric patients, specifically those younger than 4 years of age, the earlier the age of onset, the earlier is PCA involvement with resultant ischemic strokes, with infarctions rather than transient ischemic attacks, even in cases of less advanced ICA lesions.3 This supposition goes along well with the discovery by Miyatake et al. of the homozygous c.14576G>A variant of ring finger protein RNF213 predicts early-onset and severe form of moyamoya disease.6,10 Some authors have speculated that individuals diagnosed in adulthood include patients whose disease process begins in childhood, based on serial follow-up angiograms showing that the disease progresses up to adolescence but stabilizes or progresses slowly after adulthood is reached,1 and the disease does not progress in most adult patients.3,6 In that case, the results of Hishikawa et al. might be interpreted as indicating that patients with later PCA involvement survive childhood and adolescence when the lifetime vascular demand is highest, without developing ischemic symptoms, and reach adulthood (adult-onset moyamoya disease). Conversely, for children with earlier PCA involvement, such patients are more likely to develop ischemic symptoms before they reach adulthood (child-onset moyamoya disease).

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Disclosure
The authors report no conflict of interest.

References

Response: We greatly appreciate the thoughtful comments offered by Drs. Mugikura and Takahashi. The anterior circulation (AC) has been reported to be involved in the origination of moyamoya disease based on the results of historical angioarchitecture analysis.5 A complementary relationship between the AC and the external carotid artery (ECA) system is involved in the pathophysiology of moyamoya disease.1 Suzuki and Takaku’s angiographic stages were graded based on a presumption of this complementary relationship and have traditionally played the leading role in evaluating disease progression. There is also a significant interaction between AC and posterior
circulation (PC) in moyamoya disease. Under the complex pathophysiological relationship linking AC, PC, and the ECA system in moyamoya disease, one patient may exhibit ischemic complications while another exhibits hemorrhagic complications. Moreover, one patient may develop symptoms during childhood while another does so during adulthood. This multiplicity of symptomatology in moyamoya disease is mysterious, and its mechanism has been debated for many years.

The PC functions as a main collateral pathway to the AC in moyamoya disease. The impact of the PC on hemodynamic ischemia is significantly greater than that of AC, but our investigation demonstrated that PC involvement was of little relevance to hemorrhagic-type moyamoya disease. In our patient cohort, the clinical significance of PC involvement in ischemic-type moyamoya disease was similar between pediatric and adult patients: 26% of pediatric and 33% of adult patients exhibited PC involvement. The only significant difference was that pediatric patients with PC involvement had significantly less advanced AC involvement than adult patients. If the interaction between the AC and PC started in childhood and progressed uniformly, the prevalence of adult patients with PC involvement would be higher than that of pediatric patients with PC involvement. Instead, the similarity in the prevalence of PC involvement in pediatric and adult patients in this study indicates the existence of distinct patterns of interaction between the AC and PC in moyamoya disease. The theory proposed by Drs. Mugi-kura and Takahashi is a valid explanation for our results and we completely agree with it.

There could be 2 patterns of interaction between the AC and PC: one an early form of interaction and the other a delayed form. In patients with an early interaction, PC involvement complicates less advanced AC involvement and causes symptoms during childhood. In patients with a delayed interaction, PC involvement correlates with advanced AC involvement and causes onset during adulthood.

Rapid developments in the realm of genetic analysis of moyamoya disease have been seen in recent years. In particular, the discovery of the RNF213 gene is noteworthy as a new approach to pathophysiology of moyamoya disease. The homozygous c.14576G>A variant of RNF213 has been reported to be correlated with a particular clinical phenotype of moyamoya disease — namely, younger age at onset, cerebral infarction, and PC involvement. The regulation of early and delayed interaction between AC and PC involvement by this variant of RNF213 genotype is one possible mechanism that could be responsible for the distinct patterns of interaction between the AC and PC in moyamoya disease. According to this hypothesis, pediatric patients without PC involvement might develop PC involvement in adulthood if they have the delayed-interaction pattern. In the light of this point, periodic and careful observation is important in pediatric patients with moyamoya disease.

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Mammillary body angle and craniopharyngioma

To The Editor: We read with great interest the article by Pascual et al. (Pascual JM, Prieto R, Carrasco R, et al: Displacement of mammillary bodies by craniopharyngiomas involving the third ventricle: surgical-MRI correlation and use in topographical diagnosis. Clinical article. J Neurosurg 119:381–405, August 2013). We congratulate Prof. Pascual and his colleagues. They evaluated the diagnostic accuracy of MRI to define the precise topographical relationships between intraventricular craniopharyngiomas (CPs), the third ventricle, and the hypothalamus and proposed novel methods, the type of mammillary body displacement, and the mammillary body angle (MBA), to differentiate primary third ventricular CPs and primary suprasellar CPs.

We would like to address two complementary issues with respect to the change in MBA: 1) The authors mentioned “In 69% of pseudoventricular cases the value of the MBA was greater than 120° (obtuse angle) and only 6% of lesions belonging to this topographical category displayed an MBA less than 90°.” The exceptional case was described in a report by Dusick et al. In our series of CPs, there were cases in which the MBA measured less than 90° as well (Fig. 1A and B). We find the common point is that those tumors were located anterior to the pituitary stalk (preinfundibular type), which was confirmed intraoperatively in our cases. In this condition, the third ventricle floor (TVF) is intact, the tumor compresses the third ventricle anteriorly, or the origin of force...
is anterior to the infundibular recess. Then the infundibulum and the TVF are pushed posteriorly, which causes the MBA to become acute (Fig. IC). In patients with retroinfundibular pseudointraventricular CPs, the compressive force is from downward to upward, resulting in an obtuse MBA. The difference in the size of the MBA associated with preinfundibular versus retroinfundibular pseudointraventricular CPs is explained by the origin of the force that causes compression of the TVF. 2) The MBAs in secondarily third ventricular CPs were variable. This variability may be caused by the stages of tumor growth. In the early stage, upward compression may cause an obtuse MBA. In the late stage, the increased size of the tumor may cause compression in a downward direction, which results in an acute MBA.

Previously, we reported the effectiveness of 3D-FIESTA–sequence MRI in the endoscopic expanded endonasal approach for the treatment of midline skull base lesions. The TVF could be better demonstrated by the 3D-FIESTA sequence than by routine MRI as well. For example, in Fig. ID and E, which show 3D-FIESTA images obtained in one of our patients with a CP (Case 1), the infundibular recess is enlarged and filled with tumor, the TVF seems to be torn into two pieces and destroyed in places. Intraoperative views confirmed the damage of the TVF. Figure IF and G, which are 3D-FIESTA images obtained in 2 other patients (Cases 2 and 3, respectively), demonstrate a not strictly intraventricular CP and a strictly intraventricular CP, confirmed intraoperatively in both cases. Observation of the TVF on contiguous sagittal and coronal 3D-FIESTA sequences may provide adequate information to classify the exact type of CP in most cases. An intact pituitary stalk can be found in primary intraventricular CPs even when the optic recess and infundibular recess are filled with tumor. Functional MRI of the infundibulum, tuber cinereum, mammillary bodies, and hypothalamus, although not currently feasible, may be helpful in distinguishing the type of CP and planning the surgical approach.

From the perspective of an endonasal endoscopic approach, the not strictly intraventricular CPs could be further classified into 2 subtypes: 1) CPs involving the infundibulum with or without involving the TVF; and 2) CPs involving the TVF without involving the infundibulum. In the case of Subtype 1, the tumor and offended pituitary stalk are simultaneously exposed after opening the arachnoid membrane. In the case of Subtype 2, the tumor cannot be exposed directly after opening the arachnoid membrane.

Fig. 1.  A–B: Sagittal T1-weighted (A) and 3D-FIESTA (B) MR images from our illustrative pseudointraventricular cases in which the MBA was less than 90° (29° in A and 53° in B). The angle indicated by the white lines represents the MBA.  C: Schematic illustration showing an acute MBA associated with pseudointraventricular CP. The red line represents the compressed TVF between the infundibular recess and the optic recess; the angle formed by the blue lines represents the MBA.  D–E: Sagittal (D) and axial (E) 3D-FIESTA images from a case of a not strictly intraventricular CP. The TVF seemed to be torn into 2 pieces (thin black and white arrows, D) and destroyed in some place (thick black arrow, E) in MR images. The damage was confirmed intraoperatively.  F: Sagittal 3D-FIESTA image from a case of a not strictly intraventricular craniopharyngioma. The TVF was damaged (thick black arrow) but the pituitary stalk was intact (thin black arrow), and these imaging findings were both verified intraoperatively.  G: Sagittal 3D-FIESTA image from a case of a strictly intraventricular craniopharyngioma with intact pituitary stalk.
membrane but by crossing the pituitary stalk bilaterally or opening the laminal terminalis. In some cases, strictly intraventricular CPs can only be exposed after opening the lamina terminalis.

In summary, we appreciate the enlightening method proposed by Prof. Pascual and his colleagues.

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Disclosure
The authors report no conflict of interest.

References

Response: We want to express our gratitude for the words of appreciation and the enthusiastic attitude toward our work shown in the letter by Drs. Ye Gu and Xiaobiao Zhang. We very much appreciate their sound insights on the topographical diagnosis of CPs after testing the validity of the MBA to define the CP–third ventricle relationships with the use of high-resolution 3D-FIESTA MRI. An accurate preoperative definition of the CP–TVF relationships cannot usually be outlined with using T1- and T2-weighted MRI sequences, even with gadolinium enhancement. Craniopharyngiomas larger than 3 cm at the time of diagnosis have typically caused such a gross distortion of or damage to the thin neural layer of the infundibulum and the tuber cinereum that the integrity of these structures becomes impossible to ascertain with standard MRI sequences. With regard to this diagnostic limitation, heavily T2-weighted and 3D-FIESTA MRI sequences have proved the most valuable tools for optimizing the accuracy of the assessment of anatomical relationships of CPs to adjacent neurovascular structures.

The relative anatomical position of the hypothalamus with respect to the CP has been shown to be related to the primary position of the lesion. Three basic types of CP-hypothalamic relationships can be considered: 1) CPs located below an anatomically intact but upwardly displaced hypothalamus (suprasellar-suprasellar CPs); 2) CPs located at the level of the TVF, originally developing either within the infundibulum or within the tuber cinereum, whose central portion or equator is tightly attached to the remnants of the breached hypothalamus (extra-intraventricular or not strictly intraventricular or infundibulotuberal CPs); 3) pure or strictly third ventricle CPs that expand exclusively within the third ventricle above an intact or atrophied TVF, with the hypothalamus positioned adjacent to the lower-third portion of the tumor. This classification takes into consideration the anatomical-functional vertical axis along the pituitary gland–pituitary stalk–hypothalamus structures. Identification of the cleavage plane between the distorted or atrophied thin layer of the TVF and the border of the CP is not usually possible preoperatively. Yet, the compact and flexible mammillary bodies remain visible on MRI, and their relative displacement with respect to the brainstem represents a valid, useful clue to define the relative position of the lesion along the vertical hypophysial-hypothalamic axis.

We agree with the authors’ observations about the variations in the direction of displacement of the mammillary bodies, different from those described in our paper. In fact, we had also observed such additional CP–third ventricle anatomical variants, but we tried to restrict our classification only to 4 major topographies, along the vertical axis, with the aim of highlighting the importance of the primary involvement of the hypothalamus by CPs. Hypothalamic involvement by the tumor is probably the fundamental variable that predicts both the feasibility of a complete and safe tumor resection and the success of the procedure with respect to the outcome for the patient. Preoperative definition of the CP–third ventricle relationships is not only an issue of academic value but is directly related to the degree of adherence of the lesion to hypothalamic nuclei and, consequently, the risk of hypothalamic injury should total removal of the mass be attempted. The highest rates of complications involving irreversible hypothalamic dysfunction (that is, hyperphagia, obesity, and long-term emotional and cognitive disturbances) have been observed to be directly related to the degree of anatomical disruption of the TVF displayed in postoperative MRI studies after total removal of CPs. Anatomical defects or breaches at the level of the infundibulum and tuber cinereum seen on postoperative coronal and sagittal MR images usually do not seem to have been caused by the surgeon but are the residual marks of lesions developing primarily within the floor of the ventricle, that is, of not strictly intraventricular or infundibulotuberal CPs. Given the high risk of hypothalamic injury posed by the latter category of CPs, this group must be differentiated preoperatively from extraventricular or suprasellar lesions developing at the lower infundibulum or at the pituitary stalk, which are separated from the TVF by the arachnoid and pia mater.

Gu and Zhang report in their letter cases of extra-intraventricular CPs developing at either the anterior aspect of the pituitary stalk or at the anterior lower infundibulum, which progressively fold the infundibular recess of the TVF inward without breaking through the neural layer of tissue. The authors include these lesions within our category of pseudointraventricular CPs. Although any suprasellar CP pushing the TVF upwards, but not causing actual invasion of the third ventricle, can be defined...
as a pseudointraventricular lesion, the category of pseudointraventricular CPs refers, in the strict sense, to those CPs mimicking a primary intraventricular location—that is, to large lesions that have completely obliterated the third ventricle after folding the intact TVF inward. These pseudointraventricular CPs have a similar appearance to not strictly intraventricular or infundibulotuberal CPs but can be differentiated preoperatively by their effect of upward displacement on the mammillary bodies (as evidenced by an obtuse MBA), as opposed to the downward displacement (acute MBA) observed for most CPs developing at the level of the TVF itself. In addition to the vertical hypophysial-hypothalamic axis employed in our topographical classification, Gu and Zhang have introduced a second, horizontal or anteroposterior axis along the infundibulum, which differentiates between CPs developing in front of, next to, or behind the junction of the pituitary stalk with the infundibulum. Jakob Erdheim was the first author to link the origin of CPs to the nests of epithelial cells observed in normal individuals along the pars tuberalis, the tongue of pituitary glandular tissue covering the stalk and infundibulum. These epithelial cell nests tend to gather at two main locations within the pars tuberalis, at the point where the stalk joins the pituitary gland (lower location) and at the point where the anterior aspect of the infundibulum joins the optic chiasm (upper location). Qi et al. have described how the arachnoid sleeve enveloping the pituitary stalk becomes thicker, more compact, and more adherent to the pia mater at the recess between the ventral aspect of the chiasm and the upper anterior aspect of the upper infundibulum. Such a trabecular meningeal reinforcement of the chiasm-infundibulum junction would explain the type of third ventricle deformation caused by suprasellar CPs developing at the upper stalk, as it is displayed in the 3D-FIESTA sagittal images provided by Drs. Gu and Zhang. In this topographical category the preinfundibular or prestalk intraarachnoid CP progressively compresses the chiasm-infundibular recess, displacing it posteriorly, without invading the third ventricle. The infundibulum and stalk remain adhered at the posterolateral surface of the tumor capsule. The lesion indents or protrudes into the anteroinferior third ventricle without disturbing the tuber cinereum or modifying the MBA, as Gu and Zhang correctly remarked. However, the lack of infiltration or of intrinsic involvement of the hypothalamus can be easily observed on sagittal MR images, and for that reason lesions with this topography should not be included within the group of pseudointraventricular CPs, although that topographical category, as defined in our previous papers, shares with the suprasellar-intraventricular preinfundibular lesions described by Gu and Zhang the characteristic of preserving the anatomical integrity of the TVF.

Topographical classifications of CPs that use as a principal criterion the relative position of the tumor with respect to the pituitary stalk have been proposed from the surgical perspective of the endoscopic endonasal approach. Kassam et al. have designed a comprehensive scheme that highlights the true anatomical relationships of the tumor to the vital neurovascular structures situated at the basal brain surface, especially the undersurface of the optic chiasm, the infundibulum, and the tuber cinereum. The Type I, or preinfundibular CP of Kassam’s classification, corresponds to the examples in Fig. 1A and B in Gu and Zhang’s letter. Apart from the maintenance of an acute MBA, the importance of this topography is the absence of hypothalamic infiltration or tight CP adherence to the TVF present in this group of lesions. This category also corresponds to the “EA” pattern described by Qi et al., one example of such a pattern being shown in Fig. 5 of their paper. In contrast, the CP Types II (transinfundibular) and III (retroinfundibular) are tumors usually breaking through the floor into the third ventricle; therefore these two types, which displace the mammillary bodies caudally against the brainstem, are the lesions associated with the highest rate of hypothalamic disturbances and the highest risk of irreversible surgical injury to the hypothalamus.

In addition to the relative location of the tumor either along the hypophysial-hypothalamic vertical axis or around the circumferential, horizontal area of the infundibulum, a third axis that indicates the depth of development of the lesion within the neural tissue can be taken into consideration to accurately define the topography of a CP. Following Erdheim’s embryological theory for the origin of CPs, Ivan S. Ciric has considered that the different CP-hypothalamus relationships depend on the relative initial position of the epithelial remnants of the hypophysial duct with respect to the leptomeningeal layer (pia and arachnoid mater) covering the pituitary stalk–infundibulum-TVF complex. Migration of hypophysial duct epithelial cells into the floor of the diencephalic vesicle before the formation of the pia mater will cause the inclusion of such cells within the floor and the potential development of infundibulotuberal or not strictly intraventricular CPs, lesions tightly adhered to the basal hypothalamus. This type of CP can develop either retroinfundibularly, within the tuber cinereum, or preinfundibularly, at the junction of the infundibulum with the optic chiasm. In the latter location, CPs will become extra-intraventricular tumors (tumors that are both extra- and intraventricular) causing a breach at the junction of the optic chiasm with the infundibulum while separating the two structures. This preinfundibular variant of not strictly intraventricular CP will not cause such a severe downward displacement of the mammillary bodies as the retroinfundibular or intra–tuber cinereum type reported in our classification scheme. The lesion, usually cystic, will accommodate to the elliptical space of the third ventricle, with its solid basal apex poking through below the optic chiasm. It corresponds to the “IA2 + SA” pattern in the classification scheme by Qi et al., and one example of this pattern is shown in Fig. 6 of their paper. To conclude, besides a classification scheme centered in the type of third ventricle involvement by the lesion, depending on its original position along the vertical axis, different topographical variants can be described according to the origin of the lesion along the horizontal axis followed by the optic chiasm, infundibulum, and tuber cinereum, and mammillary bodies. These variants will produce specific degrees of displacement of the mammillary bodies towards particular directions, which can be better observed with the use of 3D-FIESTA MRI sequences as reported by Gu and Zhang.
Neurosurgical forum

The concept of the secondary involvement of the third ventricle by CPs, that is, the invasion of the third ventricle by lesions originally developing at a sellar or suprasellar position below an intact TVF, is a controversial issue in the literature. In a recent letter to the editor, Juraj Steno et al. pointed out the rarity of such type of lesions, while remarking on the predominance of extra-intraventricular lesions causing an initial, primary disruption of the TVF, with a symmetrical intra- and extraventricular expansion.11 In our response to their letter, we provided a detailed list of differential features that help clinicians and neuroradiologists to differentiate between CPs developing primarily at the TVF (not strictly intraventricular CPs) and CPs invading the floor from an original extraventricular position.11 A total infiltration of the pituitary stalk (90% of cases), a multilobulated shape (67%), the complete occupation of the suprasellar cistern (67%), and the presence of tumor extensions into basal cisterns (50%) and/or into the sella turcica (50%) are the morphological features associated with secondary intraventricular CPs. For their part, not strictly intraventricular CPs are characterized by their round or elliptical shape (100% of cases), lack of involvement of the sella turcica and/or basal cisterns (83%), only partial involvement of the pituitary stalk and/or the suprasellar cistern (53%), and displacement of the mammillary bodies causing an acute MBA in 100% of cases. Extraventricular lesions with secondary invasion of the third ventricle will show the most variable range of displacement of the mammillary bodies, as is properly mentioned in Gu and Zhang’s letter. Craniopharyngioma variables such as the tumor’s consistency and shape, its speed of growth, and, above all, the specific area of compression along the floor will all influence the mechanical resistance offered by the TVF against tumor penetration. The process of progressive atrophy and gliosis of the neural layer of the TVF will also play a role, as well as adherences of the floor and meningeal layers to the lesion. As a consequence, some CPs will cause an upward displacement of the TVF before breaking through it, showing an upward displacement of the mammillary bodies, while others will invade the floor at earlier stages of development without causing a significant displacement to the physiological acutal value of the MBA.

The heavily T2-weighted and 3D-FIESTA sequences represent helpful, worthwhile MRI sequences for preoperative identification of the accurate topographical relationships between CPs and the third ventricle.11,11 Both methodologies should be employed to improve CP classification methods and to recognize preoperatively the lesions causing a primary or secondary anatomical disruption of the hypothalamus. In particular, the striking and thorough 3D-FIESTA methods used by Gu and Zhang have proved invaluable to implement the identification of the mammillary bodies and their displacements associated with specific CP topographies.

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Assessment of intracranial dynamics in hydrocephalus

To The Editor: I read with interest the article by Bottan et al.1 (Bottan S, Schmid Daners M, de Zelicourt D, et al: Assessment of intracranial dynamics in hydrocephalus: effects of viscoelasticity on the outcome of infusion tests. Laboratory investigation. J Neurosurg 119:1511–1519, December 2013). The authors are to be congratulated for postulating and trying to prove the underestimation of the CSF outflow resistance (R₀) values by the bolus lumbar infusion method, as compared to the constant-flow and constant-pressure methods. They have postulated that the viscoelasticity of the craniospinal space may account for the discrepancy, and they have succeeded in proving this in the phantom model in an experimental laboratory setting.

However, in the clinical setting the bolus lumbar infusion method is simpler, easy to perform, and less time consuming. Though the constant-pressure and constant-flow methods are more accurate, they are more time consuming and less suitable for routine clinical application. I have improvised the bolus lumbar infusion method for routine use in the clinical setting and have been using it for more than 10 years. I have found it is very useful for routine clinical application, can be done with readily available material, and can be performed in a short period. The R₀ obtained by this method correlates well clinically and is very useful in the management of normal-pressure hydrocephalus, postmeningitic hydrocephalus, and posttraumatic hydrocephalus and useful in the diagnosis of idiopathic intracranial hypertension.2 Since the clinician is going to use only one method for R₀ measurement, the lower value obtained by the bolus lumbar infusion method, compared with the other methods, may not matter in the ultimate analysis.

The article by Bottan and colleagues has to be commended for identifying a possible reason for the lower R₀ value by the bolus lumbar infusion method as compared to the constant-pressure and constant-flow methods.

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Disclosure
The author reports no conflict of interest.

References

RESPONSE: We thank the author for pointing out the successful application of bolus infusion testing in the management and diagnosis of hydrocephalus. Indeed, when a clinic uses its own infusion protocol and its own thresholds for R₀, the correct absolute value of the outflow resistance may be irrelevant. However, one of the reasons why infusion testing has not found more widespread clinical use is the lack of generally accepted R₀ threshold values. In order to compare clinical results obtained at multiple centers, even a relative reading of R₀ requires prior calibration to a common base value. A very reasonable choice for this base value is the correct absolute outflow resistance.

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Glioblastoma in elderly patients

To The Editor: We read with interest the recent article by Tanaka and colleagues, who have reported their observations of the presentation, management, and outcome of newly diagnosed glioblastoma (GBM) in a series of elderly patients treated at the Mayo Clinic (Tanaka S, Meyer FB, Buckner JC, et al: Presentation, management, and outcome of newly diagnosed glioblastoma in elderly patients. Clinical article. J Neurosurg 118:786–798, April 2013).1 The authors correctly point out that this group of patients has been under-represented in previous studies and therefore optimum treatment decisions remain a challenge. Tanaka and colleagues should be commended for shedding further light on this area, especially when we consider that elderly patients are forming an ever-increasing proportion of our neurosurgical caseload.

Treating high-grade glioma in the elderly population remains an interest of our group based at the James Cook University Hospital in Middlesbrough in the United Kingdom. We would like to bring to the attention of Tanaka and colleagues our published observations and outcomes of 71 patients with high-grade glioma followed up at our neuro-oncology clinic during 2004, which included 34 patients aged 65 years and older.2 In the ≥65-years group, we observed that those patients who received radical resection with or without adjuvant treatment had a median overall survival (OS) of 7 months as compared with 3 months in those patients who underwent biopsy with or without adjuvant treatment (p = 0.003). Unfortunately, temozolomide (TMZ) was not available from the National Health Service in 2004. The findings of our study reflect those of Tanaka and colleagues in advocating an aggressive management strat-
neurological surgeon.

Despite criticism for selection biases, the findings of both studies suggest that the gold standard of maximal safe surgical resection with adjuvant radiotherapy and chemotherapy should be offered to patients with high-grade glioma irrespective of age, when safe to do so. They also demonstrated that gross resection was associated with improved OS (11.5 months) when compared with biopsy (6.5 months), although the benefit was only evident in those who received adjuvant treatment. A multivariate analysis revealed that younger age, single lesion, resection, and adjuvant treatment were associated with better OS, but only adjuvant treatment was significantly associated with prolonged PFS. Despite being a poor prognostic factor, advanced age alone should not necessarily dictate the treatment offered to a patient with high-grade glioma.

Determining whether such improved survival translates into acceptable quality of life for elderly patients remains a topic for further enquiry. Nevertheless, such findings surely herald the end of the ageist neuro-oncological surgeon.

References


Disclosure

The authors report no conflict of interest.
mm Hg that was uncontrolled with standard therapy; all of these patients had a fatal outcome. Moreover, 12 (86%) of 14 KCH patients had evidence of spreading depolarizations compared to 5 (31%) of 16 VCU patients, and smaller craniotomies were weakly associated with a higher incidence of depolarizations; this suggests a pathophysiological link between raised ICP and depolarizations.

The finding of such a significant difference in the 6-month outcomes of patients undergoing early evacuation of a mass lesion (71% poor at KCH compared to 69% good at VCU) is intriguing. The authors have eloquently outlined the several confounding factors (such as differences in the baseline characteristics, early postoperative care, and rehabilitation) that limit our ability to draw firm conclusions regarding the superiority of primary DC. Nevertheless, we believe that the study findings strongly support the hypothesis that the ability to control ICP and brain swelling with a primary DC may improve the outcome of severely head injured patients undergoing evacuation of an ASDH. A recently published nonrandomized cohort comparison study of patients undergoing evacuation of an ASDH demonstrated that the standardized morbidity ratio was lower in individuals who underwent a primary DC (0.75; 95% CI 0.51–1.07) compared to those treated with a craniotomy (0.90; 95% CI 0.57–1.35). Although the confidence intervals overlapped, this study also suggests that a primary DC could be more effective than a craniotomy.

We wholeheartedly agree with the authors on the need for a randomized trial of primary DC. The 2006 Brain Trauma Foundation guidelines for the surgical management of ASDH have identified the issue of DC versus craniotomy for primary evacuation of an ASDH as a top priority for future research. In addition, the growing appreciation of the morbidity associated with the subsequent cranioplasty makes such a study even more essential. In late 2011, a collaborative group of neurosurgeons, neurointensive care physicians, and trial methodologists was formed with the aim of answering the following question: “What is the clinical- and cost-effectiveness of decompressive craniectomy, in comparison with craniotomy for adult patients undergoing primary evacuation of an ASDH?” The proposed RESCUE-ASDH (Randomised Evaluation of Surgery with Cranietomy for patients Undergoing Evacuation of Acute Subdural Haematoma) trial is an international, multicenter, pragmatic, parallel group randomized trial that aims to determine the effectiveness of primary DC versus craniotomy in the “real world”: hence, following consent, surgeons will decide on the suitability for randomization of individual patients intraoperatively, similar to routine practice. More than 35 sites from several countries (UK, Australia, Canada, Greece, Hungary, Italy, Norway, Singapore, Spain, and US) have already expressed their interest in collaborating. The study will start with an internal pilot phase in July 2014, which, if successful, will be followed by the substantive phase. We are actively seeking expressions of interest from more sites; if you are interested in finding out more about the study, please get in touch by emailing ak721@cam.ac.uk and pjah2@cam.ac.uk.

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Disclosure

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RESPONSE: We thank the authors of this letter for their support of our conclusions and are pleased that our comparative-effectiveness study has proven timely. The authors, who are all leading experts in neurotrauma in the world today, have eloquently explained how our data support the argument that a large primary DC in patients with ASDH may improve outcomes through preemptive control of ICP and preservation of neuronal function. In our study, loss of cortical activity and breakdown of ionic membrane gradients, caused by spreading depolarizations, were less common in patients with severe neurotrauma who underwent primary DC than in those who underwent traditional craniotomy, and outcomes were significantly better. Spreading depolarizations are a recently discovered and heterogeneous mechanism that may partly explain the delayed deterioration and poor outcome experienced by many, but not all patients after ASDH.

With great interest we note that these experts have proposed a new randomized controlled trial, RESCUE-ASDH (Randomised Evaluation of Surgery with Cranietomy for Patients Undergoing Evacuation of Acute Subdural Haematoma), to assess the effectiveness of primary DC versus traditional craniotomy. We of course strongly endorse their proposal and wish to emphasize that ASDH is not only the most frequently surgically treated type of mass lesion in patients with severe TBI, but this is also the subgroup of patients who do the worst. It is gratifying to see that 35 sites from several countries around the world have already expressed interest in participating in RESCUE-ASDH and that the infrastructure of RESCUEicp (Randomised Evaluation of Surgery with Cranietomy for Uncontrollable Elevation of Intra-Cranial Pressure) can be used to facilitate this new trial.

In parallel to this initiative, 3 major neurotrauma centers in the US have begun another ASDH trial, Hypothermia for Patients requiring Evacuation of Subdural hematoma (HOPES). Herman Hospital (University of Texas, Houston); Jackson Memorial Hospital (University of Miami); and University of Pittsburgh Medical Center will collaborate to randomize comatose ASDH patients to normothermia versus preoperative hypothermia induced by indwelling vena cava catheters (34°C–35°C). Power analysis based on post hoc analysis of 2 large prospective clinical trials (National Acute Brain Injury Study: Hypothermia II [NABIS: H II] and brain hypothermia therapy [B-HYPO] in Japan) suggests that approximately 200 patients will potentially yield a meaningful answer in this trial.

It is only through refinement of research efforts based on recommendations from past clinical trial failures that improvement in outcome can be achieved for severe TBI. In this regard, studies focused on specific pathoanatomical subtypes and situations of well-defined clinical equipoise, as in RESCUE-ASDH and HOPES, represent a significant advance and hold renewed promise for detecting significant treatment effects. We note that further refinement of clinical trials can be achieved by measuring mechanisms and surrogate markers of injury progression. Monitoring of spreading depolarizations now offers this capability as a technique focused on vulnerable brain and based on well-characterized cellular mechanisms. Efforts should be made to incorporate such monitoring in clinical trials to gain mechanistic insight into causes of trial success and failure, and therefore continue the refinement of future trial design.

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Anterior petrosal approach

TO THE EDITOR: We enjoyed reading the article by Gupta and Salunke (Gupta SK, Salunke P: Intradural anterior petrosal approach for petroclival meningiomas: a new surgical technique and results in 5 patients. Technical note. J Neurosurg 117:1007–1012, December 2012), in which they present a variation of the seminal anterior petro-
rosectomy described by Kawase et al. We also read with great interest the Letter to the Editor by Tatagiba et al. on this article.

Gupta and Salunke described 5 patients with petroclival meningiomas who underwent an intradural anterior petrosectomy tailored according to tumor extension. They claimed that bone removal was significantly minimized, which was addressed in their anatomical study. We believe the advantage of intradural (subdural) drilling of the petrous apex is that the surgeon can see the tumor and its extension prior to anterior petrosectomy. The surgeon can map the operative field and minimize the drilling area, thereby decreasing the risk of drilling-related injury. Since the extradural approach results in a formal anterior petrosectomy, which may be too extensive and pose an unnecessary risk to critical structures, the authors propose that in some cases, drilling of the entire anterior petrous bone may not be required. The authors stated that the extradural approach has some risks, such as traction injury of the greater superficial petrosal nerve (GSPN), leading to facial nerve palsy, interference with the vein of Labbé, and excessive bone removal, thereby increasing the risk of injury to the cochlea, semicircular canals, and petrous internal carotid artery (ICA). However, based on our experience of more than 100 cases of anterior petrosectomies spanning 10 years, we point out the risks of extradural anterior petrosectomy indicated by Gupta and Salunke, and the difference between the intradural and extradural approaches.

As opposed to what is indicated by Gupta and Salunke in their article, we show that the risk of damage to the seventh and eighth cranial nerves is very low in the extradural approach. In more than 100 cases of anterior petrosectomy performed at our institution for different pathologies, such as petroclival meningioma, epidermoid tumors, trigeminal schwannoma, and abducens schwannoma, we experienced only 3 cases of transient facial nerve paresis postoperatively. Two of these cases resolved after 2 weeks, and the third resolved after 1 year (unpublished data).

The GSPN is identified and preserved in most cases by using the correct maneuver. The GSPN is the most reliable landmark of Kawase’s triangle and is useful in identifying the geniculate ganglion and internal auditory canal. Semicircular canals are also preserved in all cases. Although the eminentia arcuata as a landmark is not always identified clearly, careful drilling at the posterior margin of Kawase’s triangle can preserve this bone structure. Intraoperative navigation system assistance is helpful in preventing injury to the bony structures.

The risk of injury to the vein of Labbé is quite low in the anterior petrosal approach because the extradural anterior petrosectomy makes a surgical corridor with minimal temporal lobe retraction. Moreover, we can retract the temporal lobe on the dura after cutting the tentorium. The vein of Labbé and temporobasal vein injury are not involved in this maneuver. Our approach requires a small temporal craniotomy and is less invasive than an orbitozygomatic craniotomy. Epidural drilling of the petrous bone is safer than subdural drilling. There are many important structures around the anterior part of the tentorial incisura exposed during subdural drilling. They are at a higher risk of injury during subdural drilling.

We can modify dural cutting and the extradural anterior petrosectomy (Kawase’s approach) in cases with specific venous drainage patterns from the superficial middle cerebral vein (SMCV), for example, the sphenopetrosal sinus route (8%–19%) and sphenobasal vein pattern (18%–31%), to preserve the venous drainage route and avoid the risk of venous congestion. The SMCV drainage route should be determined preoperatively and preserved if at all possible. There is a risk of brain injury caused by wide opening of the sylvian fissure and venous congestion or thrombosis by cutting the drainage route of the SMCV to the sphenoparietal sinus. Since the SMCV and sphenoparietal sinus are important drainage routes in some cases, we would like to know how often Gupta and Salunke cut the route to the SMCV in their procedure.

**Disclosure**

The authors report no conflict of interest.

**References**


**RESPONSE:** I have read with interest the views expressed by Tomio et al. Kawase’s approach is one of the
most elegant skull base surgical techniques. However, for tumors that have a large suprasellar extension, we find it difficult to reach the superior-most extent of the tumor. Many petroclival meningiomas in this region are fibrous and are not easily suckable or amenable to removal by CUSA (Cavitron Ultrasonic Surgical Aspirator). This requires tumor manipulation, and the vessels and nerves on the medial and superior aspect of the lesion, which are hidden from the surgeon’s view, may be stretched and at risk of injury. In addition, tumors that extend superiorly more than 2 cm from the tentorial edge may require more temporal extension, putting draining veins at risk of injury. In the intradural approach, the surgical corridor is along the long axis of the tumor and structures on both the medial and lateral aspects of the tumor are visible.

We agree with Tomio et al. that in about 10% of patients the venous drainage can be through the sphenopetrosal sinus and cutting the superficial middle cerebral vein may lead to venous congestion. Luckily in our short series of 5 cases, we did not encounter this problem. We believe that preoperative assessment of venous drainage pattern should be done in all patients before planning the surgical approach.

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