Neurosurgical forum
Letters to the editor

Insular gliomas and lenticulostriate artery position

To The Editor: We read with great interest the article by Moshel et al. (Moshel YA, Marcus JD, Parker EC, et al: Resection of insular gliomas: the importance of lenticulostriate artery position. Clinical article. J Neurosurg 109:825–834, November, 2008).1 The authors are to be really congratulated for performing this well-conducted study providing insight into newer surgical considerations for insular gliomas based on location of lenticulostriate arteries (LSAs) in relation to the tumor noted on stereotactically performed digital subtraction angiograms and MR images. They concluded that insular gliomas with an MR imaging–defined tumor volume located lateral to the LSAs on stereotactic angiography displace the LSAs medially and are easy to resect with minimal neurological morbidity compared with tumors that appear to surround the LSAs and are associated with higher rates of neurological morbidity if aggressive resection is pursued. However, it would be interesting to know the relation between the extent of resection and development of postoperative neurological deficits with histopathological diagnosis. The authors have not separately defined the histological findings of the tumors in Groups I and II. There were 10 patients in this series with high-grade gliomas (anaplastic, glioblastoma multiforme, and gliosarcoma). If these patients constituted the majority of those in Group II, the extent of resection would be highly dependent on the histology of the tumor as the high-grade gliomas by their very infiltrating nature do not lead to a shift in LSA position, precluding gross-total resection without afflicting neurological damage. Similarly the possibility of involvement of the majority of low-grade gliomas in Group I, which grow by displacement of the LSA and are amenable to resection cannot be ruled out, which if true would make performing digital subtraction angiography redundant. If both groups are well matched in terms of histopathological features, then it is worth assessing LSA position preoperatively, more so if in a noninvasive manner on MR imaging as described recently by Saito et al.2

Manish Kumar Kasliwal, M.B.B.S. 
Ashish Suri, M.B.B.S., M.Ch., D.N.B. 
All India Institute of Medical Sciences, New Delhi, India

Response: We appreciate the interest in our paper. In our experience, lateral LSA position was unrelated to histology, and histology was unrelated to postoperative neurological status. However, readers can conclude what they will from Tables 1 and 2.

Assuming pristine opening of the sylvian fissure, morbidity in the resection of subinsular gliomas is usually due to lenticulostriate vessel injury. This is more likely to occur if these vessels are located superficial to or within the tumor rather than deep to or displaced medially by the tumor. The concept that high-grade gliomas grow by invasion while low-grade tumors grow by displacement within that parenchyma), some high-grade lesions can do this also. Most gliomas, low- and high-grade, grow by invasion of motile isolated tumor cells in the extracellular spaces of intact and functioning parenchyma.

TABLE 1: Insular glioma pathology in 38 patients*

<table>
<thead>
<tr>
<th>Tumor Type</th>
<th>Group I (confined lateral to LSAs)</th>
<th>Group II (extending medial to LSAs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LG</td>
<td>16 (64)</td>
<td>11 (85)</td>
</tr>
<tr>
<td>LG astrocytoma</td>
<td>2 (8)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>GGN</td>
<td>4 (16)</td>
<td>2 (15)</td>
</tr>
<tr>
<td>LG mixed glioma</td>
<td>6 (24)</td>
<td>3 (23)</td>
</tr>
<tr>
<td>ganglioglioma</td>
<td>4 (16)</td>
<td>0</td>
</tr>
<tr>
<td>oligodendroglioma</td>
<td>0</td>
<td>4 (31)</td>
</tr>
<tr>
<td>glioneurocytoma</td>
<td>0</td>
<td>1 (8)</td>
</tr>
<tr>
<td>anaplastic</td>
<td>5 (20)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>anaplastic mixed glioma</td>
<td>2 (8)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>anaplastic astrocytoma</td>
<td>3 (12)</td>
<td>0</td>
</tr>
<tr>
<td>malignant</td>
<td>4 (16)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>glioblastoma multiforme</td>
<td>2 (8)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>gliosarcoma</td>
<td>1 (4)</td>
<td>0</td>
</tr>
<tr>
<td>pleomorphic xanthoastrocytoma</td>
<td>1 (4)</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>25</td>
<td>13</td>
</tr>
</tbody>
</table>

* GGN = ganglioglioneurocytoma; LG = low-grade; med = medial.

References

J Neurosurg / Volume 111 / December 2009

1294

References
Lumbar drainage for intracranial pressure

To The Editor: We read with interest the study by Tuettenberg et al. (Tuettenberg J, Czabanka M, Horn P, et al: Clinical evaluation of the safety and efficacy of lumbar cerebrospinal fluid drainage for the treatment of refractory increased intracranial pressure. Clinical article. J Neurosurg 110:1200–1208, June, 2009) focusing on the use of external lumbar drainage of CSF in patients with high intracranial pressure (ICP), as well as the critical accompanying editorial. It is clear that safety concerns arise when this topic is evaluated, especially when the highly experienced group from Heidelberg reports a 12% rate of cerebral herniation in their patients as well as a decrease from 98 to 82% in the number of patients with present basal cisterns.

We have used external lumbar drainage of CSF in patients with traumatic brain injury with refractory ICP following a strict protocol. In our protocol, all patients received hyperventilation and barbiturate coma or therapeutic hypothermia before using external lumbar drainage. In addition, CSF removal was performed in a controlled way. When the ICP was > 20 mm Hg, the CSF was removed continuously. After ICP decreased to < 15 mm Hg, the external lumbar drain was closed, CSF removal stopped, and patient’s head was placed at 0°. When ICP increased > 15 mm Hg the system was opened again and patient’s head placed at 30° again. Following this protocol, none of the 17 patients showed transient or fixed dilated pupils. Currently, after using external lumbar drainage in 30 patients with traumatic brain injury, we have observed the same results. We would like to highlight that our patients were previously treated (unsuccessfully) with second-level measures. When these measures fail to control ICP, mortality and severe disability increases up to 80–100%. We agree with Tuettenberg et al. that external lumbar drainage must play a role in patients with refractory ICP, and this topic should be addressed in a randomized controlled study, in patients previously treated with second-level measures and following a strict protocol.

José María Abadal, M.D.
Juan Antonio Llompart-Pou, M.D.
Javier Homar, M.D.
María Molina, M.D.
Jon Pérez-Bárzona, M.D.
Hospital Universitari Son Dureta
Palma de Mallorca, Illes Balears, Spain

References

Response: We very much appreciate the encouraging comments of Dr. Llompart-Pou and colleagues. We are glad to hear that other departments also use the concept of lumbar CSF drainage in patients with elevated ICP, a concept that is in our opinion quite successful.

Except for hypothermia, which we do not use because we do not see any evidence for it, their protocol is quite similar to ours. After ICP decreased to < 10 mm Hg, the external lumbar drain was closed, CSF removal stopped, and patient’s head was placed at 0°. When ICP increased > 15 mm Hg the system was opened again and patient’s head placed at 30° again. Following this protocol, none of the 17 patients showed transient or fixed dilated pupils. Currently, after using external lumbar drainage in 30 patients with traumatic brain injury, we have observed the same results. We would like to highlight that our patients were previously treated (unsuccessfully) with second-level measures. When these measures fail to control ICP, mortality and severe disability increases up to 80–100%. We agree with Tuettenberg et al. that external lumbar drainage must play a role in patients with refractory ICP, and this topic should be addressed in a randomized controlled study, in patients previously treated with second-level measures and following a strict protocol.

José María Abadal, M.D.
Juan Antonio Llompart-Pou, M.D.
Javier Homar, M.D.
María Molina, M.D.
Jon Pérez-Bárzona, M.D.
Hospital Universitari Son Dureta
Palma de Mallorca, Illes Balears, Spain

TABLE 2: Postoperative neurological status in 38 patients with insular gliomas

<table>
<thead>
<tr>
<th></th>
<th>1 Day Postop</th>
<th>6 Mos Postop</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LG</td>
<td>Anaplastic</td>
</tr>
<tr>
<td>neurologically intact</td>
<td>22 (81)</td>
<td>3 (50)</td>
</tr>
<tr>
<td>unchanged preop def-</td>
<td>1 (4)</td>
<td>2 (33)</td>
</tr>
<tr>
<td>icit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>hemiparesis</td>
<td>0 (0)</td>
<td>2 (33)</td>
</tr>
<tr>
<td>dysphasia</td>
<td>1 (4)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>worsened preop deficit</td>
<td>0 (0)</td>
<td>1 (17)</td>
</tr>
<tr>
<td>hemiparesis</td>
<td>0 (0)</td>
<td>1 (17)</td>
</tr>
<tr>
<td>dysphasia</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>new postop deficit</td>
<td>4 (15)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>hemiparesis</td>
<td>4 (15)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>dysphasia</td>
<td>1 (4)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>total</td>
<td>27</td>
<td>6</td>
</tr>
</tbody>
</table>
safer. We will think about adding this step to our proto-
col. (DOI: 10.3171/2009.6.JNS09873)

Jochen Tuitenberge, M.D.
Peter Schmiedek, M.D.
Elke Muench, M.D.
Universitätsmedizin Mannheim
Medizinische Fakultät Mannheim der Universität Heidelberg
Mannheim, Germany

Hypothermia

To The Editor: It is surprising that the paper by Har-
ris et al.3 (Harris OA, Muh CR, Surles MC, et al: Discrete
cerebral hypothermia in the management of traumatic
brain injury: a randomized controlled trial. Clinical ar-
refer to any more recent studies of head cooling in ado-
lescents than that of Wang et al.7 There have been several others,
reviewed last year,1 including 2 regarding traumatic brain
injury that used similar noninvasive circulating cooling
caps to the CoolSystems device.5,6

It would have been helpful to have a clear state-
ment about where the authors measured the intracranial tem-
perature. In their article it was not obvious whether it
was always measured in a ventricle (using the Camino
110-4HMT) or whether in some patients it was measured
in the parenchyma. The site of measurement could af-
fect the intracranial/bradyn temperature gradient since
sites nearer to the head surface are likely to be colder
than those deeper in the brain, particularly with active
body warming. But what is potentially more important
is that the combination of body warming and external
head cooling can produce large intracerebral temperature
gradients by comparison with systemic cooling.4 This
is not discussed, and no consideration is given to whether
this may be harmful or otherwise. The authors are not
alone in assuming that head cooling will avoid the risks
associated with systemic hypothermia, but whether this
assumption is true is not certain as we found when we
reviewed head cooling.2

There was a high refusal of consent for this study,
which is attributed to the inner city demographic and gen-
eral negativity to research. However, a single “research
assistant” obtained consent, and we are not told whether
they were a clinician or their preparation measures. Did
the research assistant have sufficient training and skills
for what is a difficult task that requires considerable sen-
sitivity?

Bridget Harris, M.Sc.
Western General Hospital
Edinburgh, United Kingdom

References

tive hypothermia in the management of traumatic brain injury.
Neurosurg Focus 25(4):E9, 2008

2. Harris BA, Andrews PJD: Direct brain cooling, in Mayer
SA, Sessler DI (eds): Therapeutic Hypothermia. New York:
Marcel Dekker, 2005, pp 323–386.

3. Harris OA, Muh CR, Surles MC, Pan Y, Rozycki G, Macleod
J, et al: Discrete cerebral hypothermia in the management
of traumatic brain injury: a randomized controlled trial. Clinical

4. Laptook AR, Shalak L, Corbett RJ: Differences in brain tem-
perature and cerebral blood flow during selective head versus

5. Liu WG, Qiu WS, Zhang Y, Wang WM, Lu F, Yang XF: Ef-
fects of selective brain cooling in patients with severe trau-
58–64, 2006

is protective in severe traumatic brain injury. J Clin Neuros-
13:995–1000, 2006

et al: Rapid and selective cerebral hypothermia achieved us-
ing a cooling helmet. J Neurosurg 100:272–277, 2004

RESPONSE: We would like to thank Ms. Harris for the
thoughtful letter regarding our paper. We would also like
to thank the editor for the opportunity to address Ms.
Harris’ questions and concerns.

First, Ms. Harris raised concern regarding our omiss-
ion of 2 recent articles1–2 that reported on trials of other
similar selective cooling devices. It is important to re-
mind Ms. Harris that the primary goal of this trial as
clearly stated, was “to determine the effectiveness of the
Discrete Cerebral Hypothermia System (“the cooling
cap”).” It was not our intention to perform a comprehen-
sive review of all available similar devices. In addition, it
must be noted that the devices referenced in the recent tri-
ts1–2 are dissimilar to those used in our study, in that they
include a component that cools the neck in addition to the
head. Our investigated device was selective to cerebral
cooling alone.

Ms. Harris also raised a concern regarding the loca-
tion of the ICP monitors placed. Our study sample con-
sisted of 25 patients (13 controls and 12 treatments). Of the
13 patients in the control group, only 1 patient received
the 110-4B intracranial pressure monitor (Camino bolt),
while 2 of the 12 patients in the treatment group received
a 110-4B intracranial pressure monitor (Camino bolt). All
other patients in this study received a 110-4HMT intra-
cranial pressure monitor (interventricular). As discussed
in our manuscript, the cooling cap neither established nor
maintained a significant cranial-bladder temperature gra-
dient. Thus we did not have sufficient data to determine
whether location of the monitors placed (intraparenchy-
mal vs interventricular) contributed to temperature dif-
fferences.

The primary outcome of this study was to evaluate
“the effectiveness of the cooling cap in reducing the pa-
tient’s internal brain temperature and in establishing a
gradient between patients’ core and brain temperatures
following TBI.” I disagree with Ms. Harris’ statement that
the authors did not give consideration as to whether this
was harmful or otherwise. Our trial and data do not sup-
port the effectiveness of the device in establishing such
a gradient. Furthermore, we have offered an extensive
discussion on the proposed merits and complications of
hypothermia and selective hypothermia. The data pre-
sented in support of Ms. Harris’ assertion that such con-
siderations may not be necessary was not included in our
analyses, as it lacked peer review.3

J Neurosurg / Volume 111 / December 2009
Ms. Harris raised a concern for our high refusal rate, and further inquired about the suitability of our research assistant. The research assistant for the study was fully qualified and trained in all appropriate institutional and national rules and regulations. Furthermore, she had extensive background within the community and had a clear understanding of the sensitive nature of patient enrollment and informed consent. Her success in this process directly reflects established recruitment successes in this population. Our manuscript provides appropriate reference to this matter.4 We also call the readers’ attention to published literature which examines the negative views on clinical studies in patients of urban centers.5 (DOI: 10.3171/2009.8.JNBS09965)

ODETTE A. HARRIS, M.D., M.P.H.
Stanford University School of Medicine
Stanford, California

References