Brain retraction injury

Jun Zhong, Manuel Dujovny, Alfred R. Perlin, Eimir Perez-Arjona, Hun K. Park and Fernando G. Diaz

Biomechanics Laboratory, Department of Neurosurgery, Wayne State University School of Medicine, Detroit, MI, USA

This paper reviews the literature of the brain retraction injury during the last century. The review focused on the instrument characteristic as well as the physiopathological and histopathological damage of the brain induced by brain retraction. It was found that lesions were induced by cerebral ischemia. We conclude that a better monitoring system needs to be developed to avoid brain injury. [Neur Res 2003; 25: 831–838]

Keywords: Brain retractor; cerebral blood flow; cerebral infarction; cerebral ischemia; cerebral perfusion pressure; complication

INTRODUCTION

Microsurgical techniques have recently become more and more common in intracranial operations, such as in cerebral aneurysms, brain tumor, vascular malformations, cerebellar hematoma, e.g., acoustic neuromas, hypophyseal tumors, etc. One of the difficulties presented in microneurosurgery is that of maintaining consistent retraction on the brain. The operative field is so small that the assistant’s hands tend to reduce accessibility for the neurosurgeon. Accordingly, the self-retaining retractor is one of the most important and indispensable instruments for neurosurgery.

In contemporary neurosurgery, during most intracranial procedures, the microscope is used to allow the surgeon to work on structures that are located deep in the brain. Under these circumstances, brain retraction is required for adequate exposure. It has been suspected, and later confirmed, that brain retraction causes secondary brain damage. As reported by Albin and Bennett, brain retraction delivers pressure to the brain surface. This pressure is transferred to the adjacent brain tissue, subsequently causing deformation and partial or total closure of blood vessels, as well as impaired oxygen delivery to brain cells. The severity of the damage to the brain depends on such factors as the brain retraction pressure distribution, geometry of the brain retractor, geometry and physical property and type of brain tissue, vascular pressure and the duration of the retraction. Secondary surgical lesions are promoted and worsened by associated systemic conditions such as hypotension, hypoxemia, hypercapnia, etc. It is estimated that there are 50,000 surgeries per year that last between 30 and 45 min (AANS report, http://www.neurosurgery.org, 2002). Various studies indicate that brain tissues are stressed after 15 min. Laha et al. found that if the mean arterial pressure exceeds the brain retraction pressure by less than 70 mmHg, the cerebrum will be damaged. When this difference is greater than 200 mmHg, the cerebrum will recover completely. Another criterion was found by Bell et al., which stated that the regional blood flow (rCBF) should be greater than 10–13 ml 100 gm⁻¹ min⁻¹, otherwise there was imminent danger of focal ischemic brain damage. To safeguard the brain during a lengthy surgery, a monitoring system is required that can ensure that one of the above criteria is met. The major obstacle in the development of a safe way of handling a delicate human tissue is the inability of present devices to measure local pressure exerted on the brain tissue.

INTRA-OPERATIVE RETRACTION MONITORING

Pressure measurement method

Brain retraction pressure monitoring, especially at an early stage of the intradural procedure, is useful in preventing brain damage or post-operative cerebral swelling. Albin et al., Hongo et al., Moss and Price, Waring et al., Andrews, Numoto et al., and Donaghy et al., all showed benefits of retraction pressure monitoring.

Mean retraction pressure method

In order to measure the mean retraction pressure, Numoto et al. and Donaghy et al. developed a brain retractor equipped with a very thin inflatable plastic bladder. Numoto’s bladder was made by heat-sealing together two thin rectangular plastic sheets. This bladder was connected through a small tube to an air pump. Inflation of the bladder was controlled by an electrical switch comprised of two metal foil strips (electrodes) glued to the opposite walls of the bladder. When the bladder was subjected to external pressure its walls did not move apart until the pressure inside would not exceed this external pressure. When the internal pressure is slightly greater than the external pressure, the walls of the bladder move apart and break the electrical circuit. Since the radius of the curvature of the wall of the bladder is very large, so according to Laplace’s formula the pressure difference across the wall is zero. In
other words, the pressure inside the bladder is equal to the pressure outside the bladder. To make a consecutive measurement, the bladder has to be deflated and started over again. This is the so-called ‘zero crossing method’ that was employed in the Codman CPM-100 Brain Retraction Pressure Monitor (Codman, Inc., Randolph, MA, USA), which has been used recently by Moss and Price\(^8\).

**Force measurement method**

Albin et al.\(^5,6\) and Bennett et al.\(^7\) developed a method for an estimation of mean retraction pressure. It is based on the measurement of the resultant force acting on the blade of the retractor. Mean retraction pressure is obtained by division of resultant force by the estimated surface area in direct contact with the tissue. Albin et al. measured resultant force by placing a strain gage on the handle of the brain retractor. To minimize the influence of change of temperature on the force measurement, a second strain gage on the opposite side of the handle was used.

**Local retraction pressure method**

Hongo et al.\(^9\) took another route to retraction pressure monitoring. They used a brain retractor manufactured by Tokai Rika Medical Electronics using a strain gage technology. This brain retractor was equipped with a catheter-tip miniature pressure transducer.

**Cerebral pulsation method**

Monitoring of cerebral pulsation is another way to make brain retraction safer. It also correlates with arterial pulse as well as with mechanical ventilation. Waring et al.\(^10\) in 1990 developed and patented a retractor that incorporated an infrared emitter and detector.

**Blood flow monitoring**

An approximation of rCBF in the area to be retracted would give the surgeon a valuable index to aid in the prevention of potential ischemic retractor injury. Wilkins et al.\(^11\) developed a technique of measuring rCBF intra-operatively by using a bolus injection of xenon 133 into the common carotid artery. The technique, however, is not continuous and had the potential for error if the tracer happened to enter the external carotid, which is more sensitive to hemispheric ischemia than it is to regional ischemia, which is more likely to be produced by brain retraction. Feindel et al.\(^12\) used a method of injecting 1% fluorescein into the carotid artery intra-operatively while taking rapid serial photographs. The flow patterns produced are again more sensitive for hemispheric ischemia, yet they are not continuous. Carter et al.\(^13\) developed a thermal diffusion probe constructed from a Peltier Stack, producing a continuous measure of rCBF. Unfortunately, irrigation or electro-coagulation near the probe producer artifact interferes with the probe’s ability to read beneath the retractor.

Although many methods of rCBF measurement can be used, only laser-Doppler\(^12\) and thermal diffusion\(^13\) have become accepted. According to autoradiographic techniques using [14C] iodoantipyrine as a tracer, laser-Doppler flowmetry (LDF) is accurate at measuring changes in rCBF, although there is some question regarding the accuracy of absolute values\(^14\). Although transcranial Doppler blood velocity monitoring has also been used intra-operatively, the spatial resolution is insufficient to be a reliable monitor of the focal ischemia resulting from brain retraction. At the present time, the available techniques are probably neither sufficiently accurate nor practical for routine intra-operative use, though rCBF monitoring could be very useful to protect against brain retraction ischemia.

**Brain electrical activity monitoring**

Monitoring brain electrical activity can be a very useful aid to cerebral protection during brain retraction. It has been well established that brain electrical activity (both spontaneous and evoked potentials) is altered and then lost at an rCBF level of 10–15 ml 100 g\(^{-1}\) min\(^{-1}\). An approximately linear relationship between somatosensory evoked potentials (SSEP) amplitude and rCBF has been observed in primates\(^15\). For extensive cranial base procedures, all three monitoring techniques, electro-encephalogram (EEG), brain-stem auditory evoked potential (BAEP), and SSEP, have been used concurrently\(^16\). The intra-operative electro-oculograms (EOG)\(^17\), brain-stem trigeminal evoked potentials (BTEPs)\(^18\), and BAEP\(^19\) can be used in posterior cranial fossa surgery when the brain stem might be retracted.

The currently available techniques of intra-operative rCBF monitoring have not reached the stage of development of electrophysiological monitoring. However, rCBF monitoring should evolve into a reliable intra-operative tool for routine use in the near future.

**Microdialytic monitoring of the cortex**

Mendelowitsch et al.\(^20\) use microdialysis to detect local ischemia, which could be caused by brain retraction. Parameters which quantified the degree of ischemia were on-line pH, ascorbic acid, uric acid, glutathione, cysteine, glucose, and lactate. The results showed that the parameter changes were all in accordance with ischemic conditions in the region of the probe; they disappeared at the end of retraction, or sometimes even before. During the bypass operations, there were no marked changes in on-line pH or in any of the measured parameters.

**BRAIN RETRACTION DAMAGE**

Secondary brain damage after retraction is not uncommon\(^11–33\). Brain injuries associated with retraction are caused by the retractor blade’s focal pressure on the brain, which causes direct damage or produces a local deformation of the brain tissue and then develops a reduction or even cessation of the local perfusion. Such a contusion or infarction is dependent on the shape and
number of the retractor blade pressure and duration of the retraction. The literature supports that permanent injury occurs with rCBF of less than 10–15 ml 100 g−1 min−1 in larger animals, particularly when the ischemia persisted for 120 min.

Generally speaking, clinically significant post-operative deficits appear in 3%–9% of cases, though there is considerable variation depending on the difficulty of the procedure, resources available (e.g., intra-operative monitoring), skill of the operating team and the criteria.

Table 1: The animal study

<table>
<thead>
<tr>
<th>Author</th>
<th>Method</th>
<th>Results</th>
<th>Conclusion</th>
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<tr>
<td>Andrews(1992)</td>
<td>Combining brain retractor blade pressure measurement with monitoring of brain electrical activity in rats</td>
<td>RP of 30 mmHg resulted in a 50% decrement in EP amplitude after 10–20 min. Recovery occurred within 5–10 min of retraction release. Early loss of EP was associated with lower blood pressure, lower rCBF, lower PaCO2 and older age</td>
<td>Retractors should be relaxed every 10–15 min (for at least 5 min), and hypotension, in particular, but also hypocapnia (hyperventilation) should not be used indiscriminately.</td>
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<td>Bennett(1977)</td>
<td>Cortical EP, neurological status and cortical histological changes were determined as a function of graded levels of brain retractor and systemic perfusion pressure in dogs</td>
<td>A reduction of 50% of the EP amplitude after 60 min brain retraction signaled with high probability the occurrence of post-operative sensory and/or motor deficits and cortical histopathology</td>
<td>Cortical EP represents a reliable indicator of the functional effects produced by applied cortical RP at several levels of systemic perfusion pressure.</td>
</tr>
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<td>Hongo(1987)</td>
<td>The temporal lobe was retracted with a strain-gauge retractor under a pressure of 20 mmHg at the beginning in mongrel dogs</td>
<td>50% reduction in RP was 6.6 min. Attenuation gradually decreased when retraction was repeated. The lower the head position, the larger the amplitude RP</td>
<td>The hardness of the retracted local brain can be judged by analyzing the retraction wave.</td>
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<td>Laha(1979)</td>
<td>Graded RPs of 10, 20, and 30 mmHg were applied for 1 h to dogs’ cerebellum in sitting position</td>
<td>The difference between systemic perfusion pressure and RP were important determinants of the outcome</td>
<td>The mechanism of damage from RP is discussed.</td>
</tr>
<tr>
<td>Lownie(1990)</td>
<td>The cat was induced hypotension and implicated with retractor at 20 mmHg for 1 h. Then BP was returned to normal either &lt; 3 or &gt; 20 min</td>
<td>There was no significant difference between the fast and slow blood restoration groups</td>
<td>Restoration of blood pressure to normal post-operatively either gradually or rapidly did not influence the degree of edema formation.</td>
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<td>Rosendal(1982)</td>
<td>The rCBF was investigated using autoradiography with 14C-iodoantipyrine at different RP in Wistar rats</td>
<td>With 20 mmHg of RP for 30 min, a rCBF reduction to 10–75 ml 100 g−1 min−1 was found; With 30 mmHg for 30 min, 0–40 ml 100 g−1 min−1; 40 mmHg for 15 min, 0–15 ml 100 g−1 min−1. The rCBF in the basal ganglia remained unchanged.</td>
<td>Even with a brain RP of 20 mmHg for 30 min, there seemed to be a risk of focal ischemic damage.</td>
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<tr>
<td>Rosenørn(1989)</td>
<td>Different pressures were placed at the cortical surface for 15 min in Wistar rats</td>
<td>In 20 mmHg group, 1/6 revealed infarction involving all cortical layers; in 30 mmHg group, 5/6 cortical infarction, partly hemorrhagic; in 40 mmHg group, 6/6 cortical damage. No damage outside the retractor area or in deeper brain was found.</td>
<td>Only if RP did not exceed 20 mmHg for 15 min could damage be avoided in this model.</td>
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<tr>
<td>Rosenørn(1987)</td>
<td>Flat, flat with rounded edges and curved retractors were compared in rats</td>
<td>Average rCBF was 80, 90 and 75 ml 100 g−1 min−1, respectively</td>
<td>The differences were not significant.</td>
</tr>
<tr>
<td>Rosenørn(1988)</td>
<td>rCBF measurements and neuromorphological studies were conducted in rats</td>
<td>A 29–31 min RP of 40 mmHg with 5–7 min intermittent and 1 min interval did not produce brain damage. But a 10 min pressure induced brain damage.</td>
<td>Intermittent RP has to be preferred to continuous RP and the time threshold is about 7 min.</td>
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<td>Waring(1990)</td>
<td>A retractor system incorporating an infrared emitter and detector that allow detection of cerebral pulsations (CP) was developed in cat model</td>
<td>Gentle contact with the surface of the brains shows CP correlate with arterial pulse and mechanical ventilation. The amplitude of CP decreases with higher RP and disappears at around 20 mmHg</td>
<td>Monitoring cerebral pulsation may prove useful in clinical neurosurgery with respect to avoiding excessive retraction, which causes brain damage.</td>
</tr>
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<td>Yokoh(1983)</td>
<td>Continuous and intermittent retraction was compared in dogs</td>
<td>In continuous group, brain damage started at RP = 30 g and fully recovered after release when RP &lt; 40 g; in intermittent group, damage was morphologically minimal with RP &lt; 50 g and recovered more promptly</td>
<td>The intermittent retraction was superior than continuous retraction.</td>
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EP, evoked potentials; RP, retractor pressure; CP, cerebral pulsations; BP, blood pressure.
used to define brain injury. Spetzler et al.\textsuperscript{37} reported an incidence of approximately 10% of complications in skullbase surgery that were likely related to the retraction. These complications included parenchymal hematomas, aphasia, hemiparesis and numbness. A study on surgery for unruptured aneurysms of the posterior circulation found that 4% of patients suffered retraction-induced brain injuries\textsuperscript{38}. With regard to pineal region tumors, 2 of 12 patients suffered permanent visual defects, which the authors attributed to occipital lobe retraction\textsuperscript{39}.

Animal studies

In 1972, Donaghy and co-workers\textsuperscript{15} built a brain retractor that could measure retraction pressure and record EEG, and an animal model of retraction injury was developed.

Dogs

Studies in dogs demonstrated that EEG did not change much until the retraction pressure exceeded 18 mmHg. It was found that temporal lobe retraction pressure in a dog at 25 mmHg for 1 h produced blood–brain barrier disruption and a change in the SSEP that persisted for 24 h. Histopathological examination 72 h after the dog was killed shows brain edema. In addition, it was found that 50 mmHg retraction pressure that lasted for 1 h had the effect of induced hypotension. In the normotensive group, SSEP amplitude remained greater than 80% when the retractor pressure was 30 mmHg. However, it dropped to less than 60%, without recovery, if the retractor pressure was 40 mmHg or more. In the induced hypotension group, SSEP were preserved only if the retractor pressure was 10 mmHg or less. The predictive value of the SSEP was borne out by the histopathological examination many days after the end of the experiments. A 50% or more reduction in SSEP amplitude after 60 min of brain retraction had occurred in most animals with neurological deficits. Two animals that appeared neurologically intact, upon examination had deep cortical lesions\textsuperscript{16,17,40}. Damages to the dog’s temporal lobe and cerebellar hemispheres were also reported by Hongo et al.\textsuperscript{41} and Laha et al.\textsuperscript{42} with retraction pressure of 10–30 mmHg. Yokoh et al.\textsuperscript{43} demonstrated that brain damage was less severe with intermittent retraction than with continuous retraction.

Rats

Studies in rats, where the rCBF was assessed by an autoradiographic technique, demonstrated that the blood flow decreased directly with increased retraction pressure and increased retraction duration. With a retractor pressure of 20 mmHg for 30 min, there seemed to be a risk of focal ischemic damage\textsuperscript{44}. A 50% decrease in the evoked potential (EP) amplitude was observed after 10–20 min of retractor pressure of 30 mmHg\textsuperscript{45}. If the retractor pressure did not exceed 20 mmHg for 15 min, damage to cortical layers could be avoided\textsuperscript{46}. It was also shown that brain retractor pressure of 40 mmHg with 5–7 min intermittent and 1 min interval did not produce brain damage while a 10 min continuous pressure induced brain damage\textsuperscript{47,59}. The shape of the retractor blade had no effect on rCBF\textsuperscript{48}.

Swine

Andrews et al.\textsuperscript{49} developed a swine model that simulates brain retraction and found that a 30 mmHg of retraction pressure usually resulted in a 50% decrement in EP amplitude within 15 min of beginning retraction. The brain’s tolerance to retraction was reduced further by the combination of hypertension and hyperventilation than with intervention alone.

Cats

Lowni et al.\textsuperscript{50} studied a cat that was induced into hypotension and then was retracted at 20 mmHg of pressure for 1 h. The blood pressure was then returned to normal either within 3 min or over 20 min. Restoration of blood pressure post-operatively either gradually or rapidly did not influence the degree of extent of edema formation. Waring et al.\textsuperscript{11} investigated retractor system in a cat model by incorporating an infrared emitter and detector that allowed detection of cerebral pulsations. Gentle contact with the surface of the brain showed cerebral pulsations correlated well with arterial pulse as well as mechanical ventilation. The amplitude of cerebral pulsations decreased with higher retractor pressure and disappeared at approximately 20 mmHg. The pressure on the surface of the brain decreased 50% in 5 min even though the position of the retractor remained constant. Monitoring cerebral pulsation may prove useful in clinical neurosurgery with respect to avoiding excessive retraction, which causes brain damage.

Baboons

Study in baboons considered that the permanent injury was caused with an rCBF of less than 10–15 ml\textsuperscript{1}100 g\textsuperscript{1} min\textsuperscript{1}, particularly when the ischemia persisted for 120 min.

Clinical study

A retrospective study analyzed retractor-related complications in 1896 patients and found that the highest incidence of the complication was in those patients with abnormalities of the chiasmal and cellular regions and the middle cranial fossa. The complication was absent in patients with aneurysm of the anterior areas of the Willis circle\textsuperscript{62}. By intra-operative brain retractor pressure monitoring in 37 patients, it was noticed that the average initial brain retractor pressure was 26.6 mmHg, when the brain was retracted safely and gently by experienced neurosurgeons. The use of CSF withdrawal reduced retraction pressure more rapidly. The use of multiple retractors may be less harmful to the brain than retraction with a single retractor\textsuperscript{63}. An investigation of 120 retraction pressure recordings in 23 patients showed an initial steep and later a more gradual slope. The pressure at the
tip was higher than at the center of the retractor. Induced hypotension with sodium nitroprusside and the administration of mannitol did not influence the current pressure beneath the brain retractors.

With positron emission tomography (PET), Yundt et al. measured the regional cerebral metabolic rate for oxygen, regional oxygen extraction fraction, and rCBF in four patients before and after right frontotemporal craniotomies for clipping of ruptured anterior circulation aneurysms. It was observed that there was a 45% reduction in regional cerebral metabolic rate for oxygen (1.87 ± 0.22 to 1.04 ± 0.28 ml·100 g⁻¹ min⁻¹) and 32% reduction in regional oxygen extraction fraction (0.41 ± 0.04 to 0.28 ± 0.03) in the region of retraction but no change in the opposite hemisphere. Brain retraction produced a focal area of tissue injury at the site of retractor blade placement, as compared to more diffuse vascular territory changes produced by vasospasm. This reduction in the cerebral metabolic rate of oxygen and the oxygen extraction fraction indicates a primary reduction in metabolism and uncoupling of flow and metabolism (luxury perfusion).

The microdialysis is a sensitive method of detecting intra-operative changes in cerebral metabolism. By placing microdialysis probes in cerebral cortex beneath the brain retractor and perfused with Ringer solution, the microdialysis vials for glucose, pyruvate, lactate, glutamate and glycerol can be analyzed. It was found that during brain retraction regional intracerebral glucose was within normal range in cortical tissue and the levels of lactate, glutamate and glycerol as well as the lactate/pyruvate ratio were considerably above normal range. The biochemical analysis shows a pronounced incompletely cerebral ischemia due to brain retraction. The increases in glutamate and glycerol indicated tissue damage and degradation of cell membranes. Intracerebral microdialysis may be a valuable tool in the development of optimal techniques for brain retraction during neurosurgical procedures. With microdialysis, it was detected that the on-line pH fell after introduction of the retractor, and rose after removal; levels of ascorbic acid, glutathione and lactate increased and glucose decreased. These changes are all in accordance with ischemic conditions in the region of the probe; they disappeared at the end of retraction, or sometimes even before.

**INTERVENTIONS TO MINIMIZE BRAIN RETRACTION INJURY**

**Technical refinements on retraction**

The advent of ZD frame system (Zamorano–Dujovny Multipurpose Neurosurgical Image Guided Localizing Unit, Striker, Portage, MI, USA) and other stereotactic retraction systems, have significantly reduced cerebral retraction allowing the surgeon to work with small craniotomy exposures. At present, there is a tendency to extensively remove the skullbase bone to facilitate exposure and minimize brain retraction lesions. Positioning the patient in such a way that the lesion is highest in the field and can aid in the exposure and reduce the need for direct brain retraction. Cerebrospinal fluid (CSF) drainage is very helpful to enhance cerebral compliance.

**Chemical brain retractor**

The chemical brain retractor concept includes the use of a total intravenous anesthesia technique, mild hypocapnia and mannitol with strict monitoring and maintenance of the global cerebral homeostasis. This contributes to decreased brain volume and intracranial pressure and allows the best possible access to the operating site, while avoiding excessive pressure under the surgical brain retractors. Neuronal protection is based on a better understanding of the biological basis of secondary brain damage; therapeutic or prophylactic techniques include the use of specific pharmacological agents, hypothermia, hemodilution and maintenance of an elevated cerebral perfusion pressure.

**Anesthesiology**

Isoflurane combined with nitrous oxide and a narcotic, which is probably the most widely used anesthetic agent for intracranial procedures, has been tried recently as a cerebroprotective agent in neurosurgical procedures. Propofol, however may cause blood pressure drop, can decrease brain retraction pressure with the advantage of reliable intra-operative SSEP recordings. It has been shown that intra-operative pentobarbitol increases latencies in both BAEP and SSEP recordings. Etomidate is a potential cerebral protection agent with the advantages of relative lack of cardiodepression and a rapid recovery from anesthesia.

Hyperventilation is frequently used to decrease brain volume and thus to improve exposure with minimal retraction. However, the alkalosis induced by hyperventilation has been shown to be detrimental to cultured cortical neurons undergoing hypoxic or excitotoxic injury. Because many neurosurgeons have had the impression that induced hypotension is particularly detrimental in conjunction with brain retraction or temporary vessel occlusion, intra-operative induced hypotension is probably less commonly used now than previously.

**Mannitol**

Although mannitol has many potential beneficial effects during brain retraction, e.g., improved blood flow in the microcirculation, free radical scavenging, improved rCBF and pH in regions of moderate ischemia and decreased brain water content or edema, it may induce acute changes when administered in high doses, including hyponatremia, decreased hemotocrit and increased osmolality as well as hypotension, acidosis and hyperkalemia. Yet, mannitol may be more cerebroprotective during brain retraction if it is administered in frequent, small doses or by continuous infusion, rather than bolus administration every 4 to 6 h.
Nimodipine
This dihydropyridine calcium channel blocker has been tried as a prophylaxis for cerebral ischemia caused by cerebral vasospasm after a subarachnoid hemorrhage and as a treatment for acute stroke. It usually elicits an increase in rCBF during both the ischemic phase and the delayed hyperperfusion phase. Nimodipine has the following beneficial effects for brain retraction ischemia: 1. cerebral vasodilation; 2. amelioration of K+-induced vasoconstriction; 3. antagonism of calcium entry into neurons via the L-channel; 4. anticonvulsant properties; and 5. reduction of acidosis. Although the data on nimodipine’s effect on rCBF with changes in PCO2 (hyperventilation) are inconclusive, there are data to support the use of nimodipine for its cerebroprotective effect in the alkalosis induced by hyperventilation.

Other pharmacological interventions
Corticosteroids are commonly used peri-operatively in neurosurgical procedures involving brain retraction, particularly in patients with pre-operative cerebral edema. Dextromethorphan, a noncompetitive N-methyl-D-aspartate receptor antagonist, has been shown to be effective in small animal models of cerebral ischemia, which appear to be particularly effective in focal ischemia and may result in improved rCBF in ischemic regions. Tromethamine, a weak base buffering agent, has been shown recently to ameliorate lactate acidosis after focal brain injury, and prove to be efficacious in conjunction with hyperventilation during neurosurgical procedures requiring lengthy brain retraction.

Hypothermia
Mild hypothermia represents a compromise that may prove to be of considerable benefit in many cases. Although the cooling and warming protocols place a burden on operating room personnel, and recovery and extubation may be delayed an hour or two, mild hypothermia has fewer complications than deep hypothermia. The magnitude of the cerebroprotective benefit of mild hypothermia is probably substantial, but its role in relationship to other intra-operative cerebroprotective interventions remains to be established.

COMPUTER MODEL
Koyama et al. developed a ‘virtual retractor’, a program to interpolate the contour of models of a retractor. The center of the displacement was determined by spatial coordinates, and the shape of the displacement of the arterial model was calculated using a cosine-based formula with representation of a brain retractor. This computer-graphics model was applied to the simulation of the displacement and morphological changes that occur when retraction is performed in the optic nerve. An illustrative case is presented, in which the optic nerve was displaced by a retractor to simulate the surgery performed in a carotid cave aneurysm of the internal carotid artery.

Platenik et al. developed a computational model to predict tissue deformation during retraction in the porcine brain. The level of quantitative agreement achieved in the experiments was encouraging for updating pre-operative images to reflect tissue deformation resulting from retraction.

CONCLUSION
The present review presents the brain retraction injury. In modern neurosurgery, the brain retracting blade has evolved to a more complicated system which includes an articulated arm, a frame and a stereotactic placement. However, with all this improvement in instrument technology, the brain retractor continues to be a significant threat to the underlying brain tissue. A better technology is needed to detect cerebral ischemia with immediate feedback to the surgeon.

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