

Emergence hypertension in patients undergoing intracranial surgery

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Abstract

Systemic hypertension is a common accompaniment during emergence from anaesthesia following intracranial neurosurgical procedures and may predispose to development of intracranial hematoma. Although various drugs have been evaluated, management of peri-extubation hypertension in this subset of patients continues to be a challenge for anaesthesiologists.

EMERGENCE HYPERTENSION IN NEUROSURGERY

Major complications after intracranial surgery may be manifold. Of these, development of postoperative hematoma is of devastating consequence to ultimate outcome and has an incidence of 0.8%-2.2%¹. The causes of development of such hematomas may be multiple², but a commonly ascribed cause is the hemodynamic and metabolic perturbations that occur during recovery from anaesthesia.

Systemic hypertension associated with emergence from anesthesia, has long been believed to contribute to intracranial hemorrhage and cerebral edema following craniotomy⁽³⁻⁶⁾. Lewelt et al. demonstrated that elevated postoperative blood pressure was a correlate of intracerebral bleeding after craniotomy⁽⁷⁾. Forster et al.⁽⁴⁾ observed that in anesthetized animals, sudden substantial increases in arterial pressure can result in breach of the blood-brain barrier. The incidence of peri-operative hypertension has been reported to be as wide as 54-91% in various studies⁽⁸⁻¹⁴⁾. Basali et al.⁽¹⁵⁾ report an incidence of 57% for postcraniotomy hypertension. The purpose of this review is to address the causes of these hemodynamic alterations in the peri-extubation period, discuss whether better hemodynamic control during this vulnerable phase could improve neurological outcomes and highlight therapeutic options based on recently concluded studies.

Causes of hypertension during recovery from anesthesia

- Pain
- Hypothermia

- Hypo-osmolality
- Anemia
- Hypercarbia and hypoxia
- Emergence excitement
- Catecholamine release or sympathetic stimulation
- Brain manipulation
- Epinephrine-containing local anaesthetic administration
- No demonstrable cause
- The final common pathway for all of these causes leading to elevations of blood pressure in patients undergoing craniotomy appears to be activation of the sympathetic nervous system. Elevations of plasma catecholamine concentrations are common in patients after craniotomy⁽¹⁶⁾.

THE ROLE OF PLASMA CATECHOLAMINES

Olsen et al.⁽¹⁶⁾ studied the effects of vasoactive modulators in the perioperative period in patients undergoing craniotomy and their relation to postoperative hypertension. They concluded that in addition to increased discharge of the sympathetic system, as evidenced by elevated levels of norepinephrine and epinephrine, activation of the renin-angiotensin aldosterone system may also play an important role in development of postoperative hypertension after craniotomy. However, norepinephrine administration has

also been shown to be associated with reductions in cerebral blood flow(17). Thus, circulating catecholamines as such cannot account for the cerebral hyperaemia seen during craniotomy. Other stress such as metabolic stress associated with cerebral activation due to surgery may also be involved.

EMERGENCE HYPERTENSION AND INTRACRANIAL HEMORRHAGE: A TEMPORAL RELATIONSHIP?

The occurrence of systemic hypertension during anesthetic recovery is quite common in neurosurgical patients undergoing craniotomies as stated above. Such occurrence has often been related to the development of intracranial hematoma postoperatively. However, this association between peri-extubation hypertension and the occurrence of intracranial hematomas has been thinly investigated.

Epidemiologically, an association is said to be more likely causal if it is strong, appropriately temporal, specific, dose-related, consistent, plausible, and coherent¹⁸.

Basali et al.,⁽¹⁵⁾ in a retrospective case-control study evaluated the relationship between surges of blood pressure in the emergence period and the occurrence of postoperative intracranial hematomas. Of the total number of patients who developed hematomas in the postoperative period, about 62% had hypertension as against 34% incidence of hypertension in the control group. However, the study did suffer from innate limitations, principally because of its retrospective design, as also because of the limited number of blood pressure readings, especially in the control group. Hence while the study did show an association, it did not demonstrate a cause-and-effect relationship between acute hypertension and intracranial haemorrhage.

Kase et al.¹⁹, and Wintzen et al.²⁰ related systemic hypertension to the occurrence of intracerebral hemorrhage in patients on anticoagulants. Similarly, Fukumachi et al.²¹, and Kalfas et al.²² related hypertension to intracerebral hemorrhage in patients with normal coagulation. Kalfas et al. reported a 16% incidence of intracranial hematomas in hypertension, but again failed to demonstrate a causal relationship between the two.

It is reasonable to expect that an increased blood pressure in the perioperative period would result in cerebral hyperaemia with a propensity for hemorrhaging intracranially. Significant hypertension might well overwhelm the reserves of cerebral autoregulation. Equally feasible is the argument that vasomotor paralysis might already have occurred at sites

of surgical manipulation, either due to surgical retraction or residual tumors. With such tenuous cerebral autoregulation in the perioperative period, any hypertension might increase the blood flow to the brain and result in a tendency to develop postoperative hematoma.

Yet, Bruder et al.¹, demonstrated increased cerebral blood flow despite well-controlled hemodynamics perioperatively. Similarly, Felding et al., used metoprolol²³ and ketanserin²⁴ to gain adequate control over blood pressure, without eliminating the increase in cerebral blood flow seen during and after craniotomies.

Thus while perioperative hemodynamics can be fairly well-controlled, whether such control would decrease surges in cerebral blood flow, and in the ultimate analysis have an impact on the incidence of intracranial hematoma in this group of patients is far from clear. There are no prospective, randomised trials clearly demonstrating benefits accrued from stringent control of blood pressure in the perioperative period, translating into reduction in incidence of intracranial hematomas. In fact, in view of above results of Bruder et al. and Felding et al., it might be suggested that control of blood pressure may not have any such effect as it has no impact on surges of cerebral blood flow in the perioperative period.

Hence, additional studies need to be performed in order to elucidate the mechanisms of post-craniotomy intracranial hematomas and elucidate whether better control of hemodynamics could improve overall neurological outcomes. It is quite possible to have other mechanisms apart from hypertension which might have a more telling effect on intracranial hemorrhage after craniotomy. It is also possible that hypertension might be an indicator of intracranial phenomena that are not yet fully understood, but which are more temporally related to intracranial hemorrhage. It is also possible that other factors like age, anticoagulation, location of intracranial space-occupying lesions and their histological type, invasion of venous sinuses, extent of tumor removal, surgical hemostasis, cerebral venous pressures, genetic predisposition, etc., may play a more significant role in post-craniotomy hemorrhage. Tracheal intubation might be associated with elevations of cerebral venous pressures due to straining on the endotracheal tube and may predispose to development of intracranial hematomas. Significantly, pharmacotherapy in the form of vasodilators to control hypertension, might actually result in cerebral vasodilation and hyperemia and may be related to causation of postoperative intracranial hemorrhage.

MANAGEMENT OF EMERGENCE HYPERTENSION IN NEUROSURGICAL PATIENTS

While none of the studies to date have been able to establish a cause-and-effect relationship between emergence hypertension and development of intracranial hemorrhage or demonstrate better neurological outcomes with better hemodynamic control, it would still seem worthwhile striving for stable hemodynamics perioperatively and attain a 'smooth' emergence at the end of any neurosurgical procedure.

Most anesthesiologists would use beta-blockers such as metoprolol, labetalol or esmolol to tide over acute elevations of systemic blood pressure often encountered during recovery from anesthesia(25). Labetalol owing to low potency, slow onset of peak effect(26) and unpredictability in dose requirements(27) may not be the most ideal agent in these circumstances. Esmolol similarly is only mildly effective and is associated with bradycardia and conduction defects(28).

Other commonly prescribed antihypertensives such as ACE inhibitors and calcium channel blockers have also been tried with reasonable success. Nicardipine is more effective than both labetalol(27) and esmolol(29) in controlling periextubation hypertension. However calcium channel blockers have been associated with dose-dependent cerebral vasodilation, inhibition of autoregulation and hypotension(30). Experience with hydralazine has not been encouraging as it increases intracranial pressure significantly(31).

Dexmedetomidine has also been evaluated by several authors as an adjuvant to anesthesia for neurosurgery(32,33,34,35) including intracranial surgery. Improved perioperative hemodynamic control has been reported in these studies as compared to placebo.

Lignocaine is often employed during emergence in order to reduce airway responsiveness and decrease the incidence of coughing and straining. Doses of 1.5mg/kg are often appropriate for this purpose.

Numerous alterations to anesthetic techniques especially during emergence have been proposed to overcome the peri-extubation hypertensive response. These include titrated doses of short-acting opioids, continuing volatile anesthetics and nitrous oxide until head dressing is over, extubating patients undergoing otherwise uncomplicated surgeries in

deeper planes of anesthesia, etc. The accent during extubation should be on minimising coughing and straining on the ET tube, optimising pain relief without obtunding the patient neurologically and aim for a 'smooth' extubation. As to what constitutes a 'smooth' extubation in these patients is still hard to get a consensus on.

CONCLUSION

Systemic hypertension during emergence from anesthesia is commonly encountered in patients undergoing craniotomies. Causes may be multiple, some identifiable, some not quite as well understood. While an association of such hypertension with the incidence of intracranial hematomas may be found, there's no scientific evidence to date which establishes a temporal relationship between the two. Further prospective randomised trials are required to identify a cause-and-effect relationship between the two. Multiple techniques have been employed for quelling the blood pressure response especially during emergence from anesthesia with varying degrees of success, but there's no doubt that peri-extubation hypertension in this subset of patients remains a challenge for anesthesiologists.

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