

# Anaesthesia for endoscopic neurosurgical procedures

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## Purpose of review

Endoscopic neurosurgical procedures are becoming more frequent and popular in the treatment of intracranial disease. When endoscopy involves the intraventricular structures, irrigating solutions are required and may contribute to sudden and sharp increases in intracranial pressure. More recently, nasal endoscopic approach has been used to perform skull base surgery for aneurysms and tumours. We have analysed published articles in order to detect anaesthesia management and perioperative complications.

## Recent findings

Sudden and dangerously low decreases in cerebral perfusion pressures do not provoke the 'traditional Cushing's response' usually associated with significantly high intracranial pressure. It is important to note that tachycardia (not bradycardia) and/or hypertension are the most frequent haemodynamic complications during neuroendoscopic procedures. With the transnasal approach severe intraoperative haemorrhage is the most important complication to consider followed by direct injury to surrounding neural structures.

## Summary

Invasive arterial blood pressure and intracranial pressure should be measured continuously during neuroendoscopies to detect early intraoperative cerebral ischaemia instead of waiting for the appearance of bradycardia which may be a late sign. General anaesthesia remains the technique of choice. Intracranial haemorrhage increases the likelihood of perioperative complications. Close postoperative monitoring is required to diagnose and treat complications such as convulsions, persistent hydrocephalus, haemorrhage or electrolytic imbalance.

## Keywords

anaesthesia, endoscopic third ventriculostomy, hydrocephalus, intracranial pressure, neuroendoscopy

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## Introduction

During intraventricular neuroendoscopy, periods of very low cerebral perfusion pressures (CPPs) have been detected with specific monitoring [1–5]. Early detection of these preischaemic events is crucial to avoid permanent and irreversible ischaemia. Recent animal studies [6\*\*] have shown that despite complete suppression of the CPP, only a modest reduction in heart rate was observed. Although cerebral ischaemia was present for several minutes, many of the animals recovered without any obvious clinical signs of cerebral insult. Histological analysis, however, showed signs of ischaemic injury with an increased number of pinocytic neurons in the hippocampus. In this study we will review and summarize the results from published animal and clinical studies focusing on the importance of CPP and perioperative complications following neuroendoscopic surgery.

## Indications for intraventricular neuroendoscopy

Indications include treatment of a variety of different types of noncommunicating hydrocephalus associated with aqueductal stenosis, communicating hydrocephalus and intraventricular diseases such as colloid and arachnoid cysts and periventricular tumours [7,8\*,9–15].

## Type of neurosurgical procedures that can be performed using a neuroendoscope

Endoscopic third ventriculostomy (ETV) is rapidly becoming the treatment of choice for noncommunicating hydrocephalus [16,17]. ETV allows the cerebrospinal fluid (CSF) to flow directly from the third ventricle to the basal subarachnoid spaces, thus bypassing the aqueduct and the CSF pathways of the posterior fossa [10]. It

has also been successfully used to treat infective hydrocephalus secondary to tuberculous meningitis [18<sup>\*</sup>], and intraventricular haemorrhage [19<sup>\*</sup>,20]. Other interventions include endoscopic removal of cysts [21<sup>\*</sup>], tumour biopsies [22], complete removal of intra and paraventricular tumours [23<sup>\*</sup>,24], intraventricular nontumoural lesions as neurocysticercosis [25<sup>\*\*</sup>], haematomas [9] and hypothalamic hamartomas [26<sup>\*</sup>]; and choroid plexus cauterization in the treatment of hydrocephalus in developing countries [9].

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### Choice of anaesthetic technique

The key goals in anaesthesia are to ensure immobility of the patient (head fixation is frequently required); prevent, detect and treat sharp increases in intracranial pressure (ICP) (a good communication with the neurosurgeons is essential); and using a technique which will allow rapid emergence for prompt neurologic assessment. In published studies inhalation anaesthesia [1,3–5,27<sup>\*\*</sup>,28,29,30<sup>\*\*</sup>,31–35] with or without N<sub>2</sub>O was the predominant technique of choice (Table 1). Derbent *et al.* [28] did not use N<sub>2</sub>O to avoid elevations in ICP. Ganjoo *et al.* [30<sup>\*\*</sup>] recommended that N<sub>2</sub>O should not be used after reporting one episode of venous air embolism (VAE).

Derbent *et al.* [28] used sevoflurane with ET<sub>CO</sub><sub>2</sub> at 30 ± 2 mmHg to reduce its cerebral vasodilatory effects, and avoided remifentanyl to maintain normotension. These authors suggest that their anaesthetic technique together with the use of 0.9% NaCl for intravenous fluid replacement produced a lower incidence of adverse haemodynamic events. Baykan *et al.* [34] initially used alfentanil and later remifentanyl as the opioid of choice, but noted no differences in using either opioid.

Many studies used nondepolarizing neuromuscular blocking agents to facilitate endotracheal intubation and for maintenance [1,3,5,36].

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### Irrigating solutions

Warmed (at body temperature) lactated Ringer solution was the most frequently used irrigation fluid [1,2,13,28,33,34,37,38]. Salvador *et al.* [39] found that the use of saline as irrigation solution produced significant changes in CSF composition. They found a significant correlation between changes in CSF (pH, oxygen and carbon dioxide partial pressures, base excess, standard bicarbonate, glucose and total calcium) and the total volume of irrigation solution used, but no correlation with the duration of neuronavigation. A cut-off point of 500 ml of saline irrigation solution (sensitivity 0.7; specificity 0.87) was associated with a reduction in CSF pH of greater than 0.2 (Table 2). Unfortunately similar studies have not been performed with Ringer's solution.

Routine use of irrigating solutions is not necessary in some uncomplicated ETV endoscopic procedures [32]. To avoid intraoperative and postoperative complications arising from the use of irrigating fluids, some surgeons take care to limit the loss of CSF and to use irrigation only when necessary [16].

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### Recommended intraoperative monitoring for neuroendoscopy

Every patient should be monitored as if ETV was a major operation rather than a minimally invasive method [34]. Beat-to-beat monitoring of heart rate (HR) and mean arterial pressure (MAP) by an indwelling arterial catheter was strongly recommended in all patients, including children, by the majority of studies reviewed [1–5,27<sup>\*\*</sup>,30<sup>\*\*</sup>,40,41]. Continuous measurement of CPP should be mandatory [3,6<sup>\*\*</sup>]. To measure CPP, ICP needs to be measured and one method of doing this is to measure the pressure inside the endoscope (PIN). A new study has demonstrated that the PIN can be a reliable surrogate measure of ICP [42<sup>\*</sup>]. Awakening without neurological deficit did not preclude histological damage in the animal model [6<sup>\*\*</sup>], and it is possible that the same could apply for humans. It was recommended that intraoperative CPP should be maintained above 40 mmHg at all times [3].

Van Aken *et al.* [1] suggested that premature babies and neonates with a low MAP are more vulnerable to cerebral ischaemia even with a modest increase in ICP. Beat-to-beat measurement of arterial blood pressure offers the most reliable warning sign of an increase in ICP during ETV. Waiting for a persistent bradycardia before alerting the surgeon of raised ICP during ETV may be too late as fatal asystole can develop.

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### Measuring intracranial pressure intraoperatively

El-Dawlatly *et al.* [32] measured ICP in the third ventricle using a pressure transducer attached at one end to the scope and the other end to the anaesthesia monitor. However, no readings can be obtained during saline irrigation and as a result only recorded three ICP measurements which were averaged (Table 1).

Fàbregas *et al.* [5] and Salvador *et al.* [42<sup>\*</sup>] measured PIN by means of a fluid-filled catheter connected to a stopcock connected to the irrigation lumen of the neuroendoscope (inflow channel) and attached to a pressure transducer zeroed at the skull base.

In the studies by Kalmar *et al.* [2,3] the outlet of the endoscope-flushing system was connected by a long pressure tube to a pressure transducer for continuous

**Table 1 Systemic and cerebral haemodynamic changes during intraventricular neuroendoscopic published studies**

Author (year)	N study/ control	Age study/ control	Diagnosis study/control	Type of surgery	Anaesthetic technique	Hypertension/ hypotension	Brady/ tachycardia	ICP	ICP > 30 mmHg (patients)	Surgery time/ endoscopy time study/control (min)
El-Dawlatly <i>et al.</i> (1999) [32]	14	34 ± 37 weeks	Hydrocephalus	ETV	Isoflurane/N <sub>2</sub> O	-/-	43% <sup>a</sup> /-	PINm all	None	-
El-Dawlatly <i>et al.</i> (2000) [31]	49	1–108 months	Hydrocephalus	ETV	Isoflurane/N <sub>2</sub> O	No/no	41% <sup>a</sup> /no	No	-	34 ± 13
Fábregas <i>et al.</i> (2000) [5]	100	39 ± 20 years	Hydrocephalus 33%; tumour 48%	ETV 54%	Isoflurane 74% cases	19%/2%	13%/3%	PIN 47%	51%	101 ± 54
Longatti <i>et al.</i> (2000) [35]	9	22–67 years	Colloid cysts; third ventricle	Fenestration	General	-	-	No	-	54–120/24–100
Fábregas <i>et al.</i> <sup>b</sup> (2001) [4]	8	19 to 63 years	Hydrocephalus 50%	ETV 50%	Isoflurane 64%	No/no	12.5%/no	PIN inlet all	64%	45–150/5–120
Anandh <i>et al.</i> (2002) [33]	20/6	11.5 ± 13.8/ 19.5 ± 10.9 years	Hydrocephalus/ cysts	ETV/not ETV	Halothane/N <sub>2</sub> O	No/no	Yes (study group)/no	No	-	-/Endoscopy: 107 ± 42/150 ± 91
Van Aken <i>et al.</i> (2003) [1]	88	12 days to 72 years	Hydrocephalus	ETV	TIVA adults; sevoflurane induction/children	6%/no	6%/11%	No	-	-
Baykan <i>et al.</i> (2005) [34]	210	2 months–2 years	hydrocephalus	ETV	Isoflurane/N <sub>2</sub> O	2.8%/no	26.8% <sup>c</sup> / 12.3%	No	-	45 ± 10
Kalmar <i>et al.</i> <sup>c</sup> (2005) [3]	17	1 month–84 years	Hydrocephalus 70%	ETV 53%	Sevo children TIVA adults	88%/no	23%/65%	PIN outlet all	-	-
Derbent <i>et al.</i> (2006) [28]	24	4 months to 47 years	Hydrocephalus 75%	ETV 75%	Sevoflurane	No/no	4%/no	No	-	47.6 ± 31/24–3 ± 30
Prabhakar <i>et al.</i> (2007) [29]	13	15–55 years	-	-	Isoflurane/N <sub>2</sub> O	No/no	No/no	IPP	100%	-
Ganjoo <i>et al.</i> (2009) [27**]	260 (247 included)	111 <2 years 128 (2–18 years) 21 >18 years	Hydrocephalus 71.5%	ETV	Isoflurane/N <sub>2</sub> O	10%/no	12%/20%	No	-	-
Ganjoo <i>et al.</i> (2010) [30**]	298 (283 included)	126 <2 y 143 (2–18 y) 29 > 18 y	Hydrocephalus 70%	ETV	Isoflurane/N <sub>2</sub> O	9.9%/0.7%	13%/30%	No	-	Surgery 10 to 120

ETV, endoscopic third ventriculostomy; ICP, intracranial pressure; IPP, intracranial intraparenchymal pressure; N, number of patients; PIN, pressure inside the neuroendoscope; PINm, ICP mean value.

<sup>a</sup>Bradycardia heart rate <80 beats per min. In all cases, bradycardia occurred at the time of perforation of the floor of the third ventricle; withdrawal of the scope resulted in successful reversal of the bradycardia.

<sup>b</sup>Transcranial Doppler wave continuously measured in all patients.

<sup>c</sup>One patient developed asystole during balloon dilatation, normal sinus rhythm was achieved with cessation of surgery + atropine + epinephrine.

<sup>d</sup>In this study CPP was measured as <15 mmHg in 41% of patients.

**Table 2 Electrolyte imbalance following intraventricular neuroendoscopic procedures**

Author (year)	N study/ controls	Type of surgery	Rinsing solution	Irrigation volume (ml)	K+	Na
Fábregas <i>et al.</i> (2000) [5]	100	ETV 50%	Saline	–	No imbalance	1% Hyponatremia; 1% hypernatremia
Anandh <i>et al.</i> (2002) [33]	20/6	ETV/endoscopy not ETV	Lactated Ringer's	573 ± 394/ 1383 ± 618*	HyperK on day 3 (study group)	No changes
El-Dawlatly (2004) [38]	50	ETV	Saline	–	No imbalance	No changes
Derbent <i>et al.</i> (2006) [28]	24	ETV and others	Lactated Ringer's	–	No imbalance	No changes
Baykan <i>et al.</i> (2005) [34]	210	ETV	Lactated Ringer's	100 ± 20	–	Hyponatremia 2.4%
Salvador <i>et al.</i> (2007) [39]	29	ETV 70%	Saline	919.6 ± 994.7	No imbalance	3.5% Hyponatremia
Ganjoo <i>et al.</i> (2010) [30**]	298	ETV	Lactated Ringer's	50–1500	HyperK 0.35%	Hypernatremia 0.7%

\*  $P > 0.001$  between ETV and non-ETV endoscopic procedure.

monitoring of the ICP. The level of foramen of Monro was used as the zero reference point. A major pitfall in using the outflow channel to measure PIN, is that a 'zero' pressure reading is obtained when blockage in the outflow lumen occurs [2,43]. Vassilyadi and Ventureyra [44] described good results using a Codman microsensors ICP monitor inserted through the working channel of the flexible endoscope. They reported more accurate measurements of ICP. An added advantage is that the microsensors can be left in place after surgery, allowing postoperative ICP monitoring. However, this system is significantly more expensive than using PIN. In addition, the presence of the sensor in the working channel may interfere with the use of instruments, and the sensor tip floating in the ventricles may make navigation through the fluid-filled structures more difficult.

Prabhakar *et al.* [29] used an intraparenchymatous catheter to measure ICP, also with good results, but this is much more invasive and therefore less acceptable as a routine practice.

Salvador *et al.* [42\*] concluded in their study that PIN measurement was very useful in evaluating ICP changes related to the neuroendoscopic procedure and moreover it was more consistent than epidural ICP at high ICP.

### Intraoperative cerebral and systemic haemodynamic changes

Endoscopic third ventriculostomy is associated with a wide range of haemodynamic effects ranging from minor changes in heart rate and blood pressure to that of near fatal cardiac arrest (see Table 1). The possible mechanisms proposed for these changes include hypothalamic stimulation or damage [31–34,45,46] and acute rise in ICP [1,3,4]. However, there is no consensus and it is not clear if the observed changes follow a predictable pattern as suggested by Ganjoo *et al.* [27\*\*].

Hypothalamic centres and medulla oblongata may become ischaemic as a result of reduced regional cerebral

perfusion during fenestration of the third ventricle [34]. Stimulation of posterior hypothalamus increases blood pressure and HR, and stimulation of preoptic area decreases HR and blood pressure [33]. Perforation of the floor of the third ventricle during ETV is usually made in front of mamillary bodies located on the posterior hypothalamus. Anandh *et al.* [33] proposed that the traction exerted on this area during perforation inhibits the sympathetic activity and results in bradycardia.

El-Dawlatly *et al.* [31] and Proano *et al.* [25\*\*] reported a significant decrease in HR during the perforation of the third ventricle. They recommended that merely alerting the surgeon to pull the scope away from the floor of the third ventricle will resolve the bradycardia without the need for pharmacological intervention. In Van Aken *et al.*'s case series [1] simultaneous increases in blood pressure and HR ('atypical' Cushing reflex) was seen in only six patients (6.8%), and in all cases was due to high ICP resulting from excess irrigation fluid (either kink of the irrigation outflow tube or forceful inflow of irrigation to clear a view blurred by blood). Baykan *et al.* [34] reported 26.8% of children developing bradycardia during balloon dilatation of the third ventricle, with HRs suddenly dropping below the 50 beats per minute. Deflation of the balloon resolved this problem in 56 cases out of 59 cases; the other 3 patients (1.41% of total) required treatment with atropine. One of the 210 patients (0.5%) developed asystole during this part of the procedure; normal cardiac rhythm returned after the balloon was deflated and epinephrine and atropine were administered with no resulting postoperative morbidity. Six (2.8%) patients developed a sudden rise in arterial pressure unaccompanied by other signs, and 26 (12.3%) developed tachycardia alone followed by bradycardia. Derbent *et al.* [28] did not report any haemodynamic variation during the procedure except for one patient who developed bradycardia during fenestration of the floor of the third ventricle. This is in contrast with Ganjoo *et al.*'s study [27\*\*] that describes hypotension/bradycardia during navigation through the foramen of Monro (FOM); tachycardia ± hypertension during manipulation

and irrigation in third ventricle; and bradycardia  $\pm$  hypertension during fenestration of third ventricle floor and inflation of Fogarty catheter. In this study 10% of patients developed hypertension associated either with bradycardia or tachycardia but never manifested hypertension alone.

Typically bradycardia, tachycardia or hypertension associated with ETV are transient and will respond to simple surgical manoeuvres such as removal of scope, reducing the speed of inflow and allowing egress of irrigant fluids [1,3–5,27<sup>\*\*</sup>,41,42<sup>\*</sup>]. However, the need to use atropine and other resuscitative measures reported in Baykan *et al.*'s [34] case series underscore the seriousness of these changes. During neuroendoscopy, if the irrigating fluid is allowed to run in too rapidly [4,5], or if the outflow line is accidentally occluded [2], or if there is insufficient space between the endoscope and the introducer sheath [29], very high ICPs up to 150 mmHg can quickly occur and may remain unnoticed especially if ICP is not continuously monitored [1–5,27<sup>\*\*</sup>,30<sup>\*\*</sup>,42<sup>\*</sup>].

### Lowest level of cerebral perfusion pressure that can be safely tolerated

In Fàbregas *et al.*'s [5] study the best PIN cut-off point for predicting the appearance of postoperative complications was 30 mmHg. In their study PIN, values greater than 50 mmHg occurred in 12 patients (25%). The average peak pressure for all 47 patients monitored was  $36.8 \pm 28.5$  mmHg, ranging from 4 to 139 mmHg. The mean peak PIN in the patients who had intraoperative complications was  $40.8 \pm 34.9$  mmHg, and in the patients who did not have intraoperative complications the mean peak pressures inside the endoscope was  $35.2 \pm 25.6$  mmHg ( $P = .54$ ).

Van Aken *et al.* [1] first recognized the significance of tachycardia and hypertension as a reflection of raised ICP. The same research group in a recent ETV study in which ICP was also measured [2,3], showed that a tachycardia and hypertension response correlated well with a decrease in CPP of less than 15 mmHg. They proposed that hypertension, seen as the initial response to a fall in CPP, was an adaptive mechanism to restore the reduced CPP, and that tachycardia occurred only when the increase in ICP was too rapid for adaptation. The authors found that tachycardia preceding bradycardia was a better indicator of impaired brain perfusion than bradycardia alone and waiting for the latter to happen during irrigation could lead to further rises in ICP with serious cardiovascular and neurological consequences. They defined an event as CPP lower than 50 mmHg; subsequent categories were defined as CPP below 50, 40, 30, 20 and 15 mmHg; and a significant haemodynamic event as a change of 20% from baseline for a minimum of

3 s. In their study eight (47%) patients had episodes of CPP below 40 mmHg and below 30 mmHg; five (29%) patients had CPP below 20 mmHg and seven (41%) had CPP below 15 mmHg. None of the patients, however, suffered any of the postoperative complications as described by Buxton *et al.* [17]. The occurrence of bradycardia was not always associated with a low CPP. Although most clinicians rely on the occurrence of bradycardia to diagnose intracranial hypertension during endoscopic neurosurgical procedures, this study showed that a simultaneous onset of hypertension and tachycardia was a better indicator of impaired brain perfusion. In all cases in which hypertension followed a moderate decrease in CPP, the rise in blood pressure was sufficient to normalize the CPP at a level between 35 and 50 mmHg. Their observations of the haemodynamic effects of the ICP and CPP show that no observable effects are seen with a CPP above 40 mmHg, independent of the ICP. They also found that an occurrence of simultaneous hypertension and tachycardia during endoscopy has a specificity of 77% in detecting a decrease in CPP below 30 mmHg.

In Prabhakar *et al.*'s [29] case series the baseline CPP had a median value of 74 mmHg (range 52–88 mmHg) and during irrigation the median value dropped to 48 mmHg (range 17–59 mmHg). However, the rise in ICP did not last for more than 3–4 s, as the surgeon was immediately informed and necessary action was taken.

Kalmar *et al.* [6<sup>\*\*</sup>] used a rat model of direct subarachnoidal volume expansion as an experimental model for intracranial hypertension during endoscopic neurosurgery. They evaluated the nature of the haemodynamic changes resulting from isolated intracranial hypertension and tried to elucidate the hydrodynamics of the irrigation fluid that was not allowed getting off the brain. Complete CPP suppression was maintained for different periods ranging from 90 to 514 s. The first group of five rats had a high mortality: three out of five developed pulmonary oedema, probably due to excessive fluid infusion and severe hypertension making it difficult to differentiate the cause of the death between brain ischaemia and hypertension. However, despite a complete suppression of CPP only a modest change in HR was observed. In order to attenuate the cardiovascular consequences of raised ICP a group of rats were pretreated with labetalol. Another group was given increasing concentrations of sevoflurane and blood pressure lowered to 55 mmHg before raising the ICP. In many cases severe tachycardia was the only constituent of the Cushing reflex that occurred. Severe bradycardia only developed after the animal was already severely hypotensive with imminent haemodynamic collapse. These authors found that during CPP suppression a significant amount of fluid was absorbed from the subarachnoid space into the circulation

**Table 3 Postoperative complications following neuroendoscopic procedures**

Author (year)	Hypothermia	Delayed awakening	Vomiting	Convulsions	Anisocoria/III or VI palsy	Temporary diabetes insipidus	Persistent hydrocephalus
Fàbregas <i>et al.</i> (2000) [5]	–	15%	3%	–	16%/6%	1%	6%
Baykan <i>et al.</i> (2005) [34]	–	–	–	0.5%	0.9%/0.5% (transient)	2.4%	3.3%
Ganjoo <i>et al.</i> (2010) [30**]	4.2%	1.1%	–	0.71%	0.71%/0.71%	–	–

(as indicated by the decrease in haematocrit). Although brain ischaemia was induced for several minutes, many of the animals recovered without any obvious sign of cerebral damage, although some had transient paresis of the hind paws, which resolved after 24 h. However, histological study, performed on day 8, showed signs of ischaemic injury with an increased number of pynocytic neurones in the hippocampus.

### Other intraoperative complications

van Aalst [47] *et al.* described a massive upward ballooning of the third ventricle floor immediately after opening the floor in four patients, and withdrawing the Fogarty catheter completely obscured the fenestration. The third ventricle was very large, and its floor herniated hindering the endoscopic view. Preoperative magnetic resonance imaging (MRI) demonstrated a similar anatomy in all four cases, consisting of hydrocephalus, extreme dilation of the third ventricle, and disappearance of the interpeduncular cistern due to a very thin, membranous floor of the third ventricle, which herniated downward, draping over the basilar artery. The authors suggest that excessive rinsing in combination with this anatomical configuration provoked the phenomenon.

Baykan *et al.* [34] reported venous bleeding controlled with irrigation (2.4%); rupture of basilar artery requiring craniotomy (0.5%); inadequate CSF drainage requiring ventriculoperitoneal shunting (VPS) in same case series (2.4%).

Ganjoo *et al.* [30\*\*] reported four episodes of major intraoperative bleeding (1.4%); 32 episodes of minor bleeding (11.3%); and one case of VAE (0.35%). In Fàbregas *et al.*'s [5] case series four cases of VAE were reported (4%).

### Postoperative complications

In Ganjoo *et al.*'s [27\*\*] recently published study evidence of third nerve injury was seen in the postoperative period in two patients (0.8%) who had bradycardia at the time of fenestration during ETV.

Buxton *et al.* [17] had complications in 17.5% of their case series of adult patients, five related to infections, one

bifrontal cerebral infarction, one intraoperative bleeding, another developed bilateral chronic subdural haematoma and three died associated with the procedure (one postoperative bleeding in a patient with glioblastoma multiform, and the other two for massive intraventricular bleeding).

Hypothermia during neuroendoscopy is seen more often in small children, caused by large exchanges of irrigating fluid and ventricular CSF and by the wetting of drapes with the returning perfusate [36].

Convulsions have been reported by several authors (see Table 3), with one case resulting from pneumoencephalus [48].

Injury to the hypothalamus may give rise to transient hypothalamic dysfunction leading to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) or diabetes insipidus with fluctuating serum electrolyte levels. A delayed hyperkalaemic response was observed in Anandh *et al.*'s [33] study. They proposed that mild hypoaldosteronism may be provoked by the altered physiologic flow of CSF across the third-ventricular floor following ETV; and that this flow may be misinterpreted by brain receptors as an increase in fluid volume. Also in this study the postoperative potassium levels were lower in control group who received more intraoperative irrigation without ETV being performed. Postoperative electrolytic changes after intraventricular neuroendoscopy have been reported in a number of studies [28,38,39] (Table 2).

Meningeal irritation, headache and high fever from an inflammatory response to irrigating fluid have been described [49]. In Longatti *et al.*'s case series [35] nine studied patients presented mild fever (35.5–38°C) for 2 days after endoscopy for colloid cyst of the third ventricle fenestration. They used lactated Ringer's solution for intraoperative irrigation. One patient developed a septic ventriculitis (*Staphylococcus epidermidis*) on postoperative day 8.

High ICP postoperatively can account for persistence of symptoms with evidence of ventricular dilatation on computed tomographic scans after ETV. Cinalli *et al.* [16] recommended that a cycle of one to three lumbar

punctures should always be performed in patients who remain symptomatic and who show increasing ventricular dilatation after ETV, before assuming that ETV has failed and implanting an extracranial CSF shunt.

After ETV close postoperative surveillance is essential in children [34,50,51\*] and in adults [7]. In Prabhakar *et al.*'s [29,52] case series all patients were monitored in a neurosurgical intensive care unit.

## Neuroendoscopic nonintraventricular procedures

Neuroendoscope is increasingly being applied to the treatment of other central nervous system diseases, such as in sylvian arachnoid cysts [53\*]; haematomas; brain abscess; cranial synostosis; cerebral aneurysms; acoustic neuromas; arteriovenous malformation; trigeminal neuralgia procedures [54]; CSF leaks [55]. When used in combination with image guidance and MRI the transnasal endoscopic approach can be used to localize and excise tumours such as anterior cranial fossa gliomas [56,57] and pituitary tumours [58\*]. In these cases, a micro-Doppler probe can be used to identify and confirm the trajectory of both carotid arteries and the medial cavernous sinus wall. Nasal complications are reduced because the endoscopic technique avoids the septal incision and other parts of the nasal passages [59]. Spinal diseases as syringomyelia [60], palmar hyperhidrosis, disk herniation, spine deformities or tumours have also been treated using the endoscopic approaches [7,9].

## Conclusion

The neurosurgical literature is replete with hundreds of studies reporting personal experience, case reports, case series and retrospective reviews of the use of neuroendoscopy. This is in sharp contrast to the small amount of neuroanaesthetic literature published on this same topic.

General anaesthesia and invasive haemodynamic monitoring, including direct or indirect measures of ICP are crucial to identify sudden and dangerous increases in ICP. Close communication between the neurosurgeon and the anaesthesiologist is vital to reduce intraoperative and postoperative complications.

## References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 672).

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- Study in a rat model of direct subarachnoidal volume expansion as an experimental model for intracranial hypertension during endoscopic neurosurgery. When haemodynamic reflexes were induced by isolated intracranial hypertension, systemic hypertension always occurred, but not bradycardia. In fact, bradycardia was absent in the presence of near complete CPP suppression. Moreover, in many cases, a severe tachycardia was the only and very distinct constituent of the induced Cushing's reflex. Episodes of severe bradycardia occurred only after multiple minutes of severe CPP suppression when total haemodynamic collapse was imminent. This partially confirms their clinical observations that systemic hypertension and tachycardia should be the first signs to look for when severe intracranial hypertension is suspected.
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