Case Report

jfms OPEN REPORTS

Journal of Feline Medicine and Surgery Open Reports
1–6

DOI: 10.1177/20551169231192287
journals.sagepub.com/home/jfmsopenreports

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Anaesthetic management in a cat undergoing emergency craniotomy for meningioma excision

Giulia Marchionne[1], Emili Alcoverro[1], Simone Spinillo[1] and Luis Filipe Louro[1,2]

Abstract

Case summary A 15-year-old female spayed domestic shorthair cat underwent an emergency craniotomy to remove an intracranial meningioma causing marked midline shift, caudal transtentorial and foramen magnum herniation. Because intracranial structures are enclosed in the cranium, any volume-occupying lesions might raise intracranial pressure (ICP), compromising cerebral perfusion.

Relevance and novel information This case report discusses the anaesthetic management of a cat that presented with marked bradycardia and concomitant hypotension. Cushing's reflex (CR) is a well-recognised cardiovascular reflex following sudden ICP increase, and it features an irregular breathing pattern and increased arterial blood pressure with reflex bradycardia. However, CR is reported to have a low sensitivity for the detection of raised ICP in humans with traumatic brain injury. In a previous study reporting seven cats undergoing surgical removal of intracranial meningioma, ICP was measured in four cases and, in these patients, CR was not observed during surgery. Because bradycardia was not secondary to hypertension, in this case, it might have been the result of direct compression of the nucleus of the vagus nerve. Based on the literature search, there is paucity of reports of cardiovascular changes in cats with increased ICP and their perianaesthetic management.

Keywords: Anaesthesia; neurosurgery; meningioma; craniotomy; Vushing's reflex

Accepted: 19 July 2023

Case description

A 15-year-old female spayed domestic shorthair cat was presented with progressive obtundation, compulsive gait, absent bilateral menace and circling to the right. Pre-referral haematology, biochemistry, electrolytes, total thyroxine and fructosamine were within reference ranges. The cat's heart rate was 140 beats per minute with a regular rhythm and synchronous peripheral pulse. Its blood pressure (BP) was not evaluated at admission. A right forebrain disease was suspected and an MRI was scheduled. The ASA (ie, American Society of Anesthesiology)[1] score assigned was 3.

Butorphanol 0.3 mg/kg and medetomidine 3 µg/kg were administered intravenously (IV), achieving a mild sedative effect. General anaesthesia (GA) was induced with alfaxalone titrated slowly IV over 1 min (total dose 1.38 mg/kg). Immediately after induction of anesthesia, lidocaine spray was applied onto the larynx. A 5-mm uncuffed endotracheal tube was placed. GA was maintained with sevoflurane vaporised in 100% oxygen delivered via a circle breathing system, maintaining an end-tidal sevoflurane concentration of 1.6%. Intermittent positive pressure ventilation (IPPV) was commenced using a Merlin Small Animal Ventilator (Vetronics Services Ltd), maintaining an end-tidal carbon dioxide

[1]ChesterGates Veterinary Specialists, Chester, UK
[2]Veterinary Anaesthesia Consultancy Services Limited, Barnsley, UK

Corresponding author:
Giulia Marchionne DVM, MSc, MRCVS, ChesterGates Veterinary Specialists, Telford Court, Units E and F, Gates Lane, Chester, CH1 6LT, UK
Email: giulia.marchionne@chestergates.org.uk

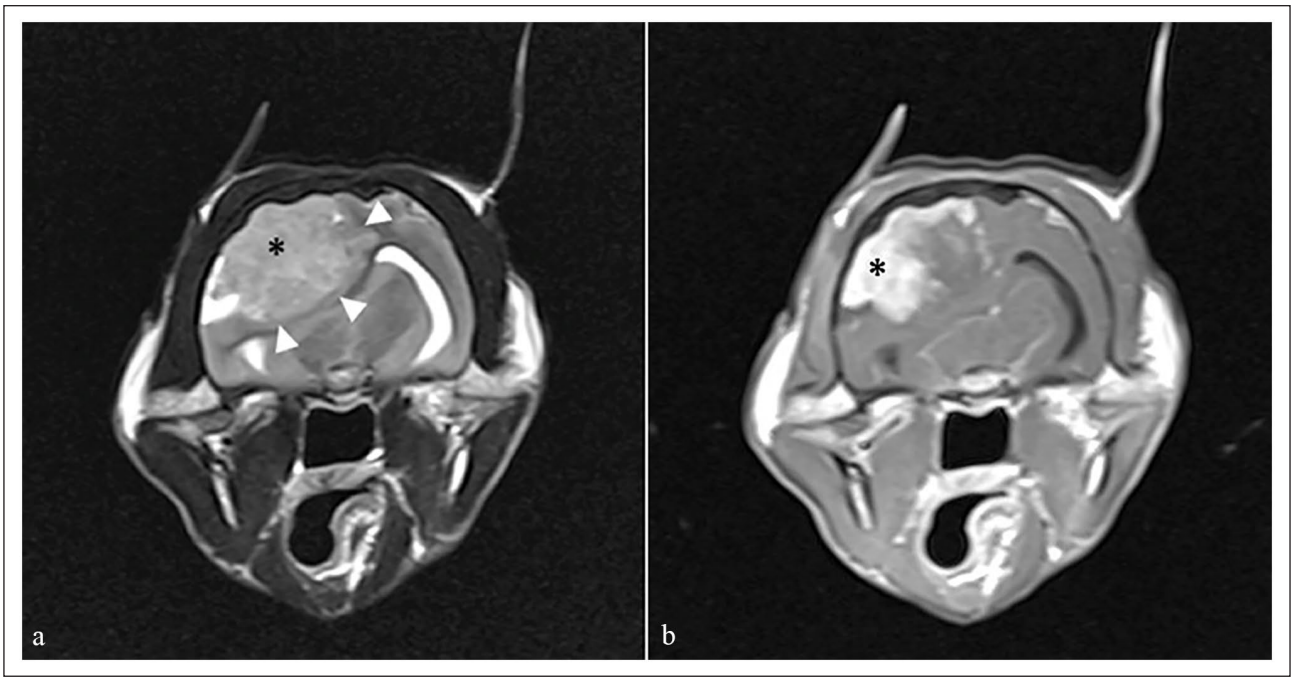

Figure 1 MRI of the brain of the cat. (a) Transverse T2-weighted and (b) post-contrast T1-weighted images at the level of the midbrain. A large, broad-based, extra-axial mass (asterisks) is noted within the right parieto-occipital cortex. It is heterogeneously hyperintense on T2W and displays strong contrast enhancement in the peripheral half of the mass. There is obliteration of the right lateral ventricle and displacement of the subjacent diencephalon and midbrain, consistent with severe mass effect (arrowheads)

partial pressure ($EtCO_2$) of 30–32 mmHg. Tidal volume was 17.2–24.1 ml/kg with a peak inspiratory pressure of 7–8 cmH_2O and a respiratory rate of 10–15 beats per min. High tidal volumes are often required owing to the high dead space and high compliance of our anaesthetic machine set up in the MRI suite. During the MRI, the patient was monitored using a multiparameter monitor continuously displaying pulse oximetry (SpO_2), non-invasive BP (model 3880; IRadimed) and capnography, including gas analysis (Compact S5; Datex-Ohmeda). IV fluid therapy was initiated after induction of anaesthesia with Hartmann's solution (4 ml/kg/h). The MRI revealed findings compatible with a meningioma with features of midline shift, caudal transtentorial and foramen magnum herniation (Figures 1-2). Dexamethasone 0.2 mg/kg and mannitol 0.5 g/kg over 30 mins were administered IV to reduce ICP. During the MRI, the median (range) pulse rate measured by the pulse oximeter was 68 (range 63–88) beats per minute, and the mean arterial pressure (MAP) was 59 mmHg (45–67). Hypertonic saline (HTS) 7% 2 ml/kg was administered IV over 10 mins, 15 mins after dexamethasone and mannitol. Bradycardia was not treated to avoid increasing cerebral blood flow (CBF) and end-tidal sevoflurane was maintained <1.6% to reduce vasodilation. Because HTS has volume-expanding properties, arterial BP improved after it was administered to treat the increased ICP.

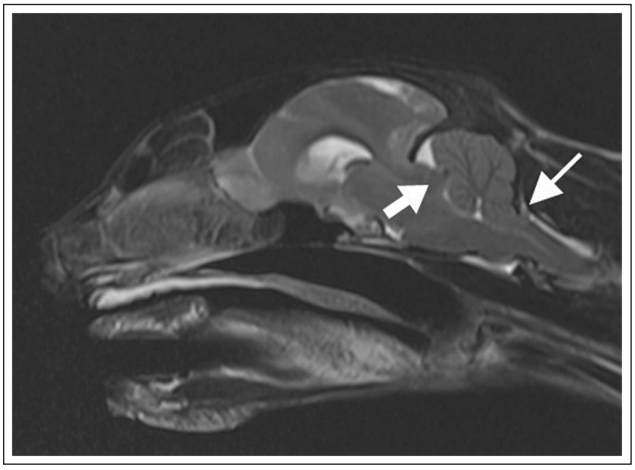

Figure 2 Mid-sagittal T2-weighted image. There is caudal transtentorial (thick arrow) and foramen magnum herniation (thin arrow)

After the MRI, emergency right rostrotentorial craniotomy was performed. Intraoperatively, capnography, spirometry, SpO_2, electrocardiogram, temperature and invasive BP were monitored. Medical air was added, achieving an inspired fraction of oxygen (FiO_2) of 0.6 and IPPV was maintained during the procedure with an $EtCO_2$ of 32 (range 26–46) mmHg. An infusion of dexmedetomidine 1 µg/kg/h was started 20 mins before surgery

Table 1 Intraoperative arterial blood gasses analysis

	Time				
	50 mins before surgery	30 mins after surgery started	60 mins after surgery started	90 mins after surgery started	195 mins after surgery started (end of surgery)
pH	7.165 L	7.138 L	7.365	7.278	7.326
pCO_2 (mmHg)	59.8 H	58.1 H	29.7	43.0 H	34.7 H
pO_2 (mmHg)	555.7 H	407.9 H	499.7 H	294.2 H	258.1 H
$cHCO_3^-$ (mmol/l)	21.6 H	19.7	17	20.1 H	18.1
BE (ecf) (mmol/l)	–7.0 L	–9.4 L	–8.4 L	–6.7 L	–7.9 L
CsO_2 (%)	100.0	99.9	100.0	99.9	99.8
Hct (%)	20 L	20 L	15 L	21 L	16 L
cHgb (g/dl)	6.6 L	6.6 L	5.2 L	7.2 L	5.6 L
Na^+ (mmol/l)	156	162	157	157	153
K^+ (mmol/l)	2.9	2.5 L	3.2	3.3	3.6
Ca^{2+} (mmol/l)	1.37 H	1.36 H	1.21	1.34 H	1.28
Cl^- (mmol/l)	125	131	127	128	127
$cTCO_2$ (mmol/l)	23.1	21.3	18	21.3	19.2
Glu (mmol/l)	8.0 H	9.6 H	8.1 H	8.2 H	9.9 H
Lac (mmol/l)	0.99	0.91	1.2	0.33 L	0.4 L
$EtCO_2$	24	46	27	35	33
V_D/V_T	0.59	0.2	0.09	0.18	0.04

V_D/V_T = dead space volume/total tidal volume, calculated with the revised Bohr equation $V_D/V_T = (PaCO_2 - P_ECO_2)/PaCO_2$, where $PaCO_2$ is the arterial partial pressure of CO_2 and P_ECO_2 is the partial pressure of CO_2 in the expired gas mixture; L = low; H = high; BE = base excess; cHCO3 = bicarbonate; cHgb = hemoglobin; CsO2 = oxygen saturation; cTCO2 = total carbon dioxide content; Glu = glucose; Hct = hematocrit; Lac = Lactate; pCO2 = partial pressure of carbon dioxide; pO2 = partial pressure of oxygen

and maintained during the whole procedure. A bolus of 2 ml/kg of HTS was administered before initiating the drilling of the skull. During surgery, the median (range) heart rate (HR) was 79 (69–93) beats per minute and MAP median (range) was 62 (52–115) mmHg. Serial arterial blood gasses analysis were performed intraoperatively (Table 1). Blood gas analysis showed metabolic and respiratory acidosis. The latter improved by adjusting the ventilator settings. Using $PaCO_2$ and $EtCO_2$ to calculate the revised Bohr equation, we can speculate that the ventilation/perfusion mismatch present is likely due to dead space ventilation. Haematocrit was consistently low before and after surgery, which might be explained by sequestration of erythrocytes outside of the circulation during anaesthesia.[2] Hyperglycaemia was present throughout the anaesthetic due to the effects of alpha-2-adrenoceptor agonists,[3] as well as surgical manipulation. High adrenaline, cortisol levels and exogenous steroid therapy likely contributed to elevated blood glucose levels. The surgery was uneventful and, subjectively, a complete removal of the mass was achieved.

Spontaneous ventilation was noted immediately after mechanical ventilation was discontinued. Dexmedetomidine constant rate infusion (CRI) was halved every 30 mins to avoid excitation during recovery. Two hours after extubation, HR improved (108 beats per minute) as did BP (MAP 80 mmHg). During the postoperative hospitalisation period, the median (range) HR was 80 (120–210) beats per min, and the median (range) systolic BP was 127 (112–220) mmHg. Postoperatively, the cat received dexamethasone 0.2 mg/kg IV q24h, cefuroxime 20 mg/kg IV q8h and three dosages of buprenorphine 0.02 mg/kg IV q8h. The cat was discharged 3 days after surgery. A tapering anti-inflammatory dose of prednisolone was prescribed and discontinued 3 weeks later. Histopathological-analysis of the mass was diagnostic of epithelioid meningioma.

Eight weeks postoperatively, neurological examination was normal, besides marginally reduced menace response on the left. Eight months after surgery, the owner reported that the cat returned to its normal behaviour with no evident neurological deficits.

Discussion

Although feline intracranial tumours might result in brain herniation without specific neurological signs,[4,5] in this case, obtundation and episodes of vacant mentation were suggestive of possible increased ICP. Therefore, a comprehensive pre-anaesthetic evaluation is important to plan an adequate anaesthetic episode.[6] The cranial cavity consists of three components: brain parenchyma, cerebrospinal fluid and blood (arterial and venous). According to the Monro–Kellie Doctrine,[7] owing to the fixed nature of the

cranium, the sum of their volume should be constant in order to maintain a physiological ICP of 5–12 mmHg.[8] The increase in ICP is the result of an increase of volume of any of the contents of the cranium, leading to alteration of cerebral perfusion pressure (CPP) and CBF.

Mathematically, CPP = MAP − ICP. Accordingly, for the brain to remain adequately perfused, MAP must be higher than ICP. In the case of increased ICP, an increase in systemic vascular resistance will raise the MAP and maintain CPP. Consequently, aortic and carotid baroreceptors are stimulated, resulting in reflex bradycardia. Compression of the brainstem owing to elevated ICP also results in distortion of the respiratory centres, causing an irregular breathing pattern. This is described as Cushing's reflex (CR).[9]

CR is a well-recognised ICP-related cardiovascular reflex; however, in human medicine, it is reported to have a low sensitivity for the detection of raised ICP.[10] Although, in dogs, higher BP and lower HR at admission have been associated with the presence of brain herniation,[11] this has not been seen in cats.[12] A retrospective study in cats undergoing surgical removal of intracranial meningioma reported the absence of CR in some patients where raised ICP was confirmed by continuous monitoring.[4]

In our case report, bradycardia and mild hypotension were consistently recorded during both MRI and surgery. BP and HR remained stable during the surgical procedure, both before and after durectomy. Bradycardia and asystole are frequently reported in humans during craniotomies, even with the absence of hypertension. Furthermore, intraoperative arterial hypotension has often been noted.[13–15] It has been suggested that in patients with increased ICP, bradycardia can result from direct compression of the vagal nucleus, located in the medulla oblongata.[16,17] The MRI showed caudal transtentorial herniation, midline shift and foramen magnum herniation, which would likely apply a high pressure on the brain stem (Figure 2).[18] Although hypotension can result in decreased brain perfusion and oxygen delivery, in this case, it may have been related to the bradycardia. It was decided not to treat it because increasing the HR might have resulted in increasing the CBF and consequently aggravating the high ICP. During surgery, the patient was consistently hypothermic (oesophageal temperature 32.7–34.2°C). Hypothermia could contribute to causing bradycardia and hypotension;[19] however, HR and BP were similar at the beginning of GA, when the temperature was recorded as normal.

The anaesthetist chose therapeutic hypothermia to lower the intracranial pressure,[20] although the risk–benefit evaluation of this treatment in human medicine remains inconclusive. Some studies suggest that oxygen delivery and consumption decrease at rectal temperatures below 35°C,[21] but maintaining a temperature of 33–34°C following decompressive craniectomy reduces ICP in patients with severe brain swelling.[20] Sakoh and Gjedde[22] propose that lowering the temperature to 32°C may confer neuroprotective benefits by restoring a physiological equilibrium between cerebral metabolism and perfusion.

Bradycardia is common after alpha-2-adrenoceptor agonist administration; however, this cardiovascular effect is dose-related.[23] The low doses administered during premedication and as a CRI during surgery were expected to have a mild effect on HR and systemic vascular resistance. It was assumed that the cardiovascular changes were likely related to the increased ICP and so treatment was aimed to reduce ICP.

Alpha-2-adrenoceptor agonists were used to take advantage of their anaesthesia-sparing effect, their sympatholytic effect and antinociceptive properties.[24] Modulating the sympathetic responses will attenuate risks of increased ICP and brain haemorrhage. In human medicine, dexmedetomidine CRI during craniotomies helps maintain the haemodynamic stability and reduce sevoflurane and opioid requirements.[25,26] Dexmedetomidine is also reported to decrease ICP by the inhibition of the hypercapnic cerebral vasodilation and venous vasoconstriction.[27]

Anaesthesia was maintained with sevoflurane. Inhalational anaesthetic agents not only present a neuroprotective action by reducing cerebral metabolism, but also cause dose-dependent vasodilation, resulting in increased ICP. In humans, sevoflurane results in the least vasodilation at less than 1 MAC (ie, minimum alveolar concentration) compared with other inhalant agents, at the same time as maintaining intact brain autoregulation.[28] The addition of dexmedetomidine CRI aimed to reduce sevoflurane requirements and reduce vasodilation. Propofol can reduce the CBF, ICP and cerebral metabolic rate of oxygen; thus, it is considered the most suitable anaesthetic drug in humans with increased ICP. However, in cats, long propofol infusions are discouraged due to the possible formation of Heinz bodies[29] and a reported longer recovery time.[30]

Hypercapnia causes cerebral vasodilation and an increase in CBF and ICP, whereas severe hypocapnia results in cerebral vasoconstriction and reduced oxygen delivery.[31] For this reason, IPPV was implemented throughout the GA, aiming for an $EtCO_2$ in the range of 30–33 mmHg.[32] After an arterial cannula was placed, a series of blood gasses where taken, adjusting ventilation according to the partial pressure of carbon dioxide (ie, $PaCO_2$).

Mannitol and HTS were administered to reduce the patient's ICP. Mannitol acts as a volume expander and osmotic diuretic. It causes fluid to move from the interstitial and intracellular spaces to the intravascular compartments and decreases blood viscosity. It also increases oxygen delivery to the brain, causing cerebral vasoconstriction and increased CBF. HTS acts in a similar way

through its osmotic effect. In this case, HTS was also used for its favourable haemodynamic properties such as increasing cardiac output and BP.[33] HTS also acts as an anti-inflammatory agent by decreasing leukocyte adhesion.[34] In human medicine, it has been suggested that HTS might be superior in decreasing ICP compared with mannitol,[35] but other reports have found no difference. During MRI, the cat received dexamethasone. Corticosteroids are reported to be beneficial in reducing peritumoural vasogenic oedema.[36]

Conclusions

During GA, although CR is commonly associated with a sudden increase in ICP, other haemodynamic changes also need to be considered as warning signs, such as bradycardia without hypertension, especially in patients that already present brain herniation prior to anaesthesia.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding The authors received no financial support for the research, authorship, and/or publication of this article.

Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*. Although not required, where ethical approval was still obtained, it is stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers) for all procedure(s) undertaken (prospective or retrospective studies). No animals or people are identifiable within this publication, and therefore additional informed consent for publication was not required.

ORCID iD Giulia Marchionne https://orcid.org/0000-0001-7969-8381

Emili Alcoverro https://orcid.org/0000-0003-2331-0872

Luis Filipe Louro https://orcid.org/0000-0002-2911-5501

References

1 American Society of Anesthesiologists. **ASA physical status classification system.** www.asahq.org/standards-and-practice-parameters/statement-on-asa-physical-status-classification-system (2020, accessed 9 August 2023).

2 Dhumeaux MP, Snead EC, Epp TY, et al. **Effects of a standardized anesthetic protocol on hematologic variables in healthy cats.** *J Feline Med Surg* 2012; 14: 701–705.

3 Fagerholm V, Haaparanta M and Scheinin M. **α_2-Adrenoceptor regulation of blood glucose homeostasis.** *Basic Clin Pharmacol Toxicol* 2011; 108: 365–370.

4 Kouno S, Shimada M, Sato A, et al. **Surgical treatment of rostrotentorial meningioma complicated by foraminal herniation in the cat.** *J Feline Med Surg* 2020; 22: 1230–1237.

5 Troxel MT, Vite CH, Winkle TJV, et al. **Feline intracranial neoplasia: retrospective review of 160 cases (1985–2001).** *J Vet Intern Med* 2003; 17: 850–859.

6 Louro LF, Maddox T, Robson K, et al. **Pre-anaesthetic clinical examination influences anaesthetic protocol in dogs undergoing general anaesthesia and sedation.** *J Small Anim Pract* 2021; 62: 737–743.

7 Monro A. **Observations on the structure and functions of the nervous system.** *Lond Med J* 1783; 4: 113–135.

8 Dewey CW. **Head trauma management.** In: Dewey CW and da Costa RC (eds). A practical guide to canine and feline neurology. Ames, IA: John Wiley & Sons, 2016, pp 237–248.

9 Cushing H. **Concerning a definite regulatory mechanism of the vasomotor centers which controls blood pressure during cerebral compression.** *Bull Johns Hopkins Hosp* 1901; 12: 290.

10 Ter Avest E, Taylor S, Wilson M, et al. **Prehospital clinical signs are a poor predictor of raised intracranial pressure following traumatic brain injury.** *Emerg Med J* 2021; 38: 21–26.

11 Her J, Yanke AB, Gerken K, et al. **Retrospective evaluation of the relationship between admission variables and brain herniation in dogs (2010–2019): 54 cases.** *J Vet Emerg Crit Care (San Antonio)* 2022; 32: 50–57.

12 Her J, Merbl Y, Gerken K, et al. **Relationship between admission vitals and brain herniation in 32 cats: a retrospective study.** *J Feline Med Surg* 2022; 24: 770–778.

13 Bilotta F, Guerra C and Rosa G. **Update on anesthesia for craniotomy.** *Curr Opin Anaesthesiol* 2013; 26: 517–522.

14 Haldar R, Prakhar G and Guruprasad B. **Isolated bradycardia due to skull pin fixation: an unusual occurrence.** *J Neurosurg Anesthesiol* 2013; 25: 206–207.

15 Vimala S and Arulvelan A. **Sudden bradycardia and hypotension in neurosurgery: trigeminocardiac reflex (TCR) and more.** *J Neurosurg Anesthesiol* 2016; 28: 175–176.

16 Tsai Y-H, Lin J-Y, Huang Y-Y, et al. **Cushing response-based warning system for intensive care of brain-injured patients.** *Clin Neurophysiol* 2018; 129: 2602–2612.

17 Cho S-M, Kilic A and Dodd-o JM. **Incomplete Cushing's reflex in extracorporeal membrane oxygenation.** *Int J Artif Organs* 2020; 43: 401–404.

18 Walmsley GL, Herrtage ME, Dennis R, et al. **The relationship between clinical signs and brain herniation associated with rostrotentorial mass lesions in the dog.** *Vet J* 2006; 172: 258–264.

19 Dahlen RW. **Some effects of hypothermia on the cardiovascular system and ECG of cats.** *Exp Biol Med* 1964; 115: 1–4.

20 Rim HT, Ahn JH, Kim JH, et al. **Therapeutic hypothermia for increased intracranial pressure after decompressive craniectomy: a single center experience.** *Korean J Neurotrauma* 2016; 12. DOI: 10.13004/kjnt.2016.12.2.55.

21 Tokutomi T, Morimoto K, Miyagi T, et al. **Optimal temperature for the management of severe traumatic brain injury: effect of hypothermia on intracranial pressure, systemic and intracranial hemodynamics, and metabolism.** *Neurosurgery* 2003; 52: 102–112.

22 Sakoh M and Gjedde A. **Neuroprotection in hypothermia linked to redistribution of oxygen in brain.** *Am J Physiol Heart Circ Physiol* 2003; 285: H17–H25.

23 Pypendop BH and Verstegen JP. **Hemodynamic effects of medetomidine in the dog: a dose titration study.** *Vet Surgery* 1998; 27: 612–622.

24 Murrell JC. **Pre-anaesthetic medication and sedation.** In: Duke-Novakovski T, de Vries M and Seymour C (eds). *BSAVA manual of canine and feline anaesthesia and analgesia*. Quedgeley: British Small Animal Veterinary Association, 2016, pp 170–189.

25 Lin N, Vutskits L, Bebawy JF, et al. **Perspectives on dexmedetomidine use for neurosurgical patients.** *J Neurosurg Anesthesiol* 2019; 31: 366–377.

26 Wang L, Shen J, Ge L, et al. **Dexmedetomidine for craniotomy under general anesthesia: a systematic review and meta-analysis of randomized clinical trials.** *J Clin Anesth* 2019; 54: 114–125.

27 Soliman RN, Hassan AR, Rashwan AM, et al. **Prospective, randomized study to assess the role of dexmedetomidine in patients with supratentorial tumors undergoing craniotomy under general anaesthesia.** *Middle East J Anesthesiol* 2011; 21: 325–334.

28 Engelhard K and Werner C. **Inhalational or intravenous anesthetics for craniotomies? Pro inhalational.** *Curr Opin Anaesthesiol* 2006; 19: 504–508.

29 Andress JL, Day TK and Day DG. **The effects of consecutive day propofol anesthesia on feline red blood cells.** *Vet Surgery* 1995; 24: 277–282.

30 Pascoe PJ, Ilkiw JE and Frischmeyer KJ. **The effect of the duration of propofol administration on recovery from anesthesia in cats.** *Vet Anaesth Analg* 2006; 33: 2–7.

31 Tameem A and Krovvidi H. **Cerebral physiology.** *Contin Educ Anaesth Crit Care Pain* 2013; 13: 113–118.

32 Leece EA. **Neurological disease.** In: Duke-Novakovski T, de Vries M and Seymour C (eds). *BSAVA manual of canine and feline anaesthesia and analgesia*. Quedgeley: British Small Animal Veterinary Association, 2016, pp 392–409.

33 Bitterman H, Triolo J and Lefer A. **Use of hypertonic saline in the treatment of hemorrhagic shock.** *Circ Shock* 1987; 21: 271–283.

34 Torre-Healy A, Marko NF and Weil RJ. **Hyperosmolar therapy for intracranial hypertension.** *Neurocrit Care* 2012; 17: 117–130.

35 Kamel H, Navi BB, Nakagawa K, et al. **Hypertonic saline versus mannitol for the treatment of elevated intracranial pressure: a meta-analysis of randomized clinical trials.** *Crit Care Med* 2011; 39: 554–559.

36 Bebawy JF. **Perioperative steroids for peritumoral intracranial edema: a review of mechanisms, efficacy, and side effects.** *J Neurosurg Anesthesiol* 2012; 24: 173–177.