Canine cerebral circulation: a review

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Abstract

The cerebral vascular system (CVS) of mammals is a complicated three-dimensional structure that supplies brain parenchyma with oxygenated blood and nutrients, drains deoxygenated blood and catabolites out from it and participates in cerebrospinal fluid (CSF) resorption, maintenance of intracranial pressure (ICP) stability, and brain thermoregulation. A thorough understanding of the morphology and function of CVS is essential for human as well as veterinary neurologists and neurosurgeons as it helps to diagnose intracranial pathological processes, to choose an optimal therapeutic approach for the specific patient concerning configuration and possible anomalies of their CVS, and to execute intracranial surgical procedures. The number of brain operations in dogs has rapidly grown, which prompted the authors to review the literature on the complex issue of canine intracranial blood vessels. The research strategy involved a PubMed, MEDLINE (Ovid), EMBASE (Ovid), and Clarivate Analytics Web of Science search from January 1960 to January 2024 using the terms 'canine brain blood vessels' and 'cerebral haemodynamics in dogs' in the English language literature; references from selected papers were also scanned, and relevant articles were included.

Dog, intracranial vascular system, anatomy, physiology

Epidemiological studies as well as everyday practice indicate that head injuries, their complications, and various pathological processes affecting the brain, meninges, and skull in humans also occur in dogs and cats (Hopkins and Wheeler 1991; Wessmann et al. 2009; Pleasas et al. 2013; Przyborowska et al. 2013; Cosme et al. 2015; Jones et al. 2018; Miller et al. 2019; Kishimoto et al. 2020). The structure and function of the mammalian central nervous system (CNS) are similar. Pigs, dogs, cats, and rats are regularly used in experiments and preclinical trials. There are owners ready to cover charges for the treatment of their pets. Therefore, intracranial interventions in cats and dogs have become more frequent. They are challenging procedures (Forward et al. 2018). One of the main hazards of craniotomy and brain surgery is an inadvertent lesion of a vital brain vessel, which can potentially cause severe complications with impending catastrophic consequences (Morton et al. 2022). These reasons inspired the authors to report on the basic structural and functional features of the cerebral vascular system of dogs.

Survey of canine intracranial blood circulation

The intracranial vascular system (IVS) of mammals consists of an arterial and venous part interconnected with cerebral capillaries, surrounded with cerebrospinal fluid (Fahmy et al. 2021). The IVS maintains not only adequate brain perfusion but also plays an important role in cerebrospinal fluid (CSF) resorption, intracranial pressure (ICP) homeostasis, and cerebral thermoregulation (Wellens et al. 1975; Bezuidenhout 2013a; Bezuidenhout 2013b; Fahmy et al. 2021; Lavinio 2022).

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Canine cerebral arterial system

The brain arteries in dogs carry oxygenated blood from the heart to intracranial structures. The wall of cerebral arteries and arterioles consists of three concentric layers. The innermost layer (tunica intima) comprises a thin sheet of squamous epithelium and the internal elastic lamina. The middle layer (tunica media) comprises circularly arranged smooth muscle cells with some elastin and collagen fibres. The outermost layer (tunica adventitia) is composed mainly of collagen fibres, fibroblasts, and (in pial arterioles and arteries) perivascular nerves (Bezuidenhout 2013a; Skerrit 2018).

Five pairs of arteries (branches of the paired internal carotid arteries - ICAs or the single basilar artery - BA) supply the canine brain. Four of them constitute the cerebral arterial circle (circulus arteriosus cerebri Willisi, the circle of Willis) situated on the ventral surface of the brain around the hypothalamus, mamillary bodies, and hypophyseal stalk (Tanaka et al. 2018). The fifth pair supplies the ventral regions of the cerebellum and brain stem via the caudal cerebellar arteries (Wellens et al. 1975; Gillilan 1976; Tanuma 1981; Skerrit 2018; Camstra et al. 2020). The rostral section of the cerebral arterial circle consists of paired rostral cerebral and middle cerebral arteries (the cranial divisions of the ICA). The lateral section of the circle consists of paired caudal communicating and caudal cerebral arteries (the caudal divisions of the ICA). The dorsal section of the cerebral arterial circle includes paired rostral cerebellar arteries originating from the dorsal communicating arteries (the terminal branches of the BA). The fifth pair of arteries supplying the canine brain are the caudal cerebellar arteries (branches of the BA). The caudalmost arterial branches of the BA are labyrinthine arteries (which supply the inner ear, as well as facial and vestibulocochlear nerves) and rami ad pontem (which supply the pons and trigeminal nerves) (Pais et al. 2009; Skerrit 2018; De Lahunta et al. 2021). In all 55 dogs studied by Tanuma (1981), the Willis circle was entirely closed, usually through the single rostral communicating artery (Fig. 1). The canine cerebral arterial system is posterior circulation dominant. Detailed surveys showed that a significant portion of oxygenated blood to the brain of dogs comes from the paired vertebral arteries (VAs) via the ventral spinal artery and the BA (the midline continuation of the ventral spinal artery) and the lesser portion comes from the common carotid arteries (CCAs) via their thinner and tortuous branches -



Fig. 1. Diagram of the main arterial supply of the canine brain; dorsal view.

internal carotid artery (a. carotis interna); 2 - basilar artery (a. basilaris); 3 - middle cerebral artery (a. cerebri media); 4 - rostral cerebral artery (a. cerebri rostralis); 5 - caudal communicating artery (a. communicans caudalis);
 caudal cerebral artery (a. cerebri caudalis); 7 - rostral communicating artery (a. communicans rostralis);
 rostral cerebellar artery (a. cerebri rostralis); 9 - caudal cerebellar artery (a. cerebellaris caudalis);
 lo - labyrinthine artery (a. labyrinthica); 11 - internal ophthalmic artery (a. ophthalmica interna); 12 - internal ethmoidal artery (a. ethmoidalis interna).

the internal carotid arteries – ICAs (Orsi et al. 2006; Skerrit 2018; Camstra et al. 2020; Ševčíková et al. 2023). There are also several extra-intracranial arterial anastomoses in the orbit (via branches of the ophthalmic arteries), in the nasal cavity (via branches of the internal ethmoidal arteries), as well as significant both-sided anastomoses between the maxillary arteries and ICAs, or between the vertebral arteries and ICAs at the neck (Orsi et al. 2006; Skerrit 2018; Camstra et al. 2020; De Lahunta et al. 2021).

Variations of the canine cerebral arterial system

The extra-intracranial arterial anastomoses in dogs and cats are significant, hence ischaemic stroke is a rare clinical entity in these animals (Gillilan 1976; Wessmann et al. 2009). The internal carotid artery divides into the rostral cerebral artery, middle cerebral artery, and the dorsal communicating artery (Tanuma 1981; Bezuidenhout 2013a; Tanaka et al. 2018). Frequently the internal carotid artery (ICA) firstly gives of the dorsal communicating artery, then divides into the rostral cerebral and middle cerebral arteries. This type of ICA division was observed in 85.5% of specimens on the right side and 81.8% on the left side (Tanuma 1981). The ICA also can first give of the dorsal communicating artery, then divide into the rostral cerebral artery and the middle cerebral artery. This type of ICA division was observed in 10.9% of specimens on the right side and in 9.1% on the left side (Tanuma 1981).

Variations of the canine circle of Willis also occur. Tanaka et al. (2018) ascertained the typical configuration of this arterial structure in 82% of dogs, aplasia of the precommunicating segment of the rostral cerebral artery in 14% of dogs, and aplasia of the rostral communicating artery in 4% of dogs (Tanaka et al. 2018). In all 55 dogs studied by Tanuma (1981), the Willis circle was entirely closed in all specimens (Tanuma 1981). The rostral cerebral arteries having a common trunk occurred in 87.3%, a single rostral communicating artery occurred in 9.1%, and a double rostral communicating artery of the diameter of the rostral cerebral arteries was observed in 14.5% of specimens (Tanuma 1981).

Canine cerebral capillary network

The brain capillaries in dogs form a dense, three-dimensional, and mutually interconnected network. Their mesh-like structure provides these tiny vessels (5–10 um in diameter) with an extremely large surface area, facilitating their vital role in nutrient exchange (Wellens et al. 1975; Bezuidenhout 2013a; Skerrit 2018). The brain capillaries have thin, nonfenestrated walls (by contrast to the rest of the extracerebral capillaries possessing micro apertures in their walls), composed of the tunica intima, but contain neither smooth muscle cells nor elastic fibres. The brain arterioles, capillaries, and venules are lined by extended astrocytic cellular processes and scattered pericytes (Bezuidenhout 2013a; Daneman and Prat 2015; Skerrit 2018). The tunica intima, consisting of a simple layer of specialized squamous endothelial cells interconnected by tight junctions encased by a basement membrane and surrounded by pericytes, creates the blood-brain barrier (BBB) separating the contents of these specific blood vessels from the brain interstitium (Wellens et al. 1975; Skerrit 2018; Procter et al. 2021). The substance movement through the cerebral capillary wall can occur paracellularly (between endothelial cells) or transcellularly (across endothelial cells). The intact BBB is permeable to oxygen, water, and small lipid-soluble molecules. Still, with the help of junctional complexes and by expression of membrane receptors and pumps, the BBB tightly regulates the movement of nutrients, wastes, and more complex molecules between circulating blood and the brain environment (Braun et al. 2017; Smith et al. 2019; Procter et al. 2021). The pericytes are also metabolic sentinels that control and help to increase blood flow when stimulated (D'Alecy and Feigl 1972; Smith et al. 2019). Disruption of the intimate cooperation between endothelial

cells and pericytes leads to alterations in cerebral blood flow, to neuroinflammation, and transcytosis, contributing to the breakdown of the BBB (Procter et al. 2021).

Canine cerebral venous system

The cerebral venules and veins collect the low-pressure deoxygenated blood from the brain capillaries (Bezuidenhout 2013b; Gada et al. 2015; Balik 2019). The cerebral veins emerge from the surface of the brain, bridge the subarachnoid space filled with cerebrospinal fluid (that is why they are also known as bridging veins), and drain into the dural venous sinuses (DVSs). The DVSs then transport the venous blood from the cranium into the systemic circulation via paired maxillary, internal jugular, and vertebral veins as well as via the internal vertebral venous plexuses (Aurboon vawat et al. 2007; Mortazavi et al. 2012; Bezuidenhout 2013b; Gada et al. 2015; Skerrit 2018; Walsh et al. 2020). The cerebral veins are valveless, thin-walled vessels reinforced by loose collagen networks but lacking tunica media and tunica adventitia (Bezuidenhout 2013b; Balik 2019). The subdural portion walls of cerebral veins are $10-600 \ \mu m$ thick, and the subarachnoid space portion walls are 50-200 µm thick (Mortazavi et al. 2012). The DVSs of dogs are located between the external (periosteal) and internal (meningeal) layers of the dura mater and, in some places, within channels inside the diploë of the skull bones (Reinhart et al. 1962; Armstrong and Horwitz 1971; Bezuidenhout 2013b a 2018). The walls of DVSs are composed of connective tissue consisting of collagen and elastin fibres with fibrocytes, sometimes also myocytes, and a dense microvascular arterial network. The inner surface of DVSs is covered by a thin layer of endothelial cells (Balik 2019; Walsh et al. 2020). The intracranial veins are connected with extracranial venous networks by emissary veins piercing the skull through bone foramina (Mortazavi et al. 2012; Bezuidenhout 2013b).

Canine DVSs form a dorsal and ventral group which are interconnected (Figs 2 and 3). The dorsal group of DVSs encompasses the dorsal sagittal sinus (DSS), the straight sinus (StS), and paired transverse sinuses (TSs). The ventral group of DVSs comprises paired cavernous sinuses (CSs), unpaired ventral and dorsal intercavernous sinuses (VIcS, DIcS), paired sigmoid sinuses (SSs), the basilar sinus (BS), the connecting ventral interbasilar sinus (VIbS) and, in some cases, also the dorsal interbasilar sinus (DIbS) and paired dorsal and ventral petrosal sinuses (DPSs, VPSs). The DSS begins with a junction of the right and left vein of the nasal cavity, approximately at the central part of the cribriform plate, and continues in the falx cerebri to the point where the DSS joins with the straight sinus (StS). The StS begins at the caudal margin of the cerebral falx (falx cerebri) as a junction of the great cerebral vein (GCV) and the vein of the corpus callosum (VCC). Caudally, inside the diploë of the occipital bone, the DSS merges with the paired transverse sinuses (TSs) and creates the confluence of the sinuses (CSs) also known as the torcular Herophili (Reinhard et al. 1962; Armstrong and Horwitz 1971; Carreira et al. 2011; Bezuidenhout 2013b; Carreira and Ferreira 2016; Skerrit 2018). From the CSs, the TSs continue laterally inside the transverse canals for about two-thirds of their lengths, then inside the transverse grooves, until they terminate by division into the temporal sinuses (TeSs) and sigmoid sinuses (SSs). The TeSs continue to the right and left temporal meatus, then empty into the right or left maxillary vein (MV). The SSs bend ventromedially and caudally on their course to the right or left jugular foramen (JF). The SSs merge with the right or left VPS to create the left or right internal jugular vein (IJV), and the right or left vertebral vein (VV). The DSS, TSs, SSs, and BSs, together with the vertebral plexus, constitute the main venous drainage system of the brain (Reinhard et al. 1962; Armstrong and Horwitz 1971; Bezuidenhout 2013b). The paired cavernous sinuses (CSs) extend from the right and left orbital fissures to the right or left petrooccipital canal. Each of the CSs communicates with the right or left ophthalmic plexus rostrally and via the right or left petrosal vein with ventral vertebral plexus caudally (Reinhard et al. 1962; Bezuidenhout 2013b).



Fig. 2. Diagram of the canine brain venous system; lateral view.

1 - nasal vein (v. nasalis); 2 - dorsal sagittal sinus (sinus sagittalis dorsalis); 3 - dorsal cerebral veins (vv. cerebri dorsales); 4 - transverse sinus (sinus transversus); 5 - confluence of the sinuses (confluens sinuum); 6 - occipital emissary vein (v. emissaria occipitalis); 7 - temporal sinus (sinus temporalis); 8 - dorsal petrosal sinus (sinus petrosus dorsalis); 9 - ventral cerebral vein (v. cerebri ventralis); 10 - sigmoid sinus (sinus sigmoideus); 11 - basilar sinus (sinus basilaris); 12 - ventral petrosal sinus (sinus petrosus ventralis); 13 - cavernous sinus (sinus cavernosus); 14 - straight sinus (sinus rectus); 15 - vein of the corpus callosum (v. corporis callosi); 15a - tributaries of the vein of the corpus callosum; 16 - internal cerebral vein (v. cerebri vein (v. thalamostriata).



Fig. 3. Diagram of the canine brain venous system; dorsal view.

1 - nasal veins (v. nasalis dextra et sinistra); 2 - dorsal sagittal sinus (sinus sagittalis dorsalis); 3 - dorsal cerebral vein (v. cerebri dorsalis); 4 - transverse sinus (sinus transversus); 5 - confluence of the sinuses (confluens sinuum);
6 - occipital emissary vein (v. emissaria occipitalis); 7 - temporal sinus (sinus temporalis); 8 - dorsal petrosal sinus (sinus petrosus dorsalis); 9 - ventral cerebral vein (v. cerebri ventralis); 10 - sigmoid sinus (sinus sigmoideus);
11 - basilar sinus (sinus basilaris); 12 - ventral petrosal sinus (sinus petrosus ventralis); 13 - cavernous sinus (sinus cavernosus); 14 - rostral intercavernous sinus (sinus intercavernosus rostralis); 15 - caudal intercavernous sinus (sinus sinue cavernosus caudalis).

Variations of the canine cerebral venous system

The physiologic variations in the size and shape of the skull in different breeds of dogs can influence the morphology of their intracranial venous system (Evans and de Lahunta 2013; Carreira and Ferreira 2016; De Simone et al. 2017). For example, the DSS in brachycephalic dogs has a triangular shape, being narrowest at its rostral part and widest at its caudal part. In mesaticephalic and dolichocephalic dogs the DSS has a butterfly shape, being widest at both ends and narrowest in the middle (Carreira et al. 2011). The TS is shortest in mesaticephalic dogs. The TS is narrowest in mesaticephalic dogs is of medium length, and it is longest in dolichocephalic dogs. The TS is narrowest in mesaticephalic dogs, in brachycephalic dogs it is widest. The left side of the TS is usually smaller than the right. In dolichocephalic dogs the TS has a caudocranial trajectory, and in the brachycephalic and mesaticephalic dogs the TS has a craniocaudal trajectory (Carreira and Ferreira 2016).

The straight sinus (StS) usually drains into the caudal part of the DSS before it enters the foramen for the DSS, but it may pursue an independent course via an adjacent accessory foramen and join the confluence of the sinuses within the occipital bone (Evans and de Lahunta 2013). The DSS also frequently bifurcates after the StS has entered it (Bezuidenhout 2013b).

The two CSs (right and left) are connected by rostral and caudal intercavernous sinuses. The smaller caudal intercavernous sinus may be absent (Bezuidenhout 2013b).

The two BSs (right and left) are the venous links between the SSs and the internal ventral vertebral venous plexus. They are connected by ventral and dorsal interbasilar sinuses. The dorsal interbasilar sinus may be absent (Bezuidenhout 2013b).

Haemodynamic parameters

The brain depends more than any other organ of the body on an adequate, relatively constant, and regulated blood supply (Schaller 2004; Grüne et al. 2015). Cerebral vessels must effectively deliver oxygen, glucose, and other nutrients to the brain, removing carbon dioxide, lactic acid, metabolic products, and wastes (Lavinio 2022). The heart rate in healthy dogs ranges from 140 to 190 beats/min (Hibino and Matsuura 1985; Mishina et al. 1997), the systolic blood pressure is 90–140 mmHg (Mishina et al. 1997; Garofalo et al. 2012), the diastolic blood pressure is 70-80 mmHg (Mishina et al. 1997). At rest, the canine brain receives approximately 14% of cardiac output, i.e., \approx 930 ml/min in an animal weighing 20 kg (Milnor and Bertrand 1958). The intracranial blood volume in dogs is about 100–130 ml (of which $\approx 15\%$ is situated in the cerebral arteries, $\approx 40\%$ in the cerebral veins and sinuses, and $\approx 45\%$ in the cerebral microcirculation) at any moment (Milnor and Bertrand 1958; Wellens et al. 1975; Gobbel et al. 1991; Lavinio 2022). There are several mechanisms preventing brain damage in physiologic conditions. Cerebral blood flow (CBF) is elevated by increased pCO₂ and decreased pH and/or pO_a. In opposite situations, the CBF is reduced by decreased pCO_a and increased pH and/or pO₂ (Itoh and Suzuki 2012; Grüne et al. 2015). Also, intravascular factors (such as red blood cell flow, leukocyte adhesion, the release of vasoactive mediators, and expression of glycoproteins on the endothelial cells) may be involved in the control of microcirculation (Itoh and Suzuki 2012; Nader et al. 2019). Autoregulation mechanisms help to maintain a relatively steady cerebral blood pressure despite fluctuations in systemic blood pressure (Busija et al. 1981; Baskurt and Meiselman 2003). The brain vascular architecture forms an effective collateral network ensuring permanent blood flow through cerebral capillaries (Lavinio 2022). The incoming oxygenated blood is conveyed by precapillary arterioles and the outcoming deoxygenated blood is drained away by venules (Gillilan 1976; Busija et al. 1981). Dilatation of small arteries and arterioles increases the microvascular pressure gradient and capillary flow (Itoh and Suzuki 2012; Gada et al. 2015; Grüne et al. 2015; Smith et al. 2019; Lavinio 2022).

Intracranial pressure and cerebral circulation

The mammal skull (cranium) represents a firm, non-expandable container with a fixed volume after its sutures and fontanelles are closed (Cushing 1925; Wilson 2016). The intracranial pressure (ICP) is determined by the reciprocal relationship between the volume of intracranial space and the volume of three essential uncompressible elements inside it. They are brain tissue, blood in cerebral vessels, and cerebrospinal fluid (Cushing 1925; Jencean and Ciurea 2008; Wilson 2016). Physiological values of ICP measured/monitored in the lateral brain ventricles in adult healthy dogs in a supine position usually range from 5 to 12 mmHg (Kolecka et al. 2019; Sturges et al. 2019). ICP fluctuates by pulsations (approximately 1 mmHg difference between systole and diastole) and breathing (approximately 5–10 mmHg difference between inspirium and expirium). At the same time, it is significantly influenced by the body's position (horizontal/vertical) and by abrupt changes in intrathoracic or intraabdominal pressure caused by barking, sneezing, straining, running, and jumping. The fluctuating subatmospheric intrapleural pressure helps to keep dilated the intrathoracic parts of caval veins (vena cava cranialis, vena cava caudalis) and supports the return of deoxygenated blood from the brain to the right heart atrium and ventricle (Hibino and Matsuura 1985; Schaller 2004; Zamboni et al. 2012; Wilson 2016; Sturges et al. 2019).

The original Monro-Kellie hypothesis/doctrine, perfected in the year 1925 by Cushing, states that the sum of brain volume plus CSF volume plus cerebral blood volume in an intact skull remains constant (Cushing 1925; Schaller 2004; Wilson 2016; Anile et al. 2021). Therefore, any increase in one of these parameters causes a reduction in one or both of the remaining two. As the venous pressure is much lower than the arterial and the total volume of blood in the intracranial veins and dural venous sinuses exceeds the volume of blood inside the brain arteries, the circulating arterial blood volume is minimally affected by changes in ICP (Wilson 2016; Anile et al. 2021). However, the pulsatile arterial blood flow generates the synchronous pulsations of the brain, which positively influence the intracranial circulation (D'Alecy and Feigl 1972; Baskurt and Maiselman 2003; Driver et al. 2020), and in addition, the intracranial pressure (ICP) provides for the stability of the diameter of thin-walled cerebral veins (Wilson 2016; Anile et al. 2021). The continuous blood flow in the intracranial venous system is maintained by the residual intracapillary pressure (vis a tergo), by the transmission of brain pulsations (vis a latere), and supported by the progressively decreasing (in a vertical position of the dog even negative) pressure in the intracranial venous sinuses, jugular veins, vertebral venous plexuses (vis a fronte) (Schaller 2004; Famaey et al. 2015; Gada et al. 2015). Several experiments in dogs have shown that CSF pressure is higher than the pressure within the DVSs and that an acute elevation of CSF pressure is not followed by increased pressure in the dural sinuses possessing considerably stronger walls (Carreira et al. 2011; Balik 2019; Walsh 2020 et al.). These data imply the presence of a pressure gradient supporting the transport of CSF to the dorsal sagittal sinus and capillaries in its arachnoid villi. It also demonstrates that the CVS, together with diploic and emissary veins, can compensate for the acute changes in CSF pressure when they act as a low-pressure runoff for the subarachnoid veins (Shulman et al. 1964; Anile et al. 2021).

The deoxygenated blood from the brain and skull of mammals is drained to the systemic circulation via the compressible and incompressible venous passages. The blood flow in the collapsible thin-walled tubes located in the superficial layers of the canine neck (e.g., maxillary veins, external and internal jugular veins) is determined by the Starling resistor mechanism in the non-collapsible vessels located inside the rigid vertebral canal (composed of vertebrae, intervertebral discs, and ligaments) by Poiseuille's law

(Holt 1959; Katz et al. 1969; Pfitzner 1976; Luce et al. 1982; Rosar and Peskon 2001; Gada et al. 2015; De Simone et al. 2017).

Poiseuille's law deals with the flow of liquids in narrow tubes with stationary diameters (Baskurt and Meiselman 2003). According to this law, the velocity of the continuous movement of the fluid in the tube (e.g., blood vessel) is directly proportional to the pressure gradient between both ends of the tube and the fourth power of its radius and is inversely proportional to the viscosity of the given fluid and the length of the tube (Pfitzner 1976; Baskurt and Meiselman 2003; Gada et al. 2015).

The Starling resistor mechanism provides not only the stability of blood flow via the thinwalled (collapsible) cerebral draining veins subjected to the influence of the vaccilating ICP, but also prevents the siphoning of blood from intracranial veins into the systemic circulation and excessive transfer of CSF from the intracranial to the spinal compartment (Luce et al. 1982; Wilson 2016; De Simone et al. 2017). The Starling resistor (also termed a choke point) is an area of constriction located at the place of interconnection between the cortical bridging vein and the DSS when the pressure inside the sinus becomes negative due to alteration of the dog's posture from horizontal to vertical. The higher CSF pressure in this situation compresses the junction of the bridging vein with the DSS and hampers venous overdrainage (Luce et al. 1982; Hibino and Matsuura 1985; Famaey et al. 2015; Gada et al. 2015). Due to the accrued backward power, the pressure in the cerebral veins before the constriction point exceeds the pressure of the CSF at the same time. Accordingly, the Starling resistor mechanism helps to maintain the pressure gradient between the liquid compartments in the brain – the arterial pressure is higher than the cerebral venous pressure, the cerebral venous pressure is higher than the CSF pressure, and the CSF pressure is higher than the pressure in the dural venous sinuses (Hibino and Matsuura 1985; Wilson 2016). Vice versa, the Starling resistor mechanism supports CSF resorption into the systemic blood via the capillaries of arachnoidal villi located inside the DSS (Hibino and Matsuura 1985; Bezuidenhout 2013b; Wilson 2016; Proulx 2021). In the vertical position of the canine body (for instance, in a sitting dog), the cervical veins collapse due to the influence of higher external (atmospheric) pressure. By contrast, the rigid vertebral column prevents the collapse of vertebral venous plexuses and spinal epidural veins in the same situation (Luce et al. 1982; Schaller 2004).

The intracranial and intraspinal CSF compartments freely communicate via the foramen magnum (FM) (Evans and de Lahunta 2013). In the vertical position of the dog the CSF pressure becomes atmospheric (zero point) at the level of FM, however, at the caudal end of the spinal dural sack (saccus durae matris spinalis) it can rise to 30 mmHg, due to the hydrostatic pressure of the CSF (Hibino and Matsuura 1985; Schaller 2004). The dura mater in the skull tightly adheres to the inner surface of the skull. In contrast, there is a 2–3 mm thick compartment between the spinal dural sack and the wall of the vertebral canal, filled with compressible adipose tissue and epidural venous plexuses. The volume of this space in big dogs can reach up to 300 cm³ (De Lahunta et al. 2021). It helps to absorb the CSF pressure fluctuations (Schaller 2004). The longitudinal axis of the skull, the vertebral canal, and the centre lines of principal veins in the atlantooccipital region in dogs meet at an obtuse angle (Schliemann 1966). When the dog's head is reclined in the supine position during general anaesthesia for abdominal surgery or imaging procedures, the jugular and vertebral veins become stretched, significantly limiting venous outflow from the head. It may cause intracranial venous congestion, increase the ICP, and damage the brain (Luce et al. 1982; Hibino and Matsuura 1985).

The valveless craniospinal venous system anastomoses with the thoracic, abdominal, pelvic and sacral veins and venous plexuses, thus providing a route for intraspinal or intracranial propagation of cancer cells or bacterial emboli and spread of neoplastic or septic metastases to the brain or spinal medulla (Gowin 1983).

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