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The anatomy of collateral venous flow from the brain and its value in aetiological interpretation of intracranial pathology

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Abstract For more than a century, available data concerning collateral venous outflow from the brain have received insufficient attention, as existing theories did not assign practical importance to them. Ideas concerning arterial blood supply and circulation of cerebrospinal fluid were considered more relevant. But available data afford a schematic model of cerebral venous outflow that does have important pathophysiological consequences. Principal outflow through the internal jugular veins can be substituted completely by the large vertebral plexuses, through communications at the cranial base. Emissary veins of the skull vault are small and few in number. Outflow from the deep venous system through the great vein of Galen can be substituted by choroidal, thalamic and striate anastomoses toward the basal vein. So-called intracerebral venous anastomoses through the centrum semiovale towards the convexity are nonexistent or negligible. Instead, a venous watershed exists separating paraventricular white matter from a layer of subcortical white matter. In most infants, the cavernous sinus is not yet connected to the cerebral veins. Once such communications have been formed, important collateral pathways exist through basal and Sylvian veins via the cavernous sinus to the pterygoid plexuses. Simultaneous hindrance of principal and collateral venous outflow will lead to elevated venous pressure and eventual insufficiency of cerebral blood flow (CBF). This will cause increased intracranial pressure, and ventricular enlargement due to periventricular atrophy. The slow phase of the two-compartment model of CBF coincides with the paraventricular white matter area of the deep venous system. In the neonate CBF was found to be still very low, and in the two compartments CBF increases at a different rate to a maximum in childhood. In hydrocephalus, measurement of CBF in the slow deep compartment, rather than the fast cortical one, will be most informative.

Key words Cerebral veins · Cerebral blood flow · Cerebrospinal fluid pressure · Hydrocephalus

Introduction

Interpretation of observations is often based on ideas which became accepted long ago. A principal scientific goal is the achievement of knowledge of fundamental determining conditions for various events and processes [1]. Science is not just a collection of concrete data, but includes abstract causal theories which should have predictive power. Relationships between observation and explanation change when a different set of determining conditions is postulated. In the natural sciences, truth is ultimately determined by the structures and

mechanisms of nature; they are not affected by dictum, vote, tradition or fashion.

Inadequate descriptions of collateral venous pathways have influenced the development of explanatory concepts in neuropathology. Charcot [2] suggested a topographical distribution of brain diseases (haemorrhage and encephalomalacia) according to the distribution of arteries; but he paid little attention to veins. Until the end of the 19th century some anatomy texts still provided a careful description of cranial and vertebral venous anatomy [3–7]. But the teachings of Charcot were very influential in regard to the initial structuring of neurological explanation and when, in the first half of the 20th century, textbooks became standardised, they gave extensive accounts of the cerebral arteries, with only a brief and incomplete description of the anatomy of the cerebral veins and vertebral venous plexuses. Insufficiency of cerebral blood flow is interpreted as disturbance of arterial inflow. That primary insufficiency of venous outflow from the brain causes a similar reduction in arterial inflow does not seem so self-evident; yet it does not require experimental proof.

In the latter half of this century, when angiography largely replaced dissection, extensive radiological descriptions of cerebral venous anatomy appeared [8–10]; however, they deal mostly with topographical landmarks, pathological displacements caused by masses, and anatomical variations. An unjustified belief that practically everywhere cerebral veins have sufficient anastomoses had evolved. This caused venous anatomy to seem irrelevant to the aetiology of disease. In the course of a century, for all practical purposes some vague model has emerged, in which cerebral venous outflow takes place through the lateral sinuses, which presumably can be substituted by scalp veins, and in which the great vein of Galen receives all blood from the deep venous system, but allegedly can be replaced by so-called "intracerebral anastomotic veins" ("medullary" or "transcerebral" veins) through the centrum semiovale. Adequate schematic description of the anatomy of collateral venous pathways, however, is physiologically most important, in regard to sufficiency or insufficiency of blood flow under pathological or experimental conditions. But results of anatomical investigations thus furnishing explanatory and predictive power have remained outside the mainstream of the literature cited and have thus not become part of general knowledge.

The vertebral plexuses and azygos system as a collateral pathway for cerebral venous outflow

In an unfinished serial work appearing around 1830, Breschet [11] described the large vertebral venous plexuses within and around the vertebral column. He also demonstrated their segmental connections, through the intervertebral foramina, with the ascending lumbar and azygos veins. During the 19th century his work achieved a prominent place in French and German anatomy texts. In 1837 Cruveilhier [4], the author of a number of important volumes on anatomy and pathology, elaborated on the work of Breschet. He considered the lumbo-azygos system to be part of the anterior external vertebral plexus. He decided that there are no valves in the azygos veins. The bilateral ascending lumbar vein he considered a lumbar azygos vein. The vertebral vein (in the transverse processes of the cervical vertebrae) and the ascending cervical vein he saw as azygos veins of the cervical region, because they also collect outflow from the vertebral plexuses. Experimenting on dogs, he found that after ligation of the jugular veins the vertebral plexuses would serve as a sufficient collateral outflow system from the cranium.

The exact beginning and end of each vessel he considered unimportant; near multiple junctions they may vary without affecting function.

Cruveilhier drew attention to analogies between the vertebral plexuses and the cranial sinuses. The anterior internal vertebral plexus would be represented by the superior and inferior petrosal, cavernous and lateral sinuses, with the transverse connections of the intercavernous sinuses and the basal plexus between them. The posterior internal vertebral plexus would be united in the midline and be represented by the superior sagittal, straight and occipital sinuses. Several bilateral foramina in the cranial base would be analogous to the intervertebral foramina, establishing communication between internal and external veins.

At the end of the 19th century Poirier [7] still gave an extensive description of the vertebral venous system. He mentioned that the vertebral plexuses can serve as a sufficient collateral pathway for the superior and inferior vena cava. According to Poirier there are valves at certain locations in the vertebral venous system. He described the collecting channels (such as the azygos and longitudinal cervical veins) in more detail. He found valves in the azygos vein always to be insufficient.

In 1940 Batson [12] explained paradoxical metastases toward vertebrae and brain as the result of reversal of flow in the internal vertebral plexuses during moments of pressure elevation in the thoracoabdominal cavity. In 1944 [13] he also described the vertebral vein system as a pathway of collateral outflow from the brain when the jugular veins or the superior vena cava are occluded. He stated that "The so-called sinuses on the skull base, such as the basilar, the cavernous, etc., are really plexiform networks which communicate with similar meshworks within the bones of the skull base, and the pterygoid plexus of either side, below the skull, and the veins of the orbit. Every nerve and arterial foramen in the skull base transmits some veins in this network. From a corrosion preparation, one could de-

scribe this intracranial, extracranial, and intraosseous network as a single venous plexus in which the bodies of the sphenoid and the occipital bones are embedded." The emissaries of the skull *vault* he considered to be quantitatively insignificant. He examined the rhesus monkey, rabbit, dog and cat and found the vertebral plexuses to be at least as large, in proportion, as in humans.

Batson examined only living animals and human cadavers. Anderson [14] supplemented these investigations by demonstrating the vertebral plexus (with azygos and ascending lumbar veins) in living human patients, by means of Diodrast injections into the femoral vein during moderate abdominal compression. The cervical venous plexuses could be shown by injecting the cephalic vein of the arm while the subject attempted a forced expiration with the glottis closed.

In his Caldwell Lecture, published in 1957, Batson [15] gave a historical survey of ancient and most recent work concerning the vertebral vein system. He pointed out that Breschet's work had become forgotten, presenting as an example an illustration from a 1903 article by Osler, a diagram of the supposed routes of collateral circulation in thrombosis of the superior vena cava. This picture had become well known, as it had often been reproduced in textbooks. Batson pointed out that the vertebral plexuses had been omitted (and even the connection of the inferior vena cava with the azygos system was not shown). Batson's work has been related mostly to the subject of paradoxical metastases, which assumed the possibility of reversal of blood flow. He showed less interest in collateral venous flow from the brain, for which the assumption of flow *reversal* in a *valveless* vertebral system is not required. Thus, in his 1957 survey, he mentioned Cruveilhier's experiments with ligation of jugular veins in dogs. But he did not refer to several recent works on the significance of the vertebral plexuses for the collateral outflow from the brain. For instance, Gius and Grier [16] demonstrated the adaptation of these collateral channels after bilateral radical neck dissection with excision of the jugular veins in patients. Sugarbaker and Wiley [17] found that, after bilateral jugular resection in patients, compression of the muscle bed in the neck caused high elevation of cerebrospinal fluid pressure (CSFP), which indicates that to a great extent collateral venous flow takes place via the *external* vertebral plexuses. Andersen et al. [18] presented a case in which a syndrome of superior caval vein obstruction (involving also the part of the vena cava connecting the azygos vein to the heart) was relieved by surgical anastomosis of the azygos vein to the right auricle. A few years after Batson's review lecture, the anatomist Clemens [19] published his investigations of the venous system in 30 normal vertebral columns (together with cranial base and pelvis) removed from adult human cadavers. The findings of Breschet were again confirmed. Dilenge and Perey [20] performed angiographic studies in monkeys and subsequently in man, demonstrating that in the upright position the vertebral plexuses, rather than the jugular veins, become the preferred pathway of venous return.

As mentioned, Batson [13] described the connections between intra- and extracranial veins at the cranial base as a single large venous plexus, and this appears to be the most practical view. The connections between the cranial and vertebral venous systems have been investigated by several workers [4, 7, 19, 21]. They can be summarised as follows:

1. The suboccipital venous plexus is the cranial beginning of the *posterior external* vertebral plexus between the dorsal muscles; it is connected to the sigmoid sinuses via the mastoid and condylar emissaries.
2. The *posterior internal* vertebral plexus receives blood from the occipital sinus and thus from the confluens sinuum (torcular). The torcular is often plexiform and asymmetrical, and when one lateral sinus is narrow or absent, the occipital sinus is larger than usual; and in neonates the occipital sinus is very large [22, 23].
3. The *anterior internal* vertebral plexus is a continuation of the basal plexus, which lies on the clivus and connects the inferior petrosal sinuses and both cavernous sinuses.
4. The *anterior external* vertebral plexus is a continuation of the large pterygoid plexuses, which receive blood from the cavernous sinuses and, via the middle meningeal veins, from the superior longitudinal sinus. The pharyngeal venous plexuses may play a role in the anterior region that should not be ignored.

Besides these affluent pathways the *outflow* channels of the vertebral plexuses can also be summarised:

1. In the *thoracolumbar* area, outflow from the vertebral plexuses takes place via the lumbo-azygos system, which also forms a collateral channel between the inferior and superior venae cavae. Valves in the azygos system are rudimentary and nonfunctioning. Obstruction of the superior vena cava cranially from the azygos vein is tolerated well, but obstruction involving the connection of the termination of the azygos vein to the heart leaves collateral flow only toward the inferior vena cava, which results in insufficient cranial venous outflow [18].
2. In the vertebral plexuses of the *cervical* area, the longitudinal collecting channels besides the internal jugular vein on either side are the deep cervical vein posteriorly between the muscles, the vertebral vein (through the transverse processes of the cervical vertebrae) and, subcutaneously, the external jugular vein. These veins join the subclavian and internal jugular veins in forming the brachiocephalic vein.

The cavernous sinus and basal vein as collateral pathways

During the 19th century, the Sylvian vein was described in various ways, for instance as ending in the great vein of Galen via the basal vein [3], in the superior sagittal sinus [4], or in the cavernous sinus directly or via the sphenoparietal sinus [5], and as connecting the superior sagittal sinus to the superior petrosal or cavernous sinus [21], or as consisting of two separate veins, the superficial and deep Sylvian vein [6], the latter originating on the insula.

These seemingly contradictory findings clearly indicate some peculiar problem. A solution can be derived from the meticulous investigations of Padget [23], published in 1956. She found that prenatally the cavernous sinus drains only the ophthalmic vein via the inferior petrosal sinus to the internal jugular vein, while the cerebral veins have no connections with the cavernous sinus, but converge into the transverse sinus. In most infants this is still the case, but in the typical adult configuration connections to the cavernous sinus have developed via medial extension of the sphenoparietal and superior petrosal sinus. The superficial Sylvian vein is then connected to the cavernous sinus directly or via the sphenoparietal sinus, and communicates with the superior sagittal and transverse sinus via the anastomotic veins now named after Trolard and Labbé. In most mammals the transverse sinus continues into the external jugular vein via a temporal foramen, while the internal jugular vein becomes attenuated [23, 24].

Problems similar to those concerning the cavernous sinus also arose in the historical development of ideas about the connections of the basal vein. In 1868 Trolard [21] stated that the basal vein springs from his *grande veine anastomotique* (i.e. the superficial Sylvian vein) and courses towards the great vein of Galen or the straight sinus. He mentions that, along this course, a branch connects it with the choroidal vein. In 1874, the influential Duret [25], working for Charcot (and chiefly describing arteries), mentioned the basal vein as an important anastomosis between Trolard's *grande veine anastomotique* and the straight sinus, but did not mention its connection to the choroidal vein.

In 1884 Browning, as cited by Merkel [6] and Hédon [26], gave the name *vein of the inferior horn* to this tributary of the basal vein. But the part of the basal vein from the region of the insula to the junction with the anterior cerebral vein he called vena fossae Sylvii (now: deep middle cerebral vein) and he wanted to distinguish it strictly from the vena cerebri media (now: superficial middle cerebral vein).

Hédon [26] named the *inferior striate veins* as tributaries of the basal vein. He stated that the basal vein is also directly or indirectly connected to the cavernous sinus, and receives a large branch from the choroidal plexus and the ventricular walls of the temporal horn, and that at its origin this choroidal tributary anastomoses with the choroidal vein which forms the internal cerebral vein.

Bedford [27] ligated the great vein of Galen in dogs, and found that collateral circulation developed through striate, thalamic and choroidal anastomoses toward the basal vein. He emphasised that in the dog the main outflow of the basal vein is towards the superior petrosal sinus, but wrongly assumed that in man outflow from the basal vein is exclusively towards the great vein of Galen.

Padget [23] found the inferior ventricular vein to originate from choroidal and hippocampal tributaries and to form a large, constant tributary of the basal vein. The large embryonic choroidal plexus is drained at one end by the inferior choroidal vein, which becomes incorporated in the basal vein and is, within the plexus anastomotically connected to the superior choroidal vein at the other end the first large tributary of the internal cerebral vein. The tributaries of the basal and internal cerebral vein ultimately complement each other. The striate anastomoses she considered less common.

Padget gave the name *lateral mesencephalic vein* to an anastomosis between basal vein and superior petrosal sinus present in man and dominant in most mammals. Ono et al. [28] later found the lateral mesencephalic vein in all human brains they examined. Padget added that, anteriorly, the basal vein connects with the superficial Sylvian vein by way of the deep Sylvian vein. Because the cerebral veins are valveless, the direction of flow can be reversed.

Thus, after the connections of the cerebral veins to the cavernous sinus have been formed, a collateral pathway towards the pterygoid plexuses exists, supplementing the cerebral venous outflow via the posterior cranial fossa. And the basal vein has then become a collateral pathway for the internal cerebral vein and great vein of Galen.

The essential collateral routes have been discussed. The basal vein also receives tributaries from the mesencephalon, diencephalon and, via the anterior cerebral vein, from the cingulate gyrus. Because of these superficial branches, Duvernoy [29] considered the basal vein to be part of the superficial cerebral venous system. The great vein of Galen also receives superficial veins, from the posterior part of the cingulate gyrus, visual cortex, and anterior cerebellum.

The venous watershed in the centrum semiovale

Charcot [2] defined a *central system* of striate and choroidal arteries, and a *cortical system* with very thin arteries serving the cerebral cortex and, as so-called medullary arteries, supplying the underlying white matter of the centrum semiovale. To provide support for Char-

cot's ideas on the distribution of brain diseases, his young assistant Duret [25] carried out investigations of the anatomical distribution of cerebral arteries, attempting to correlate them with the autopsy findings communicated to him by Charcot. Duret gave only a brief description of cerebral venous anatomy, taken from other authors, and added this to his extensive description of the arterial supply of the brain. Charcot himself did not describe venous anatomy, as he made no use of it to explain pathological findings. Duret assumed that within the centrum semiovale medullary veins from the cortex would anastomose with branches of the subependymal veins draining the ventricular walls. As support for this opinion he mentioned a Latin thesis by Ekker (misspelled Ecker, and therefore impossible to find under that name). But, remarkably, he also assumed that the striate veins drain only towards the ventricular side, and he omitted to mention the connection of the choroidal vein to the basal vein, already described by Trolard [21]. Thus the outflow of striatal, choroidal and ventricular veins would be towards the great vein of Galen, with a (hypothetical) collateral outflow only through the centrum semiovale.

In 1888, Hédon [26] graduated at Bordeaux with a thesis, reprinted the same year in Paris as a monograph. In the introduction he wrote that anatomical handbooks describe the arterial supply of the brain, due to the work of Duret, but ignore the venous part of the circulation absolutely. He added that he would limit himself to a description of venous anatomy, and that undoubtedly applications to pathophysiology would be found later.

As anastomoses from the ventricular veins to the basal vein, Hédon found and named the inferior striate veins (communicating with the superior striate veins). Citing Browning, who had recently carried out investigations in Leipzig, he also mentioned the inferior choroidal vein and the vein of the temporal horn (inferior ventricular vein) as anastomoses toward the basal vein. Hédon emphasised that he could not confirm the existence of the intracerebral anastomoses through the centrum semiovale towards the cortical veins suggested by Duret. Instead, he described anastomoses only in Charcot's central vascular system. Thus, when the internal cerebral vein is blocked, the superior choroidal vein may *receive* blood near the foramen of Monro and, through its anastomoses with the inferior choroidal vein, this blood reaches the basal vein. Many years later, the importance of the forgotten work of Browning and Hédon was confirmed by Padget [23], who considered her own contributions to supplement theirs.

In 1939, Schlesinger [30] again claimed the existence of what he called "intracerebral anastomotic veins" throughout the centrum semiovale, which would make obstruction of the deep venous system practically impossible. This was held to be proof that such an obstruction could not be the cause of hydrocephalus, and thus Schlesinger's became the most cited paper on cerebral venous anatomy. Yet, as Padget [23] pointed out, these anastomoses had apparently not been seen. The vessels in three monkeys and two human brains that Schlesinger observed can be interpreted as congested nonanastomotic radial veins, or as arteries filled retrogradely [31, 32]. Schlesinger cited Pfeifer, who claimed to have demonstrated anastomotic medullary veins, but around the time Schlesinger's article appeared it was pointed out that Pfeifer had described arteries as veins and vice versa [33]. Later publications [34–36] have been cited as supporting Schlesinger's idea of intracerebral anastomotic veins. However, in 1958 Ferner [34] investigated the internal cerebral veins and their tributaries by injection of 87 adult brains. He found a "venous watershed", with the paraventricular veins ending in the subependymal veins and the venous outflow from the cortex and subcortical white matter directed toward the convexity. It may be mentioned that in 1979 Tschabitscher [37] claimed the existence of a similar venous watershed in the cerebellum. Kaplan [35] made injection studies and, although he introduced the term *transcerebral veins*, he explicitly stated that anastomoses directly connecting the veins of the cerebral cortex with the Galenic system had not been conclusively demonstrated. Hassler [36] injected the great vein of Galen in 35 human brains with a contrast medium. In brain slices very thin radiating vessels could be observed, a few thousand in each hemisphere. The low viscosity, high pressure and long injection time were designed to reach even the smallest veins, but evidently retrograde filling of the very thin arteries which descend from the cortex into the white matter is similarly obtained. Hassler regarded the area of capillary filling (appearing whiter on radiographs of the brain slices than the surroundings) as indicating the region served by the deep cerebral veins. He considered the possible venous anastomoses too small to be functional, and expressed the belief that occlusion of the great vein of Galen will generally lead to serious effects. His diagrams of the maximum extent of the deep cerebral venous system in the centrum semiovale are clear illustrations of Ferner's "venous watershed". Ono et al. [28] published an extensive study of the deep venous system of the brain and cited a number of reports of serious consequences resulting from obstruction of the veins constituting it.

As regards the venous watershed in the centrum semiovale, the presence or absence of veins traversing the corpus callosum may be irrelevant, because the external branches will be drained by the anterior and posterior pericallosal veins, towards the basal vein and great vein of Galen respectively, and thus outflow will remain within the deep cerebral system. And the intracerebral anastomoses through the thalamus and striatum towards the basal vein are fundamentally different

from the "intracerebral anastomotic veins" through the centrum semiovale assumed by Schlesinger and Duret.

The existence of a periventricular *arterial* border zone has been refuted, as ventriculofugal arteries could not be shown in recent investigations [38]. This arterial borderzone had been used instead of the venous watershed to explain periventricular haemorrhage in premature neonates.

Collateral venous pathways and predictive explanation

The anatomy of collateral venous pathways indicates the location of areas where principal and collateral flow can be impaired simultaneously.

The most obvious example is the posterior cranial fossa, where flow both through the compressible sigmoid sinuses and towards the vertebral plexuses can be impeded by a tumour, a haematoma, meningeal fibrosis and hypoplasia of the chondrocranium. This will cause increased flow through the cavernous sinus, provided the latter has become connected to the cerebral veins. In most infants and some adults this is not the case, so that vulnerability is increased. On the other hand, other routes for venous outflow from the superficial veins may exist in the infant, namely via the occipital sinus, which is still large, and through the open cranial sutures via scalp veins, although these will always be insufficient. Venous compression by posterior cranial fossa tumours was documented by Kinal [39].

The deep venous system is not primarily vulnerable at the great vein of Galen, because channels towards the basal vein can afford sufficient collateral flow. The most vulnerable area is around the junction of internal cerebral and superior choroidal veins, because at this point principal flow and collateral flow can be impeded simultaneously. Cranial venous outflow can even be hindered at a distance, when obstruction of the superior vena cava includes the collateral outflow through the azygos vein [18].

I have discussed how disturbances of cerebral venous outflow as a whole, or from the deep cerebral system alone, can cause ventricular enlargement and elevation of CSFP [31, 32]. The CSFP depends on a hydrostatic-osmotic pressure equilibrium with choroidal capillaries and veins. Cranial enlargement will occur when CSFP greatly exceeds atmospheric pressure, while the cranial sutures are still open. The cerebral surface retains its relationship to the enlarging cranium and the size of the lateral ventricles increases. But insufficient deep venous flow also causes periventricular atrophy, manifest as ventricular dilation, when the cranial vault is not enlarged. Unlike the cranial vault, the chondrocranium does not enlarge and may even be too small.

It may be asked whether increased venous pressure causes increased CSFP, or vice versa. Shulman and Ransohoff [40] found elevation of superior sagittal sinus pressure (SSSP) in hydrocephalic infants and, when the elevated CSFP was lowered by letting some CSF escape, the SSSP fell. They postulated that elevated CSFP causes partial collapse of the dural sinuses at or near the point of venous outflow from the skull. But lowering the SSSP by letting CSF escape does not necessarily mean that the CSFP was raised independent of venous obstruction: when only the deep venous system is primarily obstructed, the resulting CSFP elevation may increase SSSP secondarily by compressing the sigmoid sinuses.

As its therapeutic effect, shunting of CSF causes improvement of cerebral blood flow (CBF), through increased perfusion pressure (CSFP determines outflow pressure at the junction of dural sinuses and the compressible subarachnoid veins). But side-effects result from brain swelling, because venous pressure is still elevated and maintains elevated interstitial fluid pressure within the brain, while counterpressure of CSF against the brain has diminished [31]. These effects are particularly marked when shunting of CSF is performed in the presence of very high venous pressure caused by arteriovenous communications. Embolisation of the arteriovenous connections, however, will treat the cause of the venous hypertension and avoid these side-effects caused by shunting of CSF. This may be regarded almost as an experimental model confirming the mechanisms described [41, 42].

Venous anatomy and the two-compartment system of CBF

The venous watershed between the periventricular and subcortical white matter is not the only indication of the separateness of the deep cerebral venous system. CBF measurements have shown isotope clearance curves consistent with a fast phase representing flow in grey matter, and a slow phase representing white-matter flow. This would be compatible with the work of White and Greitz [43], who found that the subependymal veins exclusively serving white matter showed the slowest filling during cerebral angiography.

In CBF measurements attention has been paid mainly to the fast phase, as representing cortical flow. However, in separate obstruction of the deep venous system the slow phase of the clearance curve will be primarily affected, and thus a CBF measurement of only the fast phase might mistakenly be considered normal.

The methods for measuring CBF are still surrounded by many problems, particularly in the neonatal period. In childhood, CBF amounts to almost half the basal cardiac output, and in the adult to almost one-fifth. In 1956, Kety [44] reviewed values of CBF and cerebral oxygen consumption in subjects from 5 to 93 years old. They were found to be quite high in childhood, with a rapid fall through adolescence, followed by a more

gradual reduction throughout adult life. A similar course of the continuous loss of cortical neurones was shown. An extrapolation of these data suggests that CBF would be highest in neonates. However, in contrast, Suzuki [45] reported CBF to be lowest in the neonatal period. He used the intravenous xenon-133 clearance method, in 80 normal children between 4 days and 15 years of age, and applied a two-compartment analysis. From birth on, blood flow increased. After the 1st week of life, average flow in the white matter quadrupled, reaching its highest level at 6–8 month of age, while average flow in grey matter more than tripled, towards a maximum at age 3–4 years. Suzuki considered it likely that the increase in white-matter blood flow during the first few months of life represented mainly a response to the increasing metabolic demands associated with myelination, whereas grey-matter flow reflects the establishment of higher cortical functions related to standing, walking and speaking until 3–4 years of age.

Mabe et al. [46], using a similar method, found differences in post-shunting intelligence and development quotients in hydrocephalic children, correlating with white-matter flow but not with cortical flow. They suggested that this can be explained by impairment of white-matter communicating fibres in children with hydrocephalus.

Kimura et al. [47] studied seven patients with normal-pressure hydrocephalus after subarachnoid haemorrhage, who improved after shunting, measuring CBF by stable xenon-enhanced CT. This gave better spatial resolution than other methods, improving blood flow measurement in the periventricular white matter. In all areas, CBF improved after shunting; in the periventricular white matter it even returned to normal. Other investigators had not always found restoration of CBF when clinical improvement occurred after shunting in normal-pressure hydrocephalus. One reason for this may be that the methods used reflect cortical CBF only, while blood flow in the periventricular white matter is more relevant.

Discussion

The anatomy and pathophysiology of the cerebral venous system have long been neglected, due to theoretical preoccupations with arterial blood supply and circulation of CSF. According to philosophers of science, all interpretations of data are hypotheses, which are used until they can be replaced. Interpretations used for a long time can become the basis of a vast literature, in which the postulates are taken for granted, while proof remained scanty. To make critical discussion possible, the attempts at proof they consider best must be cited specifically by anyone adhering to them. Uncritical, generalised reference to the literature can prevent open, rational discussion. Strict demands must be made not only on new interpretations, but also on old ones, which is much more difficult because of habit and loyalty.

The territory of the deep cerebral venous system offers an extremely interesting field of investigation, because as well as the periventricular white matter it includes the basal ganglia and limbic system. Important new techniques have become available to neuroradiologists, for example: various methods of venography, improved methods for assessing flow in the deep cerebral venous system, and demonstration of brain oedema and periventricular pathology of the white matter in the living subject by means of CT and MRI. Great opportunities have opened up for the acquisition of new insights.

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