Red Cerebral Veins

A Report on Arteriovenous Shunts in Tumors and Cerebral Scars*

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There is no firm anatomical evidence for the existence of direct connections between arteries and veins in the normal human brain. But at operation for a variety of lesions, cerebral veins are sometimes seen to be partly or wholly filled with a stream of red arterial blood. Such red veins are usually single or few in number, remaining restricted to the area around the lesion, and are readily distinguished by their contrasting color from nearby normal-appearing veins.6

In addition to their obvious presence in arteriovenous malformations, where they have long been familiar to neurosurgeons, red cerebral veins have now been noted in association with benign as well as malignant brain tumors, cerebral cysts and scars, and local areas of epileptogenic cortex.

Penfield15,16 first reported this phenomenon in patients undergoing operations for epileptic seizures, during which veins were temporarily filled with red blood after spontaneous or induced attacks. He interpreted this as a postictal reactive hyperemia following upon the relative ischemia of the cerebral cortex during the seizure. In studies with von Sántha and Cipriani18 on changes in local cortical blood flow, he presented evidence to support this view.

A distinction was made between the appearance of red blood in a vein draining a local region involved in focal epileptic discharge and the widespread redening of cortical veins following a convulsion. Erickson4 was able to reproduce this generalized reddening in experimental animals by temporary vascular or respiratory obstruction.

In two examples of red veins near cystic scars, also described by Penfield,16 this interpretation of postictal hyperemia did not seem applicable, since the red color of the veins was observed as soon as the dura mater was opened, and appeared independently of seizures.

In their discussion on early venous filling in angiography of malignant tumors, Tönnis and Walter27 mentioned, without further comment, that they had observed “das helrote Blut in den Venen im Bereich eines Glioblastoms.”

In 1954, two striking examples of red cerebral veins—one in a vascular glioma (Case 5) and another associated with a parietal infarct from carotid thrombosis (Case 9)—aroused renewed interest in this problem. Over the past 10 years similar examples at operation have been noted in detail and photographed in color. Since the original observations of Penfield, there has been little reference in the neurosurgical literature to red cerebral veins. We wish to direct attention to their occurrence, to describe selected examples and to consider these in relation to regional changes in blood flow through the brain.

Circumstances in which we have observed red veins can be outlined for the purpose of discussion into two main groups.

(1) Structural Arteriovenous Shunts. These lesions, with abnormal blood vessels providing direct connections between arteries and veins, include arteriovenous malformations, hemangiomas, vascular meningiomas, vascular gliomas and some metastatic tumors.

(2) Metabolic Arteriovenous Shunts. Red

Received for publication April 8, 1964.

* Reported in part at the 14th meeting of the Canadian Neurological Society, Winnipeg, Canada, June 15, 1962.
blood appears in veins that drain a local region of brain that utilizes oxygen inadequately.

This may be transient, as in the post-convulsive period, or present as a persistent change when red veins drain a local zone of permanently abnormal tissue, such as a necrotic tumor, an infarct, a scar or the region about a cyst.

Case Reports

1. Structural Arteriovenous Shunts

Case 1. G.S., aged 29 years, 3 days before admission had sudden headache, weakness and numbness of the left arm, and poor vision. Examination showed stiffness of the neck, a left hemianopia and impaired sensibility of the left side of the body. On rapid serial angiographic films, an early-filling vein in the posterior parietal region leading from a small spiral vessel indicated an arteriovenous malformation (Fig. 1).

At operation (October 1961) when the tense dura matter was reflected a vein of medium size and several of smaller size in the midparietal region were bright red. They lay on the cortex which appeared yellow and bulging over an area 5 cm. in diameter. Four cc. of old fluid blood were aspirated from this region and a moderate amount of blood clot was removed through a small incision in the overlying thin cortex. The veins below and in front of the hematoma were of normal color.

Comment. In this instance, in addition to the small arteriovenous communication there was undercutting, thinning and compression of the cortex by the intracranial hematoma. These features, characteristic of an angiomatous shunt, will also be apparent in other types of lesions to be described subsequently in which the presence of a structural arteriovenous connection was less obvious.

Case 2. E.W., aged 45 years, suffered from sub-occipital headaches and vomiting for a month. She showed cerebellar signs and early papilledema. Vertebral angiography showed a vascular blush in the distribution of the left posterior inferior cerebellar artery. No definite venous drainage could be made out on the angiogram.

At operation (December 1960) a small well-circumscribed hemangioblastoma was present in the left cerebellar hemisphere near the midline. Lateral to it was a cyst containing 10 cc. of yellow fluid under moderate pressure.

A vein coming from the superior pole of the tumor was red. Another on the opposite cerebellar hemisphere which appeared to drain the deep aspect of the tumor was also red. Two smaller veins from the inferior pole of the cerebellum and cerebellar tonsils running up in the midline were blue.

Comment. This vascular tumor was also associated with a cyst which had produced undermining and compression of the cerebellar cortex.

Case 3. U.C., aged 59 years, had progressive difficulty in speech, right-sided weakness and hemianopia for 2 months.

On the serial angiogram the arterial phase showed abnormal blood vessels in the sylvian region (Fig. 2A). The later arterial phase showed early filling of two veins corresponding to those noted to be red at operation. Another film clearly demonstrated laminar flow for a distance of several cm. along the large rolandic vein (Fig. 2B).

At operation (October 1961) abnormal tortuous vessels formed a tangle mass over the temporal cortex (Fig. 4). The vein of Labbé and a large rolandic vein were red. A cyst extended deep to the cortex in the posterior sylvian region. The tumor was a malignant glioma.
Comment. In this arteriovenous shunt in a malignant glioma, the veins selectively draining the tumor filled early on the angiogram and appeared red at operation.

Case 4.* R.R., aged 60 years, had impaired memory for 5 months and confusion with dysphasia for 3 months.

The angiogram showed a highly vascular tumor supplied mainly from the middle cerebral artery (Fig. 3). Two prominent veins filled in the early arterial phase and corresponded to those observed later at operation. The tumor, containing small cysts and necrotic tissue, was histologically a glioblastoma multiforme.

The left parietal region was exposed at operation (October 1960) by our colleague, Dr. Gilles Bertrand. The bulging parietal convolutions were widened and covered with many small tortuous vessels (Fig. 5). The large veins in front of the tumor were blue; those draining it were red. One red venous tributary was seen to join with a larger blue vein to form a main venous stem containing separate streams of red and blue blood.

Comment. Here again veins which filled early on the angiogram appeared red at operation. This example illustrates the focal nature of the shunt through the arteriovenous communications within the tumor, so that nearby cortical veins, one draining normal cortex and the other the tumor, showed a distinct difference in color.

Case 5. Y.C., aged 41 years, had focal attacks on the left side over a period of 5 years. On examination cortical sensation was impaired in the left hand and face. When the parietal region was exposed at craniotomy (March 1954) a fine

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* This case was reported briefly in a study on radioisotopic scanning for intracranial vascular lesions.
feltwork of blood vessels covered the postcentral convolutions. Two large veins containing red arterial blood drained this region and continued towards the midline. The veins anterior and posterior to the tumor appeared blue. The tumor was tough in texture and contained granules of calcium and a few small cysts. During removal, the lower margin was first separated from its pial blood supply, whereupon the red veins became blue.

On one of the limited angiograms, the inverted Y-shaped vein which appeared red at operation was more densely opacified than the veins near it.

Comment. In this case the absence of a vasoclar blush on the angiogram might have been ascribable to the small number of films. There was evidence of early filling of the veins which appeared red at operation. The change in venous color after the first stage of removal indicated that the pial arteries and not the deep arterial vessels were the source of red blood in the superficial draining veins.

Case 6. W.C., aged 34 years, had a 10-year history of focal seizures, left hemianopia and sensory loss over the left side of the body.

At operation (November 1956) when the tense dura mater was reflected, the convolutions appeared swollen and suffused. Veins on the inferior and superior margin of the tumor were red.

A large red vein from the superior margin was joined by a blue vein of the same size which drained the cortex medial to the tumor (Fig. 6). These two veins showed a striking contrast in color as they continued on the inner surface of the dura mater running toward the longitudinal sinus.

Within the tumor, a malignant glioma, there was a large cyst containing 30 cc. of clear yellow fluid.

Comment. In this example the contrasting red and blue veins drained closely adjacent regions of tissue.

Case 7. T.T., aged 50 years, complained of headache, blurred vision and vomiting for several weeks. On admission she had bilateral papilledema and left homonymous hemianopia.

Roentgenograms revealed a lesion in the right upper lobe of the lung. Serial angiography showed in the right occipital lobe a small collection of abnormal vessels filling toward the end of the arterial phase. A vein draining toward the sagittal sinus from the region of the tumor filled earlier than the adjacent parieto-occipital veins.

At operation (June 1963) a metastatic carcinoma presented on the surface of the right occipital lobe, with most of it lying within the white matter (Fig. 7). A large red vein extended from the upper edge of the tumor toward the midline. Laminar streaming of red and blue blood could be seen where a second smaller red vein joined a normal larger vein.

Analyses of blood taken by direct puncture of the large red vein from the right transverse sinus, and from the radial artery, are shown in Table 1.

Comment. The impression that the blood contained within the red cerebral veins is arterial in nature is confirmed by the blood-gas studies. Although the vascular blush as seen on the angiogram was not so conspicuous as that in some of the examples described above, the presence of some ab-

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**Fig. 4. Case 3.** Photograph at operation, showing the red draining veins which corresponded to the early filling of veins on the angiogram in Fig. 2.

**Fig. 5. Case 4.** Note the red vein on posterior part of the tumor and another on anterior margin of the tumor which joins the blue venous vessel.

**Fig. 6. Case 6.** Contrasting red and blue veins running together on the cortex and inner surface of the dura mater toward the sagittal sinus.

**Fig. 7. Case 7.** Metastatic carcinoma drained by a red vein from which samples of blood were drawn for the gas analysis shown in Table 1.

**Fig. 8.** appears on page 320

**Fig. 9. Case 8.** Photograph at operation showing vessels exposed on the cortical surface. Stimulation points 1 and 2 indicate motor cortex. Compare with Fig. 8.

**Fig. 10. Case 9.** Photograph of red veins draining an area of atrophic brain in a patient with occlusion of the internal carotid artery.
normal vessels together with the early venous filling suggests some degree of direct or structural arteriovenous communication in relation to this metastatic carcinoma.

2. Metabolic Arteriovenous Shunts

Case 8. E.J., aged 44 years, had a feeling of pins and needles in the right upper limb for several years. Just before admission a generalized seizure was followed by weakness of the right arm. An expanding lesion in the left parietal lobe (Fig. 8) was confirmed at operation (October 1961). There was slight bulging and pallor associated with lack of small vessels in the convolutions between the two large veins. This region was drained by a third smaller vein which was bright red (Fig. 9). Another slender vein behind the large posterior vein was also red.

Beneath the pale cortex marked by the red vein there was a tumor containing small cysts up to 10 cc. in all.

After removal of the neoplastic tissue, which was soft, crumbly and poorly supplied with blood, the deeper branch of the red vein became almost blue. The superficial branch remained red. Histological diagnosis was glioblastoma multiforme.

On comparing the serial carotid angiograms with the operative findings, the two large veins in the parietal region could be clearly seen. The vein which had appeared red at operation did not fill early. No abnormal vessels of the tumor were seen in the serial films.

Comment. It is suggested that much of this tumor was so necrotic that it was not utilizing oxygen—an example of a metabolic rather than a structural arteriovenous shunt.

Case 9. L.W., aged 39 years, on the day after an automobile accident noted clumsiness of the left hand and leg. A month later he had twinges of pain and tingling in the left arm and within 1½ years intermittent bouts of clonic movements which progressed to generalized seizures.

Examination 2½ years after the accident revealed a left homonymous lower quadrantic field defect, with mild spastic weakness and impaired sensibility on the left side of the body.

Both lateral ventricles were large, the right more than the left. Angiogram showed occlusion of the right internal carotid artery 1.5 cm. above the bifurcation.

Right frontoparietal craniotomy (June 1954) for excision of epileptogenic tissue showed the outer surface of the dura mater near the midline more vascular than usual. There were fine dural adhesions over the frontal and inferior parietal regions. When the dura mater was turned back, the parietal area consisted of yellow, tough, shrunken tissue (Fig. 10), with loss of convolutional pattern. The abnormal tissue extended into the first temporal and lower precentral convolutions.

The veins running toward the sagittal sinus from the surface of this scar contained red blood—those coming from the center of the scar appeared as red as arteries, those on the margins had some branches which were red and others which were blue. When two such branches joined, the red and blue streams remained distinguishable for several cm. along the main vein.

The large central vein appeared at first dark blue but in a second photograph taken 1 hour and 20 minutes later, it had a red tinge. In the interval, electrical stimulation of the cortex and corticography had been completed.

The arterial vessels running into the scar showed fewer branches than the venous vessels. There was a great reduction in the number of small vessels over the pia-arachnoidal surface as compared to normal.

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**TABLE 1**

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>PCO₂ mm.Hg</th>
<th>HCO₃ mEq./L</th>
<th>O₂ Saturation</th>
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</thead>
<tbody>
<tr>
<td>Red vein</td>
<td>7.59</td>
<td>17.2</td>
<td>22.0</td>
<td>100%</td>
</tr>
<tr>
<td>Radial artery</td>
<td>7.56</td>
<td>19.4</td>
<td>22.5</td>
<td>100%</td>
</tr>
<tr>
<td>(60 min. later)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transverse sinus</td>
<td>7.48</td>
<td>26.2</td>
<td>22.0</td>
<td>56.5</td>
</tr>
<tr>
<td>(60 min. later)</td>
<td></td>
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Histologically the abnormal tissue showed a dense glial scar with few surviving neurones. Clumps of small thickened blood vessels were present.

Comment. In this instance, it seems probable that the dense scar tissue took up little oxygen from the blood flowing through it—another example of a metabolic arteriovenous shunt. The islands of vascular granulation tissue may have provided a slight degree of structural shunting. The reddening of the venous blood became more widespread after further exposure and electrical stimulation.

Discussion

Certain anatomical and physiological problems presented by the existence of red veins can only be answered by further detailed study of the particular lesions in which they occur. But a number of comments and conclusions may be worthy of record.

Identification of Vessels as Veins. Some confusion has existed in regard to the distinction between small arterial and venous vessels within the brain as studied on histological sections. For example, in extensive investigations on intracerebral vessels in the cat, Pfeifer19 appears to have confused arteries with veins, as Campbell21 and Scharrer23 have pointed out. This makes it difficult to evaluate his description of arteriovenous communications in normal pial and intracerebral vessels. According to Scharrer23 "arterio-venous anastomoses in the brain, if they exist at all, are probably so rare that no great physiological significance can be attributed to them." But these were reported2,23 on the cerebral vessels of the cat, opossum and monkey and evidently neither support nor disprove the presence of arteriovenous anastomoses in the human brain. This question, therefore, needs further examination.

In view of this background of uncertainty, it seems useful, first of all, to verify that the red vessels described in our present cases were in fact venous in nature.

In most examples this was not difficult. The red vessels joined other venous channels which could then be traced to their point of entry into the superior longitudinal sinus. In one example (Case 3) the red vessels corresponded to the rolandic vein and the vein of Labbé with drainage into the superior longitudinal sinus and transverse sinus respectively. The smaller red vessels related to the cerebellar hemangioblastoma could be traced to a point where they joined larger cerebellar veins identified by their blue color.

Early Filling of Veins. When satisfactory angiograms were available, the red veins seen at operation corresponded to veins which filled earlier than the remaining superficial cerebral veins. One exception was provided by a case of tumor in which no blush or early filling was present but the vein proved to be red at operation (Case 8). This example was considered as a metabolic shunt.

Red draining veins in tumors may thus be related not only to the presence of a direct arteriovenous shunt as evidenced by a vascular blush and early-filling veins on the angiogram, but also to necrosis and cystic formation within the tumor.

Direct arteriovenous connections in glioblastoma multiforme were demonstrated by Nyström14 by injection of tumor vessels. Greitz2 correlated the vascular patterns and times of flow in gliomas from serial angiograms. Busch and Christensen1 found that arteriovenous anastomoses were more commonly seen in glioblastomas which they termed angioneurotic in type and their presence was considered by these authors as an indication of a high degree of malignancy with poor prognosis. On this basis their view was to discourage operation for this type of glioblastoma.

Tömni and Walter27 have contended that early filling of veins indicated malignancy when associated with tumors. Woringer et al.29 found early opacification of veins in 10 of 38 meningiomas and in 7 of 21 metastatic malignant tumors so that the association is not by any means a constant one. El-Banhawy and Walter3 reported 8 out of 151 meningiomas with early filling of the veins and noted malignant changes in this small number.

It seems clear that this question requires
further examination. Red cerebral veins which correspond in the structural arterio-
venous shunts with early-filling veins on the
angiogram can be found in conditions not
associated with malignancy and appear to be
a reflection of features of the lesion other
than its neoplastic activity.

The significance of arteriovenous shunts in
tumors remains obscure. Sanders31 has
pointed out that in transplants of tumor
there was less differentiation of capillaries
into arterioles and venules than in normal
tissue. As for the tumors noted in the present
series, the local distribution of red veins
evidently indicates vascular and metabolic
features which are distinct from the sur-
rounding normal brain. Systematic analysis
of the rates of flow and metabolic con-
stituents of blood from these red veins
would be of interest in relation to growth of
tumor. To our knowledge no such studies
have been done as yet.

Some Practical Implications of Red Veins.
Several practical points may be noted in
relation to red cerebral veins. In the first
place, their presence on the cortex can occa-
sionally point to the site of a subcortical
tumor when it may not be obvious from ab-
normality on the surface. Even when the
tumor has caused widening and discoloration
of convolutions, the distribution of the red
veins, because of their localized drainage
from the tumor, may still help to indicate the
size of the tumor and its margin with normal
surrounding brain.

Similarly, in small arteriovenous anomalies
consisting of only a few inconspicuous ab-
normal subcortical vessels (Case 1) a red
surface vein can lead to a more precise loca-
tion of the lesion. In addition, it is useful for
guiding successful excision, since any red
veins remaining on the cortex indicate that
the removal of the vascular malformation is
still incomplete.

In removal of tumors containing arterio-
venous shunts, as with angiomatous tumors,
bleeding can be controlled better by dividing the
feeding arteries before interrupting the
draining veins.

Attempts to treat brain tumors by vascu-
lar perfusion with chemical or radioactive
agents32 may be rendered less effective in
two ways by the presence of these vascular
shunts. First, the chemical agent may by-
pass the target tissue because of the prefer-
ential and rapid flow through the arterio-
venous shunt. This sort of by-pass of tissue
has been seen in a patient with an extensive
angioma.8 Intracarotid injection of Sodium
Amytal to determine dominance of speech
failed to give either the transient hemi-
paresis or disturbance of speech regularly
obtained with the same dosage in other
patients. Secondly, the amount of the agent
reaching the systemic circulation to produce
toxic effects, for example in bone marrow,
may be increased. Chemotherapeutic agents
have naturally been tried in the most
malignant types of brain tumors, including
glioblastomas, which commonly have arterio-
venous shunts. No particular recognition of
this point seems to have been made. The
vascular shunting may of course be offset by
other features, and we have found8 fairly
high differential uptakes of radioisotopes in
such tumors (e.g. in Cases 3, 4 and 7), even
when given intravenously.

Finally, radioisotopic studies indicate that
the vascular shunt through an angioma is
associated with a reduction in the circulation
through the surrounding normal cortex.5,6
In the absence of a better term, we may call
this a "cerebral steal." This effect may also
be obtained in tumors with arteriovenous
shunts. The role of these shunts in the
production of such features as the cerebral
edema associated with gliomas or metastatic
carcinomas in the brain, or in a degree of
ischemia of the surrounding tissue which
could lead to the development of paresis or
seizures requires further investigation.

Laminar Flow. Where a red venous branch
joins a normal vein to form a larger vein the
separate red and blue streams of blood could
be distinguished over a distance of several
cm. By separately compressing the red or
blue vessels with an instrument the streaming
could be appropriately altered. In some
examples, when the laminar flow occurred in
sufficiently large vessels, the streaming effect
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was also visible on serial angiograms (Case 4). This well-recognized phenomenon has been elegantly demonstrated by the motion pictures of McDonald and Potter in their experimental studies of cerebral arterial blood flow in the vertebral-basilar arterial system.

* Distinction Between Structural and Metabolic Arteriovenous Shunts. The presence of red veins in association with obvious abnormal arteriovenous communications requires little comment. A similar explanation holds true for neoplasms which exhibited on the angio-gram a vascular blush with early filling of the draining veins.

The absence of a vascular blush on the angiogram does not entirely rule out the possibility of abnormal vascularity in a tumor (Case 5).

The red color in the veins draining a local lesion could evidently result from two situations. On the one hand, with a structural arteriovenous shunt, the blood flow through the lesion would be more rapid while the metabolic uptake of the tissue might well be normal. This situation would hold with an arteriovenous malformation or a richly vascularized tumor. On the other hand, a red vein might also be present even though the speed of flow of blood through the lesion is normal, provided the usage of oxygen by the tissue within the lesion is reduced. This would be the case with scar tissue, necrotic tissue or an area taken up largely by cystic fluid. Measurements of the transit times of blood flowing through the lesion into the red veins with the use of radioisotopes or dyes may provide a means of distinguishing between metabolic and structural arteriovenous shunts.

The transient appearance of local red veins following focal seizures, first described by Penfield, might also be explained on either basis. That is to say, the blood through the local region could flow at normal speed but appear as red arterial blood in the veins because of the lack of uptake of oxygen by neurons exhausted by the epileptic dis-

charge. It is also possible that the accumulation of metabolites, especially carbon dioxide, within the tissue involved in the epileptic discharge could cause dilatation of the regional blood vessels and a temporary opening up of the local vascular bed. Arterial blood, flowing at an increased rate, would thus provide oxygen in excess of that which could be utilized by neurons even at normal metabolic rates.

Generalized reddening of cerebral veins can be noted at operation after a major convulsion involving respiratory obstruction. A similar reddening was produced by in experimental animals by clamping and releasing the large arterial vessels to the head or by temporary occlusion of the trachea. In direct observations of pial vessels no arteriovenous anastomoses were noted by Walker in normal brain of cats. But following experimental concussion he found immediate general increase in rates of flow, both arterial and venous, and further reported that "arterio-venous anastomoses appear and disappear again after five to ten minutes." Symon noted reactive hyperemia after release of a cerebral artery which had been occluded temporarily. The flow of blood then became greater than normal over a period of 15 to 45 seconds and the cerebral veins became red.

Transcerebral Vessels. In two examples, it was possible to observe that red veins became blue when part of the margin of the tumor was incised in a way that interrupted the entering arteries. Evidently, the red blood in the veins was derived from cortical rather than subcortical arterial vessels.

Moreover it was often noted that a red and blue vein ran closely together or joined to form a larger vein. This indicates that the two regions of tissue which these veins drain, though closely adjacent, remain nonetheless

quite segregated. This segregation is not a sharp one, since the two differing veins usually collected from regions separated by at least one or two convolutions. But even this is notable in view of the many collateral channels offered by surface veins. It suggests that in these circumstances there is some vertical parcellation of a given volume of cortex and white matter in relation to its local arterial supply and its venous drainage.

The anatomical basis of such a regional vascular differentiation could be provided by the vertically disposed transcerebral vessels which run from the surface toward a periventricular plexus. Although the venous side of this system has been known for some years from the description of Schlesinger, transcerebral arteries have only recently received attention. These vessels have been most clearly demonstrated in the microangiographic studies of Saunders and their profusion makes it improbable that the cortical and subcortical regional circulations are as separate as considered heretofore.

This vertical vascular pattern may offer one explanation for the localized appearance of red veins. A tumor or cyst disposed subcortically so that it interrupts the long transcerebral vessels would favor return flow then by way of venous collaterals toward the overlying cortical veins of the surface. The red vein would then be a mark of this local change in the subcortical regional circulation. Again, in localized epileptic discharge, the resulting opening up of the local vascular bed during the reactive hyperemia could direct arterialized blood into the vertical transcerebral veins, leading in turn to the appearance of red blood in the overlying veins of the surface. Support of this vascular parcellation is also provided by recent anatomical studies on the penetrating cerebral vessels in which the authors suggest the concept of a vertical arterial unit in the cortex. Clearly a closer examination of the role of the transcerebral vessels in relation to such localized circulatory changes would be worth while.

Conclusions

1. Cerebral veins filled with red arterial blood have been noted at operation in association with a variety of intracranial lesions. Usually single or few in number, they remain restricted to the area around the lesion. Examples of these are described and illustrated.

2. In one group of lesions, the red veins are the result of a structural arteriovenous shunt dependent upon a direct arteriovenous communication. They correspond to veins on the angiogram which fill early and show laminar flow. They can drain arteriovenous malformations, hemangioblastomas, vascular gliomas, meningiomas and metastatic carcinomas.

3. In a second group of lesions, red veins are related to a metabolic arteriovenous shunt because the tissue they drain does not utilize oxygen adequately. They are present as a persistent or permanent change when the lesion is a necrotic tumor, a cyst, a scar, or an infarct. They also appear as a transient local phenomenon, first described by Penfield, in association with seizures or following electrical stimulation of the cortex. Whether this is a feature unique to neuronal tissue susceptible to epileptic discharge remains an intriguing problem.

4. The red veins seen either with space-occupying lesions or postictally may be partly dependent upon preferential vascular pathways made possible by the anatomical pattern of the transcerebral vessels.

5. Red veins often provide a useful guide to the site and size of a subcortical tumor, cyst or small angioma.

6. Gas analysis of blood from a red vein in one case confirmed its arterial character.

7. The importance of recognizing red veins as evidence of arteriovenous shunts is noted in relation to chemotherapy of neoplasms.

8. Red veins offer a means of systematically studying the circulatory and metabolic changes associated with tumors and ischemic lesions of the brain.

The authors wish to thank Dr. Wilder Penfield for reviewing the manuscript and Dr. Fred Brindle for obtaining the blood-gas analyses in Case 7.
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