Isotope Angiography and CBF Patterns

To the Editor:

In an article by Awad et al., the delay of hemispheric flow, which is caused by a hemodynamically significant carotid stenosis, is discussed in an interesting way. In isotope angiography, as in digital subtraction angiography and dynamic computerized tomography, both hemispheres can be evaluated simultaneously. When examining a tight carotid stenosis or occlusion with isotope angiography, the delay of flow through a hemisphere is accompanied by a reduced activity along the neck vessels on the same side. Sometimes, however, only the reduction in the neck is seen, while the flow through the hemispheres is symmetrical. Perhaps a wide anterior communicating artery could explain this picture but only 3 cases of carotid stenosis or occlusion were found when the carotid arteries in a series of 30 such cases were examined with a Parks continuous wave directional Doppler device (Model 806C).

The files of our gamma camera registry were searched for such cases with an isolated activity reduction on one side of the neck and symmetry in the hemispheres. In 40 patients an aortic arch arteriography and a posterior view isotope angiography had been performed during the same hospital stay. In all but 2 cases the isolated reduction of neck activity matched an unusually thin vertebral artery on the relevant side. No association with a carotid lesion of hemodynamic significance or a vertebral artery stenosis or occlusion was found.

Asymmetry of the vertebral arteries is congenital and regarded as a normal variant. Sometimes, however, an unusually thin vertebral artery may get a certain significance in the development of ischemic lesions of the vertebrobasilar system, especially if associated with an anomalous circle of Willis. In our gamma camera registry of 1300 cerebrovascular events, collected mainly 1978 through 1982, this picture was found in about 15% of either infarctions or transitory ischemic attacks in the vertebrobasilar system. Infections of the vertebral artery system (including those of the posterior cerebral artery) showed this picture in 40% and TIA and TIAs in the vertebrobasilar system in nearly 30%. This suggests that the finding may be a risk factor for ischemic events in the vertebrobasilar system.

A digital subtraction angiography will give much more information about the anatomy of the vessels. Flow curves are however easily obtained in cerebral radionuclide angiography. Using a gamma camera, connected on-line to a Digital PDP-II computer with a Gamma-11 system, parts of special interest of the time activity curves, such as the arterial and venous phases, can be integrated. The values obtained from the two hemispheres can be compared by the computer and the peak delay calculated. A Basic program puts the different figures together and flags the flow pictures suggesting carotid stenosis of hemodynamic significance and vertebral artery asymmetry, thus printing out a probable diagnosis.

A careful handling and positioning of the patient during the injection efficiently avoids the interfering of venous reflux. A computer operator has to locate the areas of interest carefully over the irrigation areas of the middle and posterior cerebral arteries as well as over the neck vessels. This is done easily with the help of a movable configuration of the areas of interest. Asymmetries of flow in the posterior cerebral arteries are not commonly seen in vertebral artery asymmetry. Not seldom, however, the cases with a hemodynamically significant carotid stenosis show a delay pattern not merely embracing the middle cerebral artery but also including the area of the posterior cerebral artery.

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References


Dr. Issam Awad's response follows.

To the Editor:

I read with interest Dr. Johansson's letter. It describes an innovative use of isotope angiography, by measuring and analyzing the radioactivity over the neck and cerebral hemispheres and analyzing the data using an on-line computer. This appears to have good potential in delineating cerebral blood flow patterns. However, in the absence of direct cerebral blood flow measurements and precise angiographic delineation of vascular anatomy, there is the danger of over-interpreting the data and drawing invalid conclusions. As with all non-invasive vascular techniques, one must be acutely aware of this potential pitfall.

The author describes the phenomenon of symmetric blood flow over the cerebral hemispheres in the presence of asymmetrical activity in the neck. Such a phenomenon can be due to asymmetric flow in any of the vessels in the neck, including the internal carotid system, external carotid system, or the vertebral arteries. Artifactual activity over the neck related to swallowing or other movement can also contribute to this phenomenon. Without true measurement of cerebral blood flow, one can draw no further conclusions about the adequacy of intracranial collaterals or the pathophysiology of ischemic symptoms. The data presented by the author cannot support his implied thesis that vertebral artery asymmetry is invariably responsible for the observed phenomenon, and furthermore plays a significant role in the pathophysiology of ischemic symptoms. A review of our data (Awad, et al: Correlation of CBF and Angiographic Findings in Brain Ischemia with rCBF Measured by Xenon Inhalation Technique. Neurosurgery 11: 1-5, 1982) revealed that vertebral artery disease can play an important role in overall cerebral blood flow in the presence of significant carotid artery disease and/or appropriate anomaly of the circle of Willis. This is also shown in the references cited by the author. The mere presence of vertebral artery asymmetry in a significant number of patients with TIA or stroke in the carotid distribution does not suggest that this finding is a risk factor. Furthermore, the fact that vertebral artery asymmetry is more prevalent in patients with vertebro-basilar TIA's is not surprising, and again, does not qualify this finding as a "risk factor for ischemic events." Only with adequate control data (the incidence of a given
Letters to the Editor

Ischemic Penumbra Results in Incomplete Infarction: Is the Sleeping Beauty Dead?

To the Editor:

In their recently published studies Strong and coworkers occluded the proximal segment of the middle cerebral artery in cats for 2 hours. Over the ipsilateral parasagittal gyrus (marginal gyrus) the electroencephalogram showed a reduction in amplitude in 24 of our 26 studies, while the surface K⁺ concentration remained below 11.5 mM in 22 of 24 studies with values of 13.0 and 14.5 mM being recorded in the remaining 2 studies. This pattern of reduced electrical activity and yet preservation of essentially normal K⁺ values was taken to indicate ischemic penumbra in the marginal gyrus. Using the hydrogen clearance method they found in average a flow reduction from 34 to 27 ml/100g/min in the marginal gyrus. When originally coined the term ischemic penumbra Astrup et al referred to the rather severe level of ischemia that leads to complete abolition of all spontaneous as well as evoked electrocortical activity. Hence using the term to comprise any degree of unequivocal reduction in the electroencephalogram with preserved K⁺ concentration Strong et al probably have included milder degrees of ischemia than Astrup et al in their penumbra.

On this background it is remarkable that the histological studies of Strong and coworkers showed evidence of incomplete infarction (selective neuronal death) in the marginal gyrus. It implies, that this moderate degree of ischemic kills many nerve cells in the course of 2 hours without, nevertheless, destroying all nerve cells or glia cells and vessels: only microfoci of neuronal death and scattered nerve cell loss was seen. One cannot, obviously, predict the full extent of neuronal loss, that a longer period of ischemia would have led to. Indeed there are some indications suggesting that a vascular occlusion lasting a few hours (say 2 to 4 hours) may kill the same number of nerve cells as a more prolonged occlusion: collateral blood flow tends to increase as shown by Strong et al and if some nerve cells die, the remaining cells will have an increased O₂ delivery even at unchanged flow.

However, thus may be, the observations of Strong et al are important as they show, that even so brief a period as just 2 hours of relatively mild penumbral flow results in some degree of incomplete infarction, viz. irreversible tissue damage, that may or may not become further enhanced by a more prolonged period of vascular occlusion. That persistent penumbral ischemia results in this type of lesion is shown by the study of Mies et al. Hence it may be concluded, that moderate ischemia results in incomplete infarction (a lesion invisible on CT-scanning) and not solely in a fully reversibly state of ischemic neuronal paralyzis as implied by the term penumbra: already after 2 hours many cells die.

That brief ischemia (even if it is total ischemia, zero flow) will abolish neuronal function reversibly is well known. The studies of Astrup et al defining the ischemic penumbra pointed to a fully reversible functional paralysis with a more prolonged (1 to 2 hours) moderate ischemia. Now we know that cell loss results after just 2 hours. This suggests, that a state of chronic penumbra may not exist: perhaps the condition of a “sleeping beauty,” viz. of days-weeks-month lasting borderline hypoxia with fully reversible functional deficits does not exist; the vascular surgeon could not then, as the prince in the fairy tale, awaken these cells by the expedient of reconstructive or by-pass interventions. We have seen an improvement of regional cerebral blood flow following such interventions in 4 of 15 cases. Yet, this was not associated with a clearcut clinical improvement apart from cessation of hemodynamically induced transient ischemic episodes in 1 of the 4 patients.

Therefore, at this state the observations here summarized point to the statement, that elective vascular surgery in CVD merely improves the flow reserve preventing some acute episodes. It usually does not, so it appears, enable cells to function better. This view of the value of elective vascular surgery in CVD is shared by many other investigators, a statement we shall not here try to document by references as a proper discussion would necessitate a very detailed analysis of the clinical cases operated upon. What is intended by this brief discussion is to emphasize, that the ischemic penumbra is a highly dangerous state rapidly killing many or all nerve cells as shown by Strong et al. Evidence regarding the possible existence of a chronic fully reversible penumbral state is not available. Precise clinical and experimental studies may yet disclose its existence. Yet, we are somewhat pessimistic.

Perhaps the sleeping beauty is dead?

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References
3 Astrup J, Symon L, Brantston NM and Lassen NA: Cortical evoked potential and extracellular K⁺, and H⁺ at critical levels of brain ischemia. Stroke 8: 51-57, 1977
4 Lassen NA: Incomplete cerebral infarction — focal incomplete ischemic tissue necrosis not leading to emollosion. Stroke 13: 522-523, 1982
6 Vorstrup S: Unpublished observations
surface electrodes may reflect lateral diffusion of acidity from core areas, the rapid onset of the changes we have observed, together with the fact that a substantial CSF volume is drained by the transorbital occlusion procedure, suggests that the pH reduction originates within MG. When intracortical microelectrodes are used (Astrup et al) the findings are similar.

We would like to emphasize that the cell loss observed on MG in our experiments was not quantified and it would be misleading to state even an estimate of the numbers of neurons affected; however, the abnormalities, although unequivocal, were topographically quite restricted. The inference by Drs. Lassen and Vorstrup that 'the ischaemic penumbra is a highly dangerous state rapidly killing many or all nerve cells' (our italics) may therefore overstate the case, but only to a degree. There was evidence in our experiments of instability of Ke (recurrent transient increases) in MG. Clearance of Ke transients is also delayed in the penumbra. Since Ca2+ influx is associated not only with steady state but also with transient Ke increases, and is believed to contribute to neuronal necrosis, it is possible that repetitive or protracted Ke transients ('spreading depression') play a part in the evolution of ischaemic cell change in the penumbra. Whether they represent the cause or the effect of incomplete infarction is unclear; however, we interpret our data as suggesting that K+ transient activity sustained for perhaps 2 hours is a marker of incomplete infarction and a feature supporting Drs. Lassen and Vorstrup's interpretations of penumbra (Type 2) as carrying the potential for nerve cell loss.

It seems to us that two related questions arise. First, in a gyrus with morphological evidence of say, 10% neuronal loss (Type 2), itself lying adjacent to a more severely affected territory, what is the nature of the physiological suppression? Second, what is the nature of suppression in a territory which is entirely normal histologically (Type 1)? Inhibition of neurotransmitter synthesis, a moderate reduction in pH or diaschisis might account for functional suppression in either of these situations, but we are not aware of any experimental evidence that is conclusive. Likewise, the impact of loss of a small population of neurons on function in surrounding cortex is unclear, and an unequivocal experimental study of this question is needed.

Hypotheses for functional suppression in penumbra are not mutually exclusive: selective ischaemic metabolic change and diaschisis might co-exist. What then becomes important both scientifically and clinically, is the factor which limits recovery, and we agree that one may envisage an effect of reperfusion whereby any mild metabolic changes such as borderline hypoxia were reversed (if present) but function remained suppressed on account of deafferentation by adjacent or more distant nerve cell loss.

Although the effects of reperfusion on chronic partial ischaemic neurological deficits in man require careful clinical assessment supported by detailed pre-operative studies, neurosurgeons will also be familiar with the effects of reperfusion in a different situation. Some patients who develop progressive neurological deterioration following aneurysm surgery will respond to pharmacologically induced systemic arterial hypertension: although it is argued by some that this may promote oedema and only postpone deterioration, it would probably be agreed that a proportion of patients will show some degree of recovery, at least initially. It seems reasonable to suggest that such recovery is more likely if a proportion of patients will show some degree of recovery, at least initially.

To return to Drs. Lassen and Vorstrup's attractive analogy, we share their concern about the sleeping beauty as she appears to them (Type 2); however, her sister (Type 1) is certainly asleep, but why?

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Response by Drs. Mies and Heiss:
To the Editor:

During graded ischemia the functional state of neurons was found to depend on the remaining cerebral perfusion where at a certain flow threshold the spontaneous activity and the synaptic transmission of neurons ceases but the cellular integrity remains preserved. Upon elevation of the perfusion above a certain level these functional disturbances were reversible. Since around an ischemic brain lesion such low perfused borderzones were observed 2 it was suggested that structural intact tissue in the 'ischemic penumbra' may regain function after adequate reperfusion of such regions.

From experimental investigations on cerebral ischemia we know that the acute cellular damage depends on the severity as well as the duration of ischemia. Delayed morphological damage seems to be additionally related to the selective vulnerability of individual nerve cells. Complete cerebral circulatory arrest for more than 10 min followed by 30 min of normotensive reperfusion induced irreversible damage of 1/4 of neurons in the gyrus suprasylvius. In focal cerebral ischemia the level of remaining blood flow obviously prolongs the time limit for the onset of irreversible cell damage. A common threshold for the cessation of neuronal unit activity and of persistent tissue damage seems to be a permanent flow value below 18 ml/100 g/min indicating that the functional aspect of cell activity as observed in the acute stage of focal ischemia agrees well with the morphological alterations visible in the chronic stage. According to the flow/time relationship for the onset of neuropathological processes local perfusion at a level below 10-12 ml/100 g/min will, for instance, be tolerable for about 2 hours. The possibility for neuronal survival in the vicinity of an acute focal brain lesion would then require stable perfusion above the critical flow value. Furthermore, even when conditions favour cell survival, delayed neuronal death might be the consequence of selective vulnerability. Once the cellular damage has been initiated by hypoxic or ischemic stress the cellular desintegration occurs within a certain period of time regardless of re-established perfusion.

As our own studies revealed cortical tissue around the core of completed infarction suffers from a substantial loss of neurons. This means that the 'ischemic penumbra' as suggested before does not exist in the sense of structurally intact tissue close or even more remote (gyrus extrosylvius) from the infarcted zone. The loss of neurons in peri-infarcted brain areas (20% of control) indicates severe damage to the structural integrity of the cortex. It remains questionable whether these cortical areas would ever be capable to contribute to complex functions which require an intact neuronal network rather than fragments. Striking is the fact that the relationship between neuronal density and cortical blood flow in these areas is linear indicating, that perfusion is apparently adequate for the remaining neurons. It is doubtful whether improvement of blood flow to such cortical tissue can be made responsible for occasionally observed clinical improvements.

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LETTERS TO THE EDITOR

References


Response by Dr. Astrup:

To the Editor:

The sleeping beauty is not dead, and occasionally, surgeons who perform STA-MCA anastomosis experience the role of the prince in the fairy-tale waking functionally suppressed but viable parts of the brain by a well placed surgical kiss. Six years have now passed since the ischemic penumbra was described in acute experimental stroke as a state of suppressed synaptic transmission but preserved vital cell functions such as ion homeostasis, membrane polarity and energy state. Likely, clinical recovery in TIA and minor stroke cases is due to clearing of synaptic transmission failure in an area of ischemic penumbra along with lysis of embolus or opening of collaterals. Clearly, an extension of the acute ischemic penumbra into a permanent penumbra maintaining deficits in completed stroke cases, provides the indication of choice to surgery. But do such cases exist? Lassen and Vorstrup take a rather pessimistic view. Their situation is different in experimental models of focal cerebral ischemia in smaller "membrane unstable" mammals. Mies et al and Strong et al have shown that cats with MCA occlusion develop selective neuronal necrosis in the infarct surroundings. The problem is that this neuron loss extends to regions of almost normal blood flow, and in my opinion this makes this lesion a secondary lesion which is not directly caused by ischemia. As evidenced by Strong et al's measurements of extracellular potassium concentration, transient but rather long lasting and often recurrent elevations of extracellular potassium spread from the infarct to the better perfused periphery. During these potassium transients there is influx of Na+ and of Ca2+, membrane depolarization, ATP depletion and eventually cell death. These potassium transients relates to the phenomenon of spreading depression which can easily be released in membrane unstable species like cat and other smaller mammals as mouse, rat, gerbil and rabbit. Potassium transients occur seldom in primates with focal ischemia and may not or only by exception occur in humans. Caution should accordingly be taken when relating the observation of selective neuronal necrosis in the infarct periphery in these experimental models of focal cerebral ischemia to human pathology.

The sleeping beauty is not dead. Brain cells may well sleep for a long time in a state of ischemic penumbra. The essence of the pessimistic view held by Lassen and Vorstrup is rather that such cases are few and so far have escaped a reliable preoperative detection. Cerebral blood flow measurements performed under steady state conditions are clearly insufficient for this detection. Preoperative evaluation must include clarification of cerebral hemodynamics. Preferably, such evaluation should include a hemodynamically induced blood flow increase in the ischemic area while the patient is observed one way or another for signs of neurological recovery. We need a reliable test of reversibility.

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References


Transient hyperperfusion following carotid endarterectomy.

To the Editor:

In the interesting overview "The ischemic tolerance of neural tissue and the need for monitoring and selective shunting during carotid surgery" (Stroke 14: 93-98) Dr. Thoralf M. Sundt states that the primary cause for neurological complications following endarterectomy is related to ipsilateral hyperperfusion. A marked increase in cerebral blood flow during the operative procedure was noted in most patients developing new neurological deficits. But as the CBF (intraarterial xenon technique) was measured only immediately after completion of the endarterectomy the later course of this hyperperfusion was unknown, and as the complications usually developed several days postoperatively a further rise in the perfusion can only be assumed.

Using a newly developed, mobile 10 detector system for intravenous xenon CBF measurements (Novo Cerebrograph 10a) we have made serial CBF measurements during and after carotid endarterectomy. A 55-year-old male with a severe stenosis of the left internal carotid artery and occlusion of the opposite, underwent a left carotid endarterectomy. Approximately 200% of the preoperative resting values 2-4 hours postoperatively were demonstrated immediately following endarterectomy, reaching approximately 200% of the preoperative resting values 2-4 hours postoperatively (see fig). The patient remained clinically unaffected except for headache concomitantly with a rise in systolic blood pressure to 200 from values around 110 mmHg. Immediate treatment with hypotensive drugs, resulting in a blood pressure of 130-140 mmHg systolic, normalized the condition. Unfortunately no CBF measurement were possible since the use of hypotensive drugs is contraindicated due to possible cerebral ischemia.

A CT-scan had been without signs of infarctions. At operation a gradient of 65 mm mercury was measured over the stenosis. As clamping of the artery elicited flattening and slowing of the continuously registered EEG, a shunt was inserted. A marked bilateral hyperperfusion was demonstrated immediately following endarterectomy, reaching approximately 200% of the preoperative resting values 2-4 hours postoperatively (see fig). The patient remained clinically unaffected except for a short period 24 hours postoperatively, when he developed confusion and headache concomitantly with a rise in systolic blood pressure to 200 mmHg from values around 110 mmHg. Immediate treatment with hypotensive drugs, resulting in a blood pressure of 130-140 mmHg systolic, normalized the condition. Unfortunately no CBF measurement were performed during this episode. During the following days the flow decreased to values near the preoperative recordings.

The hyperperfusion following restoration of a normal hemispheric perfusion pressure has probably been related to an impairment of the autoregulation. The sequential CBF measurements demonstrated that the autoregulation was re-adjusted within one week. It can thus be assumed, that neurological complications related to postendarterectomy hyperperfusion, may be prevented by meticulous control of the systemic blood pressure as guided by CBF measurements in selected patients.

The author replies:

The Authors have illustrated beautifully the temporal profile of the transient hyperperfusion syndrome known to occur following endarterectomy. In that they used the same technic for each measurement, the changes are most meaningful and a comparison from one measurement to the other is of course valid. They have the unique ability to correlate their intravenous technic with the intra-arterial injection of xenon at the time of surgery and it would be most interesting to learn if they have made such a correlation in the past.

To my knowledge this is the first case in which the sequential changes of hyperperfusion following endarterectomy have been documented.

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High and Low Risk TIA Patients

To the Editor:

As pointed out by Shah et al (Stroke 14: 827, 1983), the incidence of strokes reported by Muuronen and Kaste in their TIA patients is significantly lower than that observed by others.2

Results of the Italian Study on Reversible Ischemic Attacks (RIAs) (462 patients) are very close to those of Muuronen. During a follow-up of 48 months we observed 7% of fatal and non-fatal strokes and 7.1% of deaths (including 3.2% of fatal strokes) in a population relatively young (19% of patients under 45 years) and with a low incidence (29%) of cardiac abnormalities.

Since medical and/or surgical therapies do not seem sufficient to explain such a low incidence of strokes and deaths, we share the opinion of Muuronen and Kaste that outcome is influenced by age and/or cardio-embolic factors. However other variables such as incidence of risk factors, angiographic findings and clinical territory are likely to be important as predictive factors.

In spite of these limitations due to different criteria of selection of case records, reversible focal ischemic attacks might not be considered "per se" as such high predictors of stroke. Therefore stratification of RIA patients in different subgroups with high and low risk of stroke seems a primary objective of clinical studies aimed at prevention of ischemic strokes.

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for the Italian Study on RIAs

References


FIG. 1. Sequential measurements of CBF demonstrating transient hyperperfusion following carotid endarterectomy.
Hyperfusion Following Endarterectomy

To the Editor:

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The author replies:

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References


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