up to 12 times each day. It was present while she was sitting, standing, or lying, at rest or at work, was always all the vision of one eye. There were no complaints related to her speech, face, arms, or legs. She thought she might have had an occasional day free of visual symptoms but these were rare.

She was treated with warfarin with no change in her complaints. Continuous intravenous heparin during her pre-operative investigations did not stop the intermittent blindness.

In January 1983 she was re-admitted. Her right arm had normal pulses and blood pressure, there were no carotid murmurs and her hematological abnormalities were unchanged. A repeat arch aortogram and selective left carotid angiogram showed a marked increase in the stenosis of the left common carotid artery. In addition, the internal carotid artery was seen to be critically stenosed 1.0 to 1.5 cm distal to its origin (Figure 1B). An endarterectomy of the left common and internal carotid was performed. The removed tissue consisted of atheromatous plaques with some calcification, and no evidence of intra mural hemorrhage. Attached mural thrombi in various stages of organization were present. She has been free of symptoms since then.

In the seven months between the two angiograms the disease in the left internal carotid has progressed from less than 50% occlusion to about 90% occlusion. Over the same interval the left internal carotid artery disease has also progressed, although not to the same extent. The left subclavian artery which is abnormal doesn't seem to have changed.

The rate of progression of atheromatous disease has been studied by Javid and Javid et al and the relative importance of hydraulic forces, hypertension, genetic tendencies, hyperlipidemia and tobacco smoke evaluated. The natural history of intracranial internal carotid artery stenosis has been illustrated by Craig et al. They followed 58 patients for 30 months.

The risk/accelerating factors in this present case were the cigarette smoking and the high hematocrit and hemoglobin. The latter were a double hazard by accelerating atherogenesis and increasing viscosity. The hyperviscous state produces reduced cerebral blood flow and a predisposition to stroke and transient ischemic attacks.

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References


An Easy Technique For Catheterization Of The Internal Jugular Bulb

To the Editor:

Many techniques utilizing various anatomical landmarks have been published about cannulating the internal jugular bulb. However, the catheterization has been difficult and some complication, such as inadvertent carotid puncture, facial palsy and nerve injuries may occur. We describe a new technique for the percutaneous catheterization of the internal jugular bulb, using an ultrasound Doppler flow detector.

The location of the internal jugular vein was detected by an ultrasound pencil-shaped Doppler flow detector (Ultrasonic Doppler Flow Detector, Model 811, Parks Electronics Laboratory, Beaverton, Oregon, U.S.A.), following Ulman's procedure, which was used for the central venous catheterization. Briefly, the patient was placed in the head-down position with his head turned maximally to the opposite site of the catheterization, and the Doppler detector was applied vertically to the skin. A continuous low-frequency signal, called "wind-storm," was audible 1 to 2 cm apart laterally and parallel to the carotid artery, which was detected by the characteristic pulsatile sound. The procedure was repeated at least three times at the following levels: the mandibular angle, the thyroid cartilage, and 2 cm above the clavicle. The vascular points [X] and [●] were marked on the skin in ink.

A 22 gauge needle with syringe was vertically inserted, by which the position of the internal jugular vein is made sure. Then a 16 gauge central venous catheter introducer (Intramedicus catheter kit, Nippon Sherwood Co. Ltd., Japan) was fitted with a three-way stop cock and a syringe was inserted from 1 cm below the 22 gauge needle at an angle of 30° to the skin, along the line corresponding to the internal jugular vein (fig. 1). After the removal of the inner metallic needle, a radio-opaque

Figure 1. Diagram of the right neck. The internal jugular vein is indicated by the shaded areas. The symbols [X] and [●] are the vascular points of the internal jugular vein and the carotid artery, respectively, detected by the Doppler detector. 'a' represents a needle inserted, by which the position of the internal jugular vein is made sure. 'b' is the catheter introducer inserted 1 centimeter below 'a' and at a 30 degree angle to the jugular vein.
central venous catheter was introduced upward until the tip of the catheter was obstructed by the bone. Then, the catheter was drawn back 0.5 cm. The catheter tip location was verified by an anterio-posterior X-ray taken in the recovery room. This technique was successfully applied for 48 patients. The mean length from the skin insertion to the tip of the catheter was 13 ± 0.2 cm (mean ± SD). It is recognized that the technique is very easy and no complication or dislocation of the catheter tip occurred.

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Flow and Neuronal Density in Tissue Surrounding Chronic Infarction

To The Editor
We should like to congratulate Dr. Mies and his colleagues on a most elegant study (Stroke 14: 22–27, 1983), and in particular for their lucid discussion of the potential role of diaschisis in electrophysiological suppression in areas where flow is reduced but remains above infarction thresholds.

Using standard perfusion-fixation techniques we have examined the marginal (or anterolateral), suprasylvian and ectosylvian gyri in cats subjected to occlusion of the middle cerebral artery for two hours.1 Like Dr. Mies and his colleagues, we found a gradation of severity of change: ischaemic cell change of classical type was extensive and almost homogeneous on the ectosylvian gyri, more patchy but present in all experiments on the suprasylvian gyrus, and was also found on the marginal gyrus acutely following middle cerebral artery occlusion, and was also found on the marginal gyrus support this: we doubt whether it is necessary to postulate functional degeneration due to white matter infarction, the mechanism proposed by Dr. Mies and his colleagues. Indeed we are not aware of any quantitative evidence that white matter infarction causes retrograde degeneration and loss of neurones in the cerebral cortex, and would be interested to know if such evidence actually exists. We would not however dispute that functional suppression of cortex could occur on this basis. Secondly, our findings serve to emphasise that ischaemic histopathological changes may be classified broadly under two headings. First, homogeneous or uniform change resulting in a complete cystic infarct with loss of all neurons and glia; as demonstrated by Morawetz and his colleagues, the flow threshold for this change in the primate is approximately 12.5 ml/100 g/min,2 closely resembling that for massive potassium fluxes.3 Secondly, more restricted foci of ischaemic cell change may be associated with higher ischaemic flows, perhaps up to 25 ml/100 g/min. The pattern of histological change in such penumbral areas in our own studies was variable, but in one or two examples, there was a suggestion of neuronal loss in laminar distribution (layers 2 and 3) and it is possible that certain cortical layers are selectively vulnerable to penumbral cortical ischaemia in a fashion analogous to more well established instances of selective vulnerability, for example, hypoxic or epileptic damage in the hippocampus.

Dr. Mies and his colleagues make the further point that in ischaemic penumbras it is sometimes possible to restore physiological ion distribution by reperfusion, although electrophysiological function is less regularly restored. They interpret this finding as indicating ischaemic damage to deep white matter connections of the affected zone. However, there is evidence that certain synaptic mechanisms such as monoamine and acetylcholine synthesis may be sensitive to ischaemia, and recovery in these compartments may take longer than is required for recovery of normal extracellular cation concentrations. It should also be mentioned that normal or near-normal restoration of cation activity during reperfusion does not necessarily imply histological integrity; we have made some comparisons of histological appearance with the temporal profile and extent of loss of potassium homeostasis during the period of insult, and found that where potassium is restored almost to normal by reperfusion following increases above 10 mM, there is nevertheless considerable histological abnormality.

Dr. Mies and his colleagues state (p. 26) that "...the decreased number of histologically intact nerve cells in the gyrus suprasylvius and gyrus ectosylvicus can be caused by retrograde degeneration due to white matter infarction and by transient impairment of blood supply severe and long enough to cause selective ischaemic cell necrosis..." We would favour the latter explanation and our findings in the acutely ischaemic marginal gyrus support this: we doubt whether it is necessary to postulate any additional explanation.

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