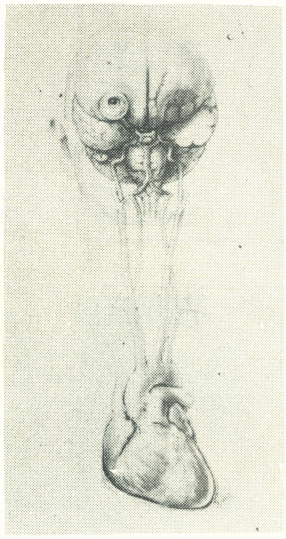
CEREBRAL THROMBOSIS

by Steven K.C.CHEUNG



Cerebral thrombosis constitutes about 60% of all cerebral vascular diseases (C.V.D peak incidence of cerebral thrombosis is in the sixth to eighth decades of life. It i common in the male

Atherosclerosis is the most common cause of cerebral thrombosis. It produces cl symptoms by causing cerebral infarction which means neural death from ischemia. The bral ischemia is the result of either a generalized or a localized prolonged reduction blood flow to the brain. If ischemia is transient, less than 10 minutes, usually no di ible neurologic deficit remains. If it lasts longer, neural damage results, producing logic dysfunction , disability and death. The atheromatous process in the cerebral ies is similar to that elsewhere in the body. The most common sites of thrombosis(fig are :

(1) At the carotid sinus of the internal carotid artery.

(2) At the first major middle cerebral bifurcation.

(3) At the junction of vertebral-basilar arteries.

(4) At the posterior cerebral artery as it winds round the cerebral peduncle.

Thrombosis in the small penetrating branches of the middle cerebral, posterior cere and basilar arteries may produce small infarcts called lacunes in the internal capsule.

tral white matter, deeper part of the basal ganglions, and brain stem. However, collateral branches of the cerebral vessels are sometimes adequate to prevent infarction. In some reports, even when a major arterial trunk was occluded, there was still no visible damage to the parenchyma.

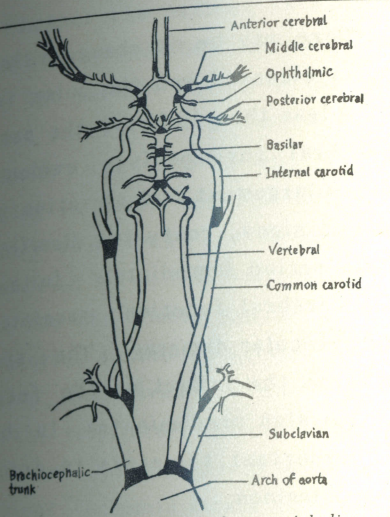


Figure 1. The darkened areas on the arterial diagram show the common site of Cerebral Thrombosis

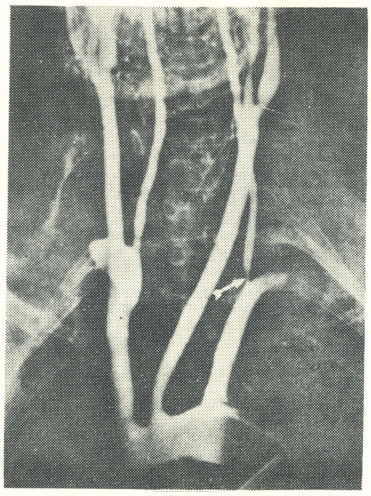


Figure 2. Vertebral artery thrombosis (arrowed) (Courtesy Dr. Bennett Stein)

CLINICAL FINDINGS

In approximately 80% of cases, the main part of the stroke is preceded by minor signs or by one or more transient, warning ischemic attacks, such as dizziness, a transient loss of speech, and visual disturbance, hemiplegia or paresthesia in one half of the body. A history of such prodromal episodes is of great importance in establishing the diagnosis of cerebral thrombosis. By the way, the most common duration of transient ischemic attacks is from a few seconds to 5 or 10 minutes. The mechanism under opthalmoscopic observation indicates that a temporary, complete or relatively complete cessation of blood flow occurs locally, possibly with microembolism. The final stroke may be preceded by hundreds of attacks or only by a single one. The stroke may come within a day of the first one or may be delayed for weeks or even months, and sometimes the attacks die away without leading to a stroke. The principle of intermittency seems to characterize the thrombotic process from the beginning to the end. The stroke, either the onset or the progression, is particularly common during sleep or shortly after arising. The generalized symptoms at the onset of the stroke include headache, vomiting, convulsion and coma. (See Table I)

Table I Incidence of Headache, Vomiting, Convulsion and Coma at the onset of Cerebral Thrombosis.

(In percentages) ------ After H. Houston Merritt.

Symptoms	Percentage
Headache #	6

Vomiting #	6
Convulsion	7
Coma	33

The figure given for this symptom indicates the percentage incidence in those patients who did not lapse into coma at the onset.

If a minor cerebral vessel is occluded by a thrombus, there may be no change in the vital sign. With occulsion of a major vessel the temperature is elevated and the pulse is increased. Abnormalities in respiration may occur only in less than 10% of the patients. Since artherosclerosis is the most frequent cause of cerebral thrombosis, evidence of sclerosis of the peripheral and retinal vessels, cardiac enlargement and elevation of the blood pressure are common findings. Mental symptoms and signs, confusion, disorientation and impairment of memory are frequently present in the period immediately following C.V.D. These are in part related to the disturbance of cerebral function associated with the vascular lesion and in part to generalized cerebral vascular diseases. The specific neurologic abnormality depends on the location and size of the infarct or the focus of ischemia. In involvement of the carotid system, unilateral sign predominates. In basilar disease, one more commonly finds bilateral signs. It is important therefore to determine if the signs and symptoms indicate unilateral or bilateral lesions.

LABORATORY FINDINGS

The cerebrospinal fluid pressure following brain infarction is always within normal limits. Cerebral thrombosis seldom causes blood in the spinal fluid, unless the infarct is especially congested. There is a slight increase in leukocytes of the spinal fluid in the early stage. The fluid protein level is normal or mildly elevated in most cases, but if the infarct is massive, it may rise above 100mg per 100ml. Roentgenograms of the skull are normal unless there is a massive cerebral hemispheric infarct, in which case the spinal shadow may be shifted to the opposite side. The electroencephalogram may show a slower frequency and lower voltage than normal. Unlike that seen in cases of cerebral neoplasm, the change is nonprogressive and will gradually resolve. Carotid arteriography provides essential information about cerebral hemodynamics. Radioactive concentration studies often show a mild positive picture over the infarcts especially in the second or third week. Serial brain scans are valuable in differentiating an infarct from a cerebral neoplasm. The pneumoencephalogram is usually normal in the early stage of cerebral infarction except in cases in which the edematous area acts as an expanding lesion and displaces the lateral ventricles to the opposite side. In the late stages of a cerebral infarct, the air study may show focal cortical atrophy dilatation of the ipsilateral lateral ventricle, or a porencephalic cyst.

COURSE AND PROGNOSIS

In the majority of cases, the neurologic deficit reaches its maximum within the first days. Old age, hypertension, coma, cardiorespiratory complication, anoxia, hypercapnia, neurologic hyperventilation are additional adverse prognostic factors, especially in the first 2 days of a verebral infarct. Some improvement may be apparent after the first 2 weeks,

by the end of 12 weeks the maximum recovery will be reached in most cases. With noticeably few exceptions, no further recovery should be expected after 6 to 9 months.

TREATMENT

Whether the patient is comatose or not, skillful nursing care is essential. Firstly, a clear airway must be immediately established. Oxygen can be adminstrated by mask if necessary. The patient should be placed in a lateral position, so that vomitus and secretions do not enter the tracheobronchial tree. Fluids and liquid nourishment should be given intravenously when the patient is unconscious. However, if the patient is conscious, oral feeding is mostly recommanded. The position of the patient in bed should be changed frequently to prevent the development of hypostatic pneumonia or bed sore. The bladder should be emptied by catheterization if necessary and the bowel kept open by enemas or cathertics. The bed sheets should be changed immediately when they are soiled by urine or feces. Antibiotics should be given if signs and symptoms of infection in the lungs or elsewhere develop. Sedatives should be used with care and the opiates avoided because they tend to depress the respiratory center. It is of great importance that the systemic blood pressure be maintained. Anemia should be corrected. Anticoagulants (dicoumarin and heparin) may prevent transient ischemic attack and postpone the arrival of an impending stroke. However, the prothrombin concentration is required to be determined regularly. In the initial days following major cerebral infarction, cerebral edema may threaten life; in such instance, dexamethasone and glycerin are helpful. Cerebral vasodilators (acetazolamide, papverine) and thrombolytic agents (fibrinolysin, profibrinolysin activator) are still of questionable value but should be tried. In surgical therapy, thromboendarterectomy or by-pass grafts have been employed. Carotid sinus, common carotid, innominate and subclavian arteries are the suitable sites for these surgical procedures. However, the operation should be undertaken at the stage of transient ischemic attacks or early in the course of thrombosis-in-evolution. When total infarction has occurred, surgery will be ineffective even though potency of the vessel is restored. Surgery is not without risk. Only a small minority of the total surgery becomes feasible.

After the patient has recovered from the stroke of the CVD, physical therapy and rehabititation should be directed toward restoration of function in the paralyzed limbs. Speech therapy is of questionable value, but they should be tried. Nevertheless, the treatment of CVD requires infinite patience on the part of the physician and persistent effort on the part of the patient.

References

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