STUDIES ON CEREBRAL CIRCULATION—I.

By Gwee Ah Leng and Yeoh Teow Seng.

The human brain is supplied by a system of arteries comprising the two internal carotids and the two vertebrals. Connecting these two sets of vessels is an interesting vascular circle first described by Thomas Willis in 1664. This is made up by the anterior communicating artery which connects up the anterior cerebral arteries, and the posterior communicating arteries which connect up the internal carotids at its emergence from the cavenous sinus with the posterior cerebral arteries. As Brain observed (1957), "the clinician is apt to look upon it (circle of Willis) as a device for maintaining as adequate as possible a blood-supply to the brain in the event of one of the main vessels being blocked", although he was of the opinion that rather than the obvious function of an anastomotic channel, the purpose served by the circle of Willis is to guarantee that whatever the position of the head in relation to gravity and to the trunk, and however, from one moment to another the relative flow through either carotid or vertebral artery may vary as a result, these variations are always compensated for distal to these vessels and within the cranial cavity, by the *freest possible anastomosis* before the brain is reached". Hamby (1952) however refers to the circle as forming only "a relatively efficient anastomotic circle between the basal cerebral arteries." Whether there is in fact the freest possible anastomosis or only a relatively efficient anastomotic channel would seem on theoretical ground to be important as having positive bearing on the deficiencies that may arise as a result of local arterial obstruction.

The findings of frequent anomalies of the circle of Willis, especially that, parts of the circle are often defective, (Padget, Fawcett and Blackford, Fetterman and Moran, Windle) would lend weight to the view that the circle as an anastomotic channel is only of some relative value, as otherwise such frequent anomalies and defects in the circle must surely be significant clinically. Obviously if the circle is frequently defective, then as a compensatory structure, the value must be frequently very small. Further, it is known also that the blood-flow in the cerebral vessels tends to have a stream effect which would confer a laterality in that the blood supply to the left side of the brain would be drawn principally from the left carotid and left vertebral and vice versa, and between the two flows, there exist the dead-points—regions of no flow. These physical phenomena are varied only in the presence of obstruction in the cerebral vessels, and not under normal circumstances (MacDonald and Potter). These findings would indicate that compensatory function of the circle as an anatomical pathway for blood-flow is of no great significance.

Animal studies (Bouchaerst and Heymans, 1935) have suggested that the cerebral blood-flow is determined chiefly by variations of systemic blood pressure, and gas dilution studies of Kety (1957) further confirmed that interruptions of cervical sympathetic pathways did not have any appreciable effect on cerebral blood flow. The paucity of muscle in the media of cerebral arteries has been cited as evidence for the improbability of neurogenic vaso-constrictive impulses playing a significant part in the control of cerebral circulation and in fact, Pickering (1955) affirmed that there was no real evidence of spasm of cerebral vessels.

However, there is a definite difference between the anatomical structures of cerebral vessels of man and animals, and the passivity of cerebral blood-flow with its dependence on systemic blood pressure remains to be proved. In fact, Kety and Schmidt showed that blood-flow in the brain is essentially unchanged in hypertension, and that this constancy is maintained by changes in the C.V.R.

It would be difficult also to imagine the presence of a rich perivascular nerve plexus serving no purpose at all! Besides, similar plexuses in the peripheral arteries have definite vaso-constrictive effects. In addition, there have also been many reported observations of visible vaso-constriction of pial vessels at human brain operation and in experimental animals. Vaso-spastic activity has been observed at operation, as also suggested by episodic fluctuations of some brain deficiency syndromes.

The position seems to be that the majority of workers accept that the cerebral blood flow is dependent passively to a large extent on the

general blood pressure, that CO_2 tension in excess of normal has strong vasodilatory action on cerebral vessels thereby affecting the cerebro-vascular resistance, and that there is little in the way of neurogenic control of cerebral circulation other than that mediated indirectly through the effect of systemic blood-flow and pressure, and that vasospasm has little part to play in cerebral deficiency syndromes.

To one of us (Gwee), these views would render difficult to explain phenomena of transient strokes, reversible cerebral deficiency syndromes after surgery of neck vessels, and vaso-spastic conditions visible in the retina under certain circumstances.

Further the cerebro-vascular resistance has been shown to be dependent on the CO_2 locally, and it seemed probable that further adjustment of a regional nature would be made dependent on regional metabolic activity through the changes in the C.V.R. This aspect has been studied by others (Adams 1964).

Accordingly, a number of studies were made to elucidate the situation further, and the following is now reported:—

METHOD

A model of the circle of Willis was constructed powered by a Sigma-motor pump. The system was filled with dextran (6% w/v solution in normal saline) so as to approximate the condition of plasma. Transparent vinyl tubings were used to facilitate observation of flow streams, and at different points, as illustrated in the diagram, provisions were made to have mercury manometer recorders to measure the pressure changes at different points simultaneously whenever desired.

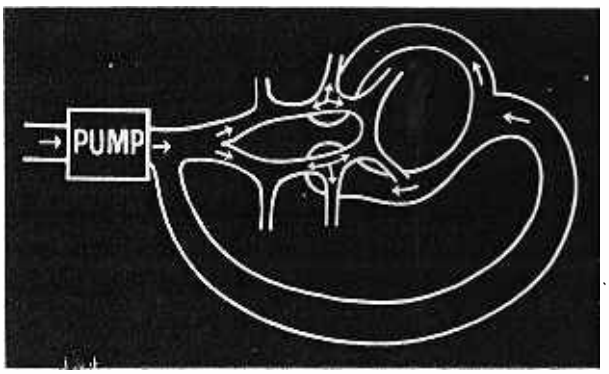

Fig. 1a.

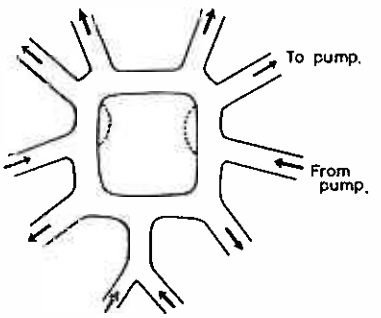

Fig. 1b.

Constrictions were produced by applying screw clips to selected tubings, and changes in the pressures were observed. Changes in flow rate were not studied as no flow-meters were available.

To observe the streaming effects, the dextran was coloured with red and blue dye when the requisite experiments were made.

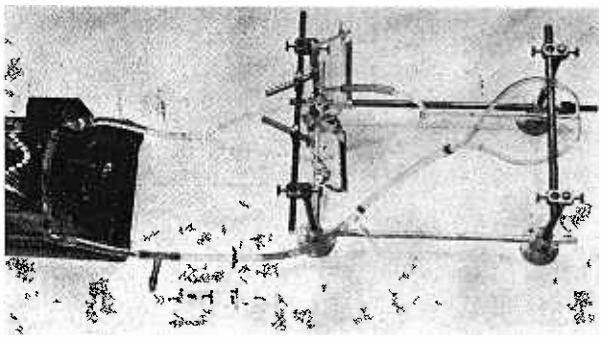

Fig. 2a.

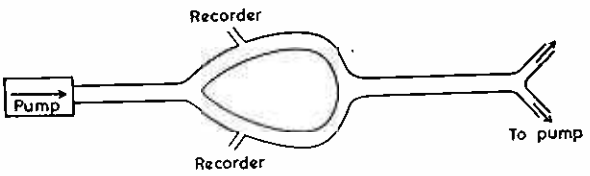

Fig. 2b.

The circulation was set up with a flow pressure of 120/60 at the vessels representing carotid and basilar arteries.

RESULTS

When the pressure head was maintained constant at 120/60, it was distributed equally to "carotids" and "basilar" system and dead-points were seen in the anterior and posterior communicating arteries as observed by Mac-Donald and Potter. On varying the pressure in the system by the application of clips to the carotids and cerebrals, either singly or in combination, the dead-points showed corresponding shifts in position in relation to the regional

changes in flow pressure. The pressure distribution in all the branches distal to the circle remained constant and similar and dependent on the pressure in the circle itself. (Figure 2)

The shift of dead-points occurred in the case of unilateral "carotid" occlusion both in the anterior and posterior communicating arteries. Measurement of pressure in arteries distal to the circle showed a very transient change of pressure which soon fell to a new level with no significant difference from the old one—the difference amounting to a few per cent only—found in *all the other vessels* distal to the circle whether on or away from the site of the applied constriction when this is proximal to the circle, that is, in the "carotids" or "basilar" arteries.

Application of constriction distal to the circle also produced a shift in the dead point in the circle, but no appreciable change in pressure was seen in the proximal vessels (Table I).

In the model, the inertia of the recording system was not adequate to reflect instantaneous changes, and hence transient changes that occurred immediately could not be regarded seriously.

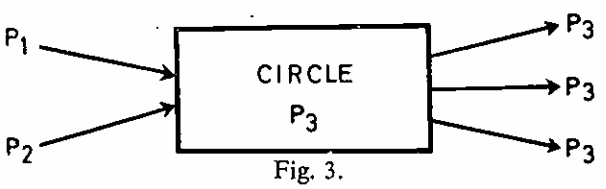

Fig. 3.

The shift of dead point, and the uniformity of pressures distal to the circle in the presence of varying constrictions, however, would suggest that the circle is an *iso-baric system* where the different pressure heads of the "carotid" and "basilar" are reequilibrated so that the blood supply distal to the circle in the absence of any local anomaly will be of the same pressure. (Figure 3)

TABLE I

	Left		Right	
	M. Cereb.		M. Cereb.	
	A. Cereb.		A. Cereb.	
Left carotid cut off at 120 systolic	114/86	105/80	105/86	106/80
Right posterior cerebral—full constriction	0/+1	0/+2	0/+1	+1/2
Right posterior cerebral—full constriction + L.M.C. full constriction—distal to pressure recording	2/13*	5/5	5/11	4/5
R.P.C. full constriction + L.M.C. (3 turns)	2/12*	5/3	14/11	4/3
R.P.C. full constriction + L.M.C. full release	2/11*	4/2	3/10	3/2
R.P.C. ½ constriction	2/11*	3/2	3/9	2/2
R.P.C. ½ constriction + L.M.C. full constriction	1/13*	4/3	4/10	3/3
R.P.C. ½ constriction + L.M.C. ½ constriction	2/12*	3/2	4/10	3/2
R.P.C. ½ constriction + L.M.C. full release	0/10*	2/0	2/9	2/0
R.P.C. full constriction + L.M.C. full constriction (proximal to manometer)	—7/+19	4/1	4/10	4/1
R.P.C. full constriction + L.M.C. ½ constriction	—6/16	3/0	2/9	3/0
R.P.C. full constriction + L.M.C. full release	—2/9	1/0	1/8	1/0
R.P.C. ½ constriction + L.M.C. full constriction	—7/+18	3/1	3/9	3/0
R.P.C. ½ constriction + L.M.C. ½ constriction	—4/+15	3/0	3/9	3/0
R.P.C. ½ constriction + L.M.C. full release	—1/+9	1/0	1/8	2/0
R.P.C. full release + L.M.C. full release	—1/+9	1/—1	0/8	1/0
R.P.C. full release + L.M.C. full constriction	—8/17	3/—1	1/14	3/0
R.P.C. full release + L.M.C. ½ constriction	—7/15	2/0	1/8	2/0

* Pressure taken proximal to constriction

There was also a marked stream effect seen in the basilar region and this was seen as a sharp partition in the mid-line of the basilar artery so that one could say for practical purposes that the left and right streams were quite distinct and this phenomenon remained unaltered as the systolic pressure head was varied up or down until it exceeded 220 mm. Hg. or fell below 80 mm. Hg. when the distinct partitioning effect between the two flowing streams became a blurred region of indefinite separation with eddying and turbulence. The latter was also seen when the pressure was kept at 120/60 and the rate of pulsation reduced to below 60/min. (Figure 1). This is not expected from the calculation of Reynold numbers according to which, the turbulence effect is dependent on a fixed function of the radius of the tube (R), the flow rate (V), the viscosity (n) and the pressure (p) expressed as RVp/n. The Reynold number for the system is well below the turbulence level throughout the observations with all the variations of pressure and rate, but the turbulence effect was clearly seen disturbing the clear-cut partitioning of the two streams. It would seem therefore that the capacity for turbulence in this particular arrangement is dependent on the pressure head with a critical range within normal variations of the systemic blood pressure.

SUMMARY AND CONCLUSION

A model was made to reproduce some of the aspects of the cerebral circulation especially with reference to the circle of Willis, and studies were made to determine the pressure changes and partitioning effect of blood stream in the presence of localised constriction.

The results suggest that the partitioning effect in the streams of blood flow may be disturbed by turbulence under conditions of normal range of pressure and rate, and there may be a critical range in existence. There is also an equilibration of pressure in the circle of Willis so that the pressure head to distal arteries is maintained, irrespective of the presence of unilateral changes of pressure in the vessels proximal to the circle. It is suggested that one of the functions of the circle of Willis is that of an iso-baric reservoir to enable the cerebral vessels to have a uniform pressure irrespective of the pressure head differences in the supplying arteries, namely, carotid and vertebral.

REFERENCES

Adams, J.H. (1964): Effects of Cerebral Hypoxia in Resuscitation and Cardiac Pacing Edited by G. Shaw, G. Smith, T.J. Thompson. Cassel, London.

Bouchaert, J.J., Heymans, C. (1935): On the Reflex of the Cerebral Blood Flow and the Central Vasomotor Tones. J. Physiol., 84:367.

Brain, R. (1957): Order and Disorder in the Cerebral Circulation. Lancet, 2, 857.

Dandy, W.E. (1944): Intracranial Arterial Aneurysms. Ithaca N.Y. Camstock.

Fawcett, E., Blackford, J.V. (1905): The Circle of Willis: An examination of 700 specimens. J. Anat. & Physiol., 40:63.

Fetterman, G.H. Moran, T.J. (1941): Anomalies of circle of Willis in relation to cerebral softening. Arch. Path., 32:251.

Hamby, W.B. (1952): The Modern Treatment of intracranial aneurysm. N.Y. State Journal of Medicine. Issue No. 20, Vol. 52, pp. 2497-2502.

Kety, S.S. (1957): Cerebral Circulation. Brit. Med. J. 1:1238.

MacDonald, D.A., Potter, J.M. (1951): The Distribution of blood to the brain. J. Physiol. 114:356.

Padget, D.H.: The Circle of Willis, its embroyology and anatomy in intracranial Aneurysm, by Dandy, W.E.

Pickering, G.W. (1955): High Blood Pressure. J.A. Churchill, London.

Windle, B.C.A. (1887): On the arteries forming the circle of Willis. J. Anat. & Physiol. 22:289.