FUNCTIONAL EFFICIENCY OF THE CIRCLE OF WILLIS

IMPLICATIONS FOR RECONSTRUCTIVE SURGERY OF MAIN CEREBRAL ARTERIES

BY

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Stenosis of one main cerebral artery such as the internal carotid is generally thought to predispose to ischaemic damage of the cerebral hemisphere of the same side, and reconstructive surgery is normally carried out on the side of the lesion. This approach implies that the circle of Willis is not a perfect anastomosis, which is an assumption difficult to confirm or deny experimentally. Dr. A. D. Thomson and I have recently measured the fluid-carrying capacity of the internal carotid and vertebral arteries in a series of necropsies, studying particularly the relation between stenosis of these arteries and high systemic arterial pressure (Dickinson and Thomson, 1959, 1960a). A close association was discovered, supporting, though not of course proving, a general hypothesis of "essential" or primary hypertension deduced on theoretical grounds (Dickinson, 1960). The original series of 80 cases was extended to include 47 consecutive unselected cases of cerebral infarction and intracerebral haemorrhage ("strokes") in which no associated cerebral disease was present other than high blood-pressure or arteriosclerosis. There were thus available for study 47 patients with normal brains at death, and the same number of patients dying with strokes, studied under the same conditions. The normal group was originally selected to cover as wide a range of age and blood-pressure as possible.

The full report is to be published elsewhere (Dickinson and Thomson, 1961); but certain aspects of the results have not been discussed, and since they throw some light on the function of the circle of Willis they seem to merit a brief report.

Methods

The technique used has been described by Dickinson (1960) and by Dickinson and Thomson (1960a). Standard cannulas were tied into the origins of each main artery in turn, the ends were cut across where the arteries entered the cranial cavity, and 5% 0.880 ammonia in tap-water was run through at a constant pressure of 140 mm. Hg to determine the maximum fluid-carrying capacity of each artery. A numerical value in ml./sec. could thus be given to describe the effective calibre of each artery individually; and by adding the values together estimates could also be made of the relative fluid-carrying capacity of both vertebral and both internal carotid arteries taken together.

Results

A clear-cut distinction between people with strokes and people with normal blood-pressure was demonstrated in terms of the fluid-carrying capacity of all four main cerebral arteries taken together, and reasons were given for suggesting that the contribution of extra-

cranial and intracranial cerebral artery stenosis to the genesis of stroke is approximately equal (Dickinson and Thomson, 1961).

The clear separation of cases of stroke from the other groups encourages the belief that the method of measurement, though crude, is capable of giving meaningful results. This belief is strengthened by the original results, showing that highly significant correlations of mean blood-pressure with total cerebral artery fluid-carrying capacity could be obtained from quite a small series of cases.

Localization of Fore-brain Lesions in Relation to the Side of Predominant Internal Carotid Artery Stenosis.—The Figure shows that for the 31 unilateral fore-brain lesions (haemorrhages and infarcts taken together) there is no appreciable tendency for stenosis to be more severe

Relative sizes of each internal carotid artery in 31 cases of stroke in which the lesion was localized to one side of the fore-brain. The artery on the side opposite the brain lesion is shown as having a fluid-carrying capacity of 0 and the right-hand column represents the number of ml./sec. by which the fluid-carrying capacity of the ipsilateral internal carotid artery was greater or less than that of the control side.

on the side of the brain lesion. The artery of the opposite side is shown on the left as "0," and the right-hand column indicates for each case the number of ml./sec. by which the internal carotid artery on the side of the lesion was greater or less in fluid-carrying capacity than that of the opposite side. The interrupted lines indicate two cases of complete internal carotid thrombosis with extension of the clot to the middle cerebral artery and infarction of the ipsilateral hemisphere. In the other case of internal carotid thrombosis in this series the lesions were bilateral, and are thus not shown in the Figure. "Appropriate" has been used to describe predominant stenosis of the internal carotid on the same side as the lesion, and "inappropriate" in cases of predominantly contralateral artery stenosis. If we ignore the two cases of spreading internal carotid thrombosis the remaining cases show no tendency for predominantly ipsilateral stenosis to be found more often. 15 cases were appropriate but 14 were inappropriate: furthermore, on average the artery on the side of the lesion had, if anything, a greater fluid-carrying capacity than that of the opposite side (+0.3 ml./sec.). This value is obviously not significantly different from zero.

Relative Degree of Stenosis of the Internal Carotid and Vertebral Arteries in Cases of Strokes Localized to the Fore- or Hind-brain.—In the 47 control cases with normal brains the fluid-carrying capacity of the vertebral arteries taken together was 38% of the total capacity of all four arteries added together. For 37 cases of spontaneous uncomplicated stroke—that is, stroke not associated with malignant hypertension or with gross circulatory disturbances—the vertebral arteries contributed on average slightly less of the total

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(35%). Analysis of the totals by χ^2 shows this difference to be significant ($0.001 < P < 0.01$). Despite this, fore-brain lesions predominated in this series, and there were only three uncomplicated cases of exclusively hind-brain lesions.

Discussion

The almost equal numbers of appropriate and inappropriate cases in the Figure suggest that the anterior part of the circle of Willis is a highly efficient anastomosis, and that, unless thrombus extends across the circle of Willis into the middle cerebral artery, unequal internal carotid stenosis, even of extreme degree, is just as likely to produce damage in the contralateral as in the ipsilateral hemisphere. It seems probable that localization of ischaemic brain lesions mainly depends on local intracranial artery disease and occlusion. Meyer and Denny-Brown (1957) showed, in monkeys, that even occlusion of the middle cerebral artery need not necessarily cause infarction of the brain if the circle of Willis is intact and if the systemic blood-pressure is adequate.

The predominance of fore-brain strokes despite significantly greater reduction in vertebral artery calibre than in internal carotid calibre might have several explanations.

1. The perfusion technique may measure vertebral arteries more accurately than internal carotid arteries—for example, because greater turbulence in the larger internal carotid arteries might lead to underestimate of fluid-carrying capacity in cases with high flow-rates.
2. Some hind-brain lesions may have been overlooked. This possibility cannot be excluded, since in most cases the brains had to be sectioned without fixing, because of undergraduate teaching requirements. However, care was taken to inspect thin coronal sections, and it seems improbable that many hind-brain lesions can have been missed.
3. The fore-brain may be intrinsically more liable to ischaemic damage because of its high blood-flow requirements, or because of its relatively poor blood-supply.

It is not possible at present to say which of these possibilities is closest to the truth, but the results, anyway, suggest a fairly free anastomosis between anterior and posterior parts of the circle of Willis. By chance the three cases of exclusively hind-brain lesions showed relatively greater internal carotid than vertebral artery stenosis, and it seems almost a pity that we have to reject the agreeably fantastic conclusion that the fore-brain is supplied by the vertebral and the hind-brain by the internal carotid arteries.

In this small series it has not been possible to show that predominantly internal carotid stenosis favours fore-brain lesions and predominantly vertebral stenosis favours hind-brain ones. The investigation of Hutchinson and Yates (1957) supported an important role for the vertebral arteries in causing hind-brain stroke. These authors recognized minor degrees of hind-brain, particularly cerebellar, softening more often than might be expected from the present series, perhaps because they were able to identify lesions better in a series of brains fixed before sectioning.

Hypertension is "extremely common" in vertebral-basilar arterial disease (Meyer *et al.*, 1960). Evidence of hypertension was found in the series of Hutchinson and Yates (1957) rather more often in cases of predominantly vertebral artery disease than in cases of predominantly internal carotid artery disease (personal communication from Dr. P. O. Yates). Dickinson and Thomson (1959, 1960a) found a higher association with

hypertension for vertebral than for internal carotid artery stenosis, and have pointed out that this fits well with the idea of a blood-pressure-regulating centre in the medulla. In addition, hind-brain strokes are probably more strongly associated with high blood-pressure than are fore-brain strokes. Russell (1954) has shown this tendency for pontine haemorrhage; Hyland and Levy (1954) and McKissock *et al.* (1960) for cerebellar haemorrhage; and Dickinson and Thomson (1960b) for strokes of all varieties in a large series.

These trends are all in the same direction, though none individually are large; and while the phenomenon appears to be a genuine one, it cannot have much significance in any individual case. It is noteworthy that, although size of the vertebral artery was rather better correlated with blood-pressure than was the size of the internal carotid artery in Dickinson and Thomson's (1960a) series, the highest negative correlation (-0.82) was found when the fluid-carrying capacity of all four cerebral arteries taken together was compared with systemic blood-pressure. Though the posterior communicating arteries are often small (<1 mm. in 22% of cases) they are only rarely completely absent on one side (0.6% of cases) and never absent on both (Alpers *et al.*, 1959).

Implications for Reconstructive Surgery of Main Cerebral Arteries.—It seems from this investigation and analysis that in most cases of cerebral ischaemia due to arteromatous stenosis of the neck arteries improvement in blood-carrying capacity of any of the four main cerebral arteries would be equally valuable, and that the indications for reconstructive surgery might be based more on the size, nature, and accessibility of the lesions rather than on their apparent relation to the site of presumed brain damage. This does not, of course, apply to acute carotid obstruction, in which cerebral ischaemia is almost always ipsilateral.

Summary

Analysis of the data from a necropsy perfusion study of the four main cerebral arteries in their cervical course suggests that stenosis of these arteries, though an important cause of stroke, has very little influence on the actual site of the lesion, which is probably determined mainly by local arterial disease within the cranial cavity.

The indirect evidence of this study suggests that the circle of Willis is a highly efficient anastomosis in man, even when considerable arterial disease is present.

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REFERENCES

- Alpers, B. J., Berry, R. G., and Paddison, R. M. (1959). *Arch. Neurol. Psychiat. (Chicago)*, **81**, 409.
 Dickinson, C. J. (1960). D.M. Thesis, University of Oxford.
 — and Thomson, A. D. (1959). *Lancet*, **2**, 46.
 — (1960a). *Clin. Sci.*, **19**, 513.
 — (1960b). *Lancet*, **2**, 342.
 — (1961). *Clin. Sci.*, **20**. In press.
 Hutchinson, E. C., and Yates, P. O. (1957). *Lancet*, **1**, 2.
 Hyland, H. H., and Levy, D. (1954). *Canad. med. Ass. J.*, **71**, 315.
 McKissock, W., Richardson, A., and Walsh, L. (1960). *Brain*, **83**, 1.
 Meyer, J. S., and Denny-Brown, D. (1957). *Neurology (Minneapolis)*, **7**, 447.
 — Sheehan, S., and Bauer, R. B. (1960). *Arch. Neurol. (Chicago)*, **2**, 27.
 Russell, D. S. (1954). *Proc. roy. Soc. Med.*, **47**, 689.