THE PATHOLOGICAL EFFECTS OF CEREBRAL ARTERIOGRAPHY

BY

T. CRAWFORD

From the Department of Pathology, St. George's Hospital Medical School, London

Cerebral arteriography is now carried out on a very large number of cases each year in the neuro-surgical centres of the world. Usually this investigation is without harmful effect and the information gleaned commonly provides a precise diagnosis and leads to the institution of palliative or curative treatment. Nevertheless, there is inevitably some degree of damage to the arterial wall and in a very small proportion of cases harmful effects ensue. It seems important therefore that those who prescribe and carry out this procedure should be aware of the degree of damage commonly inflicted and of the occasional harmful effects which may follow. The method by which the arterial puncture wounds inflicted during arteriography heal "normally" has been described already (Crawford, 1956) and requires summarizing only briefly here. After some haemorrhage the track is filled by a fibrin plug. Endothelial cells proliferate and cover the central end of the plug by about the seventh day and thereafter the plug becomes organized by a dual mechanism from the lumen and from the adventitia similar to that seen in the organization of arterial

mural thrombi (Crawford and Levene, 1952). By about the 28th day organization is complete and a fibrous scar marks the site of puncture. Over the succeeding months a few elastic and smooth muscle fibres develop in the scar but even at eight months there is no proper reconstitution of the media or of the elastic laminae (Fig. 1).

This "normal" healing process occurs in the great majority of needle-tracks but departure from the simple healing process may occur in a variety of ways and produce effects of varying significance. It is with these "abnormal" effects of arterial puncture that the present paper is concerned. The descriptions which follow are based on necropsy material from 75 subjects on whom arteriography had been carried out at intervals varying from two hours to eight months before death. This material has come from many different sources and has not been confined to the necropsies from a single neurosurgical unit.

Concealed Haemorrhage

There is always some haemorrhage from the arterial puncture wound. In most cases it leads to no more than staining of the carotid sheath for a distance of three or four centimetres above and below the puncture; but quite often the entire carotid sheath is distended with blood from the arch of the aorta to the base of the skull (Fig. 2) and occasionally the blood breaks out from the sheaths and spreads widely amongst the structures of the neck and mediastinum (Fig. 3).

It has been noticed that haemorrhage is usually minimal in young subjects and most marked in elderly hypertensives or when the vessel has been transfixd by the needle (or punctured repeatedly) with the infliction of two or more wounds (Fig. 4).

These haemorrhages are such a striking feature at necropsies on recently punctured cases that it is surprising to the pathologist to learn that significant symptoms rarely occur. Transient pain, tenderness, and dysphagia may be admitted on questioning. Persistent damage has not been seen in this study.

FIG. 1.—Site of arterial puncture made eight months before death. Elastic tissue has not regenerated. Weigert's elastic stain, $\times 65$.

FIG. 2

FIG. 4

Abscess Formation

Pyogenic infection has not been found in any of the necropsy material: but in several of the cases coming to necropsy within a week of arteriography the arterial wall was found to be heavily infiltrated with polymorphs in the neighbourhood of the needle track while a few showed sterile abscess formation in the adventitia and sheath of the artery (Fig. 5). There can be little doubt that these inflammatory reactions and sterile abscesses are due to injection of

FIG. 3

FIG. 2.—Carotid sheath distended with blood. The arterial puncture site can be seen just below the bifurcation.

FIG. 3.—Extensive haemorrhage into the tissues of the neck and mediastinum after carotid arteriography in a hypertensive woman.

FIG. 4.—Twin needle tracks in the wall of a carotid artery. Weigert's elastic stain, $\times 25$.

contrast medium into the tissues of the arterial wall and sheath—an impression confirmed by the finding of numbers of foamy macrophages amongst the polymorphs. Although harmful effects of these reactions have not been recognized, damage to the important nerves in close anatomical relationship to the carotid arteries could well result, and the further complication of thrombosis is more likely when the vessel wall has been damaged by an intramural injection.

FIG. 5.—Sterile abscess in the carotid sheath. The arterial adventitia is seen above and to the left. Picro-Mallory, $\times 280$.

Thrombosis

This is perhaps the most important complication of arterial puncture for it is important not only in its direct effects but also as the precursor of embolism.

Occlusive Thrombosis.—Occlusive thrombosis affecting both common carotid arteries has been found once. In both vessels rapidly formed thrombus extended up from the puncture as far as the bifurcation. The patient was an elderly hypertensive man with massive intracranial haemorrhage and it was impossible to distinguish the effects of the thrombosis on the course of the disease. Obstruction of one carotid artery has been found four times. The subjects were all over 55 years of age and there was advanced atherosclerosis in the affected arteries. Here again the possible effects of the arterial obstruction were obscured by the progress of the severe subarachnoid haemorrhages for the localization of which the arteriographies had been performed but in one patient dying on the third day after arteriography early softening was found in the hemisphere on the affected side.

Mural Thrombosis.—Mural thrombosis at the site of arterial puncture is a frequent finding—in fact, some minor degree of it is an almost constant occurrence. Usually it consists merely of slight lateral extensions of the fibrin plug in the needle track over the adjacent parts of the intima. A thrombus of this type will soon be incorporated into the intima and will ultimately lead to nothing more serious than a localized thickening of that layer. Sometimes, however, the mural thrombus assumes an irregularly pedunculated or polypoid form (Fig. 6). The danger of embolism is then self evident.

Cerebral Embolism

Embolism results from detachment of a mural thrombus which has formed at the site of the puncture. The effects of embolism will, of course, vary with the size and site of lodgment of the embolus and clinically they are likely to be obscured by, or mistaken for, the progress of the disease process. In the present series of cases there was one instance of fatal embolism of the basilar artery following vertebral arteriography. In five further cases necropsy revealed multiple cerebral infarcts though death was due to the progress of the primary disease. In these cases the site of carotid arteriography was the only likely site of origin for emboli. The patients

FIG. 6.—Mural thrombus with narrow attachment to the central end of the needle-track. Weigert's elastic stain, $\times 90$.

were under investigation for subarachnoid haemorrhage. They showed rapid deterioration following arteriography, and necropsy confirmed that this was not due to recurrence of haemorrhage, though in each case the original damage from haemorrhage was of a grave nature.

Aneurysm Formation

Examination of the damaged vessel after recent arteriography punctures might lead one to anticipate that aneurysmal dilatation would be a frequent sequel but in fact trouble of this kind has not been encountered in the material studied. Two types of aneurysm—both of more pathological interest than clinical significance—have, however, been seen.

Traumatic False Aneurysm.—Two examples have been seen in which a small false aneurysm had developed through the needle track. One patient, a child aged 5 years, died 110 days after arteriography and at necropsy there was a berry-like nodule 1 cm. in diameter on the wall of the common carotid artery at the site of puncture. Serial sectioning showed it to be a false aneurysm lying outside the media but communicating with the lumen by an endothelialized track (Fig. 7). The second example

FIG. 7.—Traumatic false aneurysm at the site of needle-puncture 110 days before death. Weigert's elastic stain, $\times 25$.

was a similar lesion in an elderly woman who died on the 28th day after arteriography. In neither case did the aneurysm have any clinical effects.

Traumatic Dissecting Aneurysm.—Small dissecting aneurysms, consisting of blood-containing cavities in the substance of the media, have been seen to follow arterial puncture on nine occasions. These lesions have varied from 0.5 to 4.0 cm. in length and have caused the formation of a rather prominent ridge on the intimal surface with sometimes some overlying mural thrombus. A typical example is shown in Fig. 8. In two instances, in which death

FIG. 8.—Traumatic dissecting aneurysm at the site of a needle-puncture eight days before death. Weigert's elastic stain, $\times 30$.

occurred on the 18th and 24th days respectively the dissecting spaces in the vessel wall had become extensively endothelialized. It is interesting to note that these traumatic lesions occur at a similar depth in the media to that at which spontaneous dissecting aneurysms develop.

Discussion

It is important to appreciate that the lesions which have been described in the preceding sections occurred amongst many thousands of patients subjected to arteriography. No quantitative assessment of the frequency of each type of lesion can be made for many of the specimens were submitted to the author precisely because of some unusual occurrence. Nevertheless, it is possible to recognize certain factors which increase the liability to the development of complications. Advanced age, hypertension, and severe atherosclerosis are three linked factors which come into this category and which have been present in a high proportion of the cases in which thrombosis has occurred. On the technical side two further points emerge. In the first place, multiplicity of punctures, as when the vessel is transfixated, or when repeated punctures are made, greatly increases the amount of haemorrhage and the risk of thrombosis and embolism: and

secondly, puncture close to the point of bifurcation of the common carotid artery carries a greater risk of complications than when the artery is entered a few centimetres lower down. This is associated with the fact that the region of the bifurcation is commonly the site of advanced and perhaps calcified atheromatous plaques. When the needle penetrates such a plaque an irregular gash in the wall may result and form a suitable nidus for platelet and fibrin deposition.

It would be presumptuous for the pathologist to offer unsolicited advice to the clinician and radiologist in a matter of this kind, but if advice were sought it might perhaps take the following form: reserve arteriography for the study of grave disease; beware the elderly arteriosclerotic hypertensive subject; avoid puncturing the immediate neighbourhood of the carotid bifurcation; puncture the vessel once only and do not transfix it; consider the possibility of embolism when fresh neurological manifestations follow the investigation.

Summary

Lesions resulting from arterial puncture have been studied in necropsy material from 75 patients dying at intervals varying from a few hours up to eight months after cerebral arteriography. The most serious ill effects observed have been carotid thrombosis and cerebral embolism. It has not been possible to assess the frequency of these rare misfortunes. They are more liable to occur in elderly hypertensive arteriosclerotic subjects.

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REFERENCES

- Crawford, T. (1956). *J. Path. Bact.* In the press.
—, and Levene, C. I. (1952). *Ibid.*, 64, 523.