gonad is usually intra-abdominal, most often in the position of the ovary, but not infrequently it is in the inguinal canal, when it may be associated with a hernia. Histological examination of a gonadal biopsy shows it to be a testis. The vaginal smear shows a degree of cornification such as would not be found in primary amenorrhoea due to hypogonadism; the source of the oestrogens is the testes. The paper by Grumbach *et al.* (1955) is an important contribution to the further understanding of the relationship between the various types of male pseudohermaphroditism and the gonadal dysgenesis syndrome.

A further, related variety of male pseudohermaphroditism was described by me (Swyer, 1955). By means of chromosomal sex determination techniques, I concluded that two patients presenting with primary amenorrhoea were apparently of male sex. Unfortunately, the opportunity of doing gonadal biopsies on these patients has not arisen, but it would seem that little doubt of their genetic sex need be entertained, since Greenblatt and Carmona (1955) described a very similar case of what they called a "eunuchoid female," whose chromosomal sex was male and on whom laparotomy revealed rudimentary gonads, histological examination of which showed a cortex composed of stroma without follicles and a medulla and hilus containing many "Leydig cells." These patients differ from the "classical" type of pseudohermaphrodite described above; they appear as eunuchoidal "females" with primary amenorrhoea, having normal pubic and axillary hair, little or no mammary development, and normal external genitalia except for marked enlargement of the clitoris in one of my patients and in that of Greenblatt and Carmona. The cervix is nearly normal, the uterus hypoplastic, the vaginal smear hypo-oestrogenic. Strangely enough, the oestrogen levels seemed to be definitely higher than those found in other cases of primary amenorrhoea. Both my patients responded to oestrogen treatment by producing uterine bleedings on withdrawal. The possible significance of these findings and the differential diagnosis of primary amenorrhoea of endocrine origin have already been discussed (Swyer, 1955).

It is clear that when confronted with a patient complaining of primary amenorrhoea the possibility of intersexualization must be borne in mind. It goes without saying that if such a diagnosis is established neither the patient nor her relatives should be given any grounds for suspicion that an anomaly of sexual differentiation exists. To explain that there is some degree of abnormal development of the genital organs which would prevent conception but not normal marriage (apart from child-bearing) should be quite sufficient.

REFERENCES

- Allott, E. N., and Simmons, J. H. (1951). *British Medical Journal*, **2**, 568.
 Armstrong, C. N. (1955). *Ibid.*, **1**, 1173.
 Barr, M. L., and Bertram, E. G. (1949). *Nature (Lond.)*, **163**, 676.
 — Bertram, L. F., and Lindsay, H. A. (1950). *Anal. Rec.*, **107**, 283.
 Beatty, D. C., Champ, C. J., and Swyer, G. I. M. (1953). *British Medical Journal*, **1**, 1369.
 Bishop, P. M. F. (1954). *Recent Advances in Endocrinology*, Churchill, London.
 Bongiovanni, A. M., and Eberlein, W. R. (1955). *J. clin. Endocr.*, **15**, 1524.
 Burston, R. A., and Garrod, O. (1952). *Proc. roy. Soc. Med.*, **45**, 70.
 Caughey, J. E., James, A., and Macleod, E. K. (1952). *British Medical Journal*, **1**, 1216.
 Chalmers, T. M., Fitzgerald, M. G., James, A. H., and Scarborough, H. (1956). *Lancet*, **1**, 127.
 Conn, J. (1955). *J. Lab. clin. Med.*, **45**, 3.
 Crooke, A. C. (1955). *Lancet*, **2**, 1045.
 Davidson, W. M., and Smith, D. (1954). *British Medical Journal*, **2**, 6.
 Deming, O. B., and Luetscher, J. A. (1950). *Proc. Soc. exp. Biol. (N.Y.)*, **73**, 171.
 Diezfelussy, E., Plantin, L.-O., Birke, G., and Westman, A. (1955). *Acta endocr. (Kbh.)*, **18**, 356.
 Dorfman, R. I., and Ungar, F. (1953). *Metabolism of Steroid Hormones*. Burgess, Minneapolis.
 Escanilla, R., and Lissner, H. (1942). *J. clin. Endocr.*, **2**, 65.
 Farrell, G. L., Banks, R. C., and Kolestsky, S. (1956). *Endocrinology*, **58**, 104.
 Gaunt, R., Renzi, A. A., and Chart, J. J. (1955). *J. clin. Endocr.*, **15**, 621.
 Goldberg, M. B., and Maxwell, A. F. (1948). *Ibid.*, **8**, 367.
 Gordon, E. S., Chart, J. J., Hagedorn, D., and Shipley, E. G. (1954). *Obstet. and Gynec.*, **4**, 39.
 Greenblatt, R. B., and Carmona, N. (1955). *J. clin. Endocr.*, **15**, 877.
 Gross, J., and Pitt-Rivers, R. (1952). *Brit. med. Bull.*, **8**, 136.
 Grumbach, M. M., Van Wyk, J. J., and Wilkins, L. (1955). *J. clin. Endocr.*, **15**, 1161.
 Hetzel, B. S., McSwiney, R. R., Mills, I. H., and Prunty, F. T. G. (1956). *J. Endocr.*, **13**, 112.
 Hubble, D. (1952). *Lancet*, **1**, 1123.
 — (1955). *Ibid.*, **1**, 1.

- Jailer, J. W. (1953). *Bull. N.Y. Acad. Med.*, **29**, 377.
 Jost, A. (1947). *C.R. Soc. Biol. (Paris)*, **141**, 126.
 Kendall, E. C. (1952). *Edinb. med. J.*, **59**, 1.
 Laragh, J. H., and Stoerk, H. C. (1955). Quoted by Gaunt *et al.* (1955).
 Lewis, A. G. (1952). *Proc. roy. Soc. Med.*, **45**, 63.
 Luetscher, J. A. (1952). *Ann. Rev. Med.*, **3**, 75.
 — and Axelrad, B. J. (1954). *J. clin. Endocr.*, **14**, 1086.
 Maddock, W. O., Leach, R. B., Klein, S. P., and Myers, G. B. (1953). *Amer. J. med. Sci.*, **226**, 509.
 Mader, I. J., and Iseri, L. T. (1954). *J. Lab. clin. Med.*, **44**, 895.
 Moore, K. L., and Barr, M. L. (1953). *J. comp. Neurol.*, **98**, 213.
 — (1955). *Lancet*, **2**, 57.
 — Graham, M. A., and Barr, M. L. (1953). *Swg. Gynec. Obstet.*, **96**, 641.
 Nelson, D. H., and Samuels, L. T. (1952). *J. clin. Endocr.*, **12**, 519.
 Norymberski, J. K. (1952). *Nature (Lond.)*, **170**, 1074.
 — Stubbs, R. D., and West, H. F. (1953). *Lancet*, **1**, 1276.
 Oelbaum, M. H. (1952). *British Medical Journal*, **2**, 110.
 Paschkis, K. E., and Cantarow, A. (1951). *Ann. intern. Med.*, **34**, 669.
 Polani, P. E., Hunter, W. F., and Lennox, B. (1954). *Lancet*, **2**, 120.
 Raynaud, A., and Frilley, M. (1947). *Ann. Endocr. (Paris)*, **8**, 400.
 Riis, P. (1955). *Dan. med. Bull.*, **2**, 190.
 Romatowski, H., Tolksdorf, M., and Wiedemann, H. R. (1955). *Klin. Wschr.*, **33**, 911.
 Schneider, R. W., Van Ommen, R. A., and Hoerr, S. O. (1952). *J. clin. Endocr.*, **12**, 423.
 Sheehan, H. L. (1939). *Quart. J. Med.*, **8**, 277.
 — and Summers, V. K. (1949). *Ibid.*, **18**, 319.
 — (1952). *British Medical Journal*, **1**, 1214.
 Simmonds, M. (1914). *Dtsch. med. Wschr.*, **40**, 322.
 — (1916). *Ibid.*, **42**, 190.
 — (1918). *Ibid.*, **44**, 852.
 Simpson, S. A., Tait, J. F., Wettstein, A., Neher, R., von Ew. J., Schindler, O., and Reichstein, T. (1954a). *Experientia (Basel)*, **10**, 132.
 — (1954b). *Helv. chim. acta*, **37**, 1163.
 — (1954c). *Ibid.*, **37**, 1200.
 Slessor, A. (1952). *Proc. roy. Soc. Med.*, **45**, 67.
 Steiger, M., and Reichstein, T. (1937). *Helv. chim. acta*, **20**, 1164.
 Summers, V. K. (1952). *British Medical Journal*, **1**, 787.
 Sun, L. C. Y., and Rakoff, A. E. (1956). *J. clin. Endocr.*, **16**, 55.
 Swyer, G. I. M. (1955). *British Medical Journal*, **2**, 709.
 Vogt, M. (1943). *J. Physiol.*, **102**, 341.
 Wilkins, L., and Cara, J. (1954). *J. clin. Endocr.*, **14**, 287.
 — Crigler, J. F., Silverman, S. H., Gardner, L. I., and Migeon, C. J. (1952a). *Ibid.*, **12**, 277.
 — (1952b). *Ibid.*, **12**, 1015.
 — (1952c). *Ibid.*, **12**, 257.
 — Grumbach, M. M., and Van Wyk, J. J. (1954). *Ibid.*, **14**, 1270.
 Williams, D. I. (1952). *British Medical Journal*, **1**, 1264.

CEREBRAL ANGIOGRAPHY IN A NEUROSURGICAL SERVICE

BY

STEVENS DIMANT, M.B., F.R.C.S.

Senior Registrar, Neurosurgical Department, Manchester
Royal Infirmary

C. P. MOXON, M.R.C.S., L.R.C.P., D.M.R.D.

Consultant Radiologist, the Midlands Centre for
Neurosurgery and Queen Elizabeth Hospital, Birmingham

AND

N. A. LEWTAS, M.B., M.R.C.P., D.M.R.D.

Consultant Radiologist, United Sheffield Hospitals and
Rotherham Hospitals

(From the Departments of Neurosurgery and Neuro-
radiology, Manchester Royal Infirmary)

Carotid angiography has become the main auxiliary method in the diagnosis of intracranial lesions, and systematic accounts of its technique as well as the radiological appearances have been given by many authors (Engeset, 1944; Wickbom, 1948; Bull, 1949; Lima, 1950; Dowling, 1950; Curtis, 1951). The procedure has become so standardized that its performance is not beyond the skill and facilities of many x-ray departments—quite apart from specialized neuroradiological departments working close to a neurosurgical unit. For this reason alone some account of its place in diagnosis is desirable. Furthermore, the development and elaboration of methods of isotope localization (Sweet and Brownell, 1955) make it essential that a careful assessment be made of the value of angiography in a large series of cases presenting as neurosurgical problems.

The investigation has been limited to a consecutive four-year series, so that the figures in no way represent our total experience in any pathological group. In the

Manchester Royal Infirmary, 1,556 carotid angiograms were performed on 1,007 patients referred to the neurosurgical department during the period January, 1950, to March, 1954. The object of this paper is to review these cases, with particular regard to the indications for angiography, the accuracy of the information so obtained, and the discomforts and complications that were engendered.

General Considerations

The practical application of cerebral angiography has resulted in considerable changes in the pattern of current neurosurgical practice. The frequent need for surgical treatment in cases of subarachnoid haemorrhage, and the growing recognition of the importance of fully investigating suspected cases of brain tumour, have increased greatly the pressure on neurosurgical services. This has been met partly by the introduction of out-patient angiography, many patients thereby being adequately examined without admission to hospital. Patients referred from hospitals elsewhere are frequently seen by appointment, and, after clinical examination, pre-arranged angiography is performed when indicated. These patients can return the same day to their own hospitals unless urgent surgery is required. In this way the limited number of neurosurgical beds available is used mainly for acute cases and for the operative period itself. The accompanying histogram shows the relative increase in angiography, and especially out-patient angiography, during the period under review.

Histogram showing relative increase in angiography.

General anaesthesia is ideal for both the patient's comfort and the radiologist's ease. Certainly in children and in patients restless from fear, or uncooperative because of a slightly depressed conscious level, a general anaesthetic is employed. In cases of raised intracranial pressure a local analgesic is safer (about 90% of our examinations are made in this way), and it is also preferable for out-patients. The percutaneous technique as described by Lindgren (1947) is now used exclusively. Age itself is not a limiting factor: with diodone as the

contrast medium sensitivity tests have not been found necessary, as no serious reactions have been encountered. For diagnosis satisfactory films in antero-posterior and lateral projections are needed, and if both of these are not obtained the examination is regarded as a failure; it is repeated at a later date. In this department 3% of all angiograms were thus classed as failures during 1953. They were successfully repeated at the second attempt.

Angiographic diagnosis is made on the appearances of abnormal vessels, displacements of normal vessels, or a combination of both, and as given here is unrevised by any subsequent investigation. In assessing the value of angiography as a diagnostic procedure, the abnormalities are divided into two major groups—vascular disorders and space-occupying lesions.

Vascular Disorders

The value of angiography in vascular disorders is obvious. It has revolutionized the surgical approach to intracranial aneurysms and angiomas. In degenerative vascular disease it may demonstrate thromboses of major vessels, but where the aetiology of the clinical picture is in doubt the main value of angiography is in the exclusion of tumours.

In this series 281 patients were shown angiographically to have a vascular lesion. A final diagnosis of a vascular disorder was made in a further 133 in whom angiography was negative or did not reveal the full pathology. It should be noted, however, that this apparently large number of cases includes 78 cases classified as cerebrovascular disease. Of these, two were diagnosed as cerebral thrombophlebitis. The remaining 76 were thought to have arteriosclerosis, because in many the age, clinical appearances, and blood pressure suggested that. Both pneumoencephalography in the more doubtful cases and subsequent observation for periods up to four years have supported that diagnosis. The composition of the foregoing figures, each under the final diagnosis, is shown in Table I.

TABLE I

Aneurysms :	
Diagnosed angiographically	196
Missed (found at necropsy—see below)	2
Angiomas :	
Diagnosed angiographically	36
Intracerebral clots (diagnosed angiographically) :	
Due to aneurysm (included above)	(8)
" " angioma (" ")	(2)
" " other causes	3
Subarachnoid haemorrhage with normal or failed angiograms :	
Satisfactory films	45
Failed angiograms	4
Subdural haematomas :	
Diagnosed angiographically :	
(a) Due to trauma	2
(b) " " ruptured cerebral aneurysm (included above)	(2)
Missed (bilateral frontal clots found at operation)	1
Degenerative vascular disease :	
Cerebral thrombosis seen angiographically	27
Internal carotid thrombosis seen angiographically	17
Mistaken angiographically for olfactory groove tumour	3
Clinical cerebrovascular disease with	
(a) Satisfactory angiograms	73
(b) Failed "	5
	281
	133

Aneurysms

In most cases where aneurysms exist their situation is accurately shown. Either a very small or thrombosed aneurysmal sac or an origin from the vertebral system is likely to account for most of those that escape detection. So far as we know, three aneurysms have been overlooked in

this series. (1) A small anterior communicating aneurysm which bled again and caused death before angiography had been completed. The aneurysm could not be seen on the lateral film, which, however, demonstrated severe spasm of the anterior cerebral artery. At this stage angiography had to be abandoned, so that A.P. films were not obtained and the angiograms are classed as failures. (2) A small carotid aneurysm (which had not bled) in a patient with severe arteriosclerosis who died following the rupture of an anterior cerebral aneurysm which was clearly demonstrated in the angiograms. (3) A basilar aneurysm which caused death by rupture into the third ventricle. Vertebral angiograms were not made, as carotid angiograms in the acute stage of subarachnoid haemorrhage demonstrated a carotid aneurysm which was successfully treated by "trapping," and which at operation showed clear evidence of recent leakage.

Angiomas

The reliability with which these malformations can be precisely shown is well recognized. Attention should be drawn, however, to difficulties that may arise when they have caused a large intracerebral clot. Compression or thrombosis may then cause the fistulous vessels to be partly obliterated, so that only the closest scrutiny may show their remnants, or they may not be visible at all. Thus in this series there were two patients in whom the initial angiogram showed only the vascular displacement due to a clot. In both the clot was evacuated through a burr-hole as an emergency. A subsequent injection demonstrated a deep temporal angioma in each case.

Intracerebral Clots

In 13 instances angiography indicated the site of an intracerebral clot that was surgically evacuated. Among these there were, in addition to the two angiomas just alluded to, eight cases due to ruptured aneurysms that were demonstrated simultaneously at the first injection. Of the remaining three, arteriosclerosis with hypertension existed in one, while the aetiology remained obscure in the other two.

Electroencephalography has been an important adjunct to clot location. Of the six patients on whom it was performed precise location by slow delta activity with phase reversal was obtained in four. In the other two less accurate information was obtained, so that the correct distinction between the posterior subfrontal and the anterior temporal situations of intracerebral clots was not made.

Subarachnoid Haemorrhage

It has been the practice here to perform bilateral carotid angiography in all cases at the earliest opportunity, unless there were clear contraindications (see below). Although a comprehensive examination of the cerebral vessels requires vertebral angiography, and to be absolutely certain bilateral vertebral as well as bilateral carotid angiography, we have not attained the ideal of making vertebral angiograms in all cases of subarachnoid haemorrhage. The main reason for this failure is that we have not as yet perfected a safe and sure method of percutaneous vertebral angiography. It is performed when bilateral carotid angiograms are negative—(a) in severe or repeated subarachnoid haemorrhage, especially in young people, and (b) when there is clinical evidence of a lesion in the posterior fossa, particularly when an angioma is suspected. It is in these cases of posterior fossa angiomas that hypertrophy of the vertebral artery makes percutaneous puncture of that vessel almost always successful.

Vertebral angiography has been used chiefly for vascular lesions, as we find ethyl iodophenylundecylate ("myodil") ventriculography an efficient and safe method for the demonstration of posterior fossa tumours. Of 80 patients subjected to vertebral angiography in the period under review, abnormalities have been demonstrated in eight, and each was an angioma presenting with subarachnoid haemorrhage. It is of some importance that carotid injections were per-

formed in six of those eight and in each case showed the angioma—including two that were limited to the posterior fossa.

It should be mentioned here that open vertebral angiography is a minor operation, and much safer than repeated attempts at percutaneous puncture. We believe that aneurysms of the basilar artery and its branches are uncommon, and when they are present surgery has less to offer; this is especially true in patients over the age of 50.

The selection of patients and the optimum time for angiography are major considerations in the early management of subarachnoid haemorrhage. Attention must be drawn to the importance of the diagnosis being carefully made by lumbar puncture whenever possible. Evidence of xanthochromia at the first lumbar puncture is invaluable, but a small subarachnoid haemorrhage or, more especially, an intracerebral leakage may not reveal itself by blood in the fluid obtained by early lumbar puncture. In this series, where many of the cases were investigated under difficulties elsewhere in the acute stage, the large number of normal angiograms may be partly explained by the initial diagnosis of subarachnoid haemorrhage having been mistaken.

The optimum time for angiography is a difficult problem; some (Bull, 1949) have advocated it at once in all cases, others (Petit-Dutaillis *et al.*, 1952) have recommended a deliberate delay. It is the ever-present risk of further haemorrhage that has led us to advise angiography at the earliest moment provided the patient is not in deep coma and is, of course, fit to travel if that is necessary.

The key to this problem may rest in the state of the patient rather than in the period of time that has followed subarachnoid haemorrhage. Our experiences indicate that active bleeding with or without focal signs does not contraindicate immediate arteriography if the level of consciousness remains good; indeed, it is our impression that the intra-arterial injection of diodone itself may encourage thrombosis in the aneurysmal sac at the site of rupture. There is a strong suggestion of clotting in the sudden cessation of pain and recession of third-nerve palsy that occasionally follows the injection that has revealed a carotid aneurysm. Although our observations show that when coma is deepening angiography causes a further deterioration, sometimes in such cases an intracerebral or even a subdural clot may be demonstrated as the critical factor, removal of which aids recovery. It may be in the best interests of the patient, however, that clots diagnosed by focal signs such as hemiparesis associated with drowsiness and located by a focus of delta activity on the E.E.G. should be evacuated by means of a burr-hole before angiography is undertaken. Even when the bulk of the clot has been solid, usually a few millilitres of semi-fluid blood can be removed through a large (3-mm.) cannula, and the pressure is so reduced that the patient will recover sufficiently for angiography and formal surgery.

It is particularly important in cases of subarachnoid haemorrhage, especially with active bleeding, that a clean carotid puncture should be made. We believe that vascular spasm occurring in the region of ruptured vessels and aneurysms with attendant cerebral ischaemia can be seriously aggravated by rough injections, perhaps by reason of intramural haemorrhages in the punctured carotid (Yates, 1954). We also find that with slow injections of contrast medium better filling of the vessels is obtained. Localized spasm may itself indicate that an aneurysm lies in that area and may therefore be an important sign. In such a case it is wise to repeat the angiogram at a later date, as the aneurysm will usually be revealed when the spasm has passed off; furthermore, it will often be necessary to have a clear view of the vessel relationships at the aneurysmal origin, to facilitate surgical treatment.

Subdural Haematomas

Five cases of subdural haematoma are included. Three were of the chronic variety, with no emphasis in the history on head injury, and presented clinically as vascular or space-

occupying lesions. The demonstration of two further subdural clots was obtained in cases of clinically clear subarachnoid haemorrhage where the responsible aneurysm was also shown.

The appearances may be dramatic and unexpected. On the other hand, small bilateral anterior clots are easily overlooked, as occurred in one of our five cases. The lesion is most readily diagnosed in antero-posterior projection, but good phlebograms are essential. This is even more necessary when it is realized that there may be no displacement of the anterior cerebral artery if a haematoma overlies each hemisphere. Accordingly, angiography is not a proper substitute for bilateral burr-hole exploration.

Degenerative Vascular Disease

In cases where vascular disease is difficult to distinguish from brain tumour, angiography may be extremely helpful. On the other hand, cerebral angiography can be hazardous in arteriosclerosis and arterial hypertension. This is because spasm and thrombosis may be precipitated in diseased vessels, and may endanger an already precarious collateral circulation. In addition, it is known that diodone, an organic iodine compound, produces cerebral oedema of varying degree which may endanger relatively ischaemic vital areas (Broman and Olsson, 1948). In this latter respect is "thorotrast," a suspension of a weakly radioactive thorium salt, considered to be a superior contrast medium (see below).

In 27 patients intracranial cerebral thrombosis was diagnosed angiographically. Obliteration of major branch vessels was diagnostic in some, although in that connexion it must be remembered that normal variants exist in the circle of Willis. For example, both anterior cerebral arteries may fill only from one side. That may cause the false impression of a thrombosis of the anterior cerebral artery on contralateral injection. In a further 73 there was no visible thrombosis, but the radiological features of arteriosclerosis such as lengthening, tortuosity, and variations in the calibre of the vessels were commonly seen. In a further three the deformity was of such a degree that it was regarded as a vessel actually displaced by tumour. The same mistake was made each time with reference to a tumour of the anterior fossa. Here the gradual straightening of the curves of the carotid siphon due to atheroma was accompanied by a deep bend, concave forwards, of the origin of the anterior cerebral artery. That misinterpretation led to one unnecessary craniotomy, and clearly shows the need for air studies in cases of doubt.

Internal carotid thrombosis is revealed by filling of the external carotid system only. Caution, however, is needed in that interpretation. It is well known that spasm can simulate this appearance. In addition to this, faulty technique can cause a partial intramural injection that may temporarily occlude the lumen (Sirois, Lapointe, and Côté, 1954). Therefore, in all cases of suspected carotid thrombosis, films must be taken of the neck after injection of the common carotid artery to show the position of the needle, the carotid bifurcation, and the stump of the occluded vessel. It is of interest, and of great importance in view of the possibility of replacement grafts in selected cases (Eastcott *et al.*, 1954), that the initial radiological appearances of a total block are not necessarily conclusive. Thus repeated carotid injections, or injection of the internal carotid vessel itself, may indicate that there exists a local narrowing at the usual site just beyond the bifurcation which may so reduce the flow that injection may easily suggest a total block beyond it. In the period under review we have demonstrated internal carotid thrombosis in 17 cases. Although cerebral vascular disease had been considered, in only two had clinical examination strongly suggested the exact diagnosis, and each patient came to angiography as a suspected case of brain tumour.

In the 73 cases without visible thrombosis the final clinical assessment has been cerebral arteriosclerosis in 71. For convenience we include here the other two, each considered to have had cerebral thrombophlebitis.

Altogether, therefore, 120 suspected cases of tumour were satisfactorily examined by carotid angiography in this group considered under degenerative vascular disease. Other investigations, which included air studies in many, together with observation for periods of up to four years, have not so far disclosed a cerebral tumour among this number.

Space-occupying Lesions

Angiography is a safe and reliable means of demonstrating many brain tumours. In addition to assisting in locating the tumour, in many cases it shows vascular patterns within the tumour that provide specific evidence of the pathology. Thus benign meningiomas on the one hand, and malignant glioblastomas on the other, may usually be distinguished. When the intracranial tension is high, angiography is a safer investigation than encephalography or air ventriculography.

The results of angiography in the diagnosis of 356 intracranial space-occupying lesions in this series are shown in Table II. These were all carotid angiograms. The reliability

TABLE II

Angiographic evidence of intracranial space-occupying lesions	313	87.9%
Normal angiograms in extracerebral tumours related to the skull base	13	
Normal angiograms in intracerebral brain tumours due to misinterpretation	4	5.6%
Normal angiograms in intracerebral brain tumours	3	
Failed angiograms (no films or inadequate films) in proved tumour cases	23	6.5%
Site alone diagnosed accurately	262	74%
Site and pathology diagnosed accurately	137	39%

with which tumours of the brain stem and cerebellum have been demonstrated by ethyl iodophenylundecylate ventriculography has led to the choice of vertebral angiography only when an angioma has been suspected.

Ideally the angiographic picture of lesions in this group gives details of position, size, shape, and pathology, but usually only part of this information is obtained. The value of previous plain radiography is shown by the fact that almost conclusive information of intracranial tumour pathology was derived from the plain films in 10 cases in which angiography was normal. These, including three meningiomas, are indicated later in the consideration of normal angiograms in intracranial tumours. In our series the group of 355 patients with a space-occupying lesion included neoplasms, cysts, abscesses, granulomas, and cerebral oedema from any cause (but not blood clot, which is discussed under vascular disorders). Angiographic evidence of a space-occupying lesion was recognized in 313 cases (88%). In some of these the information was not precise. Thus, the method indicated only which hemisphere was involved in 31 patients; usually this was already known, but in several cases the contralateral hemisphere was suspected on clinical grounds.

Locating the Lesion

The review showed that brain tumours were correctly located in terms of lobes and depth in 262 cases (74%). The characteristics of vessel displacement which help in locating and perhaps in giving the size and shape of a tumour, the sites in which angiography is most useful, and the comparative locating value of angiography and ventriculography have previously been described (Wickbom, 1948; Bull, 1949; Torkildsen, 1949; Cairns and Jupe, 1951).

Difficulty in locating and in estimating the size arose under several circumstances. (1) The lesion may be in an angiographically blind area. (2) Destructive and infiltrating tumours which replace brain tissue may produce surprisingly little or even no alteration of vessel position. (3) Oedema may be the only factor producing vascular displacement. It may be secondary to thrombophlebitis or cerebral thrombosis, and such appearances can be indistinguishable from those of avascular tumours. Associated with tumour or

abscess, oedema may produce an erroneous impression of bulk. (4) Avascular areas may be present owing to degenerative changes in tumours which show pathological vessels elsewhere.

Small degrees of hydrocephalus do not show angiographically, but hydrocephalus was recognized in 38 cases of this series. In 26 it was the only angiographic evidence of tumour (the remaining 12 being due to congenital and degenerative causes). Such evidence was often of great value in indicating a lesion of the posterior fossa. In these cases with few localizing signs angiography is well tolerated even when the pressure is high. For more precise localization we then proceed to ethyl iodophenylundecylate ventriculography, which is much safer than air ventriculography in obstructive hydrocephalus.

Pathology

The demonstration of tumour vessels may make it possible to recognize the type of neoplasm. The tumours that most frequently show pathological vessels are glioblastomas, meningiomas, and metastases. Wickbom (1953) has stressed the importance of establishing whenever possible the relationship of a tumour to the brain—that is, whether extracerebral, intracerebral, or intraventricular. The type of tumour filling of these neoplasms may not be characteristic, but when taken in conjunction with their situation—for example, intracerebral for glioblastoma and metastasis, and extracerebral for meningioma—a correct pathological diagnosis may be possible. Where abnormal vascularity due to tumour is present, certain criteria are used to assess the local angiographic picture. Of special importance is the angiographic evidence of a high degree of malignancy. Even confirmatory biopsy may then be unnecessary, especially if the tumour is in an inoperable situation.

Those features that we have found the most usual may be summarized thus:

1. *Metastatic Tumours* (32)—9 diagnosed correctly, 16 diagnosed only as tumour (no tumour-filling), 3 diagnosed as glioblastomas, 3 diagnosed as meningiomas, 1 considered normal (see below).—If tumour-filling occurs it is usually malignant in type and impossible to distinguish from glioblastoma. Very occasionally a meningioma is suggested. Multiple tumours certainly favour the diagnosis of metastasis, although it has to be remembered that filling may occur in separate areas of any one tumour.

2. *Glioblastomas* (138)—72 diagnosed correctly, 52 diagnosed only as tumour (no tumour-filling), 6 diagnosed as astrocytomas, 6 diagnosed as meningiomas, 2 considered normal (see below).—Irregular tumour vessels (sinusoids), arteriovenous fistulae, and a tendency for the contrast material to pass rapidly through the tumour are the chief factors. The draining veins may pass from the centre of the tumour. Avascular areas associated with these appearances may imply degenerative or haemorrhagic change: vascularity and large size with little displacement of vessels suggests a destructive infiltration. Stretched cortical veins obstructed by pressure, in the depths of sulci and displaced radially from the tumour by oedema giving a "windswept" appearance, are commonly present in cases of compression due to glioblastomas. Twenty-six glioblastomas that were suspected clinically showed gross typical changes angiographically, and were accepted as such without operative confirmation. In all other cases the diagnosis was proved by biopsy.

3. *Astrocytomas* (32)—7 diagnosed correctly, 19 diagnosed only as tumour (no tumour-filling), 2 diagnosed as glioblastomas, 3 diagnosed as meningiomas, one considered normal (see below).—Astrocytomas grow more slowly and less destructively than the other malignant tumours, so that their invasiveness is nearly always accompanied by expansion. Consequently the displacement of adjacent brain is usually seen angiographically although tumour-filling is often absent.

4. *Meningiomas* (68)—49 diagnosed correctly, 13 diagnosed only as tumour (no tumour-filling), 2 diagnosed as

glioma, 4 considered normal (see below).—Regular tumour vessels, often in radial distribution, are seen in the arterial phase. Central "star figures" due to tumour-filling were not common in this series, although they are characteristic of meningiomas when they occur. The homogeneous opacity of the meningioma "blush," caused by the contrast medium lingering in the capillary bed of the tumour, typifies the venous phase of the angiogram. In particular, of course, do these tumours characteristically displace and stretch adjacent vessels over a length of their circumference. Of special value in diagnosis when other features were absent was the appearance of well-formed surface veins, often in the later arterial phase, which make one or two loops curved to the surface of the tumour before running to an adjacent sinus. The two meningiomas diagnosed radiologically as gliomas were both parasagittal. Vascular displacement was slight, and, in one of these, only a second angiogram three months later showed the deflected pericallosal artery previously described as "an anatomical variant" to be associated with adjacent faint tumour-filling. In each case the tumour circulation was barely shown. Gliomas adherent to the falx may of course resemble meningiomas by virtue of their site, and it is here that a very careful assessment of the type of tumour-filling should be made.

5. *Other Intracranial Space-occupying Lesions*.—These are usually demonstrated angiographically by vascular displacement alone, and where the correct pathology was given in these cases it was by inference from the clinical features, the site, and occasionally from bone changes on the plain films. In the 137 cases where the pathological diagnosis was given correctly we have included 8 in which one alternative diagnosis was suggested.

Normal Angiograms in Intracranial Tumours

Twenty angiograms were considered normal where an intracranial tumour existed. Review of the films shows that four of that number did in fact have definite angiographic evidence of brain tumours. In three a symmetrical hydrocephalus—due to a tumour either at the back of the third ventricle or in the posterior fossa—was demonstrated, but these were overlooked until air ventriculography had been undertaken. The fourth showed some lateral displacement due to a diffuse hemispherical malignant growth; and that also required ventriculography before it was recognized. Of the 16 tumour cases in which no angiographic abnormality was detectable, only three tumours were intracerebral. Two were rapidly growing malignant lesions, deeply situated, and one was a parieto-frontal astrocytoma. Subsequent pneumoencephalography showed them all. The remaining 13 were all extracerebral and related to the skull base (see Table III).

TABLE III

No.	Situation	Pathology	Plain X-ray Changes
4	Cavernous sinus	Carcinoma	Present in 3
1	"	Sarcoma	Absent
3	Pituitary	Adenoma	Enlarged sellae
1	Suprasellar	Rathke pouch cyst	Thinned dorsum sellae
1	"	Meningioma	None
2	Sphenoidal ridge	Meningioma en plaque	Sclerosis and hyperostosis in both
1	Clivus	Meningioma	Clivus erosion

Not only is the importance of plain x-ray changes evident in these cases, but normal angiographic appearances are also valuable, as they indicate little or no intracranial extension of growth. The exceptions to that were the suprasellar and clivus tumours, verified by either air encephalography or surgical exploration.

To our knowledge no tragedies have followed the misinterpretation of angiograms in the diagnosis of brain tumours in this series. In all cases of doubt burr-hole biopsies, aided considerably in many cases by location by isotope, using ³²P (Morley and Jefferson, 1952), or actual craniotomies have been done to verify the diagnosis.

Technique in Tumour Cases

High-quality films in several phases of vascular filling are of the utmost importance. This is especially so in suspected tumour cases if small pathological vessels are to be demonstrated and where the abnormal features may be transitory. Good vascular filling may be difficult to obtain where raised intracranial pressure slows the cerebral circulation. It is here that a slower injection of a larger volume of contrast medium often succeeds. The danger, however, of producing cerebral oedema in such cases is real (Broman and Olsson, 1948), so that the larger injections should be withheld from those patients already severely compressed. Sometimes thorotrast is to be preferred in restless and confused patients, because it causes less pain than diodone. Owing to the slight risk of malignancy many years later (MacMahon *et al.*, 1947) thorotrast is better avoided, especially in young people. In addition, care is required with the injection of that substance, since there is the danger of progressive fibrosis should an accidental perivascular injection occur (Amory and Bunch, 1948).

Cerebral Atrophy

In using the term "atrophy" we include here disease that, by degeneration, chronic inflammation, or incomplete development, results in diminished volume of brain substance. Compared with pneumoencephalography, angiography is an unreliable method for its demonstration. Without air studies, minor degrees of atrophy affecting the convexities of the hemisphere and localized ventricular dilatations often do not show at all. When atrophy causes angiographic changes those appearances may be misleading unless all the clinical data are known. Thus the isolated finding of vascular displacements from side to side can obviously be due either to pressure from an expanding lesion on the larger side or to traction from atrophy and gliosis on the smaller side.

Out of six cases mistakes were made by misinterpretation of the angiograms in two. In the first a porencephalic cyst communicating with the lateral ventricle could not be seen without air encephalography. In the second, syphilitic atrophy predominating in one hemisphere was mistaken for a space-occupying lesion in the other.

Progress Angiograms

The chief value of angiography after operation is in the out-patient follow-up clinic. The reduction in size or the obliteration of aneurysms after treatment can be seen. The fate of arteriovenous fistulae can be followed. The extent of tumour removal, and particularly tumour recurrence, are also questions that angiography can often answer when clinical doubt exists. Care must be taken, of course, to distinguish distortions caused by the operation from those due to the initial disease. Oedema or scarring may in that way give an impression of tumour displacement. The most deceptive appearances can be produced by the effect of decompressive craniotomies on the angiographic picture. Thus the further growth of a tumour can be accommodated by the decompression and consequently cause no displacement of the anterior cerebral arteries in the antero-posterior projections. Particularly as a result of this, we overlooked, amongst 29 post-operative studies, one large recurrent meningioma that was obvious clinically.

Progress angiography is also of value in the continued observation of suspected cases of brain tumour. Reference has already been made to a parasagittal meningioma diagnosed by angiography repeated after three months. Another tumour, a frontoparietal astrocytoma, was brought to light by a second angiogram performed two years after the initial films, though of poor quality, had been considered normal.

In addition to all the cases enumerated above, 178 patients had negative angiograms. Some of these remain tumour suspects and are being followed up. In others the final diagnosis included a variety of conditions such as "idiopathic" epilepsy, migraine, and encephalitis. This group includes three failures.

Complications

The amount of discomfort arising from percutaneous angiography with a local analgesic varies a great deal, while it is much reduced if adequate premedication is permissible. Many patients are relatively unaffected and are ready to leave the department within an hour of completing the procedure. Others suffer pain and anxiety that may leave them exhausted for some hours afterwards. The hot flush that follows the diodone injection and the final pressure that is necessarily applied to the neck to stop bleeding are the common causes of immediate discomfort. For some time after angiography the majority feel unwell, owing partly to the effects of blood absorbed from the bruised neck.

The occurrence of haematomas bears some relationship to the experience of the operator. Although a small extravasation of blood follows percutaneous carotid angiography, it is seldom serious owing to its quick dispersal down the mediastinum. Commencing swelling in the neck is usually controlled quite simply by digital pressure at the injection site and sitting the patient up. When the side of the neck becomes distended by haematoma continued vigilance is necessary, and that was the chief cause of overnight admission in some 5% of our out-patients.

Large haematomas causing dyspnoea are rare. Increasing distension will cause respiratory obstruction by displacement of the larynx and trachea. Laryngeal intubation is then urgently called for. In conscious patients we use a throat spray of lignocaine hydrochloride, a Mackintosh laryngoscope, and a "portex" tube. We advocate this early rather than late, and find the tube well tolerated by conscious patients for the three to four hours necessary while the haematoma disperses. Although in this series three such cases required admission for more than one night, we have not regarded clot evacuation as necessary in any. In a further two superficial infection of the neck required treatment with antibiotics.

Two patients died following angiography. One, in whom cardiovascular disease was not suspected, suffered severe anginal pain a few minutes after the angiogram and then collapsed and died. Necropsy revealed a haemorrhagic infarction of the cardiac septum: Petit-Dutaillis *et al.* (1952) have drawn attention to the danger of angiography in heart disease. The other patient was a known hypertensive in whom carotid spasm from a diodone injection was shown at necropsy to have added a fatal infarction to an already degenerated cerebral hemisphere. The carotid arteries were patent (Yates, 1954).

There were 16 other cases in which complications occurred with carotid angiography. Six of these were varying degrees of hemiparesis that took place when cerebral aneurysms or arteriovenous fistulae were under investigation. Three cleared up in a few hours. Of the three that were left with some permanent hemiparesis active subarachnoid bleeding was in progress at the time angiography was considered necessary. It may also be significant that in two of them compression of the opposite carotid during injection was employed to demonstrate collateral circulation. Four tumour cases with raised intracranial pressure suffered a temporary increase in pre-existing focal signs. In another two patients, in whom malignant gliomas involved the basal ganglia and mid-brain, a further fall in consciousness followed angiography, and that deterioration persisted. The remaining four were cerebral arteriosclerotics who developed hemipareses. In two of these recovery was incomplete.

The complications of vertebral angiography comprise two cases of brachial neuritis. Technical difficulty was experienced in each, and the symptoms, which took some months to resolve, were attributed to trauma of the brachial plexus roots.

Conclusions

The part played by angiography in the neurosurgical diagnosis of 1,007 patients has been reviewed. By increasing the number of out-patient investigations by

angiography many admissions to a neurosurgical department for purely diagnostic measures are avoided. With over half our angiograms performed as an out-patient procedure the occupancy of beds by patients not requiring operation is reduced and the volume of operative surgical work increased. Both air encephalography and ventriculography necessitate admission to hospital.

Angiography can provide the early, accurate, and safe diagnosis of emergencies, and that applies particularly to subarachnoid haemorrhage. It is a relatively safe procedure in brain tumours with high intracranial pressure. The benign may be distinguished from the malignant, and in the above series the pathological type of tumour was correctly diagnosed in 39%. Angiography has given us the accurate situation in 74% of brain tumours and in a further 14% has indicated their presence. That compares well with pneumoencephalography. In the remainder the use of ethyl iodophenylundecylate and air as contrast media was necessary.

The mortality of 0.2% has not involved any case of raised intracranial pressure. These cases where the intracranial pressure is extremely high as a result of obstructive hydrocephalus, and mistakenly investigated at first by angiography, are in our experience less disturbed by that and the ethyl iodophenylundecylate ventriculogram that follows than by air ventriculography in the first instance alone.

We wish to thank particularly Mr. R. T. Johnson for his advice and encouragement in the preparation of this paper, and with him Dr. R. G. Reid for making the material available. We are grateful to Sir Geoffrey Jefferson and Dr. E. Duff Gray for their helpful criticisms. To the Department of Medical Illustration under Dr. R. G. W. Ollerenshaw, and to both Mrs. V. Crowley and Mr. E. G. Mercer, we also express our thanks.

REFERENCES

- Amory, H. I., and Bunch, R. F. (1948). *Radiology*, **51**, 831.
 Broman, T., and Olsson, O. (1948). *Acta radiol. (Stockh.)*, **30**, 326.
 Bull, J. W. D. (1949). *Proc. roy. Soc. Med.*, **42**, 880.
 Cairns, H., and Jope, M. H. (1951). In *Textbook of X-ray Diagnosis*, edited by S. C. Shanks and P. Kerley. London.
 Curtis, J. B. (1951). *Brit. J. Surg.*, **38**, 296.
 Dowling, J. L. (1950). *Aust. N.Z. J. Surg.*, **20**, 11.
 Eastcott, H. H. G., Pickering, G. M., and Rob, C. G. (1954). *Lancet*, **2**, 994.
 Engeset, A. (1944). *Acta radiol. (Stockh.)*, Suppl. 56.
 Lima, P. A. (1950). *Cerebral Angiography*. London.
 Lindgren, E. (1947). *Brit. J. Radiol.*, **20**, 326.
 MacMahon, H. E., Murphy, A. S., and Bates, M. I. (1947). *Amer. J. Path.*, **23**, 585.
 Morley, T. P., and Jefferson, G. (1952). *British Medical Journal*, **2**, 575.
 Petit-Dutaillis, D., Pertuiset, R., and Rougerie, J. (1952). *Presse méd.*, **60**, 1091.
 Sirois, J., Lapointe, H., and Côté, P. E. (1954). *J. Neurosurg.*, **11**, 112.
 Sweet, W. H., and Brownell, G. L. (1955). *J. Amer. med. Ass.*, **157**, 1183.
 Torkildsen, A. (1949). *Acta psychiat. (Kbh.)*, Suppl. 55.
 Wickbom, I. (1948). *Acta radiol. (Stockh.)*, Suppl. 72.
 — (1953). *Ibid.*, **40**, 529.
 Yates, P. O. (1954). *Proc. roy. Soc. Med.*, **47**, 606.

The National Society of Children's Nurseries celebrated its fiftieth anniversary by holding a jubilee conference at County Hall, London, from May 30 to June 1, at which were discussed problems concerning the care, welfare, health, and social problems of children under 5, and the part the nursery can play in giving guidance to parents. There was also a reception at the Mansion House. The conference chairmen included Professor A. A. Moncrieff and Professor R. M. Titmuss, and papers were read by Professor Fraser Brockington ("The Social Background to Child Health in the Mid-twentieth Century"), Professor W. S. Craig ("The Contribution of Nurseries to Child Health in the Home under Conditions of Modern Society"), and Dr. Kenneth Soddy ("The Nursery and the Psychological Needs of the Child"). An important aspect of the society's work has been the training of a large number of young girls so that when they marry they shall be better able to care for their own children.

MATERNAL DEATH FROM ASPIRATION ASPHYXIA

BY

R. B. PARKER, M.B., M.R.C.O.G.

Lecturer to the Department of Obstetrics and
Gynaecology, University of Birmingham

Recent reports have shown that aspiration of stomach contents during anaesthesia is responsible for a number of maternal deaths in Great Britain (Gilliatt, 1949; Jeffcoate, 1953; Parker, 1954) as well as for a number of near fatalities (Hausmann and Lunt, 1955). Although the incidence of mortality from this cause has been recorded from many centres in the United States of America, there has been no assay of the frequency of the condition in this country, and this report represents an attempt to make good the deficiency.

Birmingham Figures

The figures in Table I refer to women resident in the City of Birmingham, which in the years under review had an average population of approximately 1 million.

TABLE I.—City of Birmingham

	Confinements	Domiciliary	Institutional	Deaths, Including Abortion	Anaesthetic Deaths
1943	20,829	11,214	9,615	35	1
1944	23,318	12,196	11,122	31	1
1945	20,685	9,890	10,795	29	1
1946	23,980	11,472	12,508	20	1
1947	25,540	12,225	13,315	24	1
1948	23,067	10,005	13,062	11	1
1949	21,463	9,274	12,189	10	—
1950	19,555	8,492	11,063	16	—
1951	18,850	7,871	10,979	14	2
1952	17,831	7,018	10,813	15	—
Total	215,118	99,657	115,461	205	8 (3.9%)

From 1943 to 1952, inclusive, there were approximately 215,000 confinements and 205 maternal deaths (mortality 0.9 per 1,000 confinements). Included in the 205 deaths are 41 deaths from abortion and 6 due to ectopic gestation; deaths from associated causes are excluded (as with the national figures for England and Wales).

In 8 of the 205 cases (3.9%) I believe death was due, largely or entirely, to aspiration asphyxia, and in two other cases it seems more than possible that aspiration played a major part in the fatal outcome. This represents 1 death for every 27,000 births approximately. The eight deaths occurred in four different hospitals in the city, and summaries of their case histories are presented.

In Cases, 1, 2, 6, 7, and 8 there can be no doubt about the cause of death. In Cases 3, 4, and 5 the reference to anaesthetic difficulty in the records is brief, but, as emphasized before (Parker, 1954), regurgitation may not easily be recognized. Some trouble was experienced in the three cases; and in all of them, shortly after conclusion of the anaesthesia, the classical sequence of cyanosis and dyspnoea leading to acute pulmonary oedema was observed. In a previously fit woman with this history there is no alternative diagnosis.

Summary of Case Histories

Case 1.—A primigravida aged 38, in whom it was thought necessary to undertake craniotomy and delivery because of the risk of uterine rupture in an obstructed labour. At