Pituitary Crisis.—This may occur as the terminal event in a prolonged pituitary torpor or develop rapidly after a short period of post-operative "decline." The signs of approaching crisis are a rapid deterioration in consciousness, passing into coma; a falling blood pressure which does not respond satisfactorily to intravenous infusions or cardiovascular stimulants; a rapid, thready pulse; and shallow slow respirations. The skin becomes cold, and a patchy pallor and cyanosis appear in the lips, cheeks, and extremities. These ominous signs advance rapidly and death occurs in a matter of hours.

The serum chemistry in one patient in pituitary crisis (Case 3) was as follows: sodium 288 mg., chloride 540 mg., and potassium 18.2 mg. per 100 ml.; the serum CO₂ combining power was 51 vols. per 100 ml. These figures support the hypothesis that such patients may die in acute or sub-acute adrenocortical failure.

Sheehan and Summers (1949) have fully described what they call "hypopituitary coma," occurring in cases of chronic hypopituitarism. There is a great similarity between the state they describe and the torpor and crisis of the surgical cases. Some points of difference—for example, degree of hypoglycaemia, temperature, and pulse rate—may be eliminated by further study.

It may be argued that the syndrome is due to frontal-lobe damage at operation, especially in view of the constancy of incontinence as a sign. The recovery of Case 7 from a week's progressive torpor and incontinence, twenty-four hours after instituting hormone therapy, suggests that frontal damage does not play a large part in the syndrome.

In short, it seems likely that the syndrome of post-operative pituitary torpor and crisis is the hypopituitary coma of Sheehan and Summers, modified by surgical trauma.

Summary

An investigation has been carried out into the part played by adrenocortical deficiency as a cause of death after operation in cases of pituitary adenoma and cyst.

A scheme for pre-operative assessment of adrenocortical activity is described; and some steps to improve the prospects of surgical treatment are outlined. Some of the wider implications of the reported observations are briefly discussed.

Two syndromes of post-operative pituitary insufficiency are described, for which the terms "pituitary torpor" and "pituitary crisis" are used. Their relation to hypopituitary coma (Sheehan and Summers, 1949) is discussed.

From the results in the series described it seems permissible to infer that death from pituitary deficiency is largely preventable.

I am indebted to Mr. G. L. Alexander and Mr. D. G. Phillips for encouragement in this work and for facilitating these observations on their cases. Professor C. B. Perry, of the University of Bristol, helped materially in allocating supplies of corticotrophin furnished by the Medical Research Council. Mr. Yeoman and the staff of the hospital laboratory willingly undertook a multitude of investigations which were not initially of routine nature, and grateful acknowledgments are due to them.

REFERENCES

- Bakay, L. (1950). *J. Neurosurg.*, 7, 240.
 Caughey, J. E., and Garrod, O. (1954). *British Medical Journal*, 2, 554.
 — James, A., and Macleod, E. K. (1952). *Ibid.*, 1, 1216.
 Chalmers, T. M., and Lewis, A. A. G. (1951). *Lancet*, 2, 1158.
 Grant, F. C. (1948). *J. Amer. med. Ass.*, 136, 668.
 Henderson, W. R. (1939). *Brit. J. Surg.*, 26, 811.
 Houghton, E. A. W. (1953). *British Medical Journal*, 2, 372.
 Jefferson, G. (1940). *Proc. roy. Soc. Med.*, 33, 433.
 Robertson, J. D., and Kirkpatrick, H. F. W. (1951). *Lancet*, 2, 54.
 Robinson, F. J., Power, M. H., and Kepler, E. J. (1941). *Proc. Mayo Clin.*, 16, 577.
 Sheehan, H. L., and Summers, V. K. (1949). *Quart. J. Med.*, 18, 319.
 Tuerkischer, E., and Wertheimer, E. (1944). *J. Endocr.*, 4, 143.

PERIPHERAL ARTERIAL EMBOLISM*

BY

R. P. JEPSON, M.B., B.Sc., F.R.C.S.

Professor of Surgery, University of Sheffield; Honorary Surgeon, Sheffield Royal Infirmary

The treatment of acute ischaemia subsequent to the impaction of a peripheral embolus has received considerable attention in the past quarter of a century, and during this period authoritative surgical opinion has spoken for and then against embolectomy. The extensive experience of Strömbeck (1935) and Key (1936) stimulated the publication of many reports favouring embolectomy in the American and English literature. Such enthusiasm was frequently based on a small number of successful cases, and when larger series were reviewed the mortality of embolectomy was often high and the morbidity considerable. More recently the non-operative management has been stressed (Haimovici, 1950), supplemented by the use of heparin, vasodilator drugs, and sympathetic-nerve interruption. In the present paper a series of cases of lower-limb emboli treated

Site of impaction of 97 emboli.

Heart	72 cases (100%)
Associated with fibrillation	84%
" " recent cardiac infarct	16%

	20-29	30-39	40-49	50-59	60-69	70-79	Total
Males	1	1	6	9	6	2	25
Females	3	6	7	14	13	4	47

in the last seven years by the conservative regime are analysed. An attempt is made to discuss the diagnosis and physiopathology of embolism and define more accurately the indication for conservative management and for operative intervention.

Details of Cases

Although the first embolectomy performed in this country was on the brachial artery (Jefferson, 1925), emboli in the upper extremity do not often constitute a surgical problem, and such cases will not be considered, attention being solely directed to 72 patients in whom 97 lower limbs were endangered by embolic occlusion. The site of the impaction of the emboli is shown in the accompanying diagram. This distribution follows that previously reported by Warren and Linton (1948) and Haimovici (1950). The origin of the

*A lecture delivered at the Royal College of Surgeons on October 12, 1954.

emboli was in the heart in every case, and in 84% was associated with a controlled or uncontrolled auricular fibrillation; 16% of the patients had recently suffered a cardiac infarct and mural thrombosis (Table I). The sex and age incidence is illustrated in Table II. As in previously reported series, the majority of the patients were over 40 years of age.

Clinical Course

Although it is well recognized that an embolic occlusion of a major limb artery can occur painlessly (Haimovici, 1950), the vast majority (95%) of the present series complained of pain at or shortly after the onset of symptoms. The patient on clinical examination was often too distressed to be a satisfactory witness, and any detailed analysis of the type of pain is partially vitiated by this fact alone. The site of the pain occasionally corresponded with the position of the impacted embolism, as described by Rykert and Graham (1938). Thus the first symptom of an aortic embolism was ache in the back and suprapubic region, and in the case of a femoral embolism was initially felt in the groin, the artery being non-pulsatile and tender when palpated within six hours of the onset of symptoms. More commonly the reference of pain was in the periphery of the limb, and was usually described as a sudden ache, cramp, or tight band, a description characteristic of the deep pain which follows muscle ischaemia. Such patients had usually exercised the leg at or shortly before the onset of pain. The fact that the pain was referred to a non-muscular area such as the dorsum of the foot does not necessarily mean its origin was not in muscle.

The experiments of Boyd *et al.* (1949) clarified the mechanism of such reference patterns. Patients with emboli in the proximal large arteries frequently complained of disagreeable paraesthesiae beginning in the toes and spreading proximally up the limb as the initial symptom, although these were often replaced or overshadowed later in the course by pain. The onset of symptoms is not, of course, coincident with the time of arterial impaction, but follows at an interval. The arguments in support of this and also of the muscular origin of the pain are well documented by Lewis (1936). In addition to pain frequent early symptoms were numbness, coldness, weakness or paralysis of the foot or leg; and in many cases a partial or complete paralysis of knee and foot movements came on within a few minutes of the onset of symptoms. Emboli lodging at the aortic bifurcation occasionally produced the desire to defaecate or pass urine, and haematuria or spinal cord involvement developed if the thrombosis spread proximally to include the renal arteries.

On examination the signs of acute ischaemia were present. The skin was initially cold and pale, later becoming cyanosed and mottled, recovery being associated with the pink flush of a reactive hyperaemia and gangrene with brown and blackish tints. The veins were collapsed peripherally, and the sensory loss, most marked to light touch and pin-prick, spread centripetally up the limb, being greatest in the foot. The level of the changed sensation depended particularly on the site of the arterial occlusion, being approximately to the mid-thigh in aortic lesions, to the knee in common femoral, and to above the ankle in blocks of the superficial femoral or popliteal artery. Deep pain sensation disappeared last of all, and marked calf tenderness (or a positive Homan sign) was often present in an otherwise anaesthetic limb. Muscle weakness may be selective to single groups, but the most profound weakness was seen in the most distal muscles.

Immediately following the occlusion the involved muscle was flaccid, but within a few hours became swollen and firm, with an associated plantar deformity, and the patient greatly resented correction of this by splintage. Pulses distal to the occlusion were absent and oscillometric readings zero. With major emboli, such as those at the aortic bifurcation or with the sudden deterioration of the circulatory efficiency as in a recent cardiac infarct, the blood pressure often fell precipitately, a factor which must greatly reduce

the collateral arterial circulation. Multiple or visceral emboli often complicated the clinical picture. Haimovici has previously drawn attention to the frequency with which a venous thrombosis develops in the ischaemic leg owing to the stagnant flow and the damage to the endothelial wall, a complication which further endangers the viability of the limb.

Differential Diagnosis

Unless an embolus is demonstrated at necropsy or operation the diagnosis is always presumptive. There are four conditions which can simulate with convincing detail a major arterial embolism.

(a) A thrombophlebitis in the deep leg veins may on occasion be associated with extreme reflex arterial spasm and has been described as phlegmasia caerulea dolens. In such limbs intense pain, marked cyanosis, paralysis, and sensory change may be observed, and the major pulses are impalpable. The swelling of the limb is, however, greater than that seen in the early stages of an arterial embolism, and the arterial spasm usually relaxes after a sympathetic-nerve block or heavy sedation. Toe or more massive gangrene can, however, develop with purely venous lesions.

(b) Thrombosis in the femoral or popliteal arteries is much more frequent than impaction of an embolism at that site, and, by reason of the acute ischaemia which it causes, may present a similar clinical picture. Indeed, examination of the affected limb may not permit a differential diagnosis to be made. Often the clinical history will bring out symptoms of a long-standing chronic ischaemia, such as cold feet or intermittent claudication, and examination of the patient will reveal other evidence of obliterative vascular disease. The absence of a cardiac lesion, such as a recent infarct or auricular fibrillation, is strong evidence in support of a primary thrombosis. Most commonly an acute thrombosis comes on in an artery which is already diffusely narrowed and a collateral circulation is already established. The final acute thrombosis does not, therefore, present such a complete ischaemia, and severe pain and gross sensory or motor loss are usually absent. The age of the patient is not of great value, as most emboli and most thromboses develop in patients over 40 years of age. When the diagnosis is doubtful it is preferable to treat the patient for a thrombotic lesion and avoid surgery.

(c) A dissecting aneurysm of the abdominal aorta may occlude the distal part of the vessel, and the patient presents with acute ischaemia of both limbs. The absence of a source of emboli may be the only differential diagnostic criterion.

(d) *Pseudo-embolism*.—Occasional cases present the classical picture of a major artery embolism with absent major pulses and the clinical sequelae of acute ischaemia. Within a few days all the peripheral pulses have returned with normal pulse pressure and the leg has completely recovered. Atlas (1942) believes that reflex spasm of the large proximal arteries provoked by a small peripheral embolism may be the explanation, while possibly in some cases the embolism is ground to pieces by the repeated blows of the pulse wave. In some instances the further migration of an embolism to a minor vessel may reopen a major artery. Cases of pseudo-embolism are rare and deserve detailed investigation.

Physiopathology

After its initial arrest the embolic mass is subjected to the repeated blows of the pulse wave, which may cause its further disintegration and migration. At the site of final impaction the artery is moderately distended, its wall reacting by oedema and cellular infiltration. The thrust of each pulse is transmitted through the embolism, and on palpation of the impacted artery may be mistaken for a normal pulse wave (Nordentoft's sign). Distal to the embolism the artery is collapsed and pulseless and its intraluminal pressure greatly reduced. The capacity of the collateral circulation may be such that within minutes the peripheral blood flow approximates to a normal resting level, although the distal pulse wave amplitude is decreased (Shepherd, 1950).

On many occasions the expected restoration of blood flow through collateral channels fails to occur, and many factors can be mentioned which may account for this. The patient with a severe cardiac lesion may have an insufficient blood pressure to force an adequate blood flow through the increased resistance incurred by the use of collateral vessels, or the embolism may lodge at a bifurcation site, such as the division of the common femoral artery, thereby obstructing the main vessel and its major collateral channel.

Some (Reynolds and Jirka, 1944; Holden, 1952; Nystrom, 1936) have described severe and persistent arterial spasm following the impaction of an embolism. This must be uncommon, and the decrease in arterial diameter distal to the occlusion is probably more often due to lowering of arterial pressure; for Burton (1952) has shown that, regarded physically, blood vessels are distensible tubes with a critical closing pressure. Secondary thrombosis is an inevitable sequel to embolic occlusion, and occurs maximally in the artery distal to the lodgment where the axial flow is slow and possibly turbulent. The rate of the thrombotic growth is inconstant, but after 12 hours is often of such an extent as to make its removal hazardous or impossible.

Principles of Conservative Treatment

The essentials of conservative treatment are to maintain blood pressure, relieve pain by heavy sedation, and relax the peripheral vasculature by body-heating. The limb itself should be maintained horizontal with the pressure points carefully protected at an environmental temperature of about 25° C. Although it is often recommended that temperatures below this should be used we have found it difficult, with such cooling, to ensure a satisfactory peripheral vasodilatation by body-heating. Vasodilator drugs are contraindicated because of their ineffectiveness even when injected intra-arterially (Catchpole and Jepson, 1954) and their hypotensive action may further imperil the patient or the limb. A paravertebral block, if adequately performed, may be of value in the differential diagnosis and in the treatment, particularly of the more peripheral emboli. It is difficult to see how a sympathetic block can significantly influence the collateral circulation at the root of the limb. Heparin is used if conservative management is decided upon but not when embolectomy is contemplated. It possibly has some value in restricting the spread of the secondary thrombosis.

TABLE III.—*Survival and Loss Rate in 97 Limbs After Embolic Occlusion*

	Limbs Affected by Emboli	Limbs Survived	Limbs Gangrenous
Aorta	7	3	4
Iliac	9	6	3
Common femoral	38	19	19
Superficial femoral	27	16	11
Popliteal	14	12	2
Tibial	2	2	

Results of Conservative Treatment

Table III summarizes the rate of survival and loss due to gangrene of 97 lower limbs following embolic occlusion of a major artery. A common femoral embolism is arbitrarily defined as one in which it is possible to palpate the femoral pulse for not more than 2.5 cm. below Poupart's ligament. The decision to group an embolism as superficial femoral or popliteal was decided by oscillometry. It will be readily seen that 58 of the 97 limbs survived the acute ischaemic episode and 39 became gangrenous. If these cases are further subdivided into those with a proximal lodgment of the emboli (aortic bifurcation, iliac, or common femoral) and distal (superficial femoral, popliteal, or tibial), it is evident that the prognosis for limb survival is worse in the former. Table IV shows that 49% of the legs in the proximal group became gangrenous, in contrast to 30% of the distal. Although it is not easy to be certain of the exact location of emboli, the prognosis of those diagnosed as in

the popliteal region is surprisingly good, and is comparable to Haimovici's (1950) 6.6% incidence of gangrene for emboli in a similar site. Overall patient mortality was 39% within a month of entry into hospital, and in the 22 patients who lost one or more limbs through gangrene the mortality from mid-thigh amputation was 41%, death usually occurring within a few days of operation (Table V). The 19

TABLE IV.—*Incidence of Gangrene According to Site of Embolism*

Proximal group (aorta, iliac, common femoral) ..	49%
Distal group (superficial femoral, popliteal, tibial) ..	30%
Total incidence	40%

TABLE V.—*Outcome Within One Month of Admission to Hospital*

Survived	61%
Died without operation	26.3%
Died after mid-thigh amputation	12.5%

patients who did not require a mid-thigh amputation and who died within a month of entry into hospital succumbed to multiple visceral or cerebral emboli or cardiac failure.

On follow-up of the patients who survived and were discharged home it is evident that the majority of the limbs suffered from a chronic ischaemia. Disagreeably cold extremities, ulceration, severe claudication, and rest pain were often seen in the months following discharge. Using conservative treatment, Warren and Linton (1948) found that 17 out of 24 lower limbs were lost in patients with emboli; and Haimovici (1950), after excluding cases of death shortly after admission, reported a collective incidence of gangrene of 34.9% for the lower extremities. Gangrene occurred in 65% of the common iliac and 55% of the aortic bifurcation emboli.

Discussion

As it is evident that the prognosis in patients with peripheral emboli varies according to the site of impaction, treatment may be appropriately considered on this basis. It has been well established that the results of conservative treatment when emboli occlude the aortic bifurcation or the common iliac artery are very poor (Warren and Linton, 1948; Haimovici, 1950; Andrus, 1950). Albright and Leonard (1950), in a collective review, reported 193 cases of saddle embolism at the bifurcation of the aorta. They found that 144 of these patients died owing to the direct complications of the impaction; amputation was performed in many of those whose life was spared. Although aortic embolectomy seems to be the most rational treatment, it is extremely difficult to assess the mortality of the operation because of the small number of cases reported and the natural unwillingness to publish disappointing results. The treatment which is adopted for aortic embolism cannot for that reason be based on statistics. It is, however, our present policy to advise aortic embolectomy as the treatment of choice.

Of the three approaches to the bifurcation of the aorta and the iliac vessels, the transperitoneal exposure under spinal or general anaesthesia through a left paramedian incision is the most satisfactory. The intestines are packed away and the aorta and its branches controlled with tape, clamps, or digital pressure. An incision is then made into one or both common iliac arteries and the clot rubbed down with finger pressure. Madden (1951) prefers a direct incision into the aorta. Temporary release of the clamps, first distal and then proximal, confirms the completeness of removal. The incisions are securely closed without any significant constriction to the artery by a single layer of interrupted or continuous sutures of No. 5 Deknatel. More distal clot may need an additional exposure of the femoral arteries. This, if required, should be performed while the iliac arteries are accessible so that any further clot can be pushed down through the femoral incision. When the embolus has been satisfactorily extruded the distal pedal pulses are immediately palpable. Failure to return means incomplete removal of the aortic or iliac clot or the presence of a separate more distal embolism. If thought necessary the lumbar sympathetic chains can be resected at the time of the abdominal exposure. Heparin is not given post-operatively as

the risk of thrombosis at the suture lines is held to be less than the risk of massive haematomata in the wounds.

Although the retrograde removal of an aortic embolism following the bilateral exposure of the femoral arteries has been advocated (Griffiths, 1938), it is extremely difficult in many cases to dislodge the clot from below, and the intimal trauma it involves readily leads to further thrombosis. Retroperitoneal exposure of the aorta is more difficult and has no particular advantage.

The results of conservative therapy in iliac or common femoral embolism where the femoral artery pulsation, when palpable, terminates within 2.5 cm. below the inguinal ligament are not impressive (Haimovici, 1950), many patients dying of the effects of limb gangrene or suffering the troublesome complications of chronic ischaemia. The exposure of this region is simple, and can, if necessary, be performed under local analgesia. Probably the number of patients saved by femoral embolectomy will not be great. Many will succumb, as under the non-operative regime, to the complications of the cardiac lesions or further embolisms. Strömbeck (1935) reported a 63% mortality following embolectomy in the lower extremities. The results, however, for limb survival are vastly improved and the sequelae of chronic ischaemia largely avoided.

Distal emboli in the leg or emboli involving the arm are probably most suitable for the conservative regime, as the dissection necessary to expose the smaller vessels destroys collateral vessels and closure of the incision severely narrows the arterial lumen.

The two limiting factors to embolectomy are the general condition of the patient and the time interval between the embolic impaction and operation. The former often excludes surgery, and the non-operative regime is perforce adopted. Although many embolectomies have been successfully reported where the interval is longer than 24 hours, the extension of the retrograde thrombosis is so rapid and soft-tissue necrosis so developed that operations beyond 12–16 hours are apt to be unsuccessful (Pearse, 1933). It is a sad reflection that only 20 out of the 72 patients reported in this series were admitted to hospital within 12 hours of the onset of symptoms. It is probable, therefore, that in any large group of peripheral emboli the majority of the patients will be most satisfactorily managed by non-operative regime. There are, however, some patients whose general condition permits the removal of emboli arrested at the common femoral and aortic sites. The eventual prognosis for such patients who are saved is not good. Strömbeck (1935) has shown that of 61 patients discharged from hospital after successful embolectomy one-half are dead from heart failure or further emboli within two years. Many surgical lesions, however, carry an equally grave prognosis, and by prompt intervention the surgeon can save many limbs and some lives and, as is his duty, relieve much suffering.

Many of the cases reported in this paper were seen in the Manchester Royal Infirmary, and I am grateful to Professor A. M. Boyd for stressing the importance of the conservative regime. Mr. W. Metcalfe has kindly allowed me to use some of his material, and the Department of Medical Illustrations provided the diagram.

REFERENCES

- Albright, H. L., and Leonard, F. C. (1950). *New Engl. J. Med.*, **242**, 271.
 Andrus, W. De W. (1950). *Arch. Surg. (Chicago)*, **60**, 511.
 Atlas, L. N. (1942). *Surg. Gynec. Obstet.*, **74**, 236.
 Boyd, A. M., et al. (1949). *J. Bone Jt Surg.*, **31B**, 325.
 Burton, A. C. (1952). In *Visceral Circulation*, Ciba Foundation Symposium. Churchill, London.
 Catchpole, B. N., and Jepson, R. P. (1954). *Circulation*, **9**, 408.
 Griffiths, D. L. (1938). *Lancet*, **2**, 1339.
 Haimovici, H. (1950). *Angiology*, **1**, 20.
 Holden, W. D. (1952). *Acute Peripheral Arterial Occlusion*. Thomas, Springfield, Ill.
 Jefferson, G. (1925). *British Medical Journal*, **2**, 985.
 Key, E. (1936). *Brit. J. Surg.*, **24**, 350.
 Lewis, T. (1936). *Clin. Sci.*, **2**, 237.
 Madden, J. L. (1951). *Surg. Gynec. Obstet.*, **93**, 167.
 Nystrom, G. (1936). *Lectures on Embolism and Other Surgical Subjects*. Williams and Wilkins, Baltimore.
 Pearse, H. E., jun. (1933). *Ann. Surg.*, **98**, 17.
 Reynolds, J. T., and Jirka, F. J. (1944). *Surgery*, **16**, 485.
 Rykert, H. E., and Graham, D. (1938). *Amer. Heart J.*, **15**, 395.
 Shepherd, J. T. (1950). *Clin. Sci.*, **9**, 355.
 Strömbeck, J. P. (1935). *Acta chir. scand.*, **77**, 229.
 Warren, R., and Linton, R. R. (1948). *New Engl. J. Med.*, **238**, 421.

OCULAR NERVE PALSY WITH SEVERE HEADACHE IN DIABETICS

BY

W. P. U. JACKSON, M.D., M.R.C.P., D.C.H.

(From the Diabetic Clinic, Groote Schuur Hospital, and the Department of Medicine, University of Capetown, South Africa)

A diabetic patient presents with the complaints of double vision and of extremely severe headache behind the left eye and in the left frontal region. Examination reveals an isolated left-ocular-nerve palsy. No cause is found and the condition clears spontaneously. Is it a diabetic complication, pure and simple?

Isolated cranial-nerve palsies, especially of the third and sixth nerves, are known to occur in diabetics, either at the inception of the disease or later in its course.¹⁻¹² That these palsies are in fact true instances of "diabetic neuritis" seems likely where no other cause can be found and where the paralysis is only temporary. According to Joslin and his group⁷ the condition clears up as the diabetes is brought under good control, while Collier¹ reports total cure in all of his 30 cases.

Association of Ocular Palsy and Pain

We have seen several instances of diabetic cranial-nerve palsy in the past years, but have recently run into diagnostic difficulties over diabetic patients who present with severe headache in addition to their ocular palsies. Pain does not normally accompany such palsies; it is rarely mentioned in the literature. Groenouw⁴ remarks upon the occasional association of "neuralgia of the fifth nerve" with diabetic ocular paresis, and Larson and Auchincloss⁸ mention "numbness of the face" (presumably fifth nerve) without pain. Goodman *et al.*³ report one case of "widespread neuropathy, including an attack of pain in the distribution of the right trigeminal nerve," and Herson⁵ reports a case of internal and external ophthalmoplegia preceded by three weeks' bilateral frontal headache. Apart from these skimpy references all other authors either state that pain in head or face does not occur or else they are silent in this regard. In each of the present cases there was a strong suspicion of intracranial aneurysm (or similar localized lesion), but this could not be proved, and it was eventually believed that the syndrome was purely diabetic.

Case Reports

Case 1.—A coloured woman aged 48, known to have had diabetes for 18 months, not obese, and poorly controlled on around 30 units of zinc-protamine insulin daily, experienced very severe right hemicranial headache of sudden onset which was continuous and unrelieved by salicylates. There was no nausea, vomiting, or vertigo. The next morning her right eye was closed. Examination the same day revealed a complete right third- and sixth-nerve palsy and also hypoaesthesia to touch and pin, with mild paraesthesia in the distribution of the first division of the fifth nerve on the right side. She had no diabetic "complications" of vascular or other type; she neither drank nor smoked. Her past history and further examination were unrevealing. Radiographs and cerebrospinal fluid were normal; W.R. was negative in the blood and C.S.F. A right carotid angiogram was adequate and entirely normal. The headaches slowly abated; the neurological features were improving in three weeks, and had completely cleared within two months.

Case 2.—A coloured obese woman aged 50, with very mild diabetes of some nine months' duration (fasting blood sugar, 150 mg. per 100 ml.), who was controlled on diet only, suddenly developed an extremely severe right frontal