EMERGENCIES IN GENERAL PRACTICE**SUDDEN OCCLUSION OF A MAJOR PERIPHERAL ARTERY**

BY

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Sudden occlusion of an artery may be due to an injury, either open or closed, an arterial embolus, or an arterial thrombosis. Any artery in the body may be involved, but in this article only sudden occlusion of the major arteries supplying blood to the limbs is discussed.

This catastrophe does not necessarily imperil the life of the limb; in many patients the collateral circulation proves to be adequate. The popliteal artery has a particularly bad reputation and the incidence of amputation following sudden occlusion of the iliac, femoral, and axillary arteries is also high. But in the case of occlusion of other peripheral arteries gangrene is a rarity and the clinical effects often transitory, whilst slow occlusion of a vessel as large as the bifurcation of the abdominal aorta may produce surprisingly little disturbance. Eleven out of 26 of my patients with thrombosis of the aorta at this level are able to work on a medical regime of treatment, admittedly at only sedentary occupations.

Clinical Picture

A patient with sudden occlusion of a major peripheral artery calls in his doctor because the limb has become cold, pale, and perhaps numb, because agonizing pain has developed at rest, or because of the sudden onset of severe exercise pain (intermittent claudication). The severity of the symptoms varies with the rate of occlusion of the vessel, which means that they are usually more pronounced when the vessel has been blocked by an embolus than when it thromboses. There may be a previous history of intermittent claudication, a stroke, or angina pectoris, pointing to an arterial thrombosis as the reason for the new symptoms; or the patient may be suffering from auricular fibrillation or bacterial endocarditis, indicating that an embolus is the likely cause.

Pain is said to be a marked feature of an arterial embolus and usually absent when the blood flow has been interrupted by a thrombosis or an injury. This is on the whole true, but some patients develop no pain after an undoubted arterial embolus and others suffer agonizing pain from a thrombosis. Later the limb becomes numb, and, if gangrene is likely, the muscles can no longer contract and ischaemia of the nerve fibres leads to complete sensory and motor paralysis; in other patients gangrene is averted but severe rest pain develops.

The limb at first is pale and cold; in a severe case the skin looks and feels dead. Later the skin becomes cyanosed and, if gangrene is certain, a dark purple colour; usually areas of pallor are adjacent to areas of cyanosis, giving the skin a blotchy appearance. The temperature of the environment is of great importance both to the rate of onset of the various clinical signs and to the chances of recovery. If the part is kept cool, skin can withstand severe anoxia for 48 hours or more, muscle for 10 hours or so; nerve conduction ceases after about 30 minutes. Recovery from complete anoxia is much more likely after restoration of the blood flow if the part has been cooled. On the other hand, if the part has been warmed, blisters may form on the skin within 12 hours and muscle becomes gangrenous within a few hours.

The pulses are absent distal to the lesion and increased proximally. Thus a patient with a popliteal embolus will have no palpable pulsation in either the dorsalis pedis or posterior tibial arteries, but the popliteal artery will be

unduly prominent because the embolus usually rests on the bifurcation of an artery and the main vessel above appears to pulsate more forcibly than on the normal side.

Treatment

There can be few emergencies which are influenced to a greater extent by good initial medical care than sudden occlusion of the main artery to a limb. Whilst admission to hospital should be obtained if possible, the first few hours are of great importance. Incurable changes can occur during this time. Simple measures taken in the patient's own home serve to minimize the effects of such a lesion. Unfortunately there can be no doubt that many patients with sudden arterial occlusion do not receive the best treatment. In particular, heating the limb by blankets or hot-water bottles does much harm. It can be stated that a patient who receives no treatment at all is better off than one whose limb has been either heated or elevated.

Treatment is discussed under the following headings: the reduction of the metabolic needs of the part; development of the collateral circulation; anticoagulants; maintenance of the patient's general health, and the prevention of infection and deformity; and surgical operations.

Reduction of the Metabolic Needs

This is best achieved by keeping the limb cool. It should not be frozen but exposed to the air at room temperature in temperate or cool climates or cooled by a fan in the Tropics. Cooling reduces cell metabolism and less oxygen is required, so that the part has a better chance of surviving until the blood flow is restored. Rest further reduces the metabolic needs; therefore active movements of the affected limb should be avoided and if necessary a light and well-padded splint applied. Rest may also be encouraged by the relief of pain, for which purpose pethidine, 100 to 150 mg., is usually more effective than morphine, $\frac{1}{4}$ gr. (16 mg.). For those patients with minimal pain a hypnotic such as amylobarbitone, 3 gr. (0.2 g.), is of value. A good drug for this purpose is alcohol; this often relieves the pain of sudden vascular occlusion, probably because it is an antispasmodic, and at the same time it helps the patient to sleep. One to 2 oz. (28-57 ml.) of whisky or brandy four-hourly is a satisfactory dose for these patients. It is important not to elevate the limb; the position of maximum comfort which most patients adopt in bed with the head and shoulders raised on two or three pillows and the trunk and limbs flat is also the best position for an ischaemic lower limb. Some patients obtain considerable relief when the limb is dependent; in acute vascular occlusion this may be allowed, but not for long periods.

Collateral Circulation

The development of the collateral circulation may be encouraged in a number of ways. The most efficient and at the same time the simplest measure is to establish reflex vasodilatation. To obtain this the affected part should be cooled and the rest of the body heated. With the patient in bed the method we use is to arrange the bedclothes in such a way that the affected limb is exposed to the air. The patient's other lower limb should be covered with a thick woollen sock, and both it and the trunk covered with blankets; a long-sleeved sweater and vest should be worn over the pyjama jacket and the hands in particular should be warmed; this is most easily achieved by asking the patient to wear a pair of woollen gloves, although electrically heated gloves are better if available. In the case

of sudden occlusion of a main artery to the upper limb, this limb should be cooled, and the feet, trunk, and other hand warmed.

Evidence produced by Sir Thomas Lewis and others has established that the vasodilatation produced in this way equals and possibly exceeds that of either a sympathectomy or a sympathetic nerve block. In some patients reflex vasodilatation worsens pain, but this often coincides with recovery of the circulation and is therefore a worth-while price to pay. This pain can be reduced by pethidine, 100 to 200 mg., or, if less severe, by tablets of codeine or aspirin.

It is important that splints, dressings, or the patient's posture should not constrict the region of vascular occlusion around which the collateral vessels must develop. Vasodilator drugs are of value, but in my opinion no proprietary preparation is superior to alcohol as a vasodilator in patients with sudden peripheral arterial occlusion; as already stated, this should be prescribed in liberal doses. Papaverine hydrochloride, $\frac{1}{4}$ to 1 gr. (32–65 mg.), given intra-arterially, proximal to the lesion, may help to promote vasodilatation. Tobacco, on the other hand, causes peripheral vasoconstriction, and a wise patient stops smoking after sudden occlusion of a peripheral artery.

Sympathetic block with procaine hydrochloride or phenol is no better than reflex vasodilatation in promoting the development of the collateral circulation, but if performed with phenol it does enable some patients who would otherwise be bedridden to resume a sedentary occupation. It has no place in the treatment of an acutely ischaemic limb, but a real place in the treatment of elderly and poor-risk patients and of those who need prolonged sympathetic denervation but do not want a surgical operation. The technique consists in placing the patient on his side with the vertebral column straight, the third lumbar vertebra is located and a long (12-cm.) needle inserted at this level, and at a distance of 7 cm. (2½ in.) from the midline; the needle should be directed inwards and advanced until it reaches the side of the body of the vertebra, it should then be manipulated until it slides forward to lie just in front of the convex antero-lateral surface of this bone. Aspiration serves to exclude the presence of blood or cerebrospinal fluid, and then 4 ml. of 4% procaine should be injected; if this produces a sympathetic block 10 ml. of 10% phenol in water is injected and the patient told to lie still for half an hour. This type of block produces a long-lasting sympathetic denervation.

Anticoagulants

Anticoagulants have an important place in the management of patients with sudden vascular occlusion. They help to limit the extension of the thrombus, particularly into the vessel distal to the block where the blood flow is sluggish. They should be used as a routine during the acute stage in all patients with sudden occlusion of the main artery to a limb, and for long periods afterwards in certain patients with arterial thromboses. Heparin, 15,000 units, should be given at once, preferably by intravenous injection, alternatively intramuscularly, and repeated six-hourly for 18 hours. At the same time phenylindanedione, 200 mg., by mouth, should be given and repeated twice daily in doses of 100 mg. Phenylindanedione takes about 18 hours to become effective, and so this combination of drugs produces a rapid effect which can be maintained for an indefinite period by the oral route. Control is by the prothrombin time; this should be reduced to give a prothrombin level of approximately 20% of normal in the acute phase and to about 30% or slightly more when long-term out-patient treatment is used. Most patients reach this level on a dose of 75 mg. twice daily, but some require only 50 mg., and others as much as 150 mg.

Phenylindanedione, once the dose has been stabilized, does not cause wide alterations in the prothrombin level and is therefore a relatively safe drug; once the level has become stabilized, weekly prothrombin estimations are sufficient for control. Should an overdose of heparin be given

the antidote is an intravenous injection of 10 ml. of 1% protamine sulphate, and the antidote for phenylindanedione is 20 mg. of vitamin K₁ given orally. The above dosages for heparin and phenylindanedione refer to patients without a recent surgical or other wound. Both drugs are very difficult to control in patients with a recent wound, and should only be prescribed by those with considerable experience.

General Management

The general health of the patient must be maintained by ensuring an adequate intake of food and adequate sleep. Infection, an ever-present danger in ischaemic tissues, is best prevented by dressing all breaches in the skin with a careful aseptic technique and prescribing penicillin, 1,000,000 units twice daily, at the slightest sign that an infection is becoming established. Deformities may occur, particularly if a Volkmann type of contracture develops in the ischaemic muscle; these may be minimized by putting all joints through a full range of passive movement once a day. In some patients the ischaemic muscles are too painful to permit this. In these cases a light splint helps to prevent the development of gross deformities, particularly that of foot-drop.

Operative Treatment

Surgical operations, such as embolectomy, endarterectomy, arterial repair, or an arterial graft may succeed in restoring a normal blood flow. Others—for example, sympathectomy, incisions in the deep fascia, or arterectomy—may be used to assist the development of a collateral circulation. Alternatively, operation may be resorted to because conservative treatment has failed, when an amputation is necessary either for gangrene or for intolerable rest pain.

Embolectomy is rarely needed in the upper limb; the collateral circulation here is so efficient that the pulse usually returns to the wrist within 48 hours of a brachial embolus. On the other hand, embolic obstruction of the bifurcation of the abdominal aorta is best treated by immediate operation, as are many embolic obstructions of the popliteal or common femoral arteries. A great deal depends upon the appearance of the limb; in many patients the blood flow is returning by the time the patient reaches hospital, and conservative treatment with anticoagulants and reflex vasodilatation alone is effective; however, where there are good facilities popliteal or common femoral embolectomy usually pays, for by this means a normal blood flow can be restored. Arterial repair and arterial grafting have their most important indication in those patients with sudden arterial occlusion due to an injury; of obvious importance in war, these injuries when they occur in peacetime require adequate surgical treatment. For example, I have seen in the last two years three butchers who had accidentally divided their femoral arteries with their own knives; in one an end-to-end suture was possible, and in the other two an arterial graft was necessary; all now have patent vessels with a good blood flow and palpable pulses distal to the lesion.

Sympathectomy is of great value after the acute phase has passed; before this it has a place in those patients who cannot tolerate reflex vasodilatation. The object of the operation is to increase the collateral circulation around the occluded artery; therefore, those ganglia which control the appropriate area of the patient's vascular tree should be removed. For example, removal of the second and third lumbar ganglia denervates the patient from the knee downwards, and when the first lumbar ganglion has been removed the denervated area includes the upper thigh. In addition, sympathectomy makes the skin warm and dry—a valuable contribution to the welfare of a patient with an ischaemic limb. The other two operations designed to assist the development of the collateral circulation—arterectomy and incisions in the deep fascia—have in my view little real value.

Amputation, whilst being a last resort, should not be unduly delayed in patients with rest pain. The level varies

with the level of occlusion in the arterial tree and the efficiency of the collateral circulation. With experience, many patients can receive the added benefit of a conservative operation, but in others a major above-knee amputation is required. The important thing is to obtain primary healing of the skin flaps and a stump which is useful.

Conclusion

Sudden occlusion of a major peripheral artery is an emergency the outcome of which depends to a large extent upon the initial care which the patient receives. The importance of local cooling, reflex vasodilatation, and anticoagulants has been stressed. The ideal to aim at is restoration of the blood flow to normal, but this is not often possible. The alternative is to encourage the development of an efficient collateral circulation.

Next article on Emergencies in General Practice.—"Crises in Myxoedema," by Dr. Richard Asher.

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LEGAL LIABILITY OF DOCTORS

In the fourth of his series of lectures* at the City of London College on February 2, Mr. J. P. EDDY, Q.C., discussed the liability of doctors, hospitals, and dentists to actions for negligence. In recent years, he said, there had been a considerable increase in the number of actions brought against doctors and hospitals. Figures provided by the Ministry of Health showed that payments by hospital authorities in England and Wales by way of compensation of all kinds amounted in 1948-9 to £7,500, in 1950 to £23,000, in 1951 to £38,000, in 1952 to £106,000, and in 1953 to £153,000. The causes of this increase of litigation were: (1) the changed status of the hospital; (2) the payment of hospital staffs; (3) the availability of free legal aid; and (4) the decision of the Court of Appeal under which hospital authorities were responsible for the negligence of their staffs.

Changed Status of the Hospital

Formerly many members of the community devoted much time, thought, and energy to the welfare of the hospital, which was regarded as "our" hospital. Nowadays they were not taking the same personal interest and the hospitals were regarded as State hospitals. Consultants and other members of the hospital staff were formerly not paid or were paid such a modest sum that it might be described as an honorarium. Nowadays consultants, registrars, and house officers were paid a salary; they were appointees, or indeed employees of hospital management committees or regional hospital boards or boards of governors.

Again, the legal-aid scheme had made it possible for members of the public to institute actions against doctors or hospital authorities or both. The committees charged with the duty of issuing legal-aid certificates had been doing their work well, taking reasonable care to satisfy themselves that a prima-facie case existed before issuing a certificate. Where there was an allegation of negligence against a doctor or hospital a medical report was called for. In Scotland the prospective litigant had an opportunity of being heard before a certificate was granted. Mr. Eddy did not think that that

was desirable; there should be nothing in the nature of a preliminary trial, it was enough that there was a prima-facie case.

Finally, the decision of the Court of Appeal in *Cassidy v. Ministry of Health* (1951, 2 K.B. 343) had transformed the legal position in regard to liability of hospitals for negligence on the part of their medical staffs.

Duty of the Doctor

The duty owed by a doctor to his patient was laid down in 1838 in the case of *Lanphier v. Phippos*. A doctor undertook to bring to the exercise of his profession a reasonable degree of care and skill, but before the duty arose there must exist the relationship of doctor and patient. If a doctor saw a person lying injured in the street and passed him by he was not guilty of negligence, because there was no such relationship and consequently he owed no duty; but if he played the part of a good Samaritan the professional relationship was created, even though the doctor had no hope of reward. Moreover, he was liable only when he undertook the treatment of a person, not when he merely examined him at the request of someone else. A doctor incurred liability when he set out to treat a patient, but not when he expressed an opinion at the request of another person (1920, 3 K.B. 163).

In the *British Encyclopaedia of Medical Practice*, Dr. Walter Levitt, pointing out that the doctor must exercise a reasonable degree of care and skill, said:

"What is a competent degree of skill will depend on the circumstances in which the practitioner finds himself. If he practises in a remote village, away from modern sources of inspiration and knowledge, very much less will be expected of him than if he practises in a large town attached to a hospital with every possible convenience and inducement for keeping his knowledge up to date."

The duty of warning the patient of possible danger in the treatment to be undertaken was pointed out in the judgment in *Clarke v. Adams* (1950, 94 *Sok. Jo.*, 599). In an earlier action in 1934, in which the patient complained that a hypodermic needle had broken and been left in her tissues, the defendant claimed that the breaking was due to muscular spasm on the part of the patient, and the judge, while finding that there was no negligence, came to the conclusion that the doctor should have told the patient of the accident. The patient was awarded five guineas damages. Mr. Eddy cited other cases which turned upon the adequacy of the warning given to the patient.

Hospital Liability

In 1906, in the case of *Evans v. Liverpool Corporation* (1 K.B. 160) the plaintiff claimed damages on the ground that his son had been discharged from an infectious diseases hospital while still in an infectious condition and had communicated scarlet fever to his other children. It was held that the plaintiff was not entitled to recover, for the legal obligation of the defendants extended only to the provision of reasonably skilled and competent medical attendance. To-day beyond any question the hospital would have been held liable.

Mr. Eddy mentioned the cases of *Lindsey C.C. v. Marshall* (1937, A.C. 97) and of *Gold v. Essex C.C.* (1942, 2 K.B. 293). The latter did much to clarify the legal position. It turned upon the failure of a radiographer adequately to protect a child's face when Grenz rays were being given. Mr. Justice Tucker held that the defendants were not liable for negligence, but an appeal succeeded, and it was held that a whole-time servant of the hospital, in failing to provide adequate screening material, rendered the hospital liable.

But it was the decision of the Court of Appeal in the *Cassidy* case, already cited, which was one of the reasons for the increased litigation to-day. This was a case originally tried by Mr. Justice Streatfeild at Liverpool Assizes. The patient, suffering from a contracture, had been treated in a Liverpool hospital, and in the treatment his arm had

*The first lecture was reported in the *Journal* of January 22, p. 221.