the felt foot-sling. Other adaptations have been devised, one of which is to put a bandage over the foot first so as to increase friction and allow the sling to be fixed to this. This modification was suggested by the late Mr. Collier at Pendlebury many years ago.

The method of application shown in the sketch (Fig. 5) of an "artificial muscle" applied for talipes equinus is an example of an instrument maker's "improvement" which I do not recommend. It is much better to bring up a cord on each side, as shown in the dotted lines. Varying tension on either side can thus be arranged and much more accurate correction got. The use of "artificial muscles" does not, of course, do away with the need for tenotomy, tenoplasty, tarsectomy, and the various other operations needed in certain cases, but it is a valuable supplement to some of these, and in many cases it prevents the deformity from becoming "inveterate."

For paralytic conditions the elastic traction is a most useful adjunct to, or, in some circumstances, substitute for, tendon grafting. I will only mention the use of

Fig. 6.—Elastic traction applied for torticollis after division of the sterno-mastoid of the opposite side. A, Plaster-of-Paris jacket with hooks; B, head band or felt cap, also with hooks; C, the "artificial muscle." Another elastic is usually necessary at the back to prevent flexion of the head.

the artificial muscle in wryneck. It is usually employed, after tenotomy, with a jacket and some sort of cap (Fig. 6). Traction is made parallel with the unaffected muscle.

There is room for the useful employment of elastic traction and pressure in many more ways in surgery than those I have mentioned, and I strongly recommend you to think out and adapt the plan to any suitable case you may have in charge. These "artificial muscles" have been ignored or decried in some of the orthopaedic and surgical textbooks, but they are none the less most efficient and valuable implements, and may with advantage be used in very many cases in preference to the costly, heavy, and complicated apparatus usually recommended. As compared with the use of plaster-of-Paris, the elastic has the advantage of being more readily adjustable and safer, as well as that of giving a varying and active power.

[I am indebted to my present senior house-surgeon, G. Jefferson, M.B., and to my former house-surgeon, Hamilton Irving, F.R.C.S., for the sketches for this lecture and for some of the corresponding ones in the chapter on this subject in *Diseases of Children* (Ashby and Wright)]

THE next Congress of Diseases of Occupation will take place at Vienna in 1914. The following are the questions proposed for discussion: (1) Fatigue, its physiology and pathology; influence of professional occupation on the nervous system; night work. (2) Work in hot air and in moist air. (3) Anthrax. (4) Pneumoconioses (diseases caused by the inhalation of dust). (5) Accidents caused by electricity. (6) Industrial poisoning, by aniline, mercury, lead, etc. The general secretary of the Congress is Privatdocent Dr. Teleky, IX Türkenstrasse, 23, Vienna.

An Address

ON

HIGH BLOOD PRESSURE IN ARTERIO-SCLEROSIS.

DELIVERED AT THE ACADEMY OF MEDICINE, TORONTO.

BY

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THE occurrence of high blood pressure in arterio-sclerosis has been a much-discussed subject in recent years, since the clinical estimation of this pressure was made possible by von Basch in 1876.

At first high blood pressure was described as a constant symptom in arterial thickening, and even as a measure of the degree of this thickening,¹ so that, although it was admitted that a high pressure might exist without arterio-sclerosis, it was greatly doubted if any considerable degree of arterial disease could be present if the pressure were not raised.

But soon observers began to record cases of even well-marked arterial sclerosis in which the pressure was not raised, and even read below the normal, throughout the illness. Thus Groedel of Nanheim recorded a series of 500 cases of arterio-sclerosis, in 35 per cent. of which there was no rise in pressure, and in 18 per cent. only a slight one.¹ Several years ago Drs. Arthur Ellis and Bruce Robertson investigated the matter with me in St. Michael's and the General Hospitals, and found that in only about 50 per cent. of well-marked cases of arterial thickening was the blood pressure above normal, and since then I have seen several fatal cases of the disease in which the pressure was not raised.

The next view that became prevalent was that arterio-sclerosis only produced a high blood pressure when the splanchnic system of arteries was involved. A high blood pressure means a hypertrophied left heart, and Hasenfeld and Hirsch showed by very accurate weighments² that an enlargement of the left ventricle only occurred in arterial sclerosis when the disease involved the splanchnic vessels or the aorta above the diaphragm. But rise in blood pressure may occur from many other causes than arterial thickening, and because an individual happens to be arterio-sclerotic he is not exempt from such other causes. Probably one may say without much fear of contradiction that when in a case of arterial thickening a raised blood pressure is detected, such a rise may be due to one or more of the following causes:

1. Toxaemia; such toxaemia being at the same time the direct cause of the arterial disease by directly poisoning the vessel walls. A good example of such a poison is nicotine, which directly raises the blood pressure, and according to many authorities also acts directly upon the vessel wall as a poison. Probably there are very many such toxins manufactured in the alimentary tract or in the tissues of the body.

2. Some toxin which has nothing to do with the associated arterio-sclerosis, but is directly raising the pressure. One might theorize that in an arterio-sclerotic patient the adrenal tissues might be from time to time overactive, and might thus produce periodic rises in pressure, as might occur in individuals not arterio-sclerotic.

3. Compensatory. It is well known that if some vital tissues, such as those in the medulla, be rendered anaemic—say, by the pressure of a new growth—the blood pressure rises enormously, seemingly with the object of forcing blood at all hazards into the part. If such a part of the brain be supplied by a vessel the lumen of which is much narrowed by disease, it is likely that the blood pressure will rise from the same cause.

4. Simple nervousness. It is very common to find the pressure high from nervousness, and if an arterio-sclerotic happens to be also a neurasthenic, his pressure may be high from this cause alone.

5. Lastly, the raised pressure may be due to the arterial disease alone, and in this case probably the splanchnic vessels, or the aorta leading to them, are extensively diseased.

While the rôle of arterio-sclerosis in producing hyperpiesis is thus open to much limitation, the rôle of hyperpiesis as a producer of arterial disease seems to be an important one. Given a constantly or occasionally increased blood pressure within the arterial tree for any length of time and the arteries will tend to be dilated, and in order to prevent this dilatation the arterial walls will tend to hypertrophy and thicken, as will also the wall of the left ventricle. As to the exact way in which increased intra-arterial pressure brings about disease of the walls, nothing may be said here, but the fact is undoubted that, given sufficient arterial pressure, we will sooner or later get disease of the walls. This will occur very much sooner in some individuals than in others. Experimentally this has been shown by the effect of clamping the aorta even for a short time, as also by the administration of adrenalin, which greatly raises the blood pressure. In both cases the vessels soon show sclerotic changes. Clinically we see the disease in people who have undergone great exertion, physical or mental, with consequent raising of the pressure, and *post mortem* the change is seen in the pulmonary circulation in cases of mitral stenosis. Dr. T. D. Savill⁴ showed some years ago by *post-mortem* returns how the media of the arteries of the lower extremities were nearly always more hypertrophied than those of the upper; and lately Dr. O. K. Williamson⁵ demonstrated the same thing in another way. He measured the pressure of numerous arterio-sclerotic people, both in the upper and the lower extremities, and found that, with the limbs always at the heart level, the *apparent* pressure—due to the thickened walls—was always higher in the lower than the upper extremities. In normal individuals the pressure was the same in both, and he hence concludes that the higher readings from the lower limbs is due to the extra thickening of the walls here. The matter was hotly discussed at the Royal Society of Medicine, and Dr. Williamson quite held his own. We have been repeating his work in the Toronto General Hospital, and so far our results would tend to confirm his. Dr. Harry Campbell⁶ endeavours to prove that, at least in small and medium-sized arteries, which are in a condition of hypertonus, the muscular coat bears all the strain, and it is only when the muscular coat gives way that the strain can come upon the other tissues.

There have been many classifications of arterio-sclerosis, and perhaps the one that best fits in with what we see clinically is the etiological one of Sir T. Clifford Allbutt. He divides all cases into three classes: (1) Those due to hyperpiesis; (2) those of a toxic or infective nature; (3) involutionary cases. In the first class the raised blood pressure precedes and produces the sclerosis. In the second the pressure may not be raised, or may be so at a late stage, and is the result of the disease. In the third—the senile type—it is usually absent, or, if present, is an intercurrent event, and very open to dietetic and medicinal influence.

One must keep in mind also the terminal cases of arterio-sclerosis, where, with a big heart and thickened vessels, we find a low pressure. I have found a number of such cases in the records of the General Hospital, and can recall a number in my own experience. There had been high pressure in such cases for years, as shown by the great heart, but this pump was now giving out.

The question arises as to our ability to detect and measure an increased blood pressure. Many instruments have been invented within the last few years with this object, and there are several that have been proved by animal experiment to be very correct, the pressure being taken by one of them in one limb, while at the same time it was being measured in another by a manometer directly connected with the interior of a vessel. But whether we can always measure the pressure within a vessel, by seeing what external pressure is necessary to stop the pulse passing along it, is open to question.

If we were dealing with an inanimate elastic tubing it would be an easy matter, but the artery is a living structure and varies greatly in tone and hence in resistance to compression. It is a common thing to feel a vessel firmly contracted—for example, in peritonitis—and it must surely take a great deal of external pressure to eliminate the lumen of such an artery, entirely apart from the pressure within the vessel. Dr. William Russell has insisted much upon this

point,⁶ and there seems to be much in what he says. In cases of intermittent claudication the vessels of the limb may so contract that no pulse may be felt in the distal branches.¹¹ If the contraction of the artery were not *quite* sufficient to stop the pulse one would probably get an altogether too high reading of the general blood pressure if measured in such a limb, as the spastic vessel would resist the external pressure.

As to whether the sclerosis of the artery actually being used in the measurement of the pressure modifies the reading to any extent, it may be recalled that Dr. C. J. Martin about ten years ago seemed to prove by many experiments made upon sclerosed arteries removed from the body that even an extreme degree of hardening did not give rise to a resistance to compression of more than 10 mm. Hg. But more recently Drs. Herringham and Womack found that the resistance of a thickened artery might amount to from 4 to 34 mm. Hg, and Dr. O. K. Williamson found the same thing.⁵ Hence, when the vessel of the limb used for the blood pressure measurement be felt to be much thickened we must considerably discount the reading. In no case are we justified in accepting a high reading when the heart shows no signs of enlargement, nor is the aortic second sound accentuated.

When arterio-sclerosis has caused a rise in the arterial blood pressure, this rise is chiefly in the systolic pressure. This is only what we would expect, as the rigid condition of the vessel walls makes most of the force of the ventricular systole pass right on, as in a rigid tube, say a lead pipe, it would quite do.

At a high arterial pressure the distensibility of the arteries is distinctly diminished, and any increase in the systolic pulse volume must raise the blood pressure far more than a corresponding increase at lower pressure. . . . Thus, in all cases of arterio-sclerosis, where there is a rise in blood pressure, this rise is chiefly in the systolic pressure, on account of the lessened distensibility of the arterial walls, due either to the high tension or the disease. (Janeway.)

In the condition of high blood pressure from pure vaso-motor spasm, as in peritonitis, the diastolic pressure is the one chiefly raised. It would probably be safe to argue that in a case of arterio-sclerosis with increased pressure the relative difference between the systolic and the diastolic pressures would be a measure of the degree to which the increased pressure was due to the sclerosis of the arterial tract between the heart and the point at which the pulse is being felt.

TREATMENT.

We are only here concerned with the treatment of high blood pressure in arterial disease, and it might well be said that if the pressure were due actually to the sclerosis and to that alone, then its lowering could only be brought about, and that only very temporarily, by bleeding. But, as has been shown, the hyperpiesis is nearly always not directly due to such fibrous thickening, but rather to associated spasm of the arterioles, and, one might probably add, capillaries. Such spasm is in the great majority of cases of toxic origin, and it is to the lessening of this toxæmia that our attention must be directed. The nature of this toxæmia has been the object of much research, and it is generally recognized now that it is due to nitrogenous bodies. As Dr. William Russell says, the whole problem of hypertonus centres round the three factors of hereditary character, pabulum, and excretion. Some individuals inherit vessels that no toxin of dietetic origin scarcely will affect, while others suffer on the least indiscretion. As regards the pabulum, the fault may be in mere quantity of proteid taken, or in the way in which this is dealt with in the alimentary tract. In this tract certain bodies, technically known as the aromatic series, are produced by the decomposition of proteid materials; and these being absorbed may, either by direct action on the vessels or in some less direct way, produce vaso-constriction. These poisons must first pass through the liver, and, if this organ be active, may be destroyed there and do no harm. Eek's experiment well illustrates this. He found that if in animals he tied the portal vein and then connected the lower end of this vein with the inferior vena cava and fed the animals on milk, they would live for some time; but if, on the other hand, they were given even a small quantity of meat diet, they rapidly succumbed, with dyspnoea and other toxic symptoms.

Milk contains proteid, of course, but it seems to be specially the proteid of meat and eggs that produces most toxins. If the toxins once reach the blood they are chiefly got rid of by the kidneys. Hence it is that Huchard puts as the second most important factor in the production of the presclerotic stage of arterio-sclerosis insufficiency of hepatic and renal functions.¹³

Sir T. Clifford Allbutt lays down the good rule that every individual of 40 and over should at intervals of five years or so have his blood pressure measured in order that he may know in time if he be tending towards arterio-sclerosis. One may emphasize here that the so-called presclerotic stage continues long after the further stages of actual sclerosis and even heart involvement have become evident, and that in order to prevent the condition getting worse we must continue to treat the toxic condition.

One might emphasize this point by the following classification of the disease:

First Stage.—(1) Toxaemia.
Second Stage.—(1) Toxaemia; (2) organic change in walls of vessels and left heart, and probably in kidney.
Third Stage.—(1) Toxaemia; (2) organic change in walls of vessels and left heart, and probably in kidney; (3) breaking down of the diseased circulatory apparatus.

Take a man of, say, 45 in whom we find a systolic pressure of some 160 mm. Hg. He seems well, but probably has a slight increase in the amount of urine, which will be too watery and may show a trace of albumen occasionally and probably now and then a few hyaline casts.

If this patient show a family tendency to arterial disease so much the worse. If in such an individual we cut down his animal proteids, putting him chiefly on a lacto-vegetarian diet, cut out excessive smoking and the use of alcohol, and give him some saline every morning, with an occasional mercurial purge, he will probably improve very much and his pressure will come down to normal or nearly so, and, as long as he keeps to such simple rules, may remain so. If such a régime be not sufficient regular small doses of potassium iodide may help very much. Pharmacologically Stockman showed years ago that this drug had no effect in lowering blood pressure except in enormous doses, but clinically nothing seems to be more evident than its effect in hyperpiesis due to toxaemia. Probably it helps in elimination. Every clinician has seen its good effect in relieving the pain in aneurysm. Diuretics may be useful, especially theobromine.

The treatment so far mentioned is applicable to all cases of arterio-sclerosis where the pressure is high, whether the case be early or more advanced. But if, in spite of it all, the pressure remain too high, and perhaps symptoms of cerebral trouble—such as transient affection of speech or even paralysis—are showing, then severe purgatives or even bleeding may be required. But the question always arises here whether we should interfere actively with a high pressure merely because it be high—whether this pressure may not be compensatory, and the best thing under the circumstances for the crippled patient. May not his symptoms be due to an anaemia of a vital part of the brain, owing to a sclerosed and narrowed vessel of supply? Here the high pressure may be keeping him alive. Again, thrombosis may be occurring in such a vessel, and the most likely thing to increase such clotting will be to reduce the flow of blood through the artery, and to increase the coagulability of the blood, as may be done in both instances by the operation of bleeding.

It is probable that no one rule can be laid down about the care of high blood pressure in cases of advanced arterio-sclerosis beyond those of diet and other simple means applicable to the presclerotic stage.

Moreover, it is wonderful how well many people are with blood pressures that may at first surprise us, and not only well, but may remain so for years. Especially in stout individuals, we may find pressures reading even over 200 mm. Hg. in whom there may be practically no symptoms.

Occasionally, however, the pressure is producing some trouble that makes it urgent that we reduce it, and here blood-letting or the use of vaso-dilators may be of much value.

As regards blood-letting, most of us have seen good results from an epistaxis in an arterio-sclerotic, and such results are wonderfully lasting. If a normal animal be

bled, especially from a vein, we can reduce the arterial pressure easily, but a bleeding, even amounting to half of the total blood in the body, only reduces the pressure for a very short time. It is different in cases of toxaemia, and the chief cause of the very lasting results that are obtained is probably that by bleeding we are removing the toxins that are the chief cause of the raised pressure.

One sees the good effect of bleeding also in uraemia, where it is evidently by removal of toxins that it acts. Osler states the case well when he says:

In cases that come under observation for the first time with dyspnoea, slight lividity and signs of cardiac insufficiency, venesection is indicated. In some instances, with very high tension, striking relief is afforded by the abstraction of 20 oz. of blood.⁸

With regard to the use of the vaso-dilators, it is found that they usually act even in profound cases of arterio-sclerosis, and recently Drs. Wallace and Ringer⁹ have shown that their action is more prolonged here than in normal vessels. One might put them in the following order as regards their speed of action and associated evanescence of effect: Amyl nitrite, nitroglycerine, spiritus aetheris nitrosi, sodium nitrite, erythrol nitrate, and, lastly and most lasting, the nitrate of mannitol.

Dr. Edwin Matthew¹⁰ last year published some work on the action of the different nitrites in high blood pressure, which may be summarized as follows:

1. The effect of nitroglycerine in $\frac{1}{100}$ -grain doses lasts about one hour, but a tolerance is rapidly established.
2. The effects of sodium nitrite in 2-grain doses last about two hours, and no tolerance occurs.
3. Erythrol tetranitrate in $\frac{1}{2}$ -grain doses lasts about six hours, and no tolerance occurs.
4. Mannitol nitrate in 1-grain doses acts as the erythrol, but effect even more prolonged, and hence to be preferred.

With these drugs he found it possible to keep the abnormal pressure down about 30 mm. Hg. and such often kept the patient comfortable and free from pressing symptoms. It is interesting to note that he could get no results with tabloids of nitroglycerine or erythrol nitrate, but got them at once with solutions of the former and chocolate tablets of the latter.

As regards the sweet spirits of nitre, it is well to note that it is often very weak, or even inert. In a recent investigation made in the laboratory of the Inland Revenue Department of Canada it was found that 63 per cent. of samples examined were adulterated and that some contained no ethyl nitrite at all. There is no standard for strength in the British or Canadian pharmacopoeias, but in the American it must contain 4 per cent. of the ethyl nitrite when freshly prepared. When made according to the B.P. directions, it should contain from 2.75 to 1.75 per cent. Sodium nitrite also rapidly oxidizes and thus loses its action, so should be freshly prepared. Physicians probably often fail to get the action looked for with these drugs on account of these depreciations.

Various other and milder methods of reducing blood pressure in arterio-sclerosis are often used, such as baths, massage and gymnastic exercises, and static electricity; but no attempt has been here made to discuss them all, and probably most of the best known and most commonly used ones have been mentioned.

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THE Chelsea Hospital for Women has received a grant of £105 from the Court of Common Council of the City of London.

THE annual general meeting of the British Medical Benevolent Fund Guild will be held at the New Hospital for Women, 144, Euston Road, N.W., on Tuesday, November 29th. Lady Frederick Brudenell Bruce will take the chair at 3.30 p.m.