THE CLINICAL BEHAVIOR OF ARTERIOSCLEROTIC ANEURYSM OF THE ABDOMINAL AORTA: A RATIONAL SURGICAL THERAPY

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IN A GROUP of 32 cases of aneurysm of the abdominal aorta admitted to the Presbyterian Hospital in recent years, arteriosclerosis was the cause of the aneurysm in 26. These aneurysms were fully developed, fusiform in shape and arose from the abdominal aorta three or more centimeters distal to the origin of the renal arteries. Syphilis was the etiologic agent in six of the 32 cases, these aneurysms being saccular and causing vertebral bone erosion in four instances. In this latter group the aneurysm arose distal to the renal arteries in only one of the six cases. There were only two females in the entire group of 32 cases, both having arteriosclerotic aneurysm, and the average age of those with arteriosclerotic aneurysm was 60 years while it was 42 years for the syphilitic group.

One gains the impression from the literature that there is a high incidence of vertebral erosion in aneurysm of the abdominal aorta but such was not the case in the arteriosclerotic group of our series. Of the 26 cases of arteriosclerotic aneurysm, vertebral erosion was noted in only one instance, and in this case, erosion was due to aneurysmal involvement of the right common iliac artery. Aside from the fact that the arteriosclerotic aneurysm is usually fusiform, it seems likely there may be another factor accounting in part for the rarity of bone erosion, namely, elongation of the abdominal aorta. Considerable elongation of the vessel seems an invariable precursor to the formation of the arteriosclerotic aneurysm of the abdominal aorta. In fact, a study of many specimens reveals, a few centimeters below the renal arteries, a sudden change in direction of the aorta, (usually forward, but often to the left or right), and immediately distal to this angulation the fusiform aneurysm begins. It is common at operation to note an angulation of the aorta sometimes as much as 90 degrees immediately proximal to the origin of the arteriosclerotic aneurysm, the latter scarcely touching the vertebrae. What may be the added effect upon angulation by flexion of the thoracolumbar spine is a matter of conjecture; and, of equal importance, the hemodynamic water hammer effect of strain upon the lower abdominal aorta consequent to angulation of the sclerotic iliac arteries by flexion of the thighs as in the sitting position. We speak of angulation of the arteriosclerotic artery in the sense of its walls buckling rather than bending with a gentle curve, as would a normal artery.

We know from experimenting with arteriosclerotic popliteal arteries that flexion angulation causes a water hammer effect of increased strain upon the popliteal artery lying immediately proximal, unsupported in the upper popliteal space. In our opinion, this is an important factor in the development of

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arteriosclerotic popliteal aneurysms and it seems likely that a similar mechanism may play a part in the development of the arteriosclerotic aneurysm of the lower abdominal aorta. As possibly having some bearing on the above discussion, it is of interest that, as far as our hospital service is concerned, the popliteal artery and the lower abdominal aorta are the common arteries affected by arteriosclerotic aneurysm. By this we mean well-developed aneurysms—not counting, of course, dissecting aneurysms of the sclerotic thoracic aorta.

As may be expected, the difference in pathology between the syphilitic and arteriosclerotic aneurysm of the abdominal aorta, as pertains to vertebral erosion, affects the incidence and severity of pain. Thus, the four syphilitic cases with vertebral erosion had for months received opiates for the control of severe radicular pain. Complete relief of pain following wiring and electrothermic coagulation of the aneurysm could only mean that the aneurysm became smaller in size following operation, thus, relieving pressure upon the spinal nerves. Unfortunately one of the four cases having a subluxation of the vertebrae died of infection following a bone graft procedure done some months after the wiring operation. The remaining three cases are alive, active and pain free now, eleven, seven and four years, respectively, following operation.

One gains the impression from the literature that the high incidence of vertebral erosion with resultant radicular (nerve pressure) pain is so constant in aneurysm of the abdominal aorta as to constitute a diagnostic sign. Our attention is called to the fact that vertebral erosion was present in only five of the 32 cases herein reviewed and in only one of the '26 cases in which the aneurysm was due to arteriosclerosis. This suggests, that in the past medical literature of the symptomatology of aneurysm of the abdominal aorta has been built up largely from syphilitic cases—cases which give a long drawn out history of severe pain due to nerve pressure from bone erosion.

Pain is the most important symptom that makes people seek medical relief. Due to the absence of bone erosion as an early cause of pain in the arteriosclerotic aneurysm of the abdominal aorta, the unfortunate victim of this disease often goes to the brink of disaster before seeking medical aid. A recent case illustrating this may be outlined as follows: A local physician called, stating that five days previously he had seen, for the first time, a 64-year-old man who gave a history of having, from time to time, for several months, a slight pain in the low back, including the sacro-iliac regions. The pain was exaggerated by bending forward or by exertion. Suddenly the pain became intense, radiating to the posterior aspect of the legs. Three days after onset the pain had extended up the back, over the abdomen, into the groins and down the anterior aspect of the thighs. On the fourth day of onset the patient began to vomit. When I saw the man, two days later (on the 6th day of his illness) he was under the influence of sedatives; pulse 84, B.P. 120/60. He appeared anemic but not in shock. The skin was warm and dry. The abdomen was not soft but neither was it distended with gas. A large, pulsating mass was present in the region of the abdominal aorta. The scrotal and perineal tissues were ecchymotic. An hematocrit made at this time was 23.8.

This man undoubtedly had had a large arteriosclerotic aneurysm of the abdominal aorta for months before having what he considered sufficient symptoms to warrant seeing a doctor. His first real nerve pressure pain comparable to the radicular pain of early bone erosion in the syphilitic case was actually caused by rupture of the aneurysm resulting in a spreading retroperitoneal hematoma.

Essentially, the story of pain from the beginning rupture of the aneurysm as the presenting symptom of consequence was elicited in 11 of the 26 cases of arteriosclerotic aneurysm. Some of the cases came under medical care with the history of less violent and more varied initial symptoms. Loss of weight is a common complaint in arteriosclerotic, as is the case with syphilitic aneurysm of the abdominal aorta. Eleven of the 26 cases of arteriosclerotic aneurysm had sustained weight losses varying from 15 to 35 pounds.

In five of the 26 cases of arteriosclerotic aneurysm a pulsating mass in the abdomen first directed attention to a diagnosis.

A sudden episode of severe, cramp-like abdominal pain, followed by collapse, was the apparent symptom of onset in a 51-year-old physician in whom, four months later, a well-developed fusiform arteriosclerotic aneurysm was palpable.

Severe hunger pains, combined with anorexia, punctuated the symptoms in another case—a man of 55, in which the aneurysm had been observed to grow to the size of a large grapefruit over a three and one-half-month period.

Our oldest case, a man of 72, had attacks lasting several days, which simulated ileus or duodenal obstruction. One of these attacks complicated our second-stage wiring operation. The old gentleman recovered, following Miller-Abbott intubation, however, and lived some two years, finally dying of a cerebral accident. Considering the great amount of forward displacement of the third portion of the duodenum, often seen in cases of large aneurysms, one marvels at the relative infrequency of symptoms of duodenal obstruction. Postoperative edema does add to this hazard, however, and in one other case we considered it a contributory cause of death. Again, contrary to what one would expect, in the relation of the transverse colon to a large aneurysm of the abdominal aorta, we have been surprised at the rarity of bad constipation.

We may close the discussion on the symptomatology of arteriosclerotic aneurysm of the abdominal aorta with a word of warning about pain. Analysis of the 26 cases of arteriosclerotic aneurysm affords convincing evidence that the appearance of deep-seated abdominal pain, low back pain and pain radiating to the hips or legs is a warning of impending disaster. Furthermore, my experience has been that the mortality of well-developed arteriosclerotic aneurysm of the abdominal aorta is quite as great as that of syphilitic aneurysm.

THE SURGICAL TREATMENT OF ANEURYSM OF THE ABDOMINAL AORTA

The ideal surgical therapy for degenerative arterial aneurysm entails cure of the aneurysm with maintenance of the arterial blood flow through the vessel affected. Whereas the above ideal has been successfully accomplished in

so-called peripheral aneurysm, in aneurysm of the aorta, where it has been considered essential to preserve arterial blood flow, we have been forced to rely upon blood clotting within the aneurysmal sac as our main bulwark against the continued growth of the lesion.

In the year 1935, with the valued aid of Professor Barry G. King, of the Department of Physiology of the College of Physicians and Surgeons, a series of investigations in the hemodynamics of aneurysm was begun. In the first place, when the yellow elastic tissue of the aorta wall is destroyed and the media gives way, the budding aneurysm is subjected to a strain which increases *pari passu* with its growth. Blood, circulating under pressure, creates a strain on the sac wall of an aneurysm varying with the square root of its surface area and, to a lesser extent, with the rate of blood flow. Doubling the diameter of the aneurysm, for example, increases the strain on the sac 100 per cent. The stretched sac is further devitalized due to failure of blood supply to the sac wall to keep pace with the expanding sac. Thus, there is a vicious circle in a growing aneurysm of increased strain upon a sac wall of diminishing strength.

On the other hand, clotting of an aneurysm does offer the advantage of the physical laws governing solids. To illustrate: when the fluid blood of a saccular aneurysm becomes a solid, the total strain upon the aneurysmal sac wall is reduced from the aggregate pressure exerted on the total surface area of the sac wall to that of the pressure exerted on the surface area of the mouth of the aneurysm only. The true magnitude of this reduction in strain is only fully appreciated when one stops to consider that the surface area of the mouth of the saccular aneurysm is usually but a small fraction of the total surface area of the sac wall. It is not surprising, then, in accordance with the above mentioned physical laws, that authentic instances of nature's cure of saccular arterial aneurysm have accumulated in the literature of pathology for over 100 years—cases in which clotting of the aneurysm had resulted in complete inactivation of the lesion—the patients having subsequently died of some other cause. It is but reasonable to assume that removing the strain upon the sac wall, caused by the expanding pulsations of fluid blood, by converting the blood to a solid blood clot affords the strengthening processes within the sac wall an opportunity to become effective.

Our studies revealed that the initiation and propagation of mass clotting within aneurysms is dependent largely upon two important factors: namely, (1) the presence of adequate stimulus to blood clotting; and (2) the rate of blood flow through the aneurysm. We have obtained rate of blood flow measurements in approximately 150 aneurysms. It is of extreme importance to note the great variations in the rate of blood flow. For example, in two saccular aneurysms of equal size the rate of blood flow through the aneurysm may vary as much as 300 per cent. The larger the mouth of a saccular aneurysm the higher the rate of blood flow through the aneurysm. In cases of so-called "cup-shaped" saccular aneurysms in which the diameter of the mouth of the sac is equal to, or exceeds, the diameter of the sac, the rate of blood flow

through the aneurysm is extremely rapid—approximating that of fusiform aneurysms.

One may correlate the relation of the rate of blood flow to spontaneous clotting within aneurysms by simply reviewing pathologic specimens. It is rare, indeed, to find any appreciable deposit of blood clot within fusiform or wide-mouth saccular aneurysms.

Thus, we see that nature's cure of aneurysm has been relegated usually to the occasional case of small-mouth saccular aneurysm in which conditions favor complete "brimful" clotting of the aneurysm.

The first attempts to induce clotting within aneurysm by the introduction of wire date back to Moore, in 1864. Fifteen years later, Corradi suggested the use of an insulated needle through which to pass silver wire into the aneurysm. The protruding end of bare wire was then connected to the positive pole of a battery, the negative electrode was applied to the skin in the neighborhood and a galvanic current passed. The resulting ionization product deposited at the surface of the wire was depended upon to encourage the initiation of blood clotting within the aneurysm. Subsequently, Colt advocated the introduction of multiple wisps of wire into aneurysms to act as a nidus for the initiation of blood clotting. A thorough review of published cases employing these wiring methods revealed a great uncertainty in the results with an occasional brilliant success. Now that we know of the great variation in the rate of blood flow in aneurysms and the relation of rate of blood flow to blood clotting, we can readily understand their results.

We now have conclusive experimental and clinical evidence that a method of wiring aneurysms to be efficient in the induction of mass (brimful) clotting must fulfill the following requirements: (1) it must afford an adequate and efficient clot-stimulating surface to the passing blood; and (2) it must furnish a means of measuring blood flow in the individual aneurysm as a guide to the amount of wire it is necessary to introduce to impede the blood to a rate of flow at which, in the presence of an adequate clot-stimulating surface, complete mass clotting of the aneurysm will take place.

METHOD

The electrothermic method of coagulating aneurysms embraces the use of fine (34-gauge B. & S.), insulated, coin silver wire sterilized by autoclave. The wire is introduced into the aneurysm in ten-meter segments through a special needle (Fig. 1). Five meters of wire are wound on each of two aluminum spools and bent into a loop in the middle for passage through the needle. This permits the two ends of the segment of wire to protrude from the needle for the purpose of establishing electrical contact.

A source of approximately 100 volts of direct current is used for the heating apparatus (Fig. 2). The current is so regulated (Fig. 3) and calibrated against changes in the electrical resistance of the wire upon heating as to show an accurate measurement of the temperature of the wire on a ratiometer at all times, and of the rate of blood flow through the aneurysm upon initial heating. Each ten-meter segment of wire is finally heated to 80° C. for

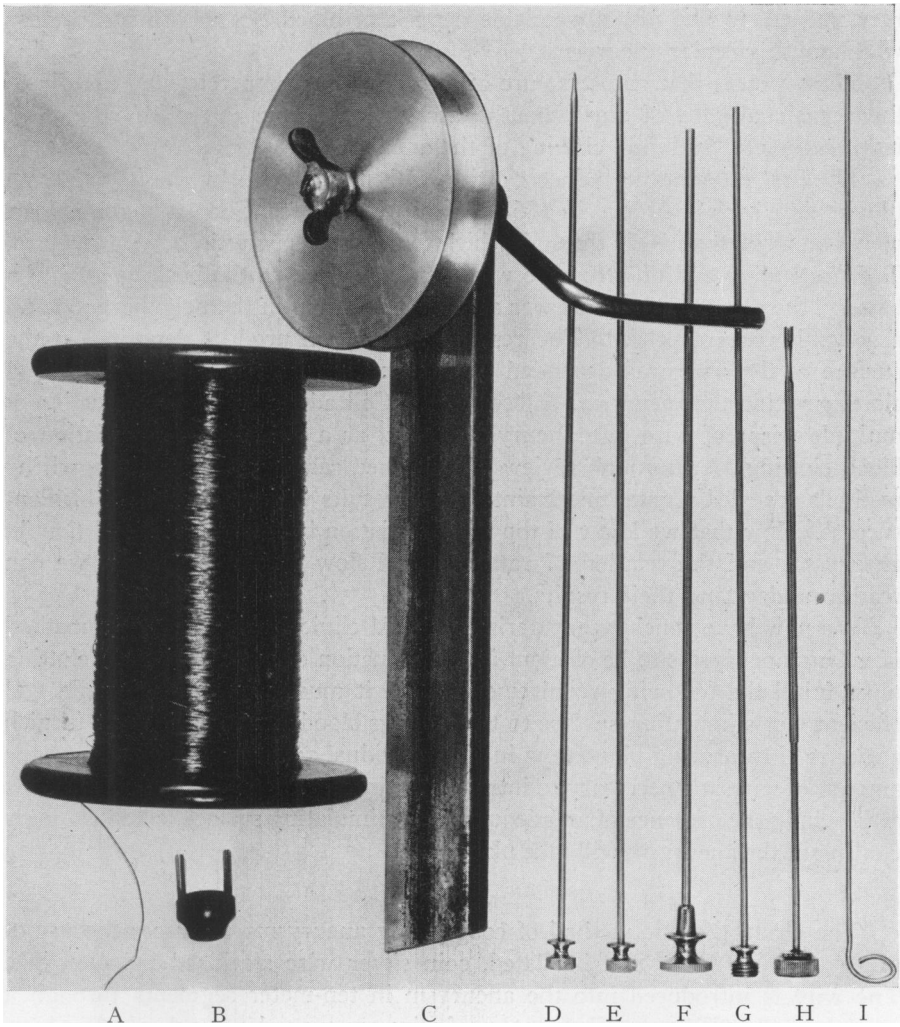

FIG. 1.—Armamentarium for Coagulating Aneurysm:

(A) Enameled wire on spool; (B) electrode cap, adapted to (G); (C) hand reel for winding wire on spools; (D) a trocar-pointed stylet adapted to (F) for traversing firm tissues and piercing the sac of the aneurysm; (E) tapered blunt stylet adapted to (F) for traversing tissue of lung; (F) 14-gauge, 6.5 in. needle made of stainless steel, with the end beveled from without in; (G) inner sheath for needle (F) which locks in place and has the end beveled from within out, fitting exactly with needle (F) to furnish a rounded nonabrasive end; (H) wire-passer adapted to (G), and (I) a blunt stylet adapted to (G).

a ten-second period. This results in the deposit of a tenacious, clot-stimulating, protein coagulum upon the wire.

On the basis of the number of amperes of current required to heat the first ten-meter segment of wire introduced into the aneurysm it is possible to determine the following: (1) the variety of aneurysm (based upon rate of blood flow); and (2) approximately the number of segments of wire it will be necessary to introduce into the aneurysm to impede the blood flow to the point of occurrence of mass clotting, namely, the requirement of three amperes to raise the temperature of a final segment of wire to 80° C.

To avoid the occurrence of "hot spots" upon heating and to promote a more even distribution of protein coagulum upon the wire throughout the segment it is desirable to attain an even distribution of the wire within the aneurysm. This is greatly facilitated by mounting the two spools upon a geared reel which,

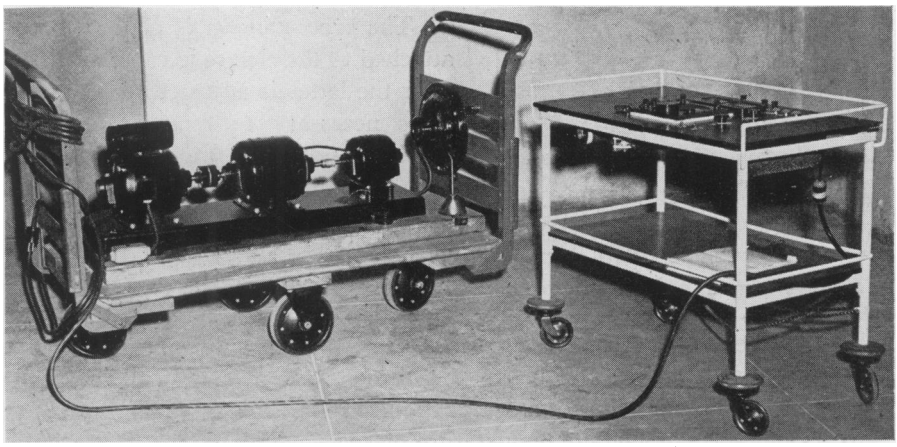

FIG. 2.—Photograph of alternating to direct current converter and heating control panel.

when turned by an assistant during the introduction of the wire, rotates the spools in opposite directions. This, in turn, twists the two wires in opposite directions, forcing them apart upon gaining the inside of the aneurysm. The wire has sufficient expansile thrust when combined with the torque imparted to the wire by the geared reel, to serve to distribute it evenly through the aneurysm.

TECHNIC

Rigid asepsis directed against air-borne as well as direct bacterial contamination must constantly be kept in mind. In cases in which a direct surgical approach is not made to expose the aneurysm, the operation is done under novocaine infiltration anesthesia with roentgenologic guidance. It is now considered best to introduce the needle through a skin incision, using towels over the skin edge held in place by skin clips. All instruments used to this point are discarded and gloves are changed before proceeding with the operation. The special needle (Fig. 1, F), is introduced through the sac wall of the

aneurysm, using the sharp trocar-pointed stylet (Fig. 1, D). The trocar-pointed stylet is replaced by the special inner sheath (Fig. 1, G) bearing a blunt stylet Fig. 1, I). A loop of wire is passed through the wire-passer (Fig. 1, H), which, for the time, is left unattached to the needle. Adequate wire is unwound from the spools mounted upon the geared reel. The loop of wire is then advanced through the needle to a point two or three centimeters beyond the end of the needle. The operator holds one wire and advances the other, thus, forcing the formation of the first loop of wire within the aneurysm. Next, the wire that has been held is gently withdrawn until the bend in the wire snugs-up to the end of the needle. This assures one that the loop thus formed and subsequent loops will land within the sac of a saccular aneurysm. After the passage of several loops of wire by hand, the wire-passer is attached to the needle and the wire quickly passed two wires at a time. The introduction of a ten-meter segment of wire requires about five minutes.

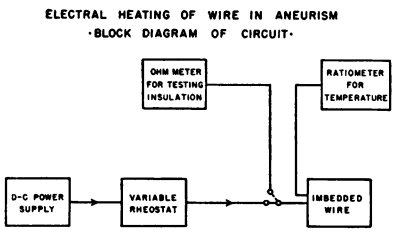

FIG. 3.—Diagram of electrical circuit.

The protruding ends of the wire are attached to the electrode cap (Fig. 1, B) after the latter is adjusted to the needle. It is necessary to remove thoroughly the insulation from the wire in contact with the binding posts of the electrode cap. A roentgenogram of the aneurysm at this point will confirm the position of the wire and reveal the amount of clot already present in the aneurysm, if any. The insulation of the wire is tested before heating, (see ohmmeter, Fig. 3). The rate of blood flow through the aneurysm is next measured by noting the number of amperes required to raise the temperature of the wire 15° C., *i.e.*, an average temperature of 53° C. The wire is finally heated to 80° C. for a period of ten seconds. After an interval the wire may be reheated for another ten-second period. Finally, the wire is clipped flush with the needle and the ends pushed into the aneurysm with a blunt stylet. (Fig. 1, I). Additional segments of wire are introduced and heated until a final ten-meter segment requires three amperes to raise the temperature of the wire to 80° C. Experience has revealed that when one or more segments of wire, well-distributed within a saccular aneurysm, requires only three amperes to heat the wire to 80° C., mass clotting of the aneurysm takes place at once. It has been a routine policy at the final operative stage to check the completeness of clotting by making an arteriogram.

Since the rate of blood flow through aneurysms of similar size may vary as much as 300 per cent, the amount of wire necessary to impede blood flow to a point conducive to mass clotting must likewise vary greatly. Each ten-meter (33-foot) segment of wire has a surface area of eight square inches. The deposit of a layer of protein coagulum by heating increases the surface area to approximately 16 square inches. Some idea of the magnitude of the clot-stimulating surface exposed to flowing blood and the impedence to blood flow effect

may be obtained by multiplying the above figure by ten, the number of ten-meter segments often required to obtain mass clotting in wide-mouth, high rate of flow aneurysms.

Adherence to the wire and to the sac wall of a clot deposited within an aneurysm is imperative and its organization desirable. Whereas wire alone may reinforce blood clot, the heating to 80° C. of wire distributed on the inside surface of an aneurysm sac, as above advocated, causes inflammation in the sac wall. Tissue subjected to a temperature of 80° C. for a few moments reacts within 24 hours with edema, vascular engorgement, and a typical inflammatory reaction on the part of the white corpuscles. The peak of the heat inflammation is reached in from four to six days. This is followed by a period of repair, in which fibroblasts appear and tissue organization takes place with the aid of a network of budding capillaries. The entire reaction occupies a period of from ten to twelve days without, at any time, the appearance of tissue slough. Experimental and clinical evidence suggests that inflammation as engendered by the electrothermic method of wiring aneurysms is an important factor in strengthening the sac wall of an aneurysm.

The electrothermic method has enabled us to attain "brimful" clotting of wide- as well as small-mouth aneurysms. Extensive clotting has been attained in high blood velocity fusiform aneurysms and in a few instances the channel available to blood flow through the aneurysm has been narrowed to approximate the diameter of the parent aorta. A fair number of cases of wide-mouth saccular aneurysms due to syphilis treated by wiring and electrothermic coagulation has now been followed for periods in excess of five years (one case ten years) without showing any return of preoperative symptoms or roentgenographic evidence of increase in size. However, a statement of physical fact was made that "brimful" clotting of saccular aneurysms reduces the strain upon the sac to that resulting from the lateral wall pressure upon the surface area of the mouth of the aneurysm only.

Since the strain is in proportion to the surface area of the mouth, other things being equal, a fully clotted, wide-mouth saccular aneurysm is subjected to a greater strain than a fully clotted, small-mouth saccular aneurysm, and, by the same token, a well, but not completely clotted, fusiform aneurysm is subjected to more strain than a "brimful" clotted wide-mouth saccular aneurysm. As would be expected from these physical facts, our results have indicated that we may expect permanent inactivation in cases of saccular aneurysm belonging to the smaller-mouth group in which "brimful" clotting may be attained (longest follow-up 11 years). Some of the wide-mouth syphilitic saccular aneurysms, however, have remained stabilized for several years, only to manifest evidence of gradual reactivation. A few of these were subjected to another wiring operation with remarkable palliation of symptoms. The wiring and electrothermic coagulation method has given a new lease on life to many cases of fusiform syphilitic aneurysm of the aorta. Many of the cases were stabilized for 4-, 6- and 8-year periods, during which they were in the great part symptom-free from shrinkage of the aneurysms following operation.

Early in our experience with wiring and electrothermic coagulation of the arteriosclerotic fusiform aneurysm of the abdominal aorta we appreciated that less inactivation of the aneurysm resulted from a given amount of clot deposited than obtained in a similarly-treated syphilitic fusiform aneurysm. This lack of resistance to expansion of the aneurysm sac is not unexpected when one considers that arteriosclerotic degeneration is likely to involve the entire thickness of the aorta wall.

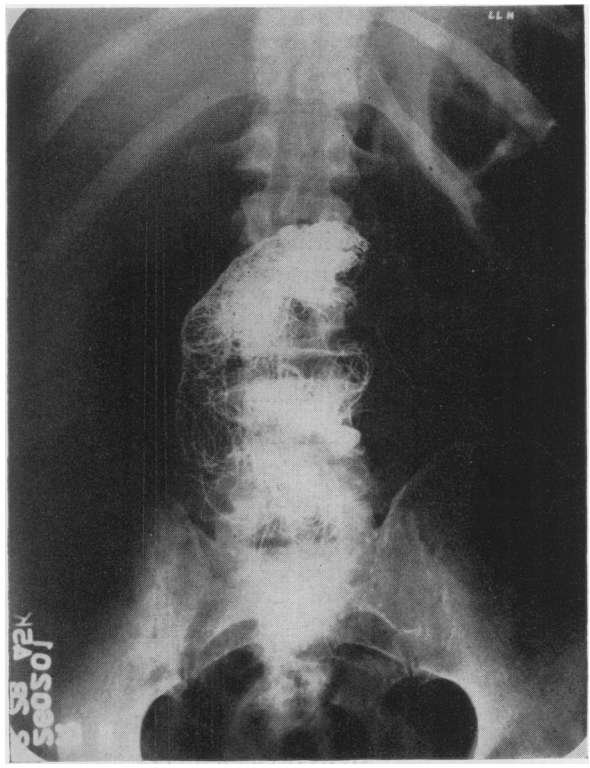

FIG. 4.—Roentgenogram of arteriosclerotic aneurysm of the abdominal aorta in a man age 55.

The fusiform aneurysm with the aorta has been occluded with wire, using the method of electrothermic coagulation and endo-arterial occlusion. The collateral circulation is adequate.

Whereas, it seemed certain that clotting the arteriosclerotic fusiform aneurysm down to a narrow channel did slow up the rapidity of growth of the aneurysm, a complete cure could only be counted upon if all blood ceased to flow through the aneurysm. The above conclusion was gradually arrived at through an experience with several cases over a 2- to 4-year period.

As previously stated in our group the arteriosclerotic aneurysm arose from the abdominal aorta some three, or more, centimeters distal to the origin of

the renal arteries in all of the cases examined. This is a fortunate finding because, following wiring with the deposit of clot within the aneurysm, there is one obstacle to hurdle to attain a cure of this vicious lesion, namely, a safe and sure way of occluding the arteriosclerotic abdominal aorta. A critical review of the total surgical experience recorded of ligation of the abdominal aorta prompted the late Dr. Mont R. Reid[1] to state that "partial or gradual occlusion of the aorta by compression (constriction) cannot succeed and should not be attempted. This applies also to the use of partial constrictions proximal to a totally occluding ligature." Brooks, Blalock and Johnson[2] have pointed out that the immediate mortality and incidence of gangrene is so high in humans as to preclude the use of a one-stage occlusion of the aorta.

An experience with some five cases of arteriosclerotic aneurysm in which gradual compression (constrictive) occlusion of the aorta was practised in conjunction with wiring and electrothermic coagulation of the aneurysm convinced me of the wisdom of Doctor Reid's remarks. A possible safe way of securing gradual occlusion of the aorta suggested itself to us in treating four cases of arteriosclerotic aneurysm of the popliteal artery by wiring and electrothermic coagulation. This was before the days of controlled anticoagulant therapy and the wiring method was decided upon because the collateral circulation was poor in these cases. The fusiform aneurysms were gradually and progressively occluded and with them the popliteal arteries, in two or three operative stages, in these four cases without mishap. This experience proved two things: (1) that in the electrothermic method we had a way of obtaining controlled blood clotting; and (2) the impedance effect of wire introduced into the popliteal artery afforded a means of gradual occlusion of the artery from within, without interference with the blood supply to the artery wall.

We thought it fitting to try this principle of endo-arterial occlusion first on a case of arteriosclerotic aneurysm of the abdominal aorta who had restored an essentially normal blood flow through the aneurysm because of cutting through of a cotton tape which had been placed upon the aorta some months previously. Not only had the tape cut through the aorta wall for most of its circumference but there was at the band site a thin-walled aneurysm, the size of a plum. First, a 33-foot segment of insulated wire was introduced into the secondary (traumatic) aneurysm and heated to 80° C. Following this, some 150 feet of No. 34-gauge (B & S) coin silver wire coated with partly hydrolyzed polyvinyl acetate was forced into the abdominal aorta immediately proximal to the beginning of the aneurysm but distal to the origin of the renal arteries. The wire was concentrated in a ball obstructing the aorta to the desired amount using the oscillometer on the leg as an indicator. Partly hydrolyzed polyvinyl acetate coating of the wire was employed primarily for its tissue-irritating effect in stimulating fibroplasia through the meshes of the wire. If this effect is not desired, *e.g.*, in a first-stage partial closure, bare wire may be employed. The combined principle of using unheated wire for gradual occlusion of the aorta immediately proximal to the origin of the aneurysm and

heated (insulated) wire directly in the aneurysm was successfully carried out in this case and it is now some three years since operation. The combined method of endo-arterial occlusion and electrothermic coagulation has now been employed successfully in three cases of fusiform arteriosclerotic aneurysm of the abdominal aorta and two cases of syphilitic fusiform aneurysm of the abdominal aorta. It is unnecessary to emphasize the necessity of using a method of inducing clotting within the aneurysm in which control of the amount of clot induced at a given time is possible. After a thorough investigation of the methods of creating clot-stimulating surfaces upon wire (electrolysis, protein coatings from blood, gelatin, *etc.*), we concluded that electrothermic coagulation, according to the technic described, is the only method which affords selective, controlled clotting within aneurysms. The electrothermic method gives selective clotting following the introduction and heating of a given amount of insulated wire simply because the clot-stimulating protein coagulum is deposited only upon those portions of the wire within the recesses of the sac of the aneurysm. Wire thus located becomes heated and, thus, coated with protein coagulum because it is not exposed to the cooling effect of the faster moving blood in the axial current. This important underlying characteristic of the electrothermic method of wiring aneurysms makes it peculiarly suited to use in combination with endo-arterial occlusion in the gradual obstruction and clotting of the parent artery with the aneurysm in those cases of aneurysm in which sudden occlusion would mean the loss of life or limb.

SUMMARY

Some important differential features in the pathology and symptomatology of aneurysm of the abdominal aorta due to syphilis and arteriosclerosis are brought out in a review of 32 cases. Arteriosclerosis was the cause of the aneurysm in 26 of the 32 cases reviewed.

The importance of pain as a warning of beginning rupture with spreading retroperitoneal hemorrhage in cases of arteriosclerotic aneurysm was emphasized. Attention is called to the fact that there is almost invariably an interval of from two to six days following beginning retroperitoneal hemorrhage before death supervenes from sudden rupture of the retroperitoneal hematoma into the general peritoneal cavity—a period during which life-saving measures may be instituted.

A method of wiring and electrothermic coagulation of aneurysms is presented and discussed. A technic is described for the surgical cure of fusiform aneurysm of the abdominal aorta employing wiring and electrothermic coagulation in conjunction with gradual endo-arterial occlusion.

CONCLUSION

It seems certain, due to the tremendous decrease in mortality from pneumonia and some other diseases, that formerly took great toll in people of the arteriosclerotic age-group, that the surgical therapy of the arteriosclerotic

aneurysm of the abdominal aorta will assume a position of increased importance.

REFERENCES

[1] Reid, Mont R., and Andrus, William DeW.: Nelson Loose Leaf Surgery, Volume I, Chapter XII, page 647. New York, Thomas Nelson & Sons.

[2] Brooks, Barney, Blalock, A., and Johnson, C. S.: Ligation of the Terminal Abdominal Aorta: An Experimental Study. Arch. of Surg., 17, No. V, 794–812, November, 1928.