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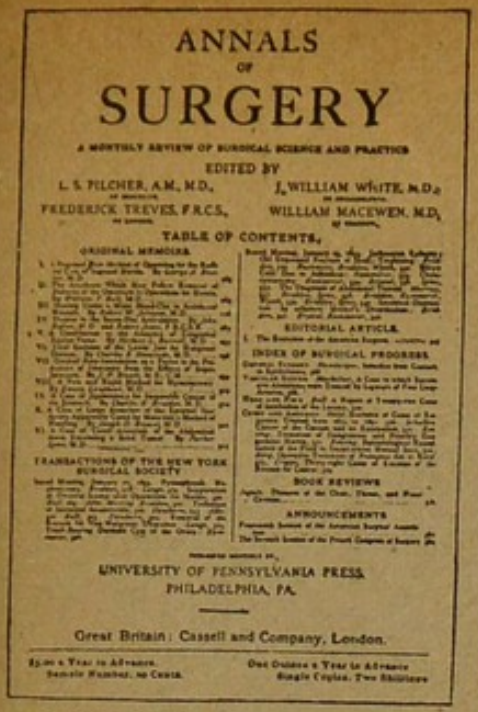

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A MONTHLY REVIEW OF SURGICAL SCIENCE AND PRACTICE

EDITED BY

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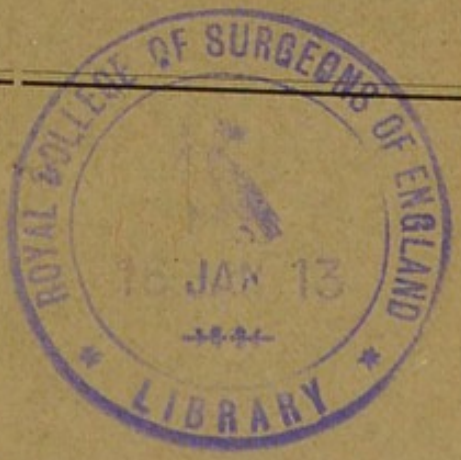
SURGICAL TREATMENT OF CERVICAL, THORACIC AND ABDOMINAL ANEURISM.

By CHARLES BEYLARD NANCREDE, M.D.

OF ANN ARBOR.

PROFESSOR OF SURGERY IN THE UNIVERSITY OF MICHIGAN.



ROYAL COLLEGE OF SURGEONS OF ENGLAND
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SURGICAL TREATMENT OF CERVICAL, THORACIC AND ABDOMINAL ANEURISM.[1]

By CHARLES BEYLARD NANCREDE, AM., M.D.,

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AS I have interpreted the request of your committee, the task assigned has appeared to me to be rather the discussion of the principles applicable in practice to the treatment of aneurismal disease in the regions concerned, and the presentation of a series of propositions for your discussion, rather than a mass of statistics. The statistical method is especially inapplicable when discussing the treatment of aneurism, because cases presenting the most diverse conditions have been treated by the same methods. Cases where cure could reasonably have been expected, and others where avowedly but temporary benefit, or "euthanasia," was aimed at, have been subjected to the same treatment and grouped together in the same tables, so that the percentages of recovery, failure or death are useless as guides to treatment. Just so soon as we have accurately determined what is the best method theoretically for each condition—mark, not variety of aneurism, as classed in the books—and in practice correctly appreciate the conditions present, will our treatment meet with its proper measure of success; but statistics, as at present constituted, cannot decide these questions. The primary object in all cases must be to prevent further enlargement of the sac, and by such means as shall not in themselves prevent nature's efforts to cure. Whatever will relieve intra-saccular tension or diminish the direct momentum of the blood, will be advantageous. The moment either of these are done, the normal

[1] Read before the American Surgical Association, May, 1893.

resiliency of the sac and the surrounding tissues tends to diminish the size of the aneurism. If, at the same time, an actual pad be supplied by the gradual deposition of a material which can actively contract, one which, if left under proper conditions for a sufficient length of time, will actually become organized into connective tissue obliterating the aneurism, we have achieved all that art theoretically need do. If permanent closure of the section of artery from which the aneurism springs can also be secured, the ideal has been attained, rendering relapse impossible.

A recapitulation of the chief points of importance concerning the formation of thrombi in the living vessels will render easier of comprehension the rationalé of certain surgical methods commonly employed in the treatment of aneurisms. Not only the physical conditions of the blood and sac, but also the vital conditions of the sac wall and of the sac contents, mutually contribute to, or interfere with, the deposition of white thrombi. Absolute or, preferably, partial physical rest of the blood favors thrombosis. These conditions can be secured by complete or partial arrest of the direct blood-current flowing through or by the sac: (1) By lessening the force and frequency of the heart's action; (2) by dilatation of the peripheral capillaries, *i.e.*, reducing blood pressure; (3) by physical interruption of direct access of blood to the sac by proximal pressure or ligation; (4) by preventing the blood passing freely out of the sac by distal mechanical obstruction of the artery. All that is effected by proximal arrest of circulation is a temporary absolute quietude of blood in the sac; with later—from establishment of the collateral circulation—only that marked diminution in velocity of the current which is most favorable, as will shortly be explained, to the formation of a thrombus. Experiments have proven that after damage of the vascular walls moderate slowing of the blood favors the accumulation of white blood cells in the plasmatic current, but that a more marked diminution in the velocity is required to allow the blood plaques to gather in numbers in this peripheral layer, while at the same time the leucocytes relatively diminish. The plaques promptly heap up at the site of injury, according to Eberth and Schim-

melbusch, owing to their rapidly undergoing "viscous metamorphosis." The primary cause of the formation of white thrombi is this accumulation of blood plaques, resulting from slowing or irregularities of current and "viscous change," induced by contact with the intima, damaged by disease or traumatism; or by the presence of foreign substances. The next step is a rapid accumulation of white cells, fibrin ferment is set free and fibrin is formed, so that the completed thrombus contains "plates, leucocytes, fibrin and included red corpuscles." It is then clear that permanent human white thrombi probably require the conjunction of marked diminution of velocity of the blood with an alteration of the vessel or sac walls, to determine the primary accumulation of blood plaques.

The structure of white thrombi, owing to their gradual formation, peculiarly fits them to undergo those subsequent changes which are necessary for their conversion into permanent tissue, and goes far toward justifying the name "active clot," given to them in the past. But is the deposition of laminated white thrombi, however disposed in an aneurism, always advantageous? do their presence by their roborant action delay the fatal issue in proportion to their amount, even if no cure can be effected? Decidedly not theoretically, and clinically numerous instances could be quoted in support of this statement. If all portions of the sac are *lined* with clot, which remains in close contact with the wall, unquestionably its resistance will be increased, but if any part of the wall is not coated with clot, the hydrostatic pressure will not be diminished upon that point, and even if not previously weaker than other portions of the sac, yet if the direct blood stream impinged originally upon it, or is now diverted to it, the deposition of clot in other portions of the aneurism will then be absolutely harmful, hastening the yielding at that point; again, if the clot does not remain in absolute contact with the wall of the aneurism, hydrostatic pressure must still continue to operate harmfully. If, on the contrary, the clot diverts the blood current from the weaker to the stronger portions of the sac, or splits up the direct momentum, as it were, by breaking the one stream into many, although no true widespread rein-

forcing of the sac by layers of fibrin can be demonstrated, the strain is actually so lessened that the same end is attained, *i.e.*, lessened distension occurs, and by the same means eddies and still bays are often produced, which favor the commencement of a white thrombosis, *if the wall be in a fit condition to induce the accumulation of blood plaques.* Thus, theoretically, any obstruction effected by a thrombus which favorably diverts the direct blood-momentum from the weaker to the stronger portions of the sac, and splits up the current, will tend to produce a cure, even if the fibrin does not line the interior of the sac, but at the outset is irregularly disposed. This fact is not merely of theoretical interest, but often explains the clinical fact that means utterly inadequate to cause a primary deposit of clot over the entire surface of the sac, and, indeed, of *themselves* to produce any bulk of thrombus, yet initiate conditions which promptly go on to complete consolidation of the aneurism. In other instances large amounts of clot are formed, yet rupture of the sac, instead of being retarded, is unquestionably hastened. The prime difficulty at present resides in the uncertainty experienced in applying these facts in practice.

Considering the question of treatment the first point to be decided is, "What is meant by the word cure?" Let us examine the conditions found in cases alleged to have been cured. In some, the sac contains much clot, varying in amount from partial or complete lining of its inner surface with a thin or thick layer up to entire obliteration of all cavity; the thrombus may or may not be in close contact with the wall of the aneurism, but whatever its relation may be, it is not structurally continuous with the wall, and is not itself converted into connective tissue. At any time blood may insinuate itself between the thrombus and the sac, terminating in a relapse. Can such a condition be considered a true cure, especially in view of the fact that relapses long after apparent cure do thus occur? While such a condition often does lead to genuine cure, it frequently does not so result, and except in one of the conditions mentioned, viz., entire filling of the sac with clot, can only act as a temporary barrier against further enlargement, unless the throm-

bus undergoes secondary changes presently to be described. It is vain to contend that filling the sac with firm coagula is potentially a cure, if relapses can and do occur, for we are not studying how to effect a temporary arrest in growth of aneurisms, but their *cure*. Examination of other specimens will show that the thrombi remain either partly, or throughout their whole extent, adherent to the lining membrane of the aneurism. At these points, or surfaces of contact, plasma cells penetrate the thrombus, probably passing along routes previously channeled out by invading white cells, if recent observations on experimental thrombi in bloodvessels are to be relied on. Still later, the thrombus will be found to have been replaced by a mass of cells —not leucocytes—which, becoming vascularized, and developing into connective tissue, shrinks, until a mere nodule of fibrous tissue remains, and a *real* cure is effected. This result is all the more prompt and certain if the sac-thrombus so extends as to occlude the mouth of the aneurism, and the artery above and below, because the blood *cannot* insinuate itself between the sac and the clot, which separation would at once arrest the process of organization just outlined. The failure of this obliteration of the artery in the immediate neighborhood of the mouth of the sac explains the temporary benefit which so often is all that results after apparent complete consolidation of an aneurism, because the blood separates the thrombus from the sac ; if the blood thus introduced coagulates, organization can now be effected, provided no further disturbances occur, and this secondary thrombosis can sometimes be recognized post-mortem.

The absolute certainty with which a clot which has caused the cessation of all the physical evidences of aneurism—except the tumor—will become disintegrated, with return of all the symptoms, unless it is allowed to remain in close contact with the sac, must be strongly insisted upon. The possibility of temporary thrombosis, separation of the clot from the sac, and secondary clotting with final organization must not be forgotten, as has just been pointed out. It is then manifest why soft, red thrombi are less desirable than white, laminated ones, not only because they are not physically so stable, but because they must

contract, thus either wholly or partially ceasing to remain in contact with the aneurismal wall. For this reason they are incapable of penetration by organizable cells. Such methods of cure are both theoretically and clinically the best which most fully combine the following requisites, viz., such a marked slowing of the circulation, or breaking up of its direct momentum, that first plaques can collect on some diseased or damaged portion of the lining of the sac, then white cells can accumulate—so that fibrin may form—and that such conditions shall exist as will allow of no mechanical disturbance of the absolute contact of thrombus and sac wall. The necessary change of the interior surface of the aneurism, which under favorable circumstances initiates white thrombosis is, probably, very often effected by the vascular disease productive of the aneurism; but may not the failures so frequently met with, of inducing the formation of white thrombi in aneurisms, be due to the insufficient extent and nature of this change, since "not every part of the internal wall of the vessel" was "covered with a thrombus" in Welch's experiments, "but such parts, even when severely lacerated," may be "entirely free from thrombi?"[1] Each successful method does not combine in the highest degree all the essentials described as requisite. Let us analyze the various methods of treatment in vogue, in order to ascertain upon what their success depends. Proximal arrest of the circulation promptly lessens the local blood pressure, and relieves all the direct momentum of the blood, and often allows, after the establishment of the collateral circulation, of just the proper rate of blood flow through the sac to favor accumulation of plaques and all the other processes requisite to the formation of a white thrombus. This method rarely permits of disturbance of the relations between the clot and sac, but when failure results it comes from mechanical disturbances of the thrombus, because of the establishment of an unusually free collateral circulation, and, probably, also from the coincident lack of such an alteration of the sac-lining that the initiatory steps toward the formation of white thrombi can occur. My colleague, Professor Lom-

[1] The Structure of White Thrombi, W. W. Welch, M.D., p. 18, Reprint from Transactions of the Pathological Society of Philadelphia, Vol. XIII, 1887.

bard, has suggested that after ligation interference with the vaso-motor and blood supply to the sac from constriction of the vaso-vatorum, may account for some of the failures, because of nutritive alterations in the sac-wall.

How does distal arrest of the circulation act? The effect of the direct momentum of the blood is diminished, but in a less efficient manner than by proximal arrest; distal arrest does not directly lead to the formation of white thrombi,[1] but may do so secondarily by creating eddies and still bays by the mechanical effects of soft coagula; moreover, the contact of thrombus and wall is more apt to be disturbed. Unless the sacculation is unusually favorably located, distal ligation for innominate and aortic aneurism will usually only temporarily arrest the disease, but cannot produce what I have laid down as a genuine cure. Are there no other means but proximal or distal arrest of circulation whereby the same mechanico-pathological conditions can be secured? Certainly; if in any way the direct momentum of the blood current can be mechanically broken up into minor currents, eddies and relatively quiet embayed collections of blood will result which, if the other chief requisite be present, *i. e.*, change of the sac wall, will give rise to white clot formation; these thrombi, if maintained in contact with the altered sac wall, can now go on to organization. This mechanical action on the blood current seems to me the far most reasonable explanation of the good effect produced by the introduction of catgut, coiled wire and electrolytic needles into the aneurism, which can only determine *at first* the formation of red thrombi. When no further changes occur after such therapeutic efforts, either no perceptible effect is produced, possibly rupture of the sac is hastened, or only temporary benefit results until the soft clot disappears. The absence of sufficiently extensive alteration of the lining of the sac explains why these methods, including acupuncture, so commonly fail, and why Macewen's "needling" has been followed by the surprising measure of success which has been reported. This is because the latter plan is a combination of interference with, and

[1] This is no doubt partly due to interference with the free access of blood plaques.

partial splitting up of the direct momentum of the blood, with such a preparation of the interior surface of the sac as certainly induces accumulation of blood plaques, followed by the formation of that relatively immovable white thrombus most capable of going on to organization. The necessity for extensive irritation and consequent plastic infiltration of the lining coat of the aneurism is proved by the observations of Macewen, that but little thrombus forms around the needle punctures, but does upon all those portions of the sac where the needle has continuously scratched for many hours, *i. e.*, has inflicted such a kind and extent of injury as will insure a plastic inflammation of the sac-wall. Again, it must be remembered that this process is repeated until it is believed that all accessible portions of the sac have undergone change; that by this means those weak spots of the wall which are about to yield can be reinforced first, something of which no other plan of treatment holds out any certainty. Still further, the residence of needles from ½ to 1½ mm. in circumference, for periods of even so long as forty-eight hours, must exert a very decided effect upon the blood momentum and current, an effect the extent of which I hardly think can be appreciated unless actually demonstrated by experiment.

To recapitulate somewhat, Macewen's "needling" should supersede the introduction of catgut, wire, etc., because this method does not give rise to the formation of soft, red thrombi; it does cause the deposition of white thrombi, which will remain in contact with a lining membrane prepared to induce organizations of the clot; the clot can be deposited upon the weakest portions of the sac-wall, and eventually upon all accessible portions, not in a haphazard manner, which as I have explained, often serves to precipitate the fatal issue; the cure, if accomplished at all, must fulfill the conditions laid down as requisite for a true cure; by careful exploration of the sac with the needles, information can be obtained permitting of the needles being so placed as to divert the direct momentum of the blood from the weakest portions of the sac, for from a few hours to even forty-eight hours, and that, too, while a thrombus is forming at these weakened spots.

In conclusion, permit me to submit for your discussion the following propositions, which the present state of our knowledge relating to aneurisms seems to warrant. Future clinical results can alone determine whether I have correctly indicated them.

May I ask the Fellows to first discuss each proposition separately, according to each only the weight its importance deserves, rather than to consider them together, which can only lead to confusion.

The Treatment of Cervical Aneurisms.

(1) All methods should be supplemented by recumbency and diet.

(2) Proximal compression, when feasible, should always be tried, and where the arterial coats are seriously diseased should supersede ligation.

(3) "Needling" should supplement pressure when the case is progressing rapidly; possibly it is advisable in all cases suitable for compression, and is certainly to be employed where this method fails in cases with highly atheromatous vessels.

(4) Proximal ligation, having been rendered much safer of late by the use of aseptic precautions, less absorbent ligatures and the avoidance of all injury to the arterial walls by employing the "stay-knot," is permissible when the arterial walls are relatively sound until experience decides whether or not "needling" is superior in its results.

(5) Since recurrence after proximal ligation almost certainly results from non-deposition of white thrombi, and their maintenance in contact with the aneurismal wall from lack of proper changes of its lining, "needling" is then clearly indicated.

(6) Where the location prevents proximal arrest of the blood current "needling" is the best operation; possibly distal compression—rarely feasible—might aid in the deposition of thrombi.

(7) For the reasons already given, although occasionally successful, the indications for the permanent introduction of such foreign bodies as wire, horse-hair, etc., into aneurismal sacs are so much better met by "needling" that such procedures had better not be adopted.

(8) The modern revival of the older method of extirpation of aneurisms should not be attempted for spontaneous cervical aneurisms.

The Treatment of Thoracic Aneurisms.

(1) All methods should be aided by the employment of rest in bed and proper diet.

(2) The permanent introduction of foreign substances should not be employed.

(3) "Needling" should be tried, aided by distal compression, when feasible, during the use of the needles; if this fails, distal ligation should be resorted to.

(4) Distal interruption of the blood current by simultaneous ligation of the carotid and subclavian arteries may be tried.

(5) "Needling" is indicated when complete or partial failure follows distal ligation.

The Treatment of Abdominal Aneurisms.

(1) All methods should include recumbency and diet.

(2) "Needling," when this can be done without injury to the hollow viscera, is the most promising plan.

(3) Proximal or distal compression may be tried, with or without "needling," but to be effectual must be done under anæsthesia.

(4) The permanent introduction of foreign bodies into the sac is unadvisable (see Proposition No. 7, Carotid Aneurisms).