DISSECTING ANEURYSM

GRAND ROUNDS,

JANUARY 28, 1960

CARLETON B. CHAPMAN, M.D.

I. PRELIMINARY

DISEASE PROCESSES WHICH ARE SEEN FREQUENTLY BUT WHICH ARE ETIOLOGICALLY AND PATHOGENETICALLY OBSCURE SEEM TO BE FAVORITE TOPICS FOR REVIEW ARTICLES. DISSECTING ANEURYSM IS A CASE IN POINT. IN THE LAST CENTURY THERE HAVE BEEN NUMEROUS REVIEWS OF THE SUBJECT, BUT SIGNIFICANT ADVANCE IN BASIC KNOWLEDGE CONCERNING ITS ETIOLOGY IS CONSPICUOUSLY LACKING. MACALLUM'S COMPLAINT (WITH REGARD TO ARTERIOSCLEROSIS) THAT CURRENT LITERATURE (AS OF 20 YEARS AGO) CONSISTS LARGELY OF REVIEWS OF REVIEWS SEEMS APT. THE REVIEW TO END ALL REVIEWS ON DISSECTING ANEURYSM IS THAT BY HIRST, JOHNS AND KIME, WHICH RUNS TO 62 PAGES AND PROVIDES 346 REFERENCES BEGINNING WITH GALEN AND ENDING WITH PAPERS PUBLISHED IN 1956. IN THE LAST FEW YEARS, THE CONTENT OF ARTICLES ON THE SUBJECT HAS BEGUN TO REFLECT RENEWED INTEREST, NOT BECAUSE OF BETTER BASIC UNDERSTANDING, BUT BECAUSE SUCCESSFUL SURGICAL TREATMENT HAS BEEN REPORTED. THE QUESTION AS IT NOW STANDS DOES NOT INVOLVE A COMPARISON OF SURGICAL TREATMENT WITH MEDICAL MANAGEMENT. APART FROM REST IN BED, RELIEF OF PAIN, AND OXYGEN THERAPY THERE IS NO SPECIFIC MEDICAL MANAGEMENT. THE REAL QUESTION IS: CAN THE SURGEON, WITH DARING BUT IMPERFECT TECHNIQUES, DECREASE MORTALITY IN A DISEASE WHICH IS USUALLY, BUT NOT ALWAYS, RAPIDLY FATAL? THE QUESTION PROBABLY IS ANSWERABLE IN THE AFFIRMATIVE.

HISTORICAL ASPECTS (WHICH, IN THIS INSTANCE AS IN MOST OTHERS, MEANS EVERYTHING COMMUNICATED BEFORE OSLER'S TIME) ARE WELL COVERED BY HIRST, ET AL. WE KNOW THAT VESALIUS OBSERVED A CASE, THAT GEORGE II DIED IN 1760 (WHILE STRAINING AT STOOL) PROBABLY OF A DISSECTING ANEURYSM FOUND AT AUTOPSY, THAT MORGAGNI (1761) PROVIDED A GOOD GROSS DESCRIPTION OF THE PATHOLOGY OF THE DISEASE, AND THAT THE TERM WE NOW APPLY TO IT CAME FROM THE CELEBRATED LAËNNEC. IN OUR OWN TIME, THE LATE DR. JAMES PAULLIN OF ATLANTA ACHIEVED THE IMPRESSIVE BUT UNENVIABLE DISTINCTION OF BEING THE FIRST TO DIAGNOSE THE DISEASE IN HIMSELF AND TO PREDICT ITS SITE OF INTRATHORACIC RUPTURE.

THE NORMAL AORTIC WALL IS AN INTENSELY TOUGH AND DYNAMIC STRUCTURE, THE TENSILE STRENGTH OF WHICH IS ENORMOUS. THE INTIMA, IN THIS SENSE, IS WEAK; ITS PURPOSE IS TO PROVIDE A SMOOTH SURFACE, HIGHLY RESISTANT TO THROMBOTIC PROCESSES, FOR THE OUTWARD TRANSMISSION OF BLOOD. THE STRENGTH AND MARVELOUS ELASTIC PROPERTIES OF THE STRUCTURE ARE SUPPLIED MOSTLY BY THE MEDIA COMPOSED MOSTLY OF ELASTIC TISSUE AND SMOOTH MUSCLE. THE STRENGTH OF THE NORMAL WALL IS SUCH THAT RUPTURE OF FRESH POST-MORTEM SPECIMENS REQUIRES PRESSURES OF 1000 MM. HG. OR MORE. IT HAS ALSO BEEN SHOWN THAT NORMALLY, THE INTIMA IS NOT EASILY SEPARABLE FROM THE MEDIA. IN ANY EVENT, NATURALLY OCCURRING AORTIC PRESSURES NEVER REACH LEVELS SUFFICIENT TO ENDANGER THE INTEGRITY OF THE NORMAL AORTIC WALL. IT FOLLOWS THAT DISSECTION OF THE AORTIC WALL DOES NOT OCCUR IN FULLY NORMAL SUBJECTS EXCEPT, PERHAPS, IN ASSOCIATION WITH TRAUMA AND EVEN THIS IS DUBIOUS.

THE CLINICAL IMPORTANCE OF CORRECT DIAGNOSIS HAD, UNTIL RECENTLY, TO DO SOLELY WITH DIFFERENTIAL DIAGNOSIS. DISSECTING ANEURYSM MAY MIMIC MANY OTHER ACUTE, PAINFUL DISEASES OF THE THORAX AND ABDOMEN. IT ALSO MAY MASQUERADE AS ARTERIAL EMBOLISM, NEUROLOGICAL DISEASE, OR DISEASE OF THE GU TRACT. MOST

FREQUENTLY, IT IS CONFUSED WITH ACUTE MYOCARDIAL INFARCTION. A CORRECT DIAGNOSIS IS IMPERATIVE SINCE TREATMENT OF THE TWO DISEASES IS VERY DIFFERENT. IT MAY ALSO COMPLICATE PREGNANCY. CORRECT ANTE-MORTEM DIAGNOSIS, ALTHOUGH INCREASINGLY IMPORTANT, IS OFTEN DIFFICULT OR IMPOSSIBLE. IN MOST PUBLISHED SERIES, CORRECT DIAGNOSIS WAS MADE IN LESS THAN 40 PER CENT OF CASES (AS DIAGNOSED AT AUTOPSY) ALTHOUGH ONE AUTHOR MADE IT CORRECTLY IN 10 OF 12 CASES.

BACKGROUNDS PREDISPOSING TO AORTIC DISSECTION ARE NOT ENTIRELY CLEAR. CERTAINLY, HYPERTENSION EITHER PREDISPOSES TO THE PROCESS OR TENDS TO CONVERT MINOR INTIMAL OR MEDIAL DEFECTS TO FATAL DISEASE. EQUALLY CERTAIN IS THE FACT THAT DISSECTION IS 2 - 3 TIMES MORE FREQUENT IN MEN THAN IN WOMEN. RACE AND OCCUPATION CANNOT BE SHOWN TO PREDISPOSE TO THE DISORDER. CERTAIN TYPES OF CONGENITAL CARDIOVASCULAR DISEASE CLEARLY PREDISPOSE TO AORTIC DISSECTION. ARACHNODACTYLY, OFTEN IN ASSOCIATION WITH AORTIC VALVULAR DISEASE (MARFAN'S SYNDROME) IS NOW FIRMLY LINKED WITH AORTIC DISSECTION. WHEN THE DISORDER IS SEEN IN YOUNG PEOPLE, SOME ASPECT OF MARFAN'S SYNDROME IS OFTEN PRESENT (PYGOTT, GOYETTE ET AL). OTHER CONGENITAL ANOMALIES HAVE BEEN ASSOCIATED WITH AORTIC DISSECTION, NOTABLY COARCTATION OF THE AORTA, AORTIC HYPOPLASIA, AND BICUSPID AORTIC VALVES (GORE AND SEIWERT).

II. PATHOGENESIS

THE REIGNING ARTICLE IN THIS AREA IS CURRENTLY THAT BY GORE AND SEIWERT. THE BASIC LESION AND PRIMARY MORPHOLOGIC CHANGE IN AORTIC DISSECTION OF THE USUAL TYPE IS SOME FORM OF MEDICAL DISEASE, A POINT ON WHICH THERE SEEMS TO BE NO DISAGREEMENT. THE POSSIBLE EXCEPTION IS THE LIMITED DISSECTION, USUALLY IN THE ABDOMINAL AORTA, THAT SOMETIMES ACCOMPANIES SEVERE AORTIC ATHEROSCLEROSIS. BREAKS IN THE INTIMA ARE OFTEN FOUND AROUND CALCIFIED PLAQUES AND SOME DISSECTION MAY OCCUR. IT MAY STILL BE ARGUED, HOWEVER, THAT MEDICAL DISEASE MUST OCCUR SINCE DISSECTION DOES NOT ALWAYS FOLLOW INTERRUPTION OF THE INTIMAL SURFACE.

AGREEMENT ON THE NATURE OF THE MEDIAL DISEASE IS NOT UNIFORM. THAT ASSOCIATED WITH ERDHEIM'S NAME (ORIGINALLY DESCRIBED, LESS FULLY, BY GSELL) WAS FORMERLY ACKNOWLEDGED AS "THE CAUSE" OF AORTIC DISSECTION. GSELL DESCRIBED FOCAL NECROSIS OF MEDIAL MUSCLE, FOLLOWED BY ELASTIC TISSUE AND COLLAGEN DEGENERATION SO THAT CLEFTS, OFTEN CONTAINING MUCOID MATERIAL, RESULT. SCARRING, SOMETIMES WITH INGROWTH OF THIN-WALLED ARTERIAL BRANCHES, THEN TAKES PLACE. ERDHEIM ELABORATED ON THE THEME AND NOTED THAT THE LESIONS ARE USUALLY FOUND IN THE MIDDLE AND OUTER THIRDS OF THE MEDIA AND ARE LIMITED TO THE ASCENDING PORTION AND TOP OF THE AORTIC ARCH. GORE'S CONTRIBUTION IS THAT MEDIONECROSIS MAY BE OF TWO TYPES:

(1) DEFECT MAINLY IN ELASTIC TISSUE AND OCCURRING IN YOUNGER PATIENTS

(2) DEFECT MAINLY IN SMOOTH MUSCLE AND OCCURRING IN OLDER AGE GROUPS

THE PATHOGENESIS OF THE DISORDER IS ALSO DEBATED. GRANTING THAT DISEASE OF THE MEDIA MUST BE PRESENT, TWO PATHOGENETIC POSSIBILITIES ARE TO BE CONSIDERED:

(1) RUPTURE OF VASA VASORUM INTO AN AREA OF MEDIONECROSIS, FOLLOWED BY RUPTURE OF THE INTIMA INTO THE AORTIC LUMEN.

(2) COLLAPSE OF MEDIA IN A NECROTIC AREA WITH BULGING AND RUPTURE OF OVERLYING INTIMA.

IN EITHER EVENT, AN INTIMAL TEAR RESULTS AND IS USUALLY TRANSVERSE. IT MAY OCCUR JUST ABOVE THE AORTIC VALVE, NEAR THE ORIGINS OF THE GREAT VESSELS, OR (MORE RARELY) IN THE DESCENDING ARCH OR THORACIC AORTA. DISSECTION USUALLY PROCEEDS PROXIMALLY AND DISTALLY BUT MORE THE LATTER THAN THE FORMER. <u>REENTRY</u> INTO THE AORTIC LUMEN AT A POINT DISTAL TO THE INITIAL INTIMAL RUPTURE OFTEN OCCURS AND IS ASSOCIATED WITH IMPROVED PROGNOSIS. MOST OF THE PATIENTS WHO SURVIVE MONTHS OR YEARS, HAVE DOUBLE-BARRELLED LESIONS CREATED BY REENTRY. THE OUTER CHANNELS COME TO BE LINED WITH ENDOTHELIUM IF THE PATIENT LIVES LONG ENOUGH. IN ANY CASE, REENTRY SEEMS TO RETARD FURTHER DISSECTION.

EXPERIMENTAL PRODUCTION OF TRUE DISSECTING ANEURYSM IS DIFFICULT. BLANTON AND CO-WORKERS PRODUCED IT IN DOGS BY INCISING THE INTIMA OF THE AORTIC ARCH AND CREATING MEDIAL LESIONS. DISSECTION OCCURRED IN 13 OF 26 DOGS WHEN BLOOD AT NORMAL AORTIC PRESSURE WAS ADMITTED INTO THE PREPARED SEGMENT OF AORTA. DISSECTION WAS NEVER PROXIMAL (UNLIKE THE DISEASE AS SEEN IN HUMAN BEINGS) AND USUALLY EXTENDED AT LEAST TO THE DIAPHRAGM. THE DISSECTION PROCESS WAS MORE VIOLENT AND EXTENSIVE IF THE ANIMAL WAS GIVEN VASOPRESSOR DRUGS. ANEURYSMAL SACS (DISSECTING HEMATOMAS) WERE LARGE ENOUGH AT TIMES TO BLOCK FLOW THROUGH THE AORTIC LUMEN; WHEN REENTRY OCCURRED THE SACS TENDED TO COLLAPSE, REOPENING THE ORIGINAL LUMEN. THE DYNAMICS INVOLVED SEEM CLEAR: AORTIC PRESSURE WAVES, ENTERING THE MEDIA THROUGH AN INTIMAL TEAR, ENCOUNTER A RESISTANCE AND CAN TRAVEL NO FURTHER. THE BATTERING OF THE PRESSURE WAVES, AND THEIR INITIAL REFLECTION BY THE MEDIA, GENERATES ENOUGH FORCE TO SPLIT THE LAYER. SPLITTING MAY CONTINUE INDEFINITELY UNTIL AND UNLESS REENTRY OCCURS. IF THIS HAPPENS, PRESSURE WAVES TRAVEL OUT OF THE AORTIC LUMEN AND RETURN TO IT WITHOUT BEING SIGNIFICANTLY OPPOSED; PRESSURE ON BOTH SIDES OF THE ISOLATED INTIMAL TUBE BECOMES EQUAL AND THE TENDENCY FOR SPLITTING TO CONTINUE BEYOND THE REENTRY SITE IS LESSENED.

IT HAS ALTERNATELY BEEN PROPOSED THAT RUPTURE OF VASA VASORUM INTO THE MEDIA NOT ONLY INITIATES THE PROCESS BUT PROPAGATES IT, INTIMAL TEARS BEING LARGELY INCIDENTAL. THIS SEEMS ILLOGICAL, ESPECIALLY WHEN IT IS REALIZED THAT PRESSURE IN VESSELS AS SMALL AS THE VASA VASORUM IS PROBABLY TOO SMALL TO DO ANYTHING MORE THAN TO RUPTURE A VERY THIN, AND POSSIBLY DISEASED, VASCULAR WALL. IT IS VERY DOUBTFUL THAT SUCH A PROCESS COULD SPLIT THE MEDIA ITSELF.

III. MORTALITY

SINCE SURGICAL INTERVENTION IN DISSECTING ANEURYSM IS RELATIVELY NEW, IT HAS NOT YET HAD TIME TO INFLUENCE MORTALITY. THE FOLLOWING DATA (FROM HIRST, ET AL) ARE BASED ON UNTREATED CASES OR ON THOSE TREATED WITH MEDICAL MEASURES (OPIATES AND REST).

MORTALITY IN 425 CASES

TIME	NUMBER DEAD	PER CENT DEAD
SUDDEN DEATH	13	3
6 HOURS	35	8
12 "	57	13
18 "	73	17
24 "	91	21
2 DAYS	157	37
3 "	189	44
4 "	208	49
5 "	228	53
6 "	252	59
7 "	266	62
2 WEEKS	318	74
3 "	335	78
4 "	343	80
2 MONTHS	370	87
3 "	380	89
6 "	390	91
1 YEAR	399	93
2 YEARS	407	95
5 YEARS	422	99
9 YEARS	425	100

MANY OTHER ESTIMATES ARE AVAILABLE BUT NONE ARE BASED ON SUCH A LARGE GROUP. WHILE IT IS POSSIBLE THAT THE MATERIAL IS WEIGHTED IN SOME WAY, ITS AGE AND SEX DISTRIBUTION SEEM REASONABLE. FOUR HUNDRED AND ELEVEN (85 PER CENT) WERE AGED 40 OR MORE AND ABOUT 75 PER CENT WERE MALE. IT IS PERHAPS HIGHLY SIGNIFICANT TO NOTE THAT, AT THE END OF 24 HOURS, 79 PER CENT OF THE TOTAL MATERIAL WERE STILL ALIVE AND AFTER 2 DAYS, 63 PER CENT STILL SURVIVED. OVER ONE-HALF THE GROUP DIED IN THE FIRST 5 DAYS AND AFTER ONE MONTH ONLY 20 PER CENT WERE STILL ALIVE. THE INDICATION IS THAT THE DISEASE MAY NOT BE AS IMMEDIATELY FATAL AS WAS ONCE SUPPOSED.

CAUSES OF EXITUS VARY, DEPENDING ON THE ACUTENESS OF THE LESION. IN ACUTE CASES (SURVIVING LESS THAN 2 WEEKS), HEMORRHAGE EXTERNAL TO THE AORTA CAUSED 89 PER CENT OF THE DEATHS. MOST OF THE CASES SHOWED INTRAPERICARDIAL HEMORRHAGE (166 OF 236 ACUTE CASES); 74 WERE INTO PLEURAL SPACES, 31 INTO THE

MEDIASTINUM AND THE REST BELOW THE DIAPHRAGM. IN CHRONIC CASES (SURVIVING MORE THAN 6 WEEKS), EXTERNAL HEMORRHAGE OCCURRED IN 37 OF 66 CASES AND THE NEXT MOST COMMON CAUSE WAS CONGESTIVE HEART FAILURE (22 OF 66 CASES).

IV. CLINICAL MANIFESTATIONS

(HIRST, ET AL., CASE REP. MGH, COPPING, ET AL).

V. ROENTGENOLOGIC DIAGNOSIS

(EASTCOTT AND SUTTON; GOLDEN AND WEANS; KNUTSSON; SAVAGE).

VI. TREATMENT

MEDICAL MEASURES, APART FROM RELIEF OF PAIN, ABSOLUTE REST, AND OXYGEN (IF INDICATED) ARE CONTROVERSIAL AND PROBABLY INEFFECTIVE. IT SEEMS LOGICAL TO USE HYPOTENSIVE MEASURES IF HYPERTENSION IS PRESENT AFTER THE DISSECTION TAKES PLACE. THE MEASURE, HOWEVER, CANNOT BE SAID TO HAVE LOWERED MORTALITY VERY MUCH. ANTICOAGULANTS ARE CONTRAINDICATED.

THE RATIONALE FOR SURGICAL THERAPY RESTS SOLIDLY ON THE IMPROVEMENT IN MORTALITY ASSOCIATED WITH THE REENTRY PHENOMENON. THE METHODS PROPOSED BY DE BAKEY AND COLLEAGUES INVOLVE CREATION OF A SITE OF REENTRY AT SOME DISTANCE FROM THE INTIMAL TEAR ALONG WITH OBLITERATION OF THE DISTAL DISSECTION AND, DEPENDING ON CIRCUMSTANCES, INSERTION OF A HOMOGRAFT. THE TECHNIQUES HAVE BEEN USED ON THORACIC AND ABDOMINAL LESIONS WITH SOME SUCCESS BY DE BAKEY AND BY OTHER GROUPS (WARREN, ET AL).

THE MOST TRYING QUESTION WITH REGARD TO SURGERY IS SELECTION OF PATIENTS. THE SIMPLEST STAND FOR THE INTERNIST, AND POSSIBLY THE BEST, IS TO HOLD THAT ONCE DIAGNOSIS IS ACCOMPLISHED THE PROBLEM IS A SURGICAL ONE. THE PROBLEM, HOWEVER, IS COMPLICATED BY THE SHAPE OF THE MORTALITY CURVE; IT RISES VERY STEEPLY DURING THE FIRST 48 HOURS, SLIGHTLY LESS STEEPLY FOR THE NEXT 5 OR 6 DAYS, THEN MORE SLOWLY BUT STEADILY FOR MONTHS AND YEARS. SOME DECISION OUGHT, THEREFORE, TO BE REACHED IN THE FIRST 48 HOURS AND PREFERABLY WITHIN AN HOUR OR TWO OF ONSET. AND THERE IS NO RELIABLE WAY, IN A SEVERELY STRICKEN PATIENT, TO IDENTIFY HIM AS ONE WHO WILL CREATE HIS OWN REENTRY AND SURVIVE MONTHS OR YEARS.

IN PATIENTS WHOSE SYMPTOMS SUBSIDE QUICKLY, WHO DO NOT DEVELOP SIGNIFICANT ARTERIAL OCCLUSION (CAROTID, SUBCLAVIAN, CORONARY, OR RENAL) AND IN WHOM HYPERTENSION IS NOT MARKED OR CAN BE BROUGHT UNDER CONTROL, CONSERVATIVE MEASURES ARE PROBABLY INDICATED. IN PATIENTS WHO ARE SEVERELY ILL, IN WHOM PAIN IS DIFFICULT TO CONTROL, SIGNS OF ADVANCING DISSECTION ARE PRESENT, AND SHOCK DEVELOPS, SURGERY OFFERS A SMALL CHANCE OF SURVIVAL. THIS, HOWEVER, IS PROBABLY MORE THAN CONSERVATIVE MEASURES CAN OFFER EVEN THOUGH A FEW SUCH PATIENTS DO SURVIVE FOR A TIME WITHOUT SURGERY. SINCE TIME IS OF THE ESSENCE, THIS TYPE OF PATIENT SHOULD BE REGARDED AS A MAJOR SURGICAL EMERGENCY. IT MIGHT BE SENSIBLE TO CONSIDER THE USE OF SOME SORT OF MODIFIED HYPOTHERMIA IN THIS GROUP, WITH SURGERY ADDED IF THE DISEASE CONTINUES TO PROGRESS. THIS FORM

OF THE DISEASE IS SOMEWHAT ANALOGOUS TO OTHER MAJOR VASCULAR CATASTROPHES SUCH AS FEMORAL OR POPLITEAL EMBOLISM, IN WHICH IT IS SOMETIMES JUSTIFIABLE TO OBSERVE THE EFFECTS OF SYMPATHETIC BLOCK PROVIDED SURGERY IS IMMEDIATELY AVAILABLE IF THE MORE CONSERVATIVE MEASURE FAILS.

IF PROGRESS IN THE THERAPY OF DISSECTING ANEURYSM IS TO BE MADE, SUCH THERAPY WILL HAVE TO BE INTRODUCED EARLY AND THIS, IN TURN, WILL REQUIRE A MEDICAL-SURGICAL ORGANIZATIONAL ARRANGEMENT THAT CAN BE BROUGHT INTO ACTION WITH MINIMAL DELAY.

REFERENCES

1. Blanton, F.S., Muller, W. H., Jr., and Warren, W.D.: Experimental production of dissecting aneurysms of the aorta. Surgery 45: 81-90 (Jan.), 1959.

2. Case Records of the Massachusetts General Hospital. New England J.Med. 260: 335-339 (Feb. 12), 1959.

3. Copping, G. A., and Szenas, P.: Dissecting aortic aneurysm. Canad. M.A.J. 80: 336-339 (Mar. 1), 1959.

4. DeBakey, M.E.: Dissecting aneurysm of the aorta. Surg., Gyn., Obs. 102: 372-373 (March), 1956.

5. DeBakey, M.E., Creech, O., Jr., and Morris, G.C., Jr.: Aneurysm of thoracoabdominal aorta involving the celiac, superior mesenteric, and renal arteries. Report of four cases treated by resection and homograft replacement. Ann. Surg. 144: 549-573 (Oct.), 1956.

6. Eastcott, H.H.G., and Sutton, D.: Chronic dissecting aneurysm of the aorta diagnosed by aortography. Lancet 2: 73-75 (July 12), 1958.

7. Erdheim, J.: Medionecrosis aortae idiopathica cystica. Virchows Arch. F. Path. Anat. 276: 187-229 (Feb.17), 1930.

8. Golden, A., and Weens, H.S.: The diagnosis of dissecting aneurysm of the aorta by angiocardiography. Am. Heart J. 37: 114-122 (Jan.), 1949.

9. Gore, I.: Pathogenesis of dissecting aneurysm of the aorta. A.M.A. Arch. Pathol. 53: 142-153 (Feb.), 1952.

10. Gore, I., and Seiwert, V.J.: Dissecting aneurysm of the aorta. Pathologic aspects. An analysis of eighty-five fatal cases. A.M.A. Arch. Pathol. 53: 121-141 (Feb.), 1952.

11. Goyette, E.M., and Palmer, P.W.: Cardiovascular lesions in arachnodactyly. Circulation 7: 373-379 (March), 1953.

12. Gsell, O.: Wandnekrosen der Aorta als selbständige Erkrankung und ihre Beziehung zur Spontänruptur. Virchows Arch. F.Path. Anat. 270: 1-36 (Nov. 20), 1928.

13. Hirst, A.E., Jr., Johns, V.J., Jr., and Kime, S.W., Jr.: Dissecting aneurysm of the aorta: A review of 505 cases. Medicine 37: 217-279 (Sept.), 1958.

14. Knutsson, F.: Dissecting aneurysm of the thoracic aorta. Acta Radiol. 51: 273-277, (April), 1959.

15. PYGOTT, F.: ARACHNODACTYLY (MARFAN'S SYNDROME) WITH A REPORT OF TWO CASES. BRIT. J. RADIOL. 28: 26-29 (JAN.), 1955.

16. SAVAGE, C.R.: RETROGRADE AORTOGRAPHY IN ACUTE DISSECTING ANEURYSM OF THE AORTA. LANCET 1: 281-282 (FEB. 7), 1959.

17. WARREN, W.D., BECKWITH, J., AND MULLER, W.H.: PROBLEMS IN THE SURGICAL MANAGEMENT OF ACUTE DISSECTING ANEURYSM OF THE AORTA. ANN. SURG. 144: 530-548 (OCT.), 1956.