AORTIC INSUFFICIENCY DUE TO RUPTURE BY STRAIN OF A NORMAL AORTIC VALVE*

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IN a hospital experience of twenty-five years I had never seen a case of aortic insufficiency due to rupture of an aortic valve and, therefore, considered the following case report worth recording.

After a brief outline of the case, I shall discuss the incidence of this comparatively rare accident, classify the cases, and then discuss at some length the pathogenesis, pathology and symptomatology of the group.

CASE REPORT

H. B., aet. 33: chauffeur: admitted to the Montreal General Hospital on November 6, 1924, complaining of pain over the heart, palpitation, shortness of breath, cough and swelling of the feet.

Family History.—Negative, except that the father died at 53 from "heart disease."

Personal History.—The patient denied rheumatic fever, chorea and syphilis. He had been married one year; his wife had had one full term child and no miscarriages. He smoked to excess and used alcohol moderately. He had enjoyed perfect health previously.

Present Illness began on October 6, 1924, when one cold morning, after cranking his car for two minutes, he was suddenly seized with a sharp pain in the left upper chest and especially in the epigastrium, but with no radiation to the arm; he immediately became short of breath and began to cough. He remained at work for some hours, when he stopped on account of the pain in the epigastrium and dyspnœa. Subsequently he noted a "thrill" in the left upper chest. He remained in bed two weeks and then returned to work, but was forced to give up again on November 1, 1924, as his symptoms had become very acute. The pain then radiated down the left arm and even to the left leg. He became very orthopnœic and vomited constantly. Œdema of the feet also appeared.

Physical examination revealed a large, stout, French-Canadian male, in great respiratory distress. The colour of the face was sallow, but cyanosis of the lips, ears and finger tips was present. The thorax was very large. There was impairment of the note at both bases with suppression of the breath sounds and the presence of many moist râles. Heart: There was slight bulging of the præcordium and a widespread heaving impulse visible over the entire left chest, the point of maximum intensity of which was in the fifth interspace, 12 cm. from the midsternum. On palpation over the left base, a very intense vibratory thrill could be felt, perceptible throughout the entire cardiac cycle, but with definite systolic and diastolic intensification. The relative cardiac dullness began in the second interspace and was much increased to the right (5 cm.), but more particularly to the left (15 cm.) of the midsternal line. On auscultation, the heart sounds were replaced by murmurs, whose maximum intensity was at the third and fourth left interspaces close to the sternum. The systolic murmur was rough and vibratory and filled the entire systolic period, completely obliterating the first sound; though very widely distributed it was of maximum intensity in the second right interspace. The diastolic murmur was of higher pitch, shorter and more musical, and, though propagated to the apex and into the vessels of the neck, was of maximum intensity over the third left interspace close to the sternum. No second sound could be heard. The pulse was of the typical water-hammer character, and was rapid, with a palpable vibratory thrill. The sphygmomanometer revealed a systolic blood pressure of 130 mm., and a diastolic that varied from 40 to zero. There was a marked pulsation of the carotid, brachial and femoral arteries, as well as a definite capillary pulse. The electrocardiogram showed an inversion of the "T-wave" in all leads. Fluoroscopic examination revealed an enormous heart with no aneurysm. The liver was enlarged and tender. While there was no ascites there was œdema of the legs. In the urine there was a constant albuminuria, but no cylindruria. The blood count was normal; (75 per cent hæmoglobin; red blood cells 5,080,000), except for an occasional leucocytosis, (7,500 to 11,000). Two blood Wassermann tests were negative.

The diagnosis favoured was, either a congenital heart lesion with cardiac decompensation, or an acquired aortic insufficiency with the sudden onset of cardiac decompensation due to strain, though the possibility of a rupture of an aortic cusp was suggested by my resident physician, Dr. E. S. Mills, and was seriously considered.

The course was practically a downhill one, in spite of short periods of apparent improvement from the bed rest and digitalis. However, the cardiac distress, dyspnœa and œdema increased, and there developed, first, signs of infarction of both lungs, and finally a thrombosis of the posterior tibial vein. Death occurred on January 1st, from progressive cardiac decompensation, practically twelve weeks after the severe muscular effort.

Post-mortem.—There were, in addition to marked œdema of the lower extremities, dense adhesions about the right lung, with an old tuberculous cavity in the right apex and a scar of the left apex; recent infarcts in the right lower as well as in the left upper and left lower lobes; acute fibrinous pleurisy over the infarcted areas. The common attachment of the anterior and medial (left posterior) cusps of the aortic valve was torn away from the aortic wall, due to a transverse tear in the intima, five-eighths of an inch in length, allowing the cusps to become very lax, (see photographs). The mitral valve showed slight thickening. There was a fairly extensive sclerosis in the sinus of Valsalva, and a still earlier process in the root and arch of the aorta as well as the descending aorta. The heart weighed 640 grm., and showed marked hypertrophy with dilatation of the left ventricle and great dilatation of the right. The liver, spleen and kidneys revealed marked passive congestion. Microscopically, the aortic cusp was normal. The vasa vasorum of the aorta showed no evidence of syphilis.

* Read before the Association of American Physicians, May 6, 1925.

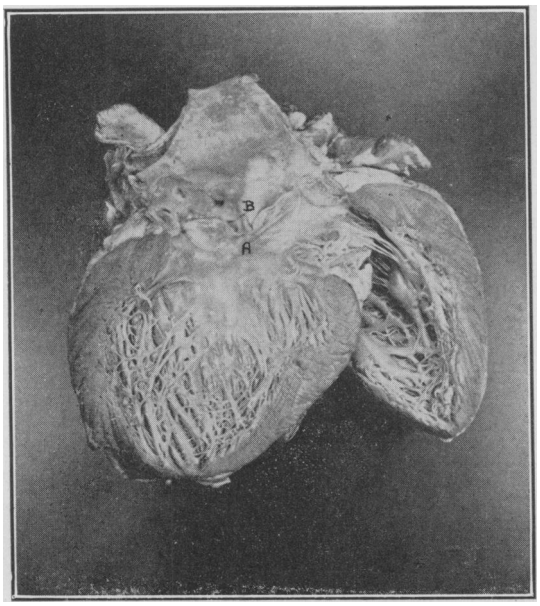

FIG. 1.—Rupture of the Aortic Valve. A—rupture of valve. B—tear in aorta.
(Montreal General Hospital A-25-1.)

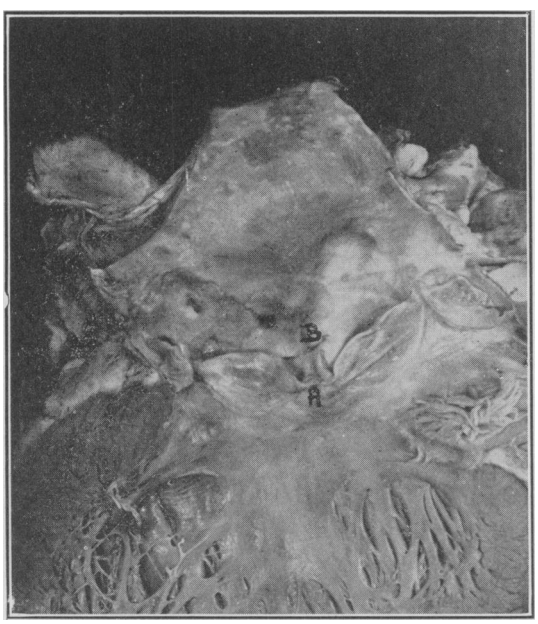

FIG. 2.—Rupture of the Aortic Valve. A—rupture of valve. B—tear in aorta.
(Montreal General Hospital A-25-1.)

INCIDENCE

Rupture of the healthy mitral and tricuspid valves due to injury from external causes has been known to pathologists since the time of Marat and Corvisart,[1] but the earliest recorded case of rupture of the aortic valve is one by Plenderleath[25] in the *London Medical Gazette* of 1820. Since then some hundred odd cases have appeared in the world's medical literature.

A prolonged search through various *referate,* as well as the *Index Medicus,* the *Surgeon-General's Catalogue,* and the *Quarterly Cumulative Index,* reveals a surprising silence on the subject in many of the text-books of medicine and even in the monographs on heart disease. However, as the writer has learnt to expect, Osler[2] in his *Principles and Practice of Medicine* mentions rupture as a very rare cause for aortic insufficiency, and some of the older writers, such as C. J. B. Williams[3] (1839), Thomas Peacock[4] (1873), Dacosta[5] (1874), Hilton Fagge[6] (1877), G. W. Balfour[7] (1898), Stern[8] (1900) and Kuelbs[9] (1909), also describe it in more or less detail. In the medical journals and theses, Quain,[33] Peacock,[44] Duroziez,[68] Barié,[70] Dreyfus[86] and Dufour,[87] have published cases of their own and collected all available reports from the literature. Dreyfus was able to find only forty-six cases up to the year 1896. The writer set himself the thankless task of combing the literature from 1830 to 1925 inclusive and succeeded in finding but 112 cases, all of which were not proven, however. The writer's case makes 113.

Cases of so-called "traumatic endocarditis" without valve rupture (Alvarenga,[10] Gerhardt,[11] Mayr,[12] Riedinger,[13] Leyden,[14] Duems,[15] Rumpf,[19] Dieckman[17]) have been excluded and, of course, cases of proven ulcerative endocarditis (Williams,[18] Packard,[19] Peacock,[20] Salter,[21] Orlebar,[22] Rosenberg,[23] Byrom Bramwell[24]), as this paper is concerned only with rupture of a healthy or diseased aortic valve from muscular effort or trauma.

CLASSIFICATION

There are two main groups to be considered: first, those due to muscular effort or *strain*; and, second, those due to *traumatism.* A third, small, group consists of cases in which the exciting factor is not stated.

The first group comprises sixty cases, of which only thirty were proved by autopsy (Plenderleath,[25] Henderson,[27] Corrigan[28] (case 2), Latham-Bence-Jones,[32] Quain[33] (case 1), Quain-Jones,[34] Peacock[37] (case 1), Peacock[41] (case 2), Meschede,[47] Foster[48] (case 1), Simpson,[52] Williams,[53] Foster[57] (case 4), Pepper,[58] Burney-Yeo[59] (case 1), Frew-Finlayson,[65] Greenhow,[66] Lindman,[67] Lewis,[77] Leyden[78] (case 1), Fraentzel,[79] Tretzel,[81] Hektoen,[82] Leyden[83] (case 2),

Jamieson,[89] Broadbent,[100] Heller,[114] Anderson[119] (case 2), Hoffmann[129] (case 4), Howard[137]). The other thirty cases, though clinically probable cannot be considered as proven in the absence of a post-mortem record in twenty-nine. (Aran,[31] Quain,[35] (case 3), Rawson,[36] Markham[39] (endocarditis?), O'Neil,[40] Peacock[44] (case 3), Peacock[45] (case 4), Foster[49] (case 2), Allbutt[54] (case 1), Burney-Yeo[60] (case 2), Peter[61] (case 1), Peter[62] (case 2), Orton-M'Aldowie,[63] Zohrab,[75] Cantley,[84] Launois,[88] Debove,[98] Ostwalt,[99] Horton-Smith,[102] Dupuis[103] (case 1), Taylor,[104] Shaw,[105] Jorns,[108] Ercklentz,[111] Oliver,[112] Anderson[118] (case 1), Allbutt[124] (case 3), Wolvins,[135] Emanuel-Roncoroni[136]). Markham's case,[39] which was the only one in this group which came to autopsy, was in my opinion one of aortic insufficiency, due to an endocarditis of the aortic valve and not to rupture. Others besides Markham have made a similar mistake. Thus, no less a clinician than Gerhardt[11] reported a case of supposed rupture of the aortic valves, which later Sinnhuber[115] stated was shown at autopsy to be due to a recurring endocarditis of the aortic valve and not to rupture. It is, therefore, wise to regard no case as acceptable without an autopsy record.

In the second group there are forty-seven cases recorded, but only fourteen were proved by autopsy: (Bouillaud - Bergeon,[30] Wilks,[46] Hayden,[50] Finnell,[51] Foster[56] (case 3), Duroziez[68] (case 1), Barié-Potain,[72] (case 3), Mader,[76] Biggs,[80] Strassmann,[107] Schmidt,[109] Tranquilli-Deganello,[120] (case 2), Steinitz,[125] Meyer[134]). The Bouillaud-Tarrall[29] case came to autopsy, but, in my opinion, the perforation of the valve was not necessarily traumatic. In the other thirty-two, no autopsy was obtained after death, or the patients were still alive at the time the case was recorded (Leroy,[64] Duroziez[69] (case 12), Barié[70] (case 1), Barié[71] (case 2), Barié[73] (case 4), Heidenhain,[85] Dreyfus,[86] Dufour,[87] Bernstein[90] (case 1), Bernstein[91] (case 2), Bernstein[92] (case 3), Lembke,[93] Schneider,[94] Guder,[95] Kantorowitz[96] (case 1), Kantorowitz[97] (case 2), Castiaux and Laugier,[101] Calwell-Campbell,[106] Cahn,[110] Sinnhuber[115] (case 1), Sinnhuber[116] (case 2), Sinnhuber[117] (case 3), Schlecht[121] (case 3), Schlecht[122] (case 4), Zulzer,[123] Hoffmann[126] (case 1), Hoffmann[127] (case 2), Hoffmann[128] (case 3), Bensaude-Monod,[130] Cramer,[131] Brossard-Heitz[132] (case 1), Brossard-Heitz[133] (case 2)).

In the third, small, group of six cases the exciting cause was not stated, though all revealed at post-mortem a rupture of one or more of the aortic valves with a resulting aortic insufficiency; (Corrigan[26] (case 1), Rokitansky[38] (case 39), Bennett,[42] Ellis,[43] Humphrey,[74] Fisher[113]). However, Fisher's case is so incomplete as to be unacceptable.

To summarize, we have records of 113 cases of rupture of the aortic valve, of which forty-nine were proven by autopsy. Of the two main groups, the "strain" group is the larger, comprising altogether thirty proven and thirty unproven cases compared with fourteen proven and thirty-three unproven cases in the traumatic group. In five acceptable cases the cause for the rupture was not stated.

PREDISPOSING FACTORS

Country and race: Among the "strain" group, sixteen proven and seventeen doubtful cases were reported from Great Britain; seven doubtful from France; eight proven and three doubtful from Germany; one doubtful case each from Italy and Holland; three proven cases from America; two proven and one doubtful case from Canada; and one proven case from Australia. As may be seen, therefore, by far the great majority of the proven and doubtful cases were reported from Great Britain.

In rather marked contrast is the geographical incidence of the "traumatic group": only four proven and one doubtful case from Great Britain; two proven cases from America; and one from Italy; while from France three proven and eleven unproven cases, and from Germany five proven and twenty-one unproven, are recorded. In this group, therefore, Germany leads, with France second. In the third group; four cases (three proven) came from Britain; and one each from America[43] and Austria.[38]

Sex: Of the forty-nine proven cases of the three groups, the overwhelming majority (98 per cent) were males. In fact, only one female is listed among the "strain" cases and that doubtful, while among the traumatic cases, there were but three females, two of which were not proven cases. Evidently, therefore, the male

sex is more exposed to rupture of the aortic valve than the female.

Age: Of the "strain" group the age was stated in twenty-six of the proven and in twenty-six of the doubtful cases. In the former it ranged between 20 and 60 years with a mean of 37.2 years; in the latter from 20 to 68 years, with a mean of 37.4 years. The most susceptible decades were the fourth and fifth in both sub-groups. In those of the "traumatic" group, in which the age was stated, the range varied from 19 to 85 years in the proven cases, a mean of 45.6 years; while in the unproven group the variation was less marked (10 to 60) years), with a mean of 35 years. The most susceptible decades here also were the fourth and fifth. In short, the point of interest is that in the strain group the mean age was 37.2 years, in contrast to a slightly higher mean age for the traumatic group of 45.6 years.

Occupation: In the strain group of the thirty proven cases, the occupation is mentioned in twenty-seven, of which all but five (physician, apothecary, cook, clerk, and bar-tender) were exposed to constant or occasional muscular effort in their daily routine; for example, five were day-labourers, two dock-labourers, one brakeman, one butcher, one chauffeur, etc. In the unproven cases, the occupation was mentioned in twenty-nine, and in only six was the occupation of the non-laborious variety; of the six latter, the most interesting case occurred in a young pregnant mother during the act of labour. Among the arduous occupations are also mentioned, two day-labourers, two farmers, a ship-wright, a carpenter, a smuggler, a poacher, a bricklayer, etc. In short, in this entire group, 81 per cent of the cases occurred in occupations which exposed the patient to great muscular effort.

In the traumatic group, among the fourteen proven cases, the occupation is not mentioned in four; in the other ten cases (80 per cent), their occupations exposed them to injury. Thus, two cases were carters, two labourers, one soldier, one ship's-cook, one jockey, one coachman, etc. In these occupations, four sustained a fall to the ground from various heights, three were kicked by a horse, two received a blow on the chest from a fist or a piece of iron, and one was squeezed between a post and a cart-wheel.

In the unproven sub-group, there was the same variety of occupation, though in the great majority it was of the arduous type: among the twenty-eight cases whose occupation is given there were four labourers, four soldiers, one miner, one sailor, one locksmith, one stone-mason, etc. Here by far the commonest cause for injury was a fall (sixteen cases) and next in order of frequency, a blow from a blunt instrument (ten cases); a not infrequent cause was a crushing injury to the chest wall (five cases).

Past medical history. The patients' history, as to alcoholic excess, syphilis, rheumatic fever, chorea or tonsillitis, and previous cardiac symptoms, is of course of great importance in determining a possible *locus minoris resistentiæ*. This question will, therefore, be discussed before taking up the more accurate pathological criteria.

In the strain group, among the thirty proven cases, alcoholic excess is admitted in three cases only, denied in three, and not mentioned in the remaining twenty-four patients. In the doubtful cases, alcoholic excess was reported in four, absent in two, and not mentioned in twenty-four.

In the traumatic group the histories are more incomplete, even in the proven cases, but an abuse of alcohol was given in three cases before, and in one case after the accident; in ten histories no mention is made of alcohol. Of the thirty-three unproven cases, no one admitted to excess of alcohol, one denied it, and in the thirty-two other reports, no mention is made of it.

In the third main group which consists of five cases, in which the exciting factor is not stated, alcohol is not mentioned. In short, neither in the strain nor traumatic group does alcoholic excess play an important rôle.

In the strain cases which were proven, venereal disease was admitted only in the Frew-Finlayson case,[65] who had a chancroid at 18 years of age, but without rash; in seven other cases it is specifically denied, and in twenty-two it is not mentioned in the history. In the unproven cases, syphilis appears five times in their records, (Zohrab,[75] Horton-Smith,[102] Dupuis,[103] Taylor,[104] Emanuel-Roncoroni[136]), though at the time of the accident the blood Wassermann was negative in one patient; in four others venereal

disease is denied, while in twenty-four histories no mention is found. Syphilis, therefore, in the strain cases does not appear as prominently as one might expect from the well-known predilection of syphilis for the aortic valve.

In the traumatic group, among the fourteen proven cases, there is no case of known lues; in three it is denied and in eleven others no mention is made of it. Among the thirty-three unproven cases, one admitted to gonorrhœa (Sinnhuber[117] (case 3)); eight denied venereal disease and in twenty-four no mention is found.

In the third main group of five cases, venereal disease is not mentioned.

Rheumatism, chorea and tonsillitis, which are so important in the etiology of endocarditis, were present in five cases, negative in eleven, and not mentioned in fourteen of the strain group proven by autopsy. Arteriosclerosis existed in one case[79] of this group and had no doubt weakened the valve. Pulmonary tuberculosis, malaria, and possibly, a chronic infection from chronic eczematous ulcers of the legs occurred in each of three cases, and may have had a predisposing influence.

Among the thirty unproven cases in the strain group, rheumatic fever was present twice and chorea once, while in thirteen cases rheumatism is denied, and in fourteen others it is not mentioned. There was, however, a history of malaria twice, sepsis twice, pneumonia twice and influenza once, diseases which might have lowered the resistance of the aortic and valvular tissues. Hence in only seven of the entire group of 60 cases was there presumably a previous rheumatic endocarditis.

In the traumatic group, even among the proven cases, there was no case with a history of the rheumatic cycle; in three it was denied and in eleven no mention is made of it. One case (Tranquilli-Deganello[120]) had furunculosis five years before the accident, and so, possibly, septicæmia. Of the thirty-three unproven cases one[66] had rheumatism since the accident; two others[87, 122] before the accident; and one[95] had rheumatic purpura; in fourteen others the disease is denied, while in fourteen histories no mention is made of it. Hence, in the traumatic group of forty-seven patients, only four had, possibly, an aortic valve damaged by a previous attack of rheumatism. Two women[29, 86] had repeated pregnancies; while two males had a previous malaria;[69, 117] one measles and influenza[86] and one influenza alone,[87] while one patient had an active tuberculosis[123] and another was in the midst of a pneumonia,[128] when he fell out of bed.

In the third main group only one patient admitted to chorea.[43]

Previous heart symptoms: Symptoms suggestive of a pre-existing valvular disease were present in only two cases[58, 79] of the strain group proven by autopsy; in eleven others they were denied; while in seventeen of this group no mention is made of their existence. Presumably, therefore, though not conclusively, in only two cases did a valvular defect of any degree exist prior to the strain. Among the thirty unproven cases of this group there was a history of previous cardiac symptoms in three[75, 84, 103] of the twenty in which this point is mentioned. Again, but a small minority had pre-existing heart disease, though, of course, only an autopsy would corroborate this belief.

Among the fourteen proven cases of the traumatic group in five it is stated previous heart symptoms were present, while in the other nine cases no mention is made of the previous state of the cardiac compensation. In the thirty-three unproven cases twenty-one definitely denied them, and in twelve no mention is made of them in the scanty case reports.

In the third main group previous heart symptoms are not mentioned.

MORBID ANATOMY

First, as to the cusp affected: Because of the great variation in the anatomical nomenclature for the aortic valve we had great difficulty at times in deciding which of the three aortic cusps was the one ruptured. The anterior aortic cusp is usually called the "anterior," sometimes the "septal" or "segment of closure"; the right posterior cusp of Gray's Anatomy is sometimes called the "posterior" and sometimes the "mitral"; the left posterior aortic cusp is frequently spoken of as the "middle" cusp. We have reduced all these terms to "anterior," "right posterior" and "left posterior" cusps. It is important to remember that from just above the anterior cusp the right coronary artery arises; and

from above the left posterior the left coronary arises.

While usually only one cusp is torn, sometimes two, and, rarely, three are affected. In the strain group, only one cusp was affected in eighteen cases, two in ten, and all three in two cases.[57, 78] When only one cusp was involved, it was the anterior in seven cases, the right posterior in four, and the left posterior in two; in five other post-mortems the ruptured cusp is not specified. When two cusps were affected, it was the anterior and right posterior in two, the anterior and left posterior in one, and the right and left posterior cusps in two; in five other protocols the two cusps affected are not mentioned.

In the traumatic group, which comprised but fourteen cases, only one cusp was involved in ten; and two cusps were affected in four. Of the single cusp cases, in five protocols the exact one is not specified, while in two it was the anterior, in two the right posterior, and in one the left posterior. When two of the cusps were involved it was the anterior and right posterior in two, and the right and left posterior in two. There was no example of traumatic tear of all three cusps.

In the third small group of cases in which no exciting factor was stated, all five came to autopsy; of these the left posterior cusp was alone affected in two, while both the anterior and left posterior were simultaneously torn in two cases, and in one case the cusp affected is not specified.

From a study of these figures the most frequent single cusp to be involved is the anterior (nine cases); next, the right posterior (six cases), closely followed by the left posterior (five cases). If one, however, considers the cusp which may be most frequently torn, either singly or in combination with one or two other cusps, we find the anterior is still first (eighteen cases), the right posterior is next (sixteen cases), and the left posterior last (fourteen cases). This finding is somewhat contrary to the early teaching that it is usually the posterior cusps that are torn, but is quite in accord with the statement of Barié,[70] who found the cusp most frequently involved to be the anterior (which corresponds directly with the interventricular septum), as was seen in one of his seven experiments.

The site of the tear: There may be a rupture of the cusp itself (eleven cases), either at its free border or at its base, and but rarely in the middle of the cusp. As a rule, however, it is a vertical or longitudinal tear of the endocardium, or, rather, of the intima of the aorta, with a consequent displacement of the cusps (thirty-seven cases). As Peacock[4] wrote in 1873, "It may be that the angles of one or more of the segments are torn from their attachment, or the convex edge of the valve may be separated from the fibrous zone, or the curtain may be torn through."

In our series the strain group revealed only three cases of rupture of the cusp itself, in contrast to nine due to a tear of the angle of attachment, and to sixteen due to a tear of the intima of the aorta near the base of the valve. In the traumatic group there were eight cases of rupture of the cusp itself, to only one of its angle of attachment, and to six of the intima of the aorta. In the small unclassified group, we find no case of rupture of the cusp itself, two of the angle of attachment, and three of the intima near the base of the valve. In the entire series, while the commonest site was a tear of the intima near the base of the cusp (twenty-five cases), rupture of the cusp itself, or of its angle of attachment, being almost equally frequent (eleven of the former and twelve of the latter), we seem justified in concluding that the cusp itself is more apt to be ruptured in direct traumatic cases, and the angle of attachment or the intima of the aorta near its base in the muscular strain group.

Now, as to the state of the valve itself at the time of the autopsy (but not necessarily at the time of the accident); in the strain group it was normal seven times, thickened or atheromatous eighteen times, once with evidence of fresh endocarditis, and four times its condition was not mentioned. In the smaller traumatic group the aortic valves were normal six times, thickened or atheromatous five times, with fresh endocarditis once, and twice their condition is not mentioned. In the third, very small, group the state of the valve is only mentioned in four cases; in three it was thickened or atheromatous, while in one it was covered with recent vegetations. What was specially interesting was that two cases re-

vealed a congenital anomaly of the valves.[43, 74]

To summarize, therefore, in only thirteen of forty-eight autopsies, or 27 per cent of the entire series, was the aortic valve reported as absolutely normal, but it was more frequently normal in the traumatic group (44 per cent) than in the strain group (23 per cent). In the great majority of the protocols the aortic valves are described as thickened, atheromatous, or even calcareous. In two cases,[33, 72] there was evidence of a fresh endocarditis in addition to the rupture, but in both cases it was probably of more recent origin.

The state of the aorta, and especially of the intima near the mouths of the coronary arteries, is of course of great interest. In the strain group the aorta was reported as normal in three cases, smooth but dilated in two and atheromatous in sixteen cases; in three of the atheromatous group there was some dilatation, and in one an aneurysm of the ascending aorta,[114] while in two[37, 83] others the mouth of one coronary was obliterated by a patch of atheroma. In the traumatic group, the aorta was both actually and proportionately more frequently normal (five cases), though in one of these it was dilated. In only four cases was there atheroma and in one of these there was also moderate dilatation. In one case of this group[109] there were multiple tears of the thoracic, and a single tear in the abdominal aorta. In the third, or unclassified, group the aorta was atheromatous in two. Unfortunately, no statement about the condition of the aorta was found in nine of the strain group, four of the traumatic and two of the unclassified. In Corrigan's[26] case only was the process in the aorta suggestive of syphilis, as the aneurysm in Heller's case[114] resembles more a dilatation than a syphilitic one. In general, therefore, the aorta was much more frequently the site of atheroma in the strain group (sixteen cases) than in the traumatic.

As to the state of the heart itself: it was reported as hypertrophied in eleven, dilated in six, and both dilated and hypertrophied in nine of the strain cases; in not a single case was it normal, though in four cases the exact state of the heart was not stated. In the traumatic group, on the other hand, it was hypertrophied in one, dilated in three and both hypertrophied and dilated in six; in one case it was said to be atrophied, while in three cases its condition is not stated. In the unclassified group it was found hypertrophied in two and not stated in three. In short, hypertrophy of one or both ventricles was the rule in all three groups, though frequently dilatation also existed. As to the condition of the myocardium itself, fatty or fibroid degeneration was reported in five cases of the strain group and two cases of the traumatic group. In addition, there was an abscess in the wall of the left ventricle in one case,[134] an aneurysm of the mitral valve in another,[37] two aneurysms of the right auricular appendage in another,[82] and an adherent pericardium in another case.[107]

The usual evidence of cardiac decompensation was found in many cases, especially venous stasis of the lungs, liver and spleen; in two cases hydrothorax, and in two other cases effusions into two or more of the serous cavities, were present. Pulmonary infarction occurred in but two cases;[67, 137] thrombosis of the right axillary and brachial artery in one case,[100] and a thrombosis of the posterior tibial vein in our own case.[137] As might have been expected in the traumatic cases, laceration of the parenchymatous organs, liver and spleen or of the intestine occurred twice[30, 109] and once[46] respectively. In this same group, fracture of the ribs,[30] dorsal vertebræ,[30, 109] or of skull was also present in the odd case. An occasional finding was healed or active tuberculosis of the lungs.

PATHOGENESIS

Muscular effort or strain may result in rupture of a healthy valve, though this is naturally a rare event according to both Romberg and Allbutt, and, as already stated, occurred only seven times in our series. Muscular effort may lead to rupture of an aortic cusp more readily in a valve previously the site of rheumatic or syphilitic or arteriosclerotic disease; in fact many authorities recognize only this group. Of course, many of the case reports are so lacking in detail as to leave themselves open to the doubt of the presence of some previously existing gross, or at least microscopical, lesion. Lastly, muscular effort or strain may result in repeated mild attacks of valvulitis, and so predispose to subsequent rupture of the valve.

In answer to the question how muscular

effort causes rupture of a valve, Barié[70] states that during the effort the thorax is filled with air but is immobilized, and the intra-aortic tension is considerable, because during each diastole of the heart the aorta has to support an extreme pressure. Pepper[58] offers a somewhat similar explanation, "Immediately before the effort, whether at striking, or lifting heavy weights, a deep inspiration is taken, which aids in filling the cavities of the heart to their utmost, and then in order to afford fixed points for the contraction of the muscles of the arms and shoulders and back, the chest is held rigidly fixed. The violent contractions of the neck which follow compress the carotid arteries, while those of the muscles connected with the arms impede the free flow of blood through the subclavians and their branches. Thus, while the tension within the chest is greatly increased and the heart is stimulated to violent contraction, there is also an enormous elevation of arterial tension. The strain which results must extend itself directly upon the walls of the left ventricle, which must over-exert themselves to press forward the blood, and indirectly upon the aortic valves, which are compelled to bear the shock of a recoil of the blood stream violent in proportion." Heller[114] believes the explanation of the fact that the predilection site of the tear during muscular effort is the first portion of the aorta is that in many persons the trachea is attached just above the aorta, and that the right bronchus in spastic respiratory arrest is pressed against the aorta. According to Potain, it requires a pressure of twenty to twenty-five c.c. of mercury to break the aortic valves. Barié's[70] experiments on the cadaver are of great interest. By increasing the intra-aortic pressure until it reached from 116 to 484 mm., of mercury, in ten cadavers dead from various non-cardiac diseases, he was successful in producing rupture of one or other of the aortic cusps in four; in two the septal segment (anterior), and in two the left segment (left posterior) were torn.

Trauma may result in rupture of an aortic valve segment, either (a) as a result of a contusion of the chest wall from a kick or a blow or compression of the chest; or (b) as a result of a violent shock to the body from a fall from a height. It must, however, be admitted, with Allbutt, that in the history of some of these accidents the distinction between the outer and inner stress cannot be made, but probably in all the mechanical process is similar, the external blow violently compressing the thoracic cage.

Barié[70] and Potain experimented on the cadaver by administering three blows from a hammer on a board fixed to the præcordium: in two out of the five cadavers rupture of the aortic valve occurred; once in the median segment (left posterior) there developed an oblique tear extending from its free border to the attachment; and once a tear of the septal (anterior) cusp.

Dufour[87] has produced in four dogs a traumatic rupture of the aortic valve. His method was the administration of one to four blows with a mallet over the præcordium; post-mortem, several ribs were found broken and in all four dogs the water-test of the aortic ring was positive; the other valves were normal. In the first dog there was a tear of the right posterior and right anterior cusps; in the second dog all three cusps were torn; in the third, rupture of the right posterior had occurred at the insertion; in the fourth dog he found rupture of the right posterior cusp, from its free border to its base, and separation of the left posterior cusp from the aorta.

Kuelbs,[9] in 1909, used a 40 cm. round meat-hammer, weighing 150 grams, and gave one to three sharp blows over the left thorax, the force of which as read by a dynamometer varied from 140 to 180 kilograms or more. The dogs revealed no evidence of pain, but merely of anxiety and shock. Two died spontaneously after the blow; the others were killed by bleeding and chloroform. The pulse was noted as rapid and irregular; heart murmurs became audible in six of the dogs. Post mortem, there were occasional small hæmorrhages into the pericardium, but more often hæmorrhages into the valvular endocardium, especially of the semilunar valves; a tear of an aortic valve occurred only once and that near the nodulus Arantii of one segment. There was often hæmorrhage into the myocardium. In short, 28 of the 34 dogs, or 82 per cent, showed valve hæmorrhages (mitral 12; aortic 7; pulmonary 4; tricuspid 5). Normal heart sounds were sometimes present when the post-mortem revealed a hæmorrhagic valve.

Further, valve hæmorrhage may occur without evidence of external injury to the skin, muscles or ribs.

SYMPTOMATOLOGY

The symptoms of rupture of the aortic valve are characterized by the suddenness of their onset and their severity, according to Broadbent.[100] The onset is sudden, as has been stated, but death does not immediately follow, as one might at first suspect. The commonest initial symptom is an agonizing pain, sometimes associated with the sense of a sudden tear within the chest. In many cases this is immediately followed by faintness, or even actual syncope, with complete loss of consciousness. When the patient regains consciousness, he usually immediately, but sometimes not for seven to fifteen days, notes palpitation of the heart, a sense of oppression, urgent dyspnœa, and the other symptoms of acute cardiac decompensation, such as cough, hæmoptysis, orthopnœa, cyanosis, restlessness, and œdema. Sometimes, too, the patient is conscious of a curious sound in his chest, which may be audible to others. In rare cases the onset of symptoms is neither immediate nor gradual, as described above, but much later in development, for in one case two years, and in another four years, intervened between the time of the strain or trauma and the actual onset of symptoms.

To summarize, therefore, the characteristic symptoms are (1) pain; (2) syncope, or faintness or vertigo; (3) oppression and dyspnœa; (4) palpitation and hæmoptysis; (5) sensation of a roaring in the chest, neck or ears, and finally; (6) the usual signs of aortic insufficiency.

The pain is acute, agonizing, tearing in character, referred to the præcordium or epigastrium, and usually with radiation down the sternum or to the neck and down the left arm and sometimes into the back between the shoulder-blades. It is said to be more frequent in the strain group than in the traumatic, and occurred forty-eight times in our series of strain cases and twenty-four times in the traumatic. There is sometimes the *anguor animi* or sense of impending death, as in true angina pectoris. With the pain there is often a sensation of "something being torn loose" in the chest (three cases); or of "something snapping in the chest" (three cases); or of "being stabbed" (one case), or more often of "something giving way" (seven cases). While usually immediate in its onset in the strain cases, it may not appear until three months later (one case), or only on exertion (one case). It was twice likened to angina pectoris.

To recapitulate: in the proven cases of the strain group pain was present in twenty-three cases (76.6 per cent); absent in two cases, and not mentioned in five cases. In the unproven cases it was present in twenty-six patients (86.6 per cent); absent in two; and not mentioned in two patients. In the proven cases of the traumatic group pain was mentioned in only five cases, in one of which it occurred with a sense of constriction in the chest and in another only on exertion. Among the thirty-two unproven cases pain occurred in nineteen and was not mentioned in thirteen. While the pain was usually noted immediately after the exertion, in four cases it did not make its appearance until a few days to ten weeks and in one case not until two years later. In short, pain is almost a constant symptom of rupture of the aortic valve due to strain (92 per cent), and is much less constant in the traumatic group (65 per cent).

Syncope or faintness or vertigo may sometimes be present, but have not been stressed by former writers. However, in eighteen of the proven strain cases, in which these symptoms are mentioned, syncope occurred in six and faintness in four; in one case weakness and in another vomiting occurred at the time of the accident. Among the twelve of the unproven strain cases, in which these were noted, syncope occurred in six patients in one of whom it lasted for eighteen hours, and in another of whom it was followed by vertigo. In three patients only faintness was noted. In the entire group, syncope, faintness or vertigo was noted, therefore, in nineteen out of sixty patients, or 31.5 per cent. In the fourteen proven traumatic cases the history is less complete, but one or other was present in three cases; in one the syncope lasted for forty and in the other for ten minutes; in the third case only faintness occurred. Curiously enough, among the thirty-two unproven traumatic group, fifteen patients suffered either from syncope, shock, faintness or vertigo. Of these

twelve had complete loss of consciousness (one of four days' duration; another of one day) which was followed in three by vertigo; one had vertigo only; one, faintness and one had shock without loss of consciousness. In the entire group of forty-seven cases, eighteen suffered from cerebral manifestations, or 39.1 per cent.

Oppression and dyspnœa: Among the thirty proven cases of the strain group, one or other of these symptoms was rather constant, being present in twenty-three, absent in three and not mentioned in four (76.6 per cent). They usually appeared immediately after the accident, and independently of further exertion, but in four patients the dyspnœa first made its appearance in from four weeks to three months. In twenty of the unproven cases of the same group dyspnœa was present, in three absent, and in seven no mention is made of it, (66.6 per cent).

Among the fourteen proven cases of the traumatic group, it was present in eight cases and not mentioned in six (57.1 per cent). In three patients it only appeared in from a few days to ten months. In the larger unproven traumatic group these symptoms were present in thirty-three, absent in two, and not mentioned in seven cases, (71.8 per cent). In five cases the onset of dyspnœa was from several days to several months after the trauma. In one case it was evidently paroxysmal in character.

Palpitation and hæmoptysis were comparatively rare symptoms, but sufficiently frequent to merit consideration. Palpitation occurred once in the proven cases of the strain group and eight times in the unproven cases; among the traumatic cases it was present once in the proven cases and eight times in the unproven cases. In the strain group hæmoptysis occurred once among the proven, and twice among the unproven, and not at all among the proven cases. Hence, it may be concluded that palpitation is more frequent than hæmoptysis, but the absence of either does not signify. The hæmoptysis is probably due to pulmonary infarction, as was noted in several of the autopsies.

Sensation of roaring in the chest, neck or ears: This is certainly a peculiar symptom, and was described by the patient as resembling the "croaking of a frog",[52] "the cooing of a dove",[63, 65] a "rumbling, rustling noise",[82] a "humming noise",[60] a "whistling noise",[75] a "musical murmur or thrill",[103, 137] a "buzzing in the chest",[104, 121] a "peculiar sound in the chest",[33, 40, 94] a "rattle in the head",[95] "a whirring noise".[58] In most patients it was constant, but most troublesome at night, disturbing their rest. In one patient it was increased by exertion. In twelve patients this sound was audible to the friends or family. This phenomenon was apparently more frequent in the strain cases, occurring among nine of the proven and eight of the unproven group. It was not mentioned among the traumatic proven cases, but was present in four of the unproven cases.

To summarize, then, a murmur was audible to the patient or his friends in nineteen of the strain series and four of the traumatic.

Signs of aortic insufficiency: The usual signs, such as a widespread area of pulsation, increased force of the heartbeat, increased area of dullness, an aortic diastolic murmur of maximum intensity at the base, a capillary pulse, Duroziez' sign, and a low diastolic blood pressure, were noted in the majority of cases. Thus in twenty-five of the proven and thirty of the unproven strain cases one or more of these signs are noted, and always, at least, the aortic diastolic murmur. Again in nine of the proven and thirty-two of the unproven traumatic cases at least a diastolic murmur was noted. In only five of the proven strain cases and five of the proven traumatic and in four of the "not stated" group is no mention made of the physical signs. The diastolic murmur was often somewhat remarkable in its quality and distribution. According to Strassmann,[107] it is often longer, more intensive, and of a peculiar tone. Foster[48] believes that the diastolic murmur frequently has a special blowing and flapping character, instead of the soft quality of the murmur of aortic insufficiency due to endocarditis. Again, it was termed "harsh" or "intense" in nine cases, "prolonged and loud," "gushing," "rumbling," "creaking," "flapping," "rough and flapping," "rasping or piping," each in one case. In six cases it was merely described as musical, without other qualification; while in nine other cases the musical quality was modified by such terms as "sibilant" (two cases), "vibrating," "tone like the vibration of a string," "piping," "buzzing," "purring,"

"like a torn sail," "siren-like" (each in one case). The murmur was audible to the physician in some cases of the strain group several inches from the chest. Thus in Quain's[33] case it was described as audible several inches from the chest, and in Tretzel's[81] at three metres; in one of Hoffmann's[129] cases it was audible "some distance" from the chest. Among the unproven strain cases, it was noted by O'Neil[40] as audible at six feet, by Burney-Yeo[59] three feet, and by Dupuis[103] fifteen to twenty centimetres. Among the traumatic cases it was audible at fifty centimetres in the Tranquilli-Deganello[120] case as well as in Barié[70] case; twenty-five centimetres in Schneider's[94] case, and "several centimetres" in Schlect's[122] patient.

A diastolic thrill was occasionally palpable over the base, being noted twice in the proven and five times in the unproven cases of the strain group. In not a single instance was it noted in the traumatic group.

An aortic systolic murmur was heard in thirteen of the proven and in fifteen of the unproven cases of the strain group. In the traumatic group it was somewhat the same, being noted in eight of the proven and twelve of the unproven cases. It was usually widely distributed over the præcordium and invariably of maximum intensity at the base, though once or twice loudest at the apex, and, possibly, therefore, due to a relative mitral insufficiency. This murmur was described as "loud" four times; "rough" four times; "musical" twice; and "harsh," "vibratory" or "flapping" once each. In addition, a systolic thrill was felt over the base in three of the proven strain cases and in two of the unproven traumatic group. It has been suggested that the presence of the systolic murmur and thrill can best be explained by the torn segment floating in the blood stream.

DIAGNOSIS

A diagnosis is possible if the previous condition of the heart is known, but, of course, without the proof of a post-mortem examination this cannot be considered as conclusive. Rupture of the heart wall, or of the aorta itself, must always be excluded, according to Dufour.[87] We are inclined to agree with the latter that there is no certainty in any case, especially when one recalls that no less a clinician than Gerhardt, of Berlin reported a case as clinically certain, but which three years later revealed an endocarditis without evidence of rupture. Meyer[134] emphasizes the absence of cardiac hypertrophy in the traumatic-rupture cases, and based his diagnosis on this point. In the series of one hundred and thirteen cases, only forty-nine came to autopsy and in only six was the clinical diagnosis made correctly, namely by Pepper,[58] Burney-Yeo,[59] Potain,[72] Tretzel,[81] Meyer,[134] and our own case by my resident, Dr. E. S. Mills.[137] In twenty-one cases no clinical diagnosis was made, while in eight the diagnosis was merely aortic insufficiency, and in one other aortic insufficiency wrongly associated with an aortic aneurysm. Strassmann[107] was content to make a diagnosis of "traumatic heart insufficiency." Six cases were absolutely wrongly diagnosed; one as indigestion, one as ulcerative endocarditis, two as aortic aneursym, one as aortitis with angina, and one as cardiac failure.

PROGNOSIS

This depends first upon the size of the defect, and, secondly, upon the efficiency of the heart muscle. In accordance with these factors, an immediate fatal syncope may result, or there may gradually develop a circulatory stasis, with increase in the size of the liver, œdema of the lower extremities from dilatation of the right ventricle, and death some weeks or months later. This is the most common course. In some cases there may be temporary comfort for a couple of weeks or even months, and then the appearance of symptoms.[100] Again, years may elapse before symptoms manifest themselves, if the previous state of the myocardium is healthy. Thus, Balfour,[7] in his monograph of 1898, writes "But I myself have seen *post mortem* cusps which had been torn from their attachments; the lesion healed and yet no serious symptoms till years subsequently. At the moment I am acquainted with a patient who more than twenty years ago had symptoms and signs apparently due to rupture of one of his aortic cusps, yet he still survives in much improved health."

Rarely, a chronic aortic insufficiency may result without symptoms. Fraentzel,[79] as well as Castiaux et Laugier,[101] have reported cases, which at autopsy showed repair of a small tear.

Foster[48] makes a nice point when he states that if the right posterior cusp is ruptured the diastolic murmur will be transmitted to the apex, when a better prognosis can be given, because there is no opening of a coronary near this aortic segment, and so there will be less interference with the nutrition of the heart muscle.

The duration of life, then, varies from a few hours or days to several years. In the thirty proven strain cases one death[28] was immediate, in another[47] it occurred in fourteen days, and in a third[77] in fifty-one days, and in the remaining twenty-two in which the duration of life is given, it ranged from three months to five and one-half years, with an average duration of life of a little more than eighteen months. In fourteen of the traumatic group proven by autopsy the duration is given for thirteen cases. In one[30] death was immediate; in another[51] a few hours, and in a third[109] but two hours intervened; one[46] survived three days. On the other hand one case[72] survived ten years, and another[125] eleven years and one month. The mean duration of this group is, accordingly, forty months for those that survived the immediate effects of the trauma. It would seem, therefore, that the prognosis is better in the traumatic than in the strain cases. There is no use in discussing the duration of the life in the unproven cases, though it is interesting to remember that in one of Barié's[75] cases, fourteen years have elapsed, and this too is a traumatic case; another of the doubtful traumatic cases[29] lived twenty years.

TREATMENT

In the event of a suspicion of rupture of an aortic valve from strain or traumatism, the indications are first absolute bed-rest, in an endeavour to avoid all causes likely to give rise to inflammation of the valve, and, second, to subdue such symptoms as may arise. Later, one must insist upon great caution in exercise and an avoidance of all forms of excitement and the use of depressant agents. Peacock, accordingly, strongly advises against the use of digitalis. Generally speaking, an ice-bag to the præcordium, and possibly venesection, will relieve the patient. Later, purgation and diuresis may be tried.

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