THE HISTORY OF MITRAL STENOSIS

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The early observations on mitral stenosis were largely made in France and Britain, and in the past its clinical diagnosis was probably more discussed than that of any other form of valvular disease of the heart.

As the study of morbid anatomy is much older than the practice of auscultation, the lesion of mitral stenosis was described more than a hundred and fifty years before its clinical recognition received the help of auscultation. Extreme constriction of the mitral orifice in a young man was recorded in 1668 by John Mayow (1640–1679), an Oxford physiologist contemporary with Richard Lower (1631–1691), whose Tractatus de corde appeared in 1669. The morbid change in a case was more fully reported in 1715 by another contemporary, Raymond de Vieussens (1641–1715) of Montpellier, with an illustration of the mitral valve, reproduced in Major’s Classic Descriptions of Disease. Morgagni (1682–1771) also recorded the lesion in 1761, under the title “ossification of the mitral valve.”

PHYSICAL SIGNS

In 1806 J. N. Corvisart (1755–1821), called by Andral “the Morgagni of France,” and in 1819 his pupil R. H. T. Laennec (1781–1826) began the description of the physical signs of mitral obstruction which, following Morgagni, they designated as ossification or calcification of the mitral valve. Corvisart gave an account of the rustling or thrill palpable over the heart as characteristic of the lesion and ascribed it to obstruction to the passage of the blood from the lungs and left auricle through the narrowed mitral orifice.

In 1809, Allan Burns (1781–1813), anatomical and surgical lecturer in Glasgow, in a report of one of three cases of mitral stenosis confirmed at necropsy, wrote: “there was a jarring when the ventricles contracted; and when the hand was laid on the side, it resembled the feel of a varicose aneurism . . . he
had unusual palpitation, jarring sensation, and a hissing noise, as of several currents meeting; the sound was often audible as in varicose aneurism." These signs were, however, ascribed to mitral regurgitation, and he went on "in all probability, it is something of this kind which is described as audible palpitation in some diseases of the heart." It thus appears that he recognized the thrill and also seems to have realized the mechanism (fluid vein) of a cardiac murmur. No reference is made to Corvisart's description of the thrill, and on account of the Napoleonic wars it is probable that he had not any opportunity of seeing Corvisart's essay. Robert Adams (1791–1875), Regius Professor of Surgery in the University of Dublin and author of an outstanding work on chronic arthritis (1857), recognized, apparently independently, this thrill in 1827.

The auscultatory signs of mitral stenosis have been a somewhat confusing battlefield of opinion. Corvisart in his aphorisms did not mention a murmur. Laennec and his followers, who attached more importance to obstruction than to incompetence of the valves in the causation of murmurs, believed that the passage of blood through a narrowed mitral orifice produced a direct, not a regurgitant, murmur, either a soft bellows (bruit de soufflet) or less often a rough rasping (bruit de scie ou de râpe) murmur. What Laennec did not do was to fix the exact point in the cardiac cycle at which this took place.

In February 1819 he examined a boy, aged 16 years, with a thrill and accompanying murmur, both ascribed to the prolonged contraction of the left auricle, the murmur being "dull but strong, exactly like that produced by the stroke of a file on wood."

Andral (1797–1876) in his annotated edition (1837) of Laennec's Traité de l'auscultation médiate drew a practical distinction in the significance of the bruit de râpe, which was evidence of organic obstruction, and of the bellows murmur. In a note on the history of valvular diseases of the heart Sir Samuel Wilks (1824–1911) in 1871 pointed out how remarkable it was that this simple view, closely resembling that of more recent times, should so soon have been abandoned in favour of its regurgitant nature; he suggested that this may have been because Laennec and others erroneously considered that the second sound of the heart was due to the auricular systole, and that when the true nature of the second sound was discovered, physicians, instead of "simply correcting the fault, swept away the whole idea of this direct mitral murmur. At least, this is what, for the most part, occurred, and the teaching of the schools was that all mitral and apex bruits were of a regurgitant nature. . . . It is a recent revival to speak of direct mitral bruits." Laennec had attributed the first sound of the heart to the ventricular systole and the second sound to the auricular systole. That the second sound was due to the closure of the sigmoid valves and not to the contraction of the auricles was shown by J. W. Turner (1790–1836) of Edinburgh in 1829, and by the experimental work between 1830 and 1836 of James Hope (1801–1841) and C. J. Blasius Williams (1805–1889), who owed this unusual Christian name to his birth on February 3, the day on which St. Blaise or Blasius underwent martyrdom in A.D. 316.

Though a relatively small matter of priority, it hardly seems reasonable that
R. J. Bertin (1767–1827), who held the same views as Laennec about the production of the heart sounds, should sometimes be picked out, rather than Laennec, to have been the first to listen to what was in reality a presystolic murmur.

Bertin reported six cases in which mitral obstruction was found at necropsy; in three of them, early in the century before Laennec invented auscultation, no mention of a murmur was made; in the other three a bellows murmur was recorded, which later in his book was said to be pathognomonic. Very likely he may have heard a presystolic murmur, but he did not describe it as rasping or presystolic.

In 1832 Hope described the second sound of the heart on the left side of the sternum in mitral stenosis as altered, i.e. losing its short, flat, clear character and becoming a more or less prolonged bellows murmur. “When the valve is permanently patescent, admitting of regurgitation, the first sound likewise is attended with a murmur.” The first above-mentioned murmur was formerly known as Hope’s early diastolic murmur. In the third edition (1839) of his Treatise Hope mentioned that he had carefully listened for a murmur due to the auricular systole, but without success. Subsequently this diastolic murmur was ascribed to dilatation of the trunk of the pulmonary artery so that its valve segments became relatively incompetent, and has been called the murmur of high blood pressure in the pulmonary artery (Graham Steell, 1888). Williams, the early colleague as an experimental cardiologist of Hope and later his rather unfriendly rival, stated in 1840 that this diastolic murmur in mitral obstruction was very rare, as he had recognized it in two or three cases only. He referred to its possible production by the suction-pump action of negative pressure due to the diastolic expansion of a well-developed left ventricle.

Fauvel and the Presystolic Murmur

The view that the murmur characteristic of mitral stenosis was due to regurgitation of blood from the ventricle into the auricle was until after 1861 (see p. 5) commonly but not universally held, and was indeed revived more than once after that date (see p. 7). In 1843, however, Fauvel (1813–1884), chief of the clinic of the Paris Faculty of Medicine at the Hôtel-Dieu, Paris, described, with five illustrative cases (three with necropsy) of mitral stenosis, the presystolic murmur.

He was careful to acknowledge that the word “presystolic” was taken from Gendrin (1796–1890), who in 1841 had divided the cycle of the heart into six periods: systole, perisystole (immediately following), presystole (immediately preceding systole); diastole, peridiastole, and prediastole (p. 32). Gendrin laid it down that obstruction to the passage of blood from the auricle to the ventricle caused a prediastolic murmur, adding that the systolic, perisystolic, and prediastolic murmurs were often continuous (p. 110). He regarded reduplication of the second sound as evidence of mitral stenosis.

Fauvel shared the fate of the prophet in his own country: Bouillaud (1796–1881) did not support his junior, being probably more interested in a triple bruit (de rappel) at the base of the heart, which he described in 1836. In 1853
Hérard (1819–1913) stated that mitral stenosis might be associated, in varying degrees of frequency, with a systolic, presystolic, or diastolic murmur. In 1862 Duroziez (1826–1897) referred to “that famous presystolic murmur of which everyone talks but no one understands.” In Germany Canstatt (1807–1850), of Ratisbon, in 1848 confirmed Fauvel’s views, and aroused criticism from Wintrich (1812–1882), who in 1849 concluded that it was a rather interesting article de luxe of physical diagnosis (Hilton Fagge). In Great Britain Fauvel’s article attracted hardly any attention, though destined eventually to become a milestone in the history of the disease. Thomas Addison (1793–1860) “never distinguished a presystolic bruit from a systolic one, and openly taught that he was quite unable to diagnose a contracted auriculo-ventricular orifice from a dilated one” (Wilks). William Stokes (1804–1878), Regius Professor of Physic in Dublin, did not welcome the new murmur; in 1854 he wrote:

“To the inexperienced, the detailed descriptions of such phenomena as the intensification of the sounds of the pulmonary valves, of constrictive murmurs as distinguished from non-constrictive, of association of different murmurs at the opposite sides of the heart, of pre-systolic and post-systolic, pre-diastolic and post-diastolic murmurs, act injuriously: . . . by conveying the idea that the separate existence of these phenomena is certain, and that their diagnostic value is certain.”
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In 1871 Hilton Fagge (1838–1883), realizing the importance of Fauvel’s description of the presystolic murmur and finding that it had not been noticed in detail by British writers, gave a full abstract of it. He pointed out that it would not be strictly accurate to assume that British physicians entirely overlooked the direct mitral murmur and its presystolic character until Gairdner’s paper in 1861. Several, such as Peter Mere Latham (1789–1875) in 1845, Bellingham (1805–1857), a Dublin surgeon, in 1853, and Markham (1818–1891) in 1854 and 1860, described it as simply diastolic. Some said it was very rare; thus in 1851 Herbert Davies (1818–1885) had never heard it, and in the same year Ormerod (1819–1873) stated that a direct mitral murmur was one of the rarest morbid cardiac sounds and that he had identified it in two cases only.

W. H. Walshe (1812–1892), born in Dublin and endowed with the brilliancy of a clever Irishman, educated medically chiefly in Paris, M.D. of Edinburgh, and physician to University College Hospital in London, appears to have been in 1851 the first in Great Britain to recognize the presystolic character of the direct mitral murmur in mitral stenosis; he spoke of the diastolic murmur as rather post-diastolic or presystolic than as coinciding precisely with the ventricular diastole.

In 1859 Austin Flint, the elder (1812–1886), then of New Orleans and later of the Bellevue hospital, New York, stated that a direct diastolic mitral murmur “follows the second or diastolic sound of the heart and precedes the systolic or first sound.” He regarded this diastolic mitral murmur as rare because the contractions of the auricle were not sufficiently powerful, but did not refer to Fauvel or actually use the adjective “presystolic” then.

Gairdner and the Auricular-systolic Murmur

In 1861 Sir William Gairdner (1824–1907), then of Edinburgh and from 1862 to 1890 Regius Professor of the Practice of Medicine in the University of Glasgow, described under the title “auricular-systolic” (A.S.) the murmur Fauvel had called presystolic. Gairdner’s “Short Account of Cardiac Murmurs,” which did not refer to Fauvel, “marked” according to G. A. Gibson (1854–1912) of Edinburgh, Gairdner’s biographer, the “commencement of a new epoch in physical diagnosis.” It was largely responsible for the eventual recognition of the presystolic murmur in Great Britain. Gairdner also described a direct mitral murmur, ventricular-diastolic in time and coincident with the filling of the ventricle by its rapid expansive action. In the second edition (1867) of his Treatise, Austin Flint preferred “auricular-systolic” to presystolic as the qualifying adjective.

After Gairdner’s important paper, opinion still fluctuated before its acceptance became general. The history and position in 1871 were exhaustively reviewed by Hilton Fagge, whose paper also contained many clinical observations, especially about the rhythm of murmurs and the state of the sounds of the heart, on 67 cases in Guy’s Hospital. In 1865 James Andrew (1829–1897), of St. Bartholomew’s Hospital, and in 1867 T. B. Peacock (1812–1882), of St. Thomas’s Hospital and a prominent authority on cardiac disease, especially on
congenital malformations, spoke of the recognition of a presystolic murmur as "one of the most difficult tasks in the physical examination of the heart, and often all but impossible, at least in the later periods of the disease." This disappearance of the murmur had to wait for an explanation until the discovery of auricular fibrillation early in this century by Sir James Mackenzie (1853–1925), who first called it "nodal rhythm" and thought it due to paralysis of the left auricle, and of the electrocardiographic and other observations of Sir Thomas Lewis. Physiologists had experimentally produced fibrillation of the heart muscle in the last century (Gaskell; MacWilliam). Hyde Salter (1823–1871), physician to Charing Cross Hospital, in a clinical lecture published in 1869 declared that the presystolic was the easiest of all cardiac murmurs to detect and that for 30 years physicians had regarded it as systolic in time.
Hilton Fagge agreed with the last statement, but gently added "it is perhaps going a little too far" (as Salter did), "even in addressing a class of students, to say that now anyone who should fail to recognize and identify this sound would not only be unfit to hold the place of an accomplished and critical physician but could hardly be considered as a decently informed member of our profession."

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Presystolic murmurs have been ascribed to causes other than ordinary organic mitral stenosis. The best established is the Austin Flint murmur at the apex beat in aortic regurgitation. Austin Flint, in describing it in 1862, explained its mechanism as follows: "in aortic incompetence the left ventricle is rapidly filled with blood regurgitated from the aorta in addition to that from the left auricle, as a result the mitral curtains are brought into coaptation, and when the auricular contraction takes place this direct mitral current passing between the curtains throws them into vibration and gives rise to the characteristic blubbery murmur. The physical condition is in effect analogous to contraction of the mitral orifice from an adhesion of the curtains at their sides." Austin Flint thus specially incriminated vibration of the valve segments, but his mention of "coaptation" does not entirely exclude the later view, namely that impingement of the regurgitant blood from the aorta on the anterior cusp of the mitral valve bulges that flap in so as to narrow the mitral orifice (Guitéras)—an inorganic stenosis; evidence of this has been adduced in the presence of endocardial thickening of the auricular surface of the mitral valve. Flint's accuracy in timing the presystolic murmur in aortic reflux uncomplicated by mitral stenosis was bluntly questioned by G. W. Balfour (1823–1903), who in 1876 warned his Edinburgh students not to make such a mistake. Flint naturally protested against this in 1884 and 1886. In his thesis for the M.D. degree at Cambridge in 1895 A. G. Phear analysed 46 cases in which the murmur was present without mitral stenosis, but associated with aortic incompetence or some other cardiac lesion. In 1901 W. S. Thayer (1864–1932) found that Flint's murmur was present in 63 per cent. of cases of aortic incompetence proved by necropsy to be free from mitral stenosis. This presystolic murmur in association with aortic reflux is now fully accepted. A presystolic murmur has also been described in cardiac conditions other than aortic incompetence or mitral stenosis: in 20 of Phear's 46 cases there was an adherent pericardium, and in some others with dilatation of the left ventricle. When a presystolic murmur has been heard in a case in which great dilatation of the left side of the heart is the chief lesion found, it has been suggested that it is due to relative or virtual stenosis, though without any absolute constriction of the mitral orifice (Rolleston and Dickinson, 1897). Another view is that the murmur may be due to the meeting of two columns of blood, one direct from the auricle, the other regurgitant from the ventricle into the auricle (Allan Burns).
Systolic, Presystolic, or Diastolic

Not long after the presystolic murmur was gaining acceptance in Britain criticism arose not about its diagnostic significance, but to the effect that it was really systolic in time. This was argued in 1864 by Ormerod, followed in 1868 by Leaming (1820-1892) of New York, Barclay (1817-1884) of St. George's Hospital, London, in 1872, F. Donaldson (1823-1891) of Baltimore in 1874, who, like Leaming, was said to have an accomplished ear for musical and other sounds, by Sir D. C. McVail (1845-1917) of Glasgow in 1879, quite briefly, by Dickinson (1832-1913), physician to St. George's Hospital, in 1887 and 1889, who wrote at length and rather provocatively on “the presystolic murmur falsely so called,” by F. C. Turner (1843-1900) of the London Hospital, in 1887, and by E. M. Brockbank in six papers between 1897 and 1910, who used the phrase “the crescendo murmur of mitral stenosis.” In 1911 Hart concluded that “in some cases at least the short crescendo murmur preceding the first sound in mitral stenosis is not due to auricular activity.” At irregular intervals there were thus revivals of the old view about the nature of the murmur, which aroused much criticism, for example from G. W. Balfour in 1872, T. D. Acland (1851-1931) in 1889, and from Sir F. Taylor (1847-1920) and Sir John Broadbent in 1909, and Sir T. Lewis in 1911. These battles long ago are now as if they had never been.

In 1866 Hayden (1823-1881) of Dublin recorded six cases with a presystolic murmur, three confirmed as regards mitral stenosis by necropsy, and stated that in mitral stenosis embarrassment of respiration and oedema are much less prominent than in mitral incompetence, and that the presystolic murmur is not transmitted to the left side of the lower dorsal spine. He also spoke of post-diastolic and post-ventricular murmurs. From cardiographic records Galabin (1843-1913) in 1875 concluded that two totally distinct murmurs may be caused by mitral obstruction: (1) the auricular-systolic and (2) a diastolic, due to the venous flow through the narrowed and roughened mitral orifice, which may be separated from the auricular-systolic murmur. James Andrew in 1877, while deprecating complicated expressions such as auricular-systolic and post-diastolic, admitted that as all direct mitral murmurs are diastolic, it was advisable to describe them in three groups according to the position of the murmurs in diastole. J. S. Bristowe (1827-1895) of St. Thomas's Hospital in 1887 followed this up by describing as the murmurs of mitral stenosis the three murmurs: (i) the early diastolic, taking the place of the second sound, thus resembling the diastolic murmur of aortic incompetence, audible on the left side of the sternum—this was later explained by pulmonary regurgitation caused by dilatation of that artery from increased venous pressure (see p. 3); (ii) the mid diastolic murmur, which may imitate a reduplicated sound, and has been ascribed to the suction action of the expanding left ventricle, a vis a fronte, as suggested by C. J. B. Williams (see p. 3); and (iii) the late diastolic or presystolic murmur.

Sir W. H. Broadbent (1835-1907) in 1886 described three stages of mitral stenosis: (a) with good compensation and a presystolic murmur and second sound audible at the apex; (b) the period of strained compensation in which the
second sound is absent at the apex and the first sound becomes short, usually very loud, and so may be erroneously regarded as the second sound and the presystolic murmur running up to it as systolic in time; (c) in the third stage, in which the compensation has completely broken down, the presystolic murmur has disappeared, probably as the result of tricuspid incompetence. It was more than twenty years later that the important factor of auricular fibrillation was established by Mackenzie and Lewis (see p. 6). Broadbent discussed the variations in the pulse from a regular rhythm in the early stage to extreme irregularity later, as Stokes had done, and described a moderately high blood pressure. He also commented on the frequency with which œdema was absent, and associated the presence of extreme œdema with the complication of tricuspid stenosis.

ASSOCIATED CONDITIONS

Tricuspid stenosis, when present, is nearly always associated with mitral stenosis which is in a more advanced stage. Isolated cases in this bilateral auriculo-ventricular stenosis were recorded by Morgagni (1761), Crüwell (1765), Corvisart (1806), Horn (1808), Allan Burns (1809), Laennec (1823), and Bertin (1824). Bedford Fenwick (1882) collected 70 such cases; Leudet (1888) found among 114 collected cases of tricuspid stenosis 103 with mitral stenosis, and Newton Pitt (1853–1929) among 109 cases of tricuspid stenosis from the records of Guy’s Hospital found that all but two showed mitral stenosis.

A ball thrombus in the left auricle in mitral stenosis was reported in 1814 by Wood in Edinburgh. Allan Burns in 1809 described an early stage of a loose ball-clot in mitral stenosis: “the left auricle contained a concretion larger than a pigeon’s egg, firmly adherent to the skin of the cavity, and composed of several portions forcibly pressed together,” and Robert Adams recorded a case in 1827. In 1909 Welch collected 19 cases associated with mitral stenosis, and it has been suggested that impaction of the ball-thrombus in the stenosed mitral may cause sudden death, but Welch’s analysis gave little support to this possibility. Cases of intra-auricular ball-thrombi and tumours without mitral stenosis may imitate ordinary mitral stenosis (Thompson and Aitchison).

Pulmonary apoplexies, described in 1819 by Laennec, appear to have been first correlated with mitral obstruction by J. A. Wilson (1795–1882), physician to St. George’s Hospital from 1829 to 1857, in a paper read before the Royal College of Physicians of London on the evening of March 22, 1830, but only reported briefly in the London Medical Gazette, in which three cases seen in a short period of six weeks were reported. This was confirmed in 1832 by Hope and about the same time by Sir Thomas Watson (1792–1882). In 1896 W. L. Dickinson (1862–1904) published in full his grandfather’s three cases, but on analysis of 70 necropsies showing the presence of pulmonary apoplexies was surprised to find that sixteen only were associated with mitral stenosis, most of the others undoubtedly showing mitral regurgitation. T. B. Peacock in 1867 argued that, as the pulmonary vessels underwent gradual dilatation, pulmonary apoplexies were not prone to occur; whereas Hyde Salter stated in 1869 that
hæmoptysis is more likely to occur in mitral obstruction than in other forms of cardiac disease.

Pressure exerted by a greatly dilated left auricle on the left bronchus and collapse of much of the left lung was recorded in 1889 by T. D. Acland, who referred to two such specimens in the Guy’s Hospital museum.

I am much indebted to Dr. Maurice Campbell for the following note. “In 1838, T. Wilkinson King (1809–1847), curator of the Museum and lecturer on morbid anatomy at Guy’s Hospital, published a paper ‘On a Morbid Flattening or Compression of the Left Bronchus produced by dilatation of the left auricle.’ He summarized his conclusions as follows:

“‘A particular morbid effect, which, as far as I am informed, has not been made known, and which, as I believe, is of rather common occurrence, is the flattening and obstruction of the left bronchus, when the left auricle is dilated so as to press upon this air-tube. Our Collection affords three specimens of this affection; and I think I may say I have remarked it many times, in different degrees. I propose to make a brief reference to the most remarkable instances of those which I have recorded; as well as to a few concurrent circumstances, which may explain what is necessary to the production of this compression, as well as what should be looked for in connexion with it. ‘‘I have been able correctly to anticipate the existence of this change, by considering the state of the heart; but I have hitherto perceived nothing distinctive in the respiratory sounds, and, indeed, should not expect to do so.’

The cases, specimens of which are still in the Guy’s Museum and show the compression of the left bronchus very clearly, were (1) a boy of 15 with rheumatic heart disease and mitral stenosis, who died with congestive failure with pleural and pericardial effusions, (2) a man of 28 with mitral stenosis and an adherent pericardium, and (3) a woman of 21 with mitral stenosis and a slighter degree of tricuspid stenosis, who died with general congestive failure. In the first two cases the great dilatation of the left auricle was emphasized; in the third case there was much dilatation of both auricles, especially the left. A fourth case was mentioned in which the left bronchus was compressed, probably from a dilated right auricle associated with a patent foramen ovale.”

Paralysis of the left recurrent laryngeal nerve, causing cough and aphonia, and associated with mitral stenosis, was described in two patients in 1897 by Ortner (1865– ). In 1920 Garland and White collected 61 cases of dilatation of the left auricle, and agreed with Fetterolf and Norris that such dilatation alone did not explain the compression of the nerve and that the left pulmonary artery played a part in the process. Enormous dilatation of the left auricle may occur without mitral stenosis (see Ewart and Owen).

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