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HISTORICAL NOTES ON THE MECHANISM OF CLOSURE OF THE ATRIOVENTRICULAR VALVES

by

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"The exact mechanism for closure of the mitral valve is unknown." This was stated in a textbook on *The heart*, published in 1978.[1] If this problem has not yet been clarified it is not through lack of effort, the work of many authors having produced various ideas since Galen.

Galen's clear appreciation of the function of the heart valves is evident from the following passage: "The common purpose of them all [the valves] is to prevent things from moving backwards again. But each kind has its own special purposes. In the cases of those which direct things out of the heart it is to prevent backflow into that organ; in the cases of those which direct things into the heart, it is to prevent any outflow from it . . . As there are altogether four openings, two in each ventricle, one of each pair leads in and the other out."[2]

The function of the cardiac valves to allow the flow of blood in only one direction while preventing movement in the opposite one was one of the features on which Harvey,[3] who quoted the above passage of Galen, based his concept of the circulation of the blood.

In the introduction of his *De motu cordis* he wrote: "If the three tricuspid valves at the entry into the right ventricle hinder return of blood into the vena cava, and if the three semilunar ones at the opening into the artery-like vein [pulmonary artery] have been made to hinder return of blood; how, when a similar arrangement holds in the left ventricle, can we say that these valves have not been made similarly to hinder forward or backward movement of the blood?" And, associating himself with Galen, ". . . of the four kinds of valve present, two subserve entry and two exit of the blood".[4]

These quotations refer to all four valves of the heart. The present paper deals only with the atrioventricular (a-v) valves, i.e. those between atria and ventricles, and structures directly involved in their position and movements, viz. the papillary muscles and their tendinous connexions to the valves, the chordae tendineae.

It is proposed to preface the discussion of the mechanism of a-v valve closure by remarks on two closely related anatomical features, and then to treat the functional aspects by discussing (1) passive closure; (2) active closure; (3) combination of the two; (4) role of atrial contraction; (5) some additional aspects.

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[1] J. W. Hurst *et al.* (editors), *The heart*, New York, McGraw-Hill, 1978, p. 1013.

[3] Galen, *De usu partium*, Lib. 6, c. 11, translated by K. J. Franklin, in William Harvey, *Movement of the heart and blood in animals*, Oxford, Blackwell Scientific Publications, 1957, p. 54.

[3] William Harvey, *Exercitatio anatomica de motu cordis et sanguinis in animalibus*, Frankfurt, William Fitzer, 1628, translated by K. J. Franklin, op. cit., note 2, above, pp. 15-16.

[4] Ibid., p. 55.

PREFATORY ANATOMICAL REMARKS

Two anatomical features seem particularly relevant for a discussion of a-v valve closure: the question of the presence of muscular fibres in a-v valves of the human heart, and the classification of the chordae tendineae. Both seem to have been first reported by Kürschner,[5] Lecturer of Medicine at the University of Marburg.

Kürschner described, as "an observation diametrically opposed to the general opinion", muscular fibres in the a-v valves of the human heart, originating from and contiguous with the musculature of the atria. In his opinion, their discovery was of paramount importance for the explanation of the mechanism of valve closure. One year before, Reid,[6] Physician at the Royal Infirmary at Edinburgh, had reported muscular fibres in a-v valves of oxen and horses but stated explicitly that he could never observe them in the human heart and warned not to mistake tendinous fibres when tinged with blood for muscular fibres. Palicki,[7] Purkyně's student who first described the "Purkyně fibres", held the same view. This observation gave rise to lively controversies.

Baumgarten,[8,9] the first to emphasize the importance of atrial contraction for a-v-valve closure (discussed below), denied the presence of muscle fibres in the human heart; he traced only a few fine strands as an inconstant finding in the posterior cusp of the tricuspid. Donders,[10] a leading physiologist and pioneer in physiological optics, and Valentin,[11] who held the Chair of Physiology in Berne for forty-five years, stated explicitly that no muscle fibres in human a-v valves could be found.

By contrast, the presence of these fibres was confirmed by Nega,[12] working under Purkyně and later Professor of Pharmacology at the University of Breslau (now Wroclaw). L. Joseph,[13] agreeing with Kürschner, termed them "Klappenmuskulatur" (valvular musculature). Gussenbauer,[14] Professor of Surgery at Liège, later Prague, devoted a paper to 'The musculature of the atrioventricular valves of the human heart', confirming and amplifying Kürschner's observations. His findings were accepted by Bernays.[15] The exaggerated view about the importance of these fibres held by

[5] [G.] Kürschner, 'Ueber die venösen Klappen des Herzens und ihre Action', *Froriep's Neue Notizen*, 1840, **15:** 113-122.

[6] John Reid, 'Heart', in *Todd's cyclopaedia of anatomy and physiology*, London, Longman, Brown, Green, Longman & Roberts, 1839, vol. 2, p. 589.

[7] B. Palicki, *De musculari cordis structura*, Vratislava, Friedlaender, 1839, pp. 14, 36; in J. E. Purkyně, *Sebrané Spisy, Opera omnia*, Prague, 1954, vol 6, pp. 10-12.

[8] A. Baumgarten, *Commentatio de mechanismo quo valvulae venosae cordis clauduntur*, Inaugural Dissertation, Marburg, 1843.

[9] A. Baumgarten, 'Ueber den Mechanismus, durch welchen die venösen Herzklappen geschlossen werden', *Arch. Anat. Physiol. wiss. Med.*, 1843, 463-470.

[10] F. C. Donders, *Physiologie des Menschen*, translated by F. W. Theile, Leipzig, Hirzel, 1856, p. 37.

[11] G. Valentin, *Versuch einer physiologischen Pathologie des Herzens und der Blutgefässe*, Leipzig and Heidelberg, Winter'sche Verlagshandlung, 1866, p. 278.

[12] V. J. Nega, *Beiträge zur Kenntnis der Funktion der Atrioventrikular-Klappen des Herzens, zur Entstehung der Töne und Geräusche in demselben und deren Deutung*, Breslau, Ferd. Hirt, 1852, p. 15.

[13] L. Joseph, 'Ueber die Ringe und Klappen des menschlichen Herzens', *Virchow's Arch. pathol. Anat. Physiol. klin. Med.*, 1858, **14:** 244-270, p. 248.

[14] C. Gussenbauer, 'Ueber die Musculatur der Atrioventricularklappen des Menschenherzens', *Sber. Akad. Wiss. Wien, mathemat.-naturwiss. Kl.*, 1868, **57,** I Abt.: 1103-1110.

[15] A. C. Bernays, 'Entwicklunsgeschichte der Atrioventricularklappen', *Morphologisches Jahrbuch*, Leipzig, Engelmann, 1876, vol 2, p. 498.

Paladino[16] is discussed below. Paladino's report of muscle fibres in the *ventricular* aspect of a-v valves, contiguous with muscle fibres of the ventricular walls appears to be one of the earliest, others being by Bernays[17] and Zuckerkandl.[18]

Between these extremes are reports by several authors describing muscle fibres in a-v valves but emphasising that they are only found in certain circumscribed areas and that they vary in extent. Thus Savory[19] wrote that these fibres terminate generally "by a tolerably well defined margin, but this varies . . .". Luschka,[20] accepting this view, regarded them as exceptional in the human heart. Henle[21] stressed the variability of their length, being greatest in the anterior cusp of the mitral valve, and according to Darier[22] extending from the attached border to about one-sixth of the diameter of the cusp. Albrecht[23] constantly found muscular fibres in a-v valves though only in areas where basal chordae tendineae entered into the valve, whereas Tandler[24] reported them as being confined to the basal third of the cusps.

In Gray's *Anatomy*, fibres derived from the atrial musculature in the basal third of each cusp of the tricuspid, and a few muscular fibres in the cusps of the mitral valve are mentioned.

Kürschner,[25] apparently also the first to describe in detail and classify the chordae tendineae, divided them into three groups. (In 1783 Senac[26] had briefly referred to a division of the "tendons" into a superior and inferior layer, the former inserting in the margin, the latter spreading along the inferior surface of the valve.) The first or primary group, originating from the papillae, insert in muscular tissue of several "lines" width (1 line = 2.5mm) of the *limbus cordis* (anulus fibrosus). Two chordae, one of them originating from each papillary muscle, are attached to each adjacent cusp. The second group originates frequently from the primary chordae at different levels, inserting at the sides of the central portions of the valve. The third group, becoming visible on unfolding the fringes of the valve, branch off from the second group, dispersing in the fringes.

Subsequently, considerable confusion arose about the division into these three groups, since later writers proposed different classifications. Henle[27] emphasized the difference between his and Kürschner's numbering of the three groups. Confusion

[16] G. Paladino, *Contribuzione all'anatomia, istologia e fisiologia del cuore*, Naples, 1876. Quoted from *Jahresber. Fortschr. Anat. Physiol.*, Leipzig, Vogel, 1878, **5**, II, p. 47.

[17] Bernays, op. cit., note 15 above.

[18] E. Zuckerkandl, 'Anatomische Beiträge. II. Ueber Muskelgewebe in der Kammerfläche der Valvula tricuspidalis', *Allg. Wien. med. Zt.*, 1877, 22 Jahrg., pp. 155-156.

[19] W. Savory, 'Observations on the structure and connection of the valves of the human heart', *Lond. Edinb. Dublin philosoph. Mag. J. Sci.*, 1852, **3**, 4th series: 304-309, p. 308.

[20] H. Luschka, *Die Anatomie des Menschen in Rücksicht auf die Bedürfnisse der praktischen Heilkunde*, Tübingen, Laupp'sche Buchhandlung, vol. 1, Abt. II, pp. 386, 387.

[21] J. Henle, *Handbuch der systematischen Anatomie des Menschen*, Braunschweig, Vieweg, 1868, vol. 3, Abt. 1, pp. 23, 24.

[22] J. Darier, 'Les vaisseaux des valvules du coeur chez l'homme à l'état normal et à l'état pathologique', *Arch. physiol. normale pathol.* 1888, IV série, **2**: 35-59, 151-180, p. 55.

[23] E. Albrecht, *Der Herzmuskel und seine Bedeutung für Physiologie, Pathologie und Klinik des Herzens*, Berlin, Springer, 1903, pp. 67-69.

[24] J. Tandler, *Anatomie des Herzens*, Jena, Fischer, 1913, pp. 97-101.

[25] Kürschner, op. cit., note 5 above, p. 115.

[26] J. B. Senac, *Traité de la structure du coeur, de son action et de ses maladies*, Paris, Méquignon, 2nd ed., 1783, vol. 1, p. 386.

[27] Henle, op. cit., note 21 above, pp. 23-24 footnote.

became compounded when some authors accepted in principle Henle's classification, e.g. Tandler,[28] Benninghoff,[29] whereas others used Kürschner's, e.g. Sée,[30] Albrecht[31] who emphasized that the division is also justified on physiological grounds, Rauber-Kopsch.[32]

MECHANISM OF A-V VALVE CLOSURE

One of the main controversies regarding the mechanism of a-v valve closure centred around the question whether this was a passive phenomenon, due only to the pressure of blood in the ventricles during ventricular systole, or an active process due to the activity of muscles other than ventricular myocardium, or a combination of the two.

1. *Passive closure*

Passive closure was assumed by Lower:[33] "These membranes [cusps of the tricuspid valve] rise from the edge of the ventricle, and completely encircle the entrance into it. Thus, when the apex of the heart is drawn nearer the base in each Systole, the papillae also move upwards and slacken their fibres to very low reins; the membranes to which they are attached follow suit, and, hanging loose, are driven upwards like bellying sails by the expulsion of blood at each systole of the Heart." In this way the opening is firmly closed. Lower realized that "not all the membranes in the right ventricle receive their fibres directly from the papillae" – foreshadowing the later classification of the chordae – but nevertheless they were able to check the backflow of blood. In diastole "the apex goes back again and draws down with it the papillae and their fibres. Hence the membranes are likewise withdrawn, and uncover at once the entrance into the heart, opening the doors, as it were, to the inflow of blood from the auricle." A similar statement can be found in Galen: "When the heart dilates, these ligaments [i.e. chordae tendineae] are tensed by its expansion and draw the membranes in their direction, reflecting them, so to speak, toward the body of the heart itself. When all these membranes are reflected upon the heart in a circle, the mouths of the vessels are thrown open and the heart easily draws in through the broad passage the material contained in them."[34] Regarding the mechanism of closure, Lower stressed the importance of the "situation and structure of the papillae". They ensure "that the membranes shall always be separated by some interval from the sides of the ventricle, and that they shall thus easily be raised by the first thrust of the blood which is driven upwards at each systole". This separation is made possible by the location of the papillae, which are "situated on the side opposite to the membrane, to which they are attached", and by the "fleshy columns" (i.e. trabeculae carneae), whose sole function is to keep the membranes at an adequate distance from the ventricular wall. The chordae tendineae

[28] Tandler, op. cit., note 24 above, p. 89.

[29] A. Benninghoff, 'Blutgefässe und Herz', in Wilhelm von Möllendorff (editor), *Handbuch der mikroskopischen Anatomie des Menschen*, Berlin, Springer, 1930, vol. 6, part 1, p. 181.

[30] Marc Sée, *Recherches sur l'anatomie et la physiologie du coeur, spécialement au point de vue du fonctionnement des valvules auriculo-ventriculaires*, Paris, Masson, 1875, pp. 48, 49.

[31] Albrecht, op. cit., note 23 above, pp. 77-80.

[32] August Rauber and F. W. T. Kopsch, *Lehrbuch und Atlas der Anatomie des Menschen*, 10th ed., Leipzig, Thieme, 1914, vol. 3, p. 248.

[33] R. Lower, *Tractatus de corde*, London, Allestry, 1669, pp. 41-45, translated by K. J. Franklin in, R. T. Gunther (editor), *Early science in Oxford*, 15 vols., Oxford, [for the subscribers], 1923-67, vol. 9 (1932).

[34] Galen, *De usu partium*, L.6, c.14, translated by M. Tallmadge May, Ithaca, New York, Cornell University Press, 1968, vol 1, p. 313.

allow the free passage of blood to the space behind the cusps, and Lower regarded the presence of blood in this space as an important factor in causing the movement of the valve toward the a-v orifice and its closure at the onset of ventricular systole, clearly recognizing the raising of the cusps during diastole: " . . . one must believe that the blood thrown into the chambers of the Heart at each diastole penetrates back between those membranes and the walls of the ventricle, and that the membranes themselves rise upwards and puff out more as the ventricular cavity becomes fuller. . . . For the blood which is thrown into the Heart and received among the fibres [of the chordae] soon fills the ventricle, and simultaneously, by its raising the membranes, prevents its own escape through them. Hence, when the Heart contracts, it must of necessity expel the blood through the open channel into the aorta." Lower also mentioned "the possibility of their [trabeculae carneae] sifting and mixing the blood".

As is evident from the first of the above quotations, Lower believed that during ventricular systole the chordae tendineae are slack, allowing the valves to be pushed upwards by the blood. While therefore he did not assume a contraction of the papillary muscles and stretching of the chordae thereby, he recognized that the valves " . . . should unfurl just as far as the relaxation of their attachments permits. These attachments nature has allowed to move far enough for the membranes to stretch out on all sides and completely to close the entrance into the ventricles." Regarding the part played by the chordae, the emphasis here is only on their allowing the cusps to move far enough to close the orifice.

The recognition of the role of the chordae to prevent excessive bulging into the atria was stressed by Perrault,[35] member of the Académie des Sciences and distinguished by his work in comparative anatomy, who also held the view of passive closure of the a-v valves. He vividly illustrated the restraining role of the chordae by comparing the valves to lock-gates:

"Now because these valves are not of a rigid material, as are the gates which resist the impulse of the water when they are joined to each other, Nature has given them another means to resist the impulses of the blood; and this comes about by means of a large number of ligaments, which are like many small cords attached to two margins of each valve, just as lock-gates are held back by chains: for, these ligaments prevent the membranes forming the main part of the valves from being pushed further forward when the blood has brought them into close proximity: for, should this occur, they could not prevent the blood from passing through and returning from whence it came. This kind of valve exists in the heart at the end of the vessels which bring the blood into each ventricle, and the arteria venosa [pulmonary vein], which is really a vein, and which returns to the left ventricle the blood which the arterial vein [pulmonary artery] has poured into the lung. The vena cava has three of these valves [cusps]; but the arteria venosa [pulmonary vein] has only two, because it does not return to the left ventricle as much blood as the cava returns to the right, since part of the blood, which the cava returns to the heart and which the vena arteriosa [pulmonary artery] distributes in the lung, is consumed for the nourishment of this part, which dissipates much of it."

[35] C. Perrault, *Essais de physique ou recueil de plusieurs traitez touchant les choses naturelles*, Paris, Coignard, 1680, pp. 258-260.

Vieussens,[36] Fellow of the Royal Society and member of the Académie des Sciences, published in the year of his death a treatise on the heart described by Major[37] as "a classic of cardiology". In it, Vieussens expressed similar views regarding passive closure and function of the chordae. He also thought that the connexion and position of the chordae prevented undue separation of the walls of the ventricles at the time they receive the blood in the cavities.

At a meeting of the Académie Royale des Sciences in 1731, where the question was discussed whether the heart shortens or lengthens during ventricular systole, Bassuel,[38] a surgeon from Paris, declared that the heart shortens, basing this opinion on the movements of the a-v valves about which he held similar views to Lower. He therefore believed that "the moment of systole is that where the chordae tendineae are relaxed" but, contrary to Lower, he thought that during ventricular diastole the valves are applied to the ventricular walls. Both propositions became controversial and finally were found to be wrong.

It is of interest that in that same meeting of 1731 systole was regarded "as a state somehow forced by the intervention of an extraneous cause, such as would be the Animal Spirits", diastole being the heart's natural state.

Passive closure was assumed by some of the leading physicians of the eighteenth and nineteenth centuries. They include: Winslow,[39] one of Albrecht von Haller's teachers; Senac,[40] Louis XV's physician, whose view was quoted by Haller,[41] and who believed that the cusps are applied to the ventricular walls when lowered during diastole; Bichat[42] also assumed passive closure but regarded narrowing of the a-v orifice as another important factor.

Sömmerring,[43] well known for his fine anatomical illustrations, described a similar mechanism of the closure of the tricuspid valve. He thought "that the tricuspid valve would probably (*"vielleicht"*) be rolled back into the right atrium unless the papillary muscles held back its border and by their contraction, which occurs at the same time as that of the heart, fixed it in such a way that, though tensed, it is not damaged." It is noteworthy that Sömmerring realized that the papillary muscles contracted synchronously with the rest of the ventricular myocardium but apparently was not quite sure about their part in restraining the valve from being thrust into the atrium.

Another aspect of the function of the trabeculae carneae (probably including the papillary muscles) was emphasized by Magendie.[44] He was "tempted to believe as did Boerhaave" that, apart from their function in valve closure, the trabeculae carneae of

[36] R. Vieussens, *Traité nouveau de la structure et des causes du mouvement naturel du coeur*, Toulouse, Guillematte, 1715, pp. 99, 102.

[37] R. H. Major, *A history of medicine*, Springfield, Ill., Thomas, 1954, vol. 1, p. 539.

[38] P. Bassuel, 'Sur le changement de figure du coeur dans la sistole', in *Histoire de l'Académie Royale des Sciences Année 1731*, Paris, Imprimerie Royale, 1733, pp. 24, 27-29.

[39] J. B. Winslow, *An anatomical exposition of the structure of the human body*, translated by G. Douglas, London, Prevost, 1733, vol. 2, Sec. IX, pp. 13, 14.

[40] J. B. Senac, *Traité de la structure du coeur, de son action et de ses maladies*, Paris, Briasson, 1749, p. 344.

[41] A. von Haller, *Elementa physiologiae corporis humani*, Lausanne, Marei-Michael, Bousquet *et al.*, 1757, vol. 1, p. 405.

[42] M. F. X. Bichat, *Traité d'anatomie descriptive*, Paris, Brosson & Gabon, 1803, vol. 4, pp. 116-121.

[43] S. T. Sömmerring, *Vom Baue des menschlichen Körpers*, Frankfurt, Varrentrapp & Wenner, 1792, Vierter Theil, p. 19.

[44] F. Magendie, *Précis élémentaire de physiologie*, Paris, Méquignon-Marvis, 1817, vol. 2, pp. 249-250.

the right ventricle also play a large part in the mixing of diverse elements of the blood.* Magendie believed that this intimate mixing is highly necessary in view of the great tendency of its constituents to separate. His view is thus a development of an idea put forward by Lower, referred to above.

An unusual view of the function of the papillary muscles was put forward by Oesterreicher.[45] After recalling two opposite opinions about the behaviour of these muscles – Haller[46] claiming to have seen the chordae tendineae relaxed during ventricular systole as the papillary muscles get nearer to the a-v orifice, and by contrast Burdach[47] stating that the contraction of the papillary muscles during systole draws the valves inwards – Oesterreicher adduced reasons for his view that during systole the a-v valves are almost as slack as during diastole. Since the effects of the contraction of the papillary muscles and of all the other fibres of the heart, contracting at the same time, cancel each other, the result of the contraction of the papillary muscles is " . . . only to reduce the slackening of the valves sufficiently so that the pressure of the blood does not thrust them into the atria". Burdach[48] criticized Oesterreicher's views.

No one could have been more firmly convinced about passive closure than David Williams. In his own words: "That the auricular valve is closed solely by the impulse of the blood cannot be doubted; but with every deference, I certainly must differ in opinion with respect to the power that opens it. It is then by the contraction of the musculi papillares that I am disposed to think the auriculo-ventricular valves to be mainly opened . . .".[49] This view about the opening mechanism is based on the unlikely proposition that the papillary muscles contract at the beginning of ventricular diastole, namely, at the moment the ventricles cease to contract.

According to E. H. Weber,[50] the co-discoverer of the cardio-inhibitory effect of the vagus, "a space necessarily remains between the ventricular walls and the fold" (i.e. cusps). He emphasized the part played by the blood in this space for effecting the closure of the a-v orifice, and described the function of the papillary muscles thus: "By these trabeculae carneae [*"Fleischkegel"*, by which term he obviously means the papillary muscles] is ensured that the chordae are always adequately stretched also during contraction of the heart; for, by the same amount by which the walls of the ventricles approach the atrial orifice during their contraction, the apex of every trabecula, connected to the chordae of the cusp, appears at the same time to recede from it by the contraction of the trabecula."

At their sixth meeting, the British Association for the Advancement of Science[51]

* Magendie does not give any reference to Boerhaave's publication; a statement to this effect is in Boerhaave's *Opera omnia medica*, 1766, p. 29, 162.

[45] J. Oesterreicher, *Versuch einer Darstellung der Lehre vom Kreislaufe des Blutes*, Nürnberg, J. L. Schrag, 1826, pp. 20-21.

[46] Haller, op. cit., note 41 above, p. 390.

[47] K. F. Burdach, *Berichte von der königlichen anatomischen Anstalt zu Königsberg. Dritter Bericht. Mit Bemerkungen über den Mechanismus der Herzklappen*, Leipzig, Dyksche Buchhandlung, 1820, p. 45.

[48] K. F. Burdach, *Die Physiologie als Erfahrungswissenschaft*, Leipzig, Leopold Voss, 1832, vol. 4, p. 210.

[49] David Williams, 'On the sounds produced by the action of the heart', *Edinb. med. surg. J.*, 1829, **32**: 297-305, pp. 302-303.

[50] E. H. Weber, in *Friedrich Hildebrandt's Handbuch der Anatomie des Menschen*, Braunschweig, Schulbuchhandlung, 1831, vol. 3, pp. 136-137.

[51] British Association for the Advancement of Science, *Report of the sixth Meeting held at Bristol in August 1836*, London, John Murray, 1837, pp. 275-283.

discussed the question "What is the precise mode in which the tricuspid and mitral valves prevent the reflux of blood? Are they floated up and stretched across the auriculo-ventricular orifices, or drawn together to a point within the cavity of the ventricle by the action of the columnae carneae?" The decision went in favour of the former, i.e. passive closure, and the papillary muscles were assigned the function which is now generally accepted. The view, expressed at that meeting, that during ventricular diastole the a-v valves are separated from the ventricular walls by a space, was shared by Valentin,[52] who stressed the importance of the blood in this location for the closure of the orifices at the very *beginning* of systole.

An idiosyncratic terminology led Spring[53] to somewhat strange views. He distinguished three phases of heart action: ventricular dilatation, ventricular contraction, rest. A forceful ventricular dilatation corresponds to the beginning of systole: " . . . the dilatation is for us precisely a systolic phenomenon in the ordinary sense of the word".[54] This phase he termed "Presystole", which also indicated contraction of the atria. The term "Presystole" was first used by Gendrin.[55] Fauvel,[56] referring to Gendrin, borrowed this term "though giving it a different semeiotic meaning", being the first to use it to describe the presystolic murmur of mitral stenosis.

During presystole the papillary muscles contract and lower the a-v valves abruptly, with strong tension of valves and chordae tendineae.[57] Systole follows presystole without interruption and consists of ventricular contraction. Closure of the a-v orifice is due to the pressure of blood, i.e. passive closure, the papillary muscles not playing any part and being relaxed during systole. The chordae tendineae, particularly of the second and third group (Kürschner's classification), restrain the valve. In order to do this it is not necessary that the papillary muscles and the chordae contract, since their elasticity suffices.[58] The valves, raised by the pressure of the blood, close the a-v orifice hermetically and remain in this position during diastole, diastole being synonymous with rest. During diastole the ventricles resemble an inert pocket, without any movement whatever, arterial and venous orifices being closed.[59]

It seems that what Spring calls diastole is the isometric relaxation phase, and what he terms presystole as part of the systolic phase is the presystolic phase of diastole during which he assumes contraction of the papillary muscles. By regarding atrial contraction and initial ventricular dilatation as part of ventricular systole, Spring claims to have overcome the difficulty experienced by some authors in believing the contraction of the papillary muscles to take place in diastole, i.e. at a time different from that of the contraction of all other ventricular fibres[60] (e.g. David Williams,[61] Laennec[62]).

[52] G. Valentin, *Lehrbuch der Physiologie des Menschen*, Braunschweig, Vieweg, 1847, vol. 1, pp. 425-426.
[53] A. M. Spring, 'Mémoire sur les mouvements de coeur, spécialement sur le mécanisme des valvules auriculo-ventriculaires', *Mém. Acad. Roy. Sci., Lettres Beaux-arts de Belgique*, 1861, **33**: 3-140, p. 46.
[54] Ibid., p. 71.
[55] M. A.-N. Gendrin, *Leçons sur les maladies du coeur et des grosses artères*, Paris, Baillière, 1841, vol. 1, pp. 31-32.
[56] A. Fauvel, 'Mémoire sur les signes stéthoscopiques du rétrécissement de l'orifice auriculo-ventriculaire gauche du coeur', *Archs gén. Méd.*, 4e série, 1843, **1**: 1-16, p. 16.
[57] Spring, op. cit., note 53 above, p. 118.
[58] Ibid., pp. 116, 118. [59] Ibid., pp. 49, 50. [60] Ibid., p. 116.
[61] Williams, op. cit., note 49 above.
[62] R.-T.-H. Laennec, *Traité de l'auscultation médiate et des maladies des poumons et du coeur*, 2nd ed., Paris, Chaudé, 1826, vol. 2, p. 419 footnote.

In the same year Austin Flint jr.[63] stressed that the a-v valves are closed by backward pressure during ventricular contraction and not by the current of blood from auricles to ventricles. The last point is at variance with the explanation of Baumgarten, who emphasized the importance of atrial contraction in the mechanism of a-v valve closure (discussed below). According to Wundt,[64] these valves begin to open at the end of ventricular systole, at a time when atrial exceeds ventricular pressure; moreover, at this stage of the cardiac cycle the pull of the still contracted papillary muscles is no longer opposed by the pressure of blood in the ventricles.

A modified view of essentially passive closure was put forward by Markham,[65] discussed below.

In the mitral valve " . . . the two small tongues placed between the two large ones play a noticeable part in the closure and, therefore, the anatomists have been wrong to overlook them ever since fashion has suppressed them in the mitres of bishops", thus Colin,[66] another adherent to passive closure. Colin's view that during diastole the valves are separated from the ventricular walls was rejected by Surmay,[67] his reasons being unconvincing.

2. *Active closure*

The first to suggest closure of the a-v valves by the activity of muscles, namely, the papillary muscles, seems to have been J. F. Meckel jr.: "Inasmuch as they [the papillary muscles] shorten at the contractions of the heart, the several portions of the valves are drawn into the cavity of the heart toward each other and in this way the orifice is firmly closed. This attachment of the valve at its otherwise free border was necessary, because it has to resist not only the weight of the blood, as the other valves, but also the action of the muscular walls of the heart, which presses the blood against it [the valve]."[68]

Mayo,[69] best known for his work on the physiology of the nervous system, held similar views. He found that if, in an opened heart, one pulls on the fleshy columns "in a proper direction", one can imitate the natural action, and the valve will close. In a later edition he also drew attention to the following two features: each fleshy column receives all the chordae tendineae of the adjacent sides of the points of the valves, and the tightening of the cords "is capable of bringing the opposite parts of the edge of the valve into contact". Furthermore, in order to close the orifice, the chordae must be drawn not only from the base of the heart toward the apex but also outwards.

The latter statement is the reverse of the opinion of others (see below), the former contrasts with that of Bouillaud.

Bouillaud[70] believed that the contraction of the papillary muscles during ventricular

[63] Austin Flint jr., 'Experimental researches on points connected with the action of the heart and with respiration', *Amer. J. med. Sci.*, new series, 1861, **42**: 341-381, p. 379.

[64] W. Wundt, *Lehrbuch der Physiologie des Menschen*, 3rd ed., Leipzig, Enke, 1873, p. 298.

[65] W. O. Markham, 'Remarks on the cause of the closure of the valves of the heart', *Br. med. J.*, 1861, **i**: 313-314.

[66] [-] Colin, in 'Discussion sur le coeur', *Bull. Acad. Méd.*, 1874, **38**: 348-350.

[67] [-] Surmay, 'De l'occlusion des orifices auriculo-ventriculaires', *J. Anat. Physiol.*, 1876, 458-485, pp. 467-471.

[68] J. F. Meckel jr., *Handbuch der menschlichen Anatomie*, Halle and Berlin, Buchhandlung des Halleschen Waisenhauses, 1817, vol. 3, pp. 22-23.

[69] H. Mayo, *Outlines of human physiology*, London, Burgess & Hill, 1829, pp. 66-68; 4th ed., London, Renshaw, 1837, pp. 42-44.

[70] J. B. Bouillaud, *Traité clinique des maladies du coeur*, Paris, Baillière, 1835, vol. 1, pp. 17-18.

systole raises and straightens the lowered cusps, since they draw them everywhere from the circumference to the centre. He therefore termed them *"muscles tenseurs, éleveurs ou adducteurs"* of the mitral valve. By this mechanism the mitral valve is hermetically closed. During this stage "the whole of the left or atrial half of the left ventricle [its inflow tract] is almost completely obliterated, whereas the other half [outflow tract] propels into the aorta the blood which it had received from the left atrium." Similar conditions prevail in the right heart. A similar view of active closure by the papillary muscles was put forward by Surmay.[71]

Parchappe,[72] psychiatrist, and founder and president of the Société médico-psychologique but also author of a book on the heart arousing a great deal of interest at the time, put forward the view that the a-v valves do not function as valves in the mechanical sense of the term, as most writers assumed. He compared the passive elements of the a-v valve closing mechanism (the valve ring and chordae tendineae) to a purse fitted with strings, and the active elements (papillary muscles) to a system of synergistic muscles. During ventricular diastole the papillary muscles are separated from each other; the a-v orifice is kept open, and the cusps are pressed against the inflow tract of the ventricular wall by the inflowing blood. During ventricular systole the papillary muscles approach each other closely, forming one single pillar and, by pulling at the circumference of the valve ring toward the midline, draw the cusps toward each other, their contact becoming even closer by the wrinkling and enmeshing of their borders, and the simultaneous narrowing of the valve ring. In this way the a-v orifice is firmly closed.

With few unimportant modifications Parchappe's ideas were accepted by several authors, e.g. Fossion,[73] Professor of Human and Comparative Physiology at Liège; Bérard,[74] Professor of Physiology at Paris; and Küss.[75] Ludwig,[76] however, had reservations, writing that the constant relations of the valves to the papillary muscles ". . . are treated only by Parchappe, with some though inadequate thoroughness".

Chauveau, Professor at the Medical Faculty at Lyons, and Faivre,[77] the first to estimate accurately the blood pressure in man by direct readings of the intra-arterial pressure, largely though not entirely rejected Parchappe's thesis, calling it *"un ingénieux roman"*. They found in horses that, by introducing a finger into the right atrium, "at the very moment when the ventricles start to contract, the tricuspid cusps

[71] [-] Surmay,'Recherches sur les mouvements et les bruits normaux du coeur pour arriver au diagnostic des bruits anormaux qui se passent aux orifices de cet organe', *Gaz. méd. Paris*, 1852, XXII Année, III[e] Séries, **7**: 653-656, 761-768, p. 767.

[72] Maximilien Parchappe, *Du coeur, de sa structure et de ses mouvements, ou traité anatomique, physiologique et pathologique des mouvements du coeur de l'homme*, Paris, Masson, 1848, preface ii, iv, pp. 21-24, 35, 36.

[73] N.-G. Fossion, 'Recherches sur les mouvements du coeur', *Mém. Acad. Roy. Méd. Belgique*, 1849, **2**: 590-630, p. 609.

[74] P. Bérard, *Cours de physiologie*, Paris, Labé, 1851, vol. 3, pp. 637-643.

[75] Emile Küss, *A course of lectures on physiology*, translated by R. Amory, Boston, Campbell; London, Baillière Tindall & Cox, 1876, p. 134.

[76] C. Ludwig, 'Ueber den Bau und die Bewegungen der Herzventrikel', *Zt. rat. Med.* 1849, **7**: 189-220, p. 200.

[77] A. Chauveau and J. Faivre, 'Nouvelles recherches expérimentales sur les mouvements et les bruits du coeur, envisagés au point de vue de la physiologie médicale', *Gaz. méd. Paris*, 1856, **11**: 365-367, 406-411, 457-459, 569-573, p. 410.

straighten, join edge-to-edge with their borders, and are stretched so much that they become convex upwards, forming a multiconcave dome above the ventricular cavity . . . . Nevertheless we are in agreement with M. Parchappe about the contraction of the auriculo-ventricular orifice, but within far narrower limits: for us, this orifice contracts sufficiently to allow the marginal edge-to-edge joining of the cusps, but not more. Their joining is, however, as perfect as possible."

Surmay[78] pointed out that the valves in close proximty to the a-v orifice and under tension have a ventricular surface consisting of two planes at an angle to each other, the bloodstream being guided along them in such a direction that "the arterial orifices appear to be a continuation of these planes".[79] Summing up, he wrote: "The auriculo-ventricular valves are thus for the auriculo-ventricular orifices instruments of occlusion, for the bloodstream instruments of transmission and direction, for the auricular cavities instruments of protection, and these instruments are put into action by the contraction of the valvular [papillary] muscles". Much of the last part of Surmay's paper[80] is taken up by an acrimonious controversy about priority with Sée, another protagonist of active closure. Parts of it are on record in letters to the Académie de Médecine and *Archives générales de Médecine*, reproduced by Surmay.

Sée,[81] at the time surgeon at the Maison municipale de Santé at Paris, developed the following ideas about the closure mechanism: Contraction of the papillary muscles, coinciding with that of the ventricular walls, produces tension of the chordae and lowering of the cusps. During left ventricular systole the contracting papillary muscles pull both cusps of the mitral valve to the left, applying them to the ventricular wall, whereby diminution of the transverse diameter of the heart plays an important part. In the neighbourhood of the a-v orifice the two cusps separate "like the two faces of a coin", leaving a triangular space between them through which blood could flow back into the atrium unless this was prevented by two accessory small tongues *("petites languettes")*. The larger cusp – the anterior having by far the more important role – as well as the accessory small "tongues" could be more or less raised by the blood and the multiconcave dome of Chauveau and Faivre, discussed above, could be produced in this way. Obliteration of the right a-v orifice is assumed to be effected by a similar pulling mechanism of the anterior and posterior cusps of the tricuspid valve. "Thus we see that on the right as on the left the muscular action of the columns have the essential if not the only role in the play of the auriculo-ventricular valves."

Moritz,[82] Professor of Medicine at the University of Cologne, who criticized Sée's work on technical grounds, included his entirely speculative views among the old theories which could definitely be abandoned.

3. *Combination of active and passive closure*

The majority of authors emphasizing the part played by muscles in the closure of the a-v valves admit that the pressure of the blood in the ventricular cavities is also an important factor.

[78] Surmay, op. cit., note 67 above, pp. 458-462.
[79] Ibid., pp. 464-465.
[80] Ibid., pp. 480-485.
[81] Sée, op. cit., note 30 above, pp. 55, 61, 63.
[82] F. Moritz, 'Physiologie und Pathologie der Herzklappen', in *Handbuch der normalen und pathologischen Physiologie*, Berlin, Springer, 1928, 7/1, p. 181.

Thus Burdach,[83] after stating that during diastole the a-v valves were pressed against the ventricular wall by the inflowing blood, went on: "During systole the papillary muscles . . . draw the valve inward, or into the shape of a funnel, but in such a way that at the apex of the funnel gaps remain between the attachments of the chordae. Since, however, owing to the movement of the valve produced by the papillary muscles, blood passes between the chordae to the outer surface of the valve, it presses inward and upward the slack lower cusps of the valve between the attachments of the chordae, in this way completing the closure which had been initiated by the papillary muscles." Burdach objected to the view, held by Lower,[84] Bassuel,[85] and Haller,[86] that the chordae become relaxed when, during systole, the apex is drawn towards the base, since this would imply that the chordae are stretched during diastole when the papillary muscles are relaxed, which he rightly regards as unlikely. Burdach also rejected the opinion that the only function of the papillary muscles was to prevent the cusps from being thrust into the venous sacks *("Venensäcke")*. Criticizing Oesterreicher's arguments (see above), Burdach[87] pointed out that, if this were the case, this could be achieved solely by the chordae and there would be no need for papillary muscles.

It is noteworthy that, according to Burdach, the papillary muscles draw the cusps inward, this being the reverse of Mayo's opinion referred to above, but in accordance with Reid and others, discussed below.

In contrast to David Williams's[88] opinion, Allen Thomson,[89] Fellow of the Royal Society and holder of the Chair of Anatomy in the University of Glasgow for twenty-nine years, subscribed to the view now generally held about the opening mechanism of the a-v orifice and the synchronicity of papillary muscle contraction with ventricular systole. He concluded ". . . that, while the tendons serve to fix the valves, the action of the columnae carneae is to draw them down, so as to allow the blood to pass behind them, and to press them together and close them in the same manner as the semilunar valves of the aorta and pulmonary artery are shut." The views of Reid[90] are practically identical, except that he thought that the papillary muscles set the valve in motion. He believed, however, that only at the commencement of systole were the chordae sufficiently tense to move the valves; as the contraction proceeds, the capacity of the heart diminishes so much that the chordae become relaxed.

Another notable adherent to the theory of a combined active and passive closure mechanism was Skoda,[91] who also reported the existence of pockets in the slack portions of the a-v valves. He thought that they were produced by the particular type of insertion of the chordae tendineae. Their existence was denied by Kürschner.[92]

[83] Burdach, op. cit., note 47 above, pp. 22, 45.
[84] Lower, op. cit., note 33 above.
[85] Bassuel, op. cit., note 38 above.
[86] Haller, op. cit., note 41 above, p. 390.
[87] Burdach, op. cit., note 48 above, p. 210.
[88] Williams, op. cit., note 49 above.
[89] A. Thomson, in *Todd's cyclopaedia of anatomy and physiology*, London, Longman, Brown, Green, Longman & Roberts, 1835-36, vol. 1, p. 656.
[90] Reid, op. cit., note 6 above.
[91] J. Skoda, *Abhandlung Über Perkussion und Auskultation*, Vienna, Ritter von Mösle & Braumüller, 1839, p. 153.
[92] Kürschner, op. cit., note 5 above, p. 115.

A somewhat different picture emerges from a paper by Kürschner,[93] the first to report muscular fibres in the human a-v valves and to classify in detail the chordae tendineae, as already discussed. He applied this newly acquired knowledge to apportion the part played in a-v valve closure by the muscular fibres in the cusps and by the blood, respectively, and to analyse the closure mechanism.

Kürschner held that during ventricular diastole the a-v valves were surrounded by blood and that "The assumption that the posterior aspect of the [mitral] valve is applied to the interior wall of the ventricle is evidently wrong".[94] The main function of the papillary muscles is assumed " . . . to hold back the unfolded cusp against the pressure of the blood in the ventricle during its systole. Therefore, they subserve the fixation of the valve and secure its position."

The function of the muscular fibres continuing from the atrium into the cusp is regarded as complex: they narrow the cusp and, in conjunction with the second group of the chordae, bring the valve into a degree of tension and into a position favourable for its unfolding. Kürschner considered the unfolding of the borders of the cusps to be very important for the firm apposition of the cusps and the complete closure of the a-v orifice. In his opinion, Skoda's failure to unfold the borders led him to the erroneous view that there were pockets in the valves. Kürschner concluded that "during the contraction of the atrium the cusp is . . . fixed on the border of the atrium as if it had not sunk down after [its] complete unfolding, but pushed together toward the *limbus cordis*".[95] He compared this mechanism to a spring which one compresses in the direction in which it is subsequently to project. Similarly, the valvular musculature moves the cusps at the circumference of the orifice in a direction that it need only be pushed forward and expanded in order to close the orifice. Summing up, he stated: "On contraction of the atrium the cusp is tightened and set, on contraction of the ventricle it is fixed in the moving mass of blood by the vigorous stretching of the tendons of the first group, with the result that it cannot budge in the [blood]stream; however, by the blood flowing along and pressing against it, the cusp is pushed forward, in all its separate parts unfolded, and spread out in front of the ostium venosum . . .".[96]

That the size of the a-v valves greatly exceeds that of the a-v orifices is attributed by Kürschner to an uneven and gradual unfolding of the valves; his explanation, given in some detail, is unconvincing.

In a later article, Kürschner[97] described the behaviour of the valve immediately following ventricular contraction. The blood flowing from the atrium separates the cusps and dilates the ventricle. "If it passed directly from the atrium into the ventricle without flowing through the canal formed by the valve, the blood would strike against a particular spot, rather than press evenly against all parts of the cavity. This appears to have been avoided in the case of the valve, since toward the end of systole the walls of the ventricle lie on the valve, the pressure of the blood coming from the atrium thus

[93] Ibid.
[94] Ibid., p. 116.
[95] Ibid., p. 117.
[96] Ibid., p. 118.
[97] G. Kürschner, 'Herzthätigkeit', in *R. Wagner's Handwörterbuch der Physiologie mit Rücksicht auf pathologische Physiologie*, Braunschweig, Vieweg, 1844, vol. 2, p. 63.

being evenly distributed and necessarily remaining evenly distributed during the successive separation of the [ventricular] wall from the valve."

Grossly exaggerated views about the function of the valvular musculature were held by Paladino.[98] He thought that the wave of contraction, passing from the atrial to the contiguous valvular fibres, exerts traction on the papillary muscles. Similarly, ventricular systole leads to a contraction of the contiguous, i.e. ventricular, layer of the valvular musculature. He therefore assumed that closure of a-v orifices is effected by raising of the valves resulting from the contraction of their own musculature. This takes place toward the end and as a continuation of atrial systole. Only this raising, separating the lamellar parts from the ventricular wall and immersing it into blood, makes it possible for the ventricular pressure to close the valves at the very beginning of systole, whereby the ventricular valvular contraction reinforces the closure. Paladino's opinion was declared as "quite extreme views" by Albrecht,[99] since it appeared to him that Paladino regarded valve closure exclusively as an "active effect of the valvular musculature".

4. *The role of atrial contraction*

Baumgarten[100] was apparently the first to put forward the idea that atrial contraction causes a-v valve closure; he investigated this problem at the suggestion of Carl Ludwig, at that time Professor of Anatomy at Marburg. Baumgarten published the results as an Inaugural Dissertation and, since only a few copies of the original Latin thesis appeared in the book trade, summarized it in German.[101]

The investigations were carried out on excised hearts of man and animals (mostly calves), with half the atria removed and the arterial orifices sealed by ligatures or preferably by wax.

At the time atrial systole begins, atria and ventricles are full of blood. Investigating the effect upon the position of a-v valves of filling the ventricles, Baumgarten found that, when carrying this out with a fluid of approximately the same specific gravity as blood, the valves swam in the fluid, forming a funnel with its apex in the middle of the ventricular cavity and its base at the sites of attachment of the valves on the a-v orifice.[102] The borders of the valves at the apex of the funnel were not in contact with each other, but were curled with their convexity toward the ventricular axis and their concavity toward the walls. Pouring in fluid *slowly*, even overfilling, did not alter the position of the valves. This represented, therefore, the condition at the start of atrial systole.

Contraction of an atrium at this stage, Baumgarten argued, will produce a movement of blood toward the ventricle. This produces some pressure of the blood in the ventricular cavity, slightly stretching the ventricular walls, the position of the valve remaining unchanged. The pressure in the ventricular cavity spreads in all directions, affecting also the blood behind and below the valve.

As soon as atrial contraction lessens or ceases, the blood in the ventricle will start to move toward the area from which previously pressure had been exerted and where this

[98] Paladino, op. cit., note 16 above.
[99] Albrecht, op. cit., note 23 above, p. 63.
[100] Baumgarten, op. cit., note 8 above.
[101] Baumgarten, op. cit., note 9 above.
[102] Ibid., p. 465.

pressure had now ceased, namely, the a-v orifice. Blood behind the valve will press it inward, and that below the valve, upward. The cusps are thereby brought into a position more parallel to the orifice and in this way its closure is effected.[103]

Baumgarten supported this view experimentally. If, in a heart preparation as described above, the ventricles were filled with fluid and then a jet of water about one foot high was momentarily poured on to a cusp or the whole a-v orifice, at the very moment the jet ceased the valve closed so firmly that the heart could be inverted without a drop of fluid leaking out. He concluded that "the cause of closure of the venous valves of the heart is the pressure of the blood in the ventricle produced by the contraction of the atrium".[104] The a-v- valves are thus closed already at the beginning of ventricular systole, which prevents regurgitation.

One of the earliest supporters of Baumgarten's theory was Hamernjk,[105] Assistant Physician at the Allgemeines Krankenhaus at Prague, who emphasized that Baumgarten's view of the closure mechanism makes it possible to discard the opinion that the venous valves are only closed in the course of ventricular systole, which would entail a waste of ventricular force spent on regurgitation. Another adherent to Baumgarten's view was Gustav Joseph,[106] who studied under Purkyně in Breslau where he became Lecturer. He too stressed that Baumgarten's analysis implied that the a-v orifice is closed *before* the beginning of ventricular systole.

The views of Halford,[107] Professor of Anatomy and Physiology at the University of Melbourne, were very similar to Baumgarten's though with one important modification. He asked: How can the force of auricular contraction be brought to bear on the undersurface of the a-v valve? He demonstrated that, if a ventricle is opened and the flaps of a-v valves are cut away from their attachments, the chordae tendineae "rise up like stems of aquatic plants from the musculi papillares, and from the side of the ventricle". With the chordae cut off close to the undersurface of the cusps, the cusps are still supported to a certain level in the fluid and their delicate borders curl upward toward the auricle. Halford attributed to the presence of elastic tissue in the valve this upward curling, which assists in the approximation of the edges of the valve; " . . . finally, the pressure exerted by the blood from the auricle brings all into play, and their closure is effected."

Markham,[108] according to Halford the first to draw attention to the importance of elastic tissue in the valves, argued that it is "manifestly incorrect" to attribute to the blood the raising of the valve during ventricular diastole, because the pressure of the blood, passing from atria to ventricles, must be as great on the atrial as on the ventricular surface, in fact, greater. Having dissected a thickish layer of elastic tissue from the "auricular" surface of a-v valves of bullocks, he firmly believed that the rise of the valves during ventricular diastole was effected "by the agency of elastic tissue".

[103] Ibid., p. 466.

[104] Ibid., p. 468.

[105] J. Hamernjk, 'Physiologisch-pathologische Untersuchungen über den Mechanismus, nach welchem die venösen und arteriösen Klappen des Herzens geschlossen werden und nach welchem die Töne der Herzgegend enstehen', *Prag. Vierteljahrsschr. prakt. Heilk.*, 1847, Vierter Jahrg., **16:** 146-176.

[106] G. Joseph, 'Geschichte der Physiologie der Herztöne vor und nach Laënnec', *Janus*, 1853, p. 517.

[107] G. B. Halford, 'On the time and manner of closure of the atrio-ventricular valves', *Med. Times Gaz.*, 1861, p. 519, quoted from *Schmidt's Jahrb.*, 1864, **121:** 113.

[108] Markham, op. cit., note 65 above.

Contrary to Baumgarten's view, Markham thought that at the end of ventricular diastole the valves are pressed down and lie flat against the inner walls, the elastic fibres being stretched. Since the specific gravity of the valves is considerably greater than that of blood, "it is evident that no other moving force than that of elastic tissue can be in action here."

Valentin[109] was rather guarded. He wrote that Baumgarten's view of closure by the rebound of blood in the ventricles, due to the velocity of the inflowing blood, was presumably correct as a rule *("vermuthlich in der Regel")*, and discussed that an alternative factor, viz. the elasticity of the stretched ventricular walls, would have the same effect, provided there is a sufficiently long interval between atrial and ventricular contraction. In these circumstances the resulting pressure would be more than adequate to close the valve. Valentin's appreciation of the part played by the length of the a-v interval preceded by half a century the recognition by later authors of its relevance to a-v valve closure (see below).

In his paper on the mechanics of the tricuspid valve, Krehl[110] emphasized that the a-v valves have to close during ventricular diastole; therefore only atrial contraction and the movement of blood in the ventricles caused thereby come into question. Accepting the view of Baumgarten, Krehl mentioned, among other factors, narrowing of the a-v ring due to muscular fibres running from the atrium into the valve, approximation of the peripheral parts of the cusps, and eddies moving the cusps upward and toward the midline.

Henderson and Johnson,[111] modifying Baumgarten's theory, pointed out that the a-v valves can close in two distinct ways.

In the normally-coordinated heart, the breaking of a jet at an ostium is regarded as the essential factor. These authors demonstrated on models, and on excised mitral or tricuspid valves of oxen,[112] that, if the flow of fluid (simulating blood) through an ostium ceases abruptly, the part of the column behind the opening stops, whereas the portion which had already passed the opening continues to flow. The latter produces a small area of negative pressure in the rear of the still forward-moving column which, drawing blood from each side, approximates the valve cusps. With this mechanism the parts of the cusp nearest their base are the first to move, the edges of the flaps being the last to be put into a position of closure. This makes closure possible without regurgitation. Atrial systole immediately preceding ventricular contraction "assures the inrolling and non-leaking mode of closure in the mitral and tricuspid valves even when the rate of the heart is slow",[113] namely, by the mechanism described by Baumgarten. With rapid heart action the a-v valves would "probably" close in the same way even without atrial systole.

If, in a slowly beating heart, ventricular systole occurred without a preceding atrial systole, the valves would close in another way, viz. by a hinge-type movement, the free edges of the cusps moving first. This would result in considerable regurgitation.

[109] Valentin, op. cit., note 11 above, p. 277.
[110] L. Krehl, 'Die Mechanik der Tricuspidalklappe', *Arch. Anat. Physiol., Physiol. Abt.*, 1889: 289-294.
[111] Y. Henderson and F. E. Johnson, 'Two modes of closure of the heart valves', *Heart*, 1912-13, **4**: 69-82, pp. 70-72.
[112] Ibid., pp. 72-77.
[113] Ibid., p. 81.

However, in the normal heart this is avoided by atrial systole, ensuring closure without regurgitation, as described above.[114] The hinge type of closure may become operative in certain types of atrioventricular dissociation.

Little[115] accepted the views put forward by Baumgarten with the modifications of Henderson and Johnson.

5. SOME ADDITIONAL ASPECTS

Denial of direct valvular action in closure mechanism

Onimus[116] rejected the view that the a-v valves play any direct part in the closure of the orifices, his rejection being based on the assumption that during ventricular systole the contracting papillary muscles lower the cusps, applying them to the ventricular wall. This would result in an open a-v orifice at a time when no communication between ventricles and atria should exist. Therefore, he argued, the cusps cannot be the factor effecting closure. He detailed the function of the a-v valves as follows: during the very short moment required for the closure of the a-v orifice they prevent the reflux of the blood which is near the orifice; they drive the blood toward the arterial orifices, also expelling blood engulfed between the cusps and the corresponding ventricular wall; and they render the contour of the a-v orifice smooth. He attributed the obliteration of the a-v orifices to the application of mobile parts, namely the lateral ventricular walls, to immobile ones, viz. the aortic cusp of the mitral valve on the left and the interventricular septum on the right, followed by a sphincter-like contraction of circular fibres of the valve ring. In order to apply firmly to each other, the superior parts of the ventricular wall – of which the aortic cusp of the mitral valve is assumed to form a part – have to be smooth. This requires an "independent aponeurosis", and nature has provided this by the valves, which therefore could be called "floating aponeuroses of the heart". In retrospect, this paper shows how a whole wrong edifice can be built on one mistaken basic assumption.

Importance of coronary sinus musculature in closing mechanism

Vintschgau,[117] Assistant to Bruecke in Vienna and later Professor of Physiology at Padua and Innsbruck, drew attention to the part played in valve closure by the muscular fibres in the coronary sinus and its valve. Being contiguous with the atrial musculature, these fibres contract at the time of atrial systole, narrowing the sinus and probably closing its orifice momentarily, thereby impeding the blood flow into the right atrium. This results in a momentary congestion of blood in the cardiac vessels and an increase in tension of the ventricular walls, reaching its maximum at the end of atrial contraction. When atrial systole begins to subside, blood starts to flow again from the coronary sinus into the right atrium, with the result that the tension in the ventricular wall diminishes, enhancing the effect of their elasticity on the pressure of the blood in the ventricular cavities. The cusps of the a-v valves are thereby brought into apposition, the a-v orifice being closed before the beginning of ventricular systole.

[114] Baumgarten, op. cit., note 9 above.

[115] R. C. Little, 'Effect of atrial systole on ventricular pressure and closure of the A-V valves', *Amer. J. Physiol.*, 1951, **166**: 289-295, p. 289.

[116] E. Onimus, 'Etudes critiques et expérimentales sur l'occlusion des orifices auriculo-ventriculaires', *J. Anat. Physiol.*, 1865, **2**: 337-381, particularly pp. 375, 381.

[117] M. v. Vintschgau, 'Einige Bemerkungen über die physiologische Bedeutung der Muskelfasern in der Wand des sinus communis venarum cardiacarum', *Pflüg. Arch. ges. Physiol.*, 1896, **64**: 79-96, pp. 89-94.

Importance of the length of the a-v interval in closure mechanism

Dean[118] recorded optically in cats the contractions of the left atrium and ventricle, and the movements of the septal cusp of the mitral valve by means of a hair which conveyed them to a light-weight lever. He found that the mechanism of closure depended on the length of the a-v interval, i.e. the interval between atrial and ventricular contraction. With an average of 0.272 sec or more, he recorded a double movement of the cusp, viz. toward the ventricle during a short period after the onset of atrial systole and, toward the end of atrial systole, a marked quick movement toward the atrium though not to a position of complete closure. It has been suggested by Erlanger,[119] who saw in one isolated instance the posterior leaflet of the mitral valve of an ox heart beating rhythmically, that this quick movement toward the atrium might have been caused by the contraction of the valve musculature. More recently, the capability of these muscle fibres to contribute to valve movements has been discussed by Sonnenblick *et al.*[120]

At the onset of atrial diastole the cusp moved quickly toward the ventricle and the orifice remained open until ventricular systole began; at its onset the cusp moved immediately upward to a position of complete closure, remaining there until the start of ventricular diastole. There were thus two periods of closure movement of the a-v valves with these longer a-v intervals, one due to atrial, the other to ventricular contraction. These two separate movements can only occur with a sufficiently long a-v interval. If this is less than 0.147 sec "the valves are in the process of closing due to the auricular effect when ventricular systole begins. Hence the cardiac event merely completes the closure already initiated by the auricle. There is in this case only a single closure movement . . .", the atrial and ventricular components being combined in one movement. Dean's paper is a forerunner of echocardiographic studies showing the more complex movements of the a-v valve cusps in connexion with closure, discussed below.

Inadequate closure of a-v valves

Several authors believed that the mitral valve closes more firmly than the tricuspid. Thus Harvey[121] thought: "To prevent the blood from slipping back into the vein-like artery [pulmonary vein] and thereby reducing the effort of the left ventricle in propelling that blood forwards to the body as a whole, the mitral valves which I have mentioned exceed in size and strength and accuracy of closure the ones situated in the right ventricle". In his view, the mitral valve has only two cusps "so that the occlusion may be more precise in accordance with the greater force . . .". John Hunter[122] stated: "I have reason to believe, that the valves in the right side of the heart do not so perfectly do their duty as those of the left, therefore we may suppose it was not so necessary."

[118] A. L. Dean jr., 'The movements of the mitral cusps in relation to the cardiac cycle', *Amer. J. Physiol.*, 1916, **40**: 206-217.

[119] J. Erlanger, 'A note on the contractility of the musculature of the auriculo-ventricular valves', ibid., 1916, **40**: 150-151.

[120] E. H. Sonnenblick, L. M. Napolitano, W. M. Daggett, and T. Cooper, 'An intrinsic neuromuscular basis for mitral valve motion in the dog', *Circulation Res.*, 1967, **21**: 9-15.

[121] Harvey, op. cit., note 3 above, pp. 103-104.

[122] John Hunter, *A treatise on the blood, inflammation, and gun-shot wounds*, London, George Nicol, 1794, Part I, Chapter II, Section II, pp. 139, 140.

Magendie[123] wrote of the nearly *("à peu près)* complete closure of the tricuspid; Burdach[124] held similar views. King[125] injected the left and right ventricles of human hearts and found that there was always complete closure of the mitral orifice but reflux through the tricuspid. He regarded this as a safety device, and the tricuspid as a combination of a valve preventing reflux, and a safety-valve protecting the lungs from the inflow of an excessive amount of blood. Mayo[126] wrote that the chambers of the right side "generally appear something larger than those of the left, which probably has in part to do with the imperfectness of the tricuspid valve". Rüdinger,[127] Professor of Anatomy in Munich and pupil of Henle, thought, like Harvey, that two cusps make firm closure more reliable than three or more; and a firm closure on the left is necessary because "the arterial back pressure of the body is greater than that on the part of the lung". Quoting King with approval, Milne Edwards[128] pointed out that in normal conditions a-v valve closure is sufficiently perfect to prevent any, or any significant, reflux into the atria. However, as mitral closure is more perfect than tricuspid, the latter allows some reflux into the right atrium if the circulation in the pulmonary vessels is disturbed.

The reverse view was held by Galen.[129] He regarded three "membranes" as best, and better than two or four, and wrote: "There was good reason, then, why two outgrowths of membranes were formed at only one orifice, that of the venous artery. For it was better for this one opening not to be accurately closed, because it was better that this one alone should give access to the lung for the fuliginous residues from the heart which necessarily accumulate there on account of the abundance of the innate heat and which have no other shorter outlet."

According to Richerand,[130] Consulting Surgeon to Louis XVIII, both the mitral and tricuspid valves close imperfectly, with some amount of blood regurgitating into the atria.

A reversal of opinion on atrial activity

A striking reversal of opinion about the activity of the atria is to be found in successive reports of the British Association for the Advancement of Science. In his "Abstract of Observations on the Motion and Sounds of the Heart", Carlisle[131] stated: "The auricles contract but little upon their contents in man and in the higher classes of animals . . ." The fifth meeting of the Association[132] arrived at the same conclusion,

123 Magendie, op. cit., note 44 above, p. 246.

124 Burdach, op. cit., note 48 above, p. 209.

125 T. W. King, 'An essay on the safety-valve function in the right ventricle of the human heart, and on the gradations of the function in the circulation of warm-blooded animals', *Guy's Hosp. Rep.*, 1837, **2**: 104-178, p. 125.

126 Mayo, op. cit., note 69 above, p. 44.

127 N. Rüdinger, *Ein Beitrag zur Mechanik der Aorten-und Herzklappen*, Erlangen, Enke, 1857, pp. 14, 15.

128 H. Milne Edwards, *Leçons sur la physiologie et l'anatomie comparée de l'homme et des animaux*, Paris, Masson, 1859, vol. 4, p. 30.

129 Galen, op. cit., note 34 above, p. 318.

130 A. Richerand, *Elements of physiology*, 4th ed., edited by J. Copland, London, Longman, Hurst, Rees, etc., 1824, pp. 170, 171.

131 H. Carlisle, 'Abstract of observations on the motion and sounds of the heart', *Report of the Third Meeting of the British Association for the Advancement of Science held at Cambridge in 1833*, London, John Murray, 1834, p. 455.

132 British Association for the Advancement of Science, *Report of the Fifth Meeting held at Dublin in 1835*, London, John Murray, 1836, p. 248.

adding "an active contraction being observable only in the appendices". By contrast, the report of the tenth meeting[133] stated that the normal systole of the auricles is energetic.

Importance of systolic narrowing of the a-v valve ring for closure of the orifice.

The importance of systolic narrowing of the a-v valve ring for the closure of the a-v orifice was emphasized by several authors, e.g. Bichat,[134] Parchappe,[135] Lian,[136] and Luciani.[137] According to Nega,[138] "a circular contraction of the venous walls of the orifices invariably takes place" simultaneously with ventricular systole and the contraction of the papillary muscles. Hesse[139] was of the opinion that "we have to attribute to the muscles surrounding the atrioventricular orifices a significant part in the closure mechanism of the venous orifices". The same movement of the anterior cusp of the mitral valve opens up the reduced space in the uppermost part of the ventricle toward the aortic orifice but closes it toward the a-v one. Similar conditions prevail in the right ventricle.[140] Several authors stressed that during the inflow of blood from atria into ventricles the a-v valves not only open up the a-v orifices but also close the arterial orifices, e.g. Sömmerring,[141] Bichat,[142] Adelon,[143] and Burdach.[144] This was denied by Bouillaud,[145] who stressed that during ventricular diastole, when the semilunar valve closes the aortic orifice, the aortic cusp of the mitral valve could not by its position at that time obstruct the aortic orifice.

Krehl,[146] amplifying his previous study of the tricuspid valve, compared the narrowing of the ventricular cavity to that of a folded filter by a constricting ring, drawing attention to folds protruding into the lumen of the ventricles. The aortic orifice is kept open during systole, since such protrusions are absent on the septum; the bloodstream moves the large cusp of the mitral valve upward, toward the centre of the a-v orifice, and away from the aortic orifice. As systole progresses, closure of the a-v orifice by the cusps is promoted by two changes: the area of the planes of the cusps, which are pressed together, increases, and the orifice becomes increasingly narrower.[147] Essentially the same conditions prevail in the right heart.[148]

Albrecht[149] seems to have over-emphasized the role of valve ring contraction: "For

133 British Association for the Advancement of Science, *Report of the Tenth Meeting held at Glasgow in August 1840*, London, John Murray, 1841, p. 204.

134 Bichat, op. cit., note 42 above.

135 Parchappe, op. cit., note 72 above.

136 C. Lian, 'Contribution à l'étude de la physiologie de l'appareil valvulaire mitral', *J. Physiol. Pathol. gén.*, 1909, **11**: 597-612, p. 604.

137 L. Luciani, *Human physiology*, translated by F. A. Walby, London, Macmillan, 1911, vol. 1, p. 193.

138 Nega, op. cit., note 12 above, p. 15.

139 F. Hesse, 'Beiträge zur Mechanik der Herzbewegung', *Arch. Anat. Physiol., Anat. Abt.*, 1880, 328-353, p. 344.

140 Ibid., pp. 351, 352.

141 Sömmerring, op. cit., note 43 above, p. 19.

142 Bichat, op. cit., note 42 above, pp. 116, 120.

143 N.-P. Adelon, *Physiologie de l'homme*, Paris, Compère Jeune, 1829, vol. 3, p. 297.

144 Burdach, op. cit., note 48 above, p. 209.

145 Bouillaud, op. cit., note 70 above, p. 356 footnote.

146 L. Krehl, 'Beiträge zur Kenntnis der Füllung und Entleerung des Herzens', *Abh. mathem.-physisch. Cl. königl. sächs. Gesellsch. Wiss.*, 1891, **17**: 339-362, p. 350.

147 Ibid., p. 359.

148 Ibid., p. 360.

149 Albrecht, op. cit., note 23 above, p. 116.

a long time the auxiliary muscular forces in the closure of the venous valves were thought to be the papillary muscles, but in error: today . . . we can regard it as completely proved that this is ensured by the constricting force of the basal parts of the heart."

Results of echocardiography

More accurate visualization of valve movements in man has become possible by the recently introduced non-invasive method of ultrasound recording: echocardiography. Its principle consists in the emission, by a transducer applied to the chest wall, of very short high-frequency sound impulses, which are partly reflected back to the transducer when the impulse encounters a tissue of different acoustic impedance, e.g. at the interface between blood and mitral valve. By this method movements of valve leaflets can be recorded on photosensitive paper.

Mitral valve movements have been extensively investigated. Apart from demonstrating the closed position of its leaflets during ventricular systole and their rapid movement toward opening at the beginning of diastole, echocardiography has shown that during and immediately after the initial rapid inflow of blood into the ventricle the cusps move toward closure. If diastole is sufficiently long, a mid-diastolic re-opening of the orifice with flow of blood into the ventricle may occur, but this is not observed if the heart rate is faster and diastole shorter. Following atrial contraction, the mitral orifice opens again as blood flows into the ventricle. With the subsequent atrial relaxation the leaflets float toward closure, which is completed with ventricular systole (Edler,[150] Feigenbaum[151]). The effect of the length of the P-R interval, i.e. the electrocardiographic record of the interval between atrial and ventricular activation, on the closure mechanism of the mitral valve has been demonstrated by Zaky *et al.*[152]

CONCLUSION

It can be said that, despite gaps in our knowledge, progress has been made over the years in our understanding of the closure mechanism of the a-v valves. It seems opportune, therefore, to summarize present views on some aspects alongside important though not necessarily first steps in their development.

Lower[153] believed that during ventricular systole the chordae tendineae relax just sufficiently to allow the cusps to close the a-v orifice completely, but the restraining action of the chordae and thus of the papillary muscles was only stated clearly a few years later by Perrault[154] in 1680. Contrary to the view of some, that the papillary muscles contract simultaneously with the rest of the myocardium was stressed by Sömmerring[155] in 1792, though according to some recent authors they start to contract before the rest of the ventricular muscle (Rushmer *et al.*,[156] Brecher and Galletti[157]).

[150] J. Edler, 'Ultrasound cardiography', in Gilbert Baum, *Fundamentals of medical ultrasonography*, New York, G.P. Putnam's Sons, 1975, pp. 310-314.

[151] H. Feigenbaum, *Echocardiography*, 2nd ed., Philadelphia, Lea & Febiger, 1976, p. 93.

[152] A. Zaky, E. Steinmetz, and H. Feigenbaum, 'Role of atrium in closure of mitral valve in man', *Amer. J. Physiol.*, 1969, **217**: 1652-1659.

[153] Lower, op. cit., note 33 above.

[154] Perrault, op. cit., note 35 above.

[155] Sömmerring, op. cit., note 43 above.

[156] R. F. Rushmer, B. L. Finlayson, and A. A. Nash, 'Movements of the mitral valve', *Circulation Res.*, 1956, **4**: 337-342.

Meckel[158] seems to have been the first to state, in 1817, that by their contraction the papillary muscles draw portions of the valve together and in this way close the orifice. The mode of attachment of the chordae to adjacent cusps, contained in Kürschner's[159] classification of the chordae (1840), forms the anatomical basis for this action, which is still regarded as an important factor. The importance of atrial contraction for a-v valve closure was first proclaimed by Baumgarten[160] in 1843, present concepts dating from his work, with the modification of Henderson and Johnson[161] being accepted by a number of workers. The action of atrial contraction to produce eddies of the blood in the ventricular cavities in a direction contributing to valve closure was emphasized by Krehl[162] and Luciani.[163] Bichat[164] appreciated that the narrowing of the a-v circumference by contraction of the muscles of the valve ring was an important factor. Whether during ventricular diastole the valves are applied to the ventricular walls or separated from them by a space was the subject of controversies over a considerable period. Finally, that during this phase of the cardiac cycle the valves rise toward partial or complete closure of the orifice, as established today by echocardiography, was known to Lower over 300 years ago.

SUMMARY

Though the exact mechanism of closure of the atrioventricular (a-v) valves is still unknown, some progress has been made in our understanding of this problem. It was thus of interest to study the historical development of views on this topic. The discussion is prefaced by that of two closely related anatomical aspects: the question of the presence of muscular fibres in the a-v valves of the human heart, and the classification of the chordae tendineae. Ideas about the mechanism of closure of the a-v valves are discussed under the headings: (1) passive closure; (2) active closure; (3) combination of the two; (4) role of atrial contraction; (5) some additional aspects.

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[157] G. A. Brecher and P. M. Galletti, 'Functional anatomy of cardiac pumping', in *Handbook of physiology*, Washington D.C., American Physiological Society, 1963, vol. II, Section 2, p. 770.
[158] Meckel, op. cit., note 68 above.
[159] Kürschner, op. cit., note 5 above.
[160] Baumgarten, op. cit., notes 8 and 9 above.
[161] Henderson and Johnson, op. cit., note 111 above.
[162] Krehl, op. cit., note 110 above.
[163] Luciani, op. cit., note 137 above.
[164] Bichat, op. cit., note 42 above.