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Review

The myocardium and its fibrous matrix working in concert as a spatially netted mesh: a critical review of the purported tertiary structure of the ventricular mass☆

Paul P. Lunkenheimer [a,*], Klaus Redmann [a], Philipp Westermann [a], Kay Rothaus [b], Colin W. Cryer [c], Peter Niederer [d], Robert H. Anderson [e]

[a] Experimentelle Thorax-, Herz und Gefäßchirurgie, Universität Münster, Munster, Germany
[b] Institut für Informatik, Universität Münster, Munster, Germany
[c] Institut für Numerische Mathematik, Universität Münster, Munster, Germany
[d] Institut für Biomedizinische Technik, ETH und Universität Zürich, Switzerland
[e] Cardiac Unit, Institute of Child Health, University College London, UK

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In memory of two old friends: William F. Whimster and Francisco Torrent-Guasp.

Summary

With the increasing interest now paid to volume reduction surgery, in which the cardiac surgeon is required to resect the ventricular myocardium to an extent unenvisaged in the previous century, it is imperative that we develop as precise knowledge as is possible of the basic structure of the ventricular myocardial mass and its functional correlates. This is the most important in the light of the adoption by some cardiac surgeons of an unvalidated model which hypothesises that the entire myocardial mass can be unravelled to produce one continuous band. It is our opinion that this model, and the phylogenetic and functional correlates derived from it, is incompatible with current concepts of cardiac structure and cardiodynamics. Furthermore, the proponents of the continuous myocardial band have made no effort to demonstrate perceived deficiencies with current concepts, nor have they performed any histological studies to validate their model. Clinical results using modifications of radius reduction surgery based on the concept of the continuous myocardial band show that the procedure essentially becomes ineffective. As we show in this review, if we understand the situation correctly, it was the erstwhile intention of the promoters of the continuous band to elucidate the basic mechanism of diastolic ventricular dilation. Their attempts, however, are doomed to failure, as is any attempt to conceptualise the myocardial mass on the basis of a tertiary structure, because of the underlying three-dimensional netting of the myocardial aggregates and the supporting fibrous tissue to form the myocardial syncytium. Thus, the ventricular myocardium is arranged in the form of a modified blood vessel rather than a skeletal muscle. If an analogy is required with skeletal muscle, then the ventricular myocardium possesses the freedom of motion, and the ability for shaping and conformational self-controlling that is better seen in the tongue. It is part of this ability that contributes to the rapid end-systolic ventricular dilation. Histologic investigations reveal that the fibrous content of the three-dimensional mesh is relatively inhomogeneous through the ventricular walls, particularly when the myocardium is diseased. The regional capacity to control systolic mural thickening, therefore, varies throughout the walls of the ventricular components. The existence of the spatially netted structure of the ventricular mass, therefore, must invalidate any attempt to conceptualise the ventricular myocardium as a tertiary arrangement of individual myocardial bands or tracts.

Keywords: Ventricular myocardial band; Spatially netted architecture; Radius reduction surgery; Antagonism

1. Introduction

At the end of the twentieth century, the prevailing concepts of cardiodynamics were such that the suggestion by Batista [1] that the failing heart could be ameliorated by resection of parts of the left ventricular walls was greeted in disbelief by many cardiac surgeons. We now know, of course, that many patients with dilated or ischaemic cardiomyopathy have benefited greatly subsequent to surgical reduction in the radius of their left ventricles. This has led some cardiac surgeons, understandably, to demand a better understanding of the structure of the organ on which they operate [2]. This, in turn, has led other cardiac surgeons

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* Corresponding author. Tel.: +49 251 83 56256; fax: +49 251 8356257.
E-mail address: redmann@uni.muenster.de (P.P. Lunkenheimer).

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[3,4] to ignore completely the evidence that shows the heart is basically arranged in the form of a modified blood vessel, and instead to promote enthusiastically the concept of the 'unique myocardial band'. In doing this, they have made no attempt to show that existing anatomic concepts have no foundation, and moreover, have made no attempt to conduct histological investigations, which might have revealed the partitions that would permit uniform and consistent dissection of the purported myocardial tract, should this tract truly exist? This is disturbing in the new millennium, where so much emphasis is placed on evidence-based medicine.

It is difficult, nonetheless, to disprove concepts that themselves have never been proven. In this chapter, therefore, we first review our own understanding of the concept of the 'myocardial band'. We then offer a brief account of the traditional anatomic understanding of the spatially netted nature of the ventricular mass. We then discuss just a little of the evidence that invalidates any concept based on a tertiary morphologic arrangement for the ventricular mass, not least the unlikely formation suggested by Torrent-Guasp and co-workers [5—7].

2. The concept of the unique myocardial band

In the late 1960s, ignoring the multiple anatomical studies which, over the centuries, had shown that the ventricular myocardium was arranged in the form of a modified blood vessel [8—27], rather than as an analogue of the skeletal musculature, the Spanish general practitioner Torrent-Guasp dissected the ventricular myocardial mass, purporting to follow the perceived predominant longitudinal orientation of myocardial aggregates. He claimed to have shown the existence of a continuous myocardial tract extending from the pulmonary trunk to the aortic root [5—7]. When making his dissections, however, he chose to ignore the essential invasive nature of the technique. Furthermore, he paid no heed to justifiable criticisms made by earlier investigators [10,17], who pointed out that any dissector seeking to follow the course of myocardial aggregates through the ventricular walls must, of necessity, destroy the essential spatially netted nature of the ventricular myocardium. Having conveniently ignored these heuristic caveats, he then strove to assign physiological functions to his perceived unique myocardial band, seeking to explain not only the systolic emptying and diastolic replenishing of the ventricles but also their phylogenetic development, and their susceptibility to distinct pathological conditions.

More recently, enthusiastic cardiac surgeons have used the purported presence of the band to justify surgical procedures for the treatment of dilated cardiomyopathy [28], even though it is now recognised that the known timecourse of ventricular activation is incompatible with the notion of the continuous myocardial tract [29]. Those who quite rightly seek to use cardiac structure as the basis for logical surgical manoeuvres [2], therefore, need also to recognise the reality of the three-dimensional arrangement of the ventricular mass. This must be revealed not only by dissections but also validated by histological studies. It is only by using supporting microscopic investigations that anatomists are able to reveal the nature of the supporting fibrous matrix, now recognised as a crucially important component of the ventricular myocardial network [30].

3. The netted nature of the ventricular mass

Any histological section taken through the ventricular wall is sufficient to demonstrate that there are no fibrous partitions segregating the myocardium into sheets or bundles (Fig. 1). This does not mean that the myocardium is devoid of a supporting fibrous matrix. On the contrary, the importance of the supporting fibrous tissue has long been recognised, with Borg and Caulfield [30] showing how the overall arrangement can be conceptualised in terms of epimysial, perimysial and endomysial components. None of these fibrous elements, however, binds together the ventricular myocytes into individual muscles in the fashion of the muscular arrangements of the limbs or the trunks, were fascial sheaths permit each individual muscle to be demonstrated in repetitive fashion. The fashion of arrangement of the ventricular myocardium is much more analogous to the structure of the tongue, with the muscular aggregates interwoven in the three orthogonal spatial orientations, and with each muscle cell attached to its neighbour, and often to several neighbouring cells.

In the heart, as in the tongue, it is possible to recognise the overall longitudinal arrangement of the aggregates of myocytes such that a 'grain' can be perceived on the ventricular surface once the supporting epicardial fibrous layer has been removed. Then, at various depths of the ventricular wall, it is possible to note an ordered gradation in the overall long axis of the myocardial aggregates relative to the equator of the ventricular mass [11,13,26,27]. It is also possible to distinguish an obvious circular array of myocardial

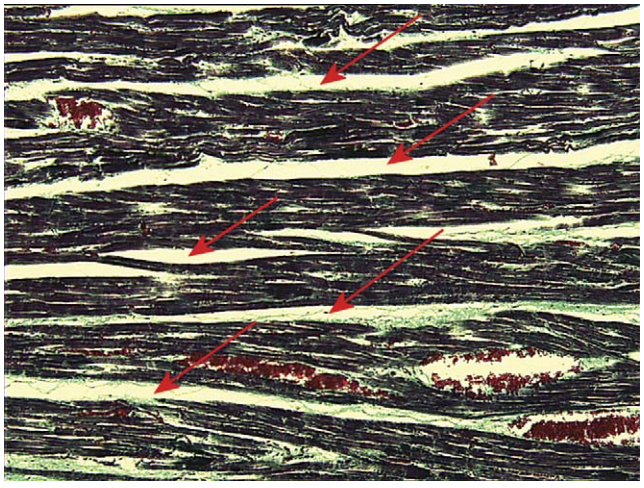

Fig. 1. Histological section (magnification of 100 times) showing the ventricular myocytes supported by a fibrous matrix, with the myocytes stained dark, erythrocytes red, and supporting connective tissue green. There are short gaps (red arrows) rarely bridged by offsprings of myocytes dividing the cells into particularly short strands. Yet, there is no continuous cleavage plane partitioning the myocytes into any discernable tertiary structure. There is, nonetheless, an overall longitudinal axis for the myocytic aggregates. The upper edge of the section is aligned parallel to the epicardium.

aggregates arranged parallel to the left ventricular equator. This circular component is the 'Triebwerkzeug' emphasised by Krehl [16]. It is then surely significant that, in hearts with right ventricular hypertrophy, such as those with tetralogy of Fallot, a layer of circular fibres corresponding to the 'Triebwerkzeug' is to be found encircling the right ventricle, an arrangement that is lacking in the normal heart [24,31]. It is the basic three-dimensional arrangement of interweaving longitudinal arrays of myocytes, most aligned tangentially, but some intruding into the ventricular wall (Fig. 2), which underscores the concept of dualistic ventricular function [32].

None of these studies, neither histologic investigation nor results obtained using magnetic resonance diffusion tensor imaging, demonstrate in unequivocal fashion any discernable tract of myocardial fibres that encircles both ventricles in 'figure of eight' fashion. Both methods, however, as with peeling and cleaving procedures, permit the investigator to follow any arbitrary selected pathway throughout the basic netted structure of the myocardium. The arrangement of the supporting connective tissue, nonetheless, is insufficiently precise to delimit any tertiary arrangement, such as the continuous band of myocardium suggested to exist by Torrent-Guasp et al. [6,7,33]. In summary, therefore, histologic studies, and studies using magnetic resonance diffusion tensor imaging, show the ventricular mass to be arranged in the form of a spatial net, made up of myocytes set in a supporting matrix of fibrous tissue. This spatial net is:

- Self-bracing, accounting for regional stiffening of the ventricular walls. In this way, certain circumscribed areas are able to serve as fixed points for the action of adjacent fibres. Inspection of the surface of the beating heart is sufficient to reveal that some distinct areas move relative to other parts, while some areas on the ventricular surface seem to remain immobile.
- Self-restraining in terms of regional shortening, and hence, able to autoregulate the onset, velocity, amount and termination of systolic wall thickening [32].
- Self-shaping with respect to the contour of the ventricular lumen and its systolic deformation, thus determining intraventricular resistance to flow.

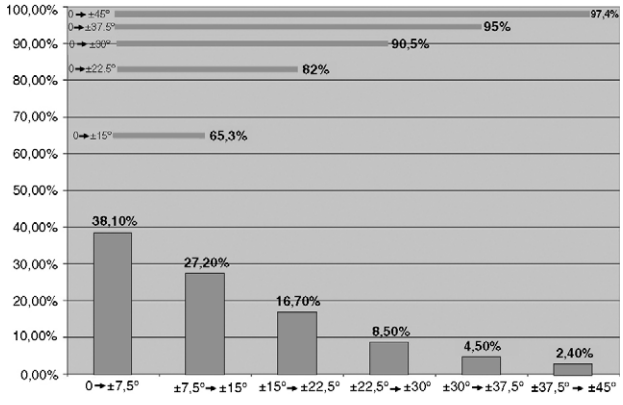

Fig. 2. Distribution of angles of intrusion in a histological section such as that shown in Fig. 1. The data summarizes measurements on 36 specimens.

4. Deficiencies in the concept of the unique myocardial band

4.1. Cardiac activation

In obvious contradiction to the established knowledge in electrophysiology, Torrent-Guasp and co-workers have claimed a sequential propagation of electrical activation along their proposed continuous band [5—7]. This ancillary hypothesis is necessary because the technique of unravelling the purported unique band destroys the integrity of the specialised ventricular conduction system. The alternative concept, however, fails to account for the well-established time-determining impact of pre- and afterload on myocardial fibres. It is the three-dimensional netting of the myocardium which serves as a substrate for disparately distributed afterloads which, according to Brutsaert et al. [34—36], cause some degree of disparity in the amount and timing of regional shortening. Gradients in onset, velocity, amount, and in termination of shortening, therefore, can readily be explained independent of any purported dephasing in the onset of activation [5—7].

4.2. The technique of dissection

Torrent-Guasp [33] had suggested initially that his preparation was produced by following a preexisting and unique plane of cleavage, which he claimed was readily discernible to the initiated investigator [6,7]. Should such a unique plane of cleavage exist, however, it would be discernible to the histologist. As we have already discussed, there is no histological evidence supporting the existence of such a discrete plane of cleavage, albeit that it is disappointing that those supporting the concept of the band so enthusiastically [3,4] have never sought the necessary histological validation of their fanciful notions. In his last publication, nonetheless, Torrent-Guasp et al. [6] weakened the claim concerning the unique plane of cleavage, arguing that the dissector needed only to follow the long axis of the myocytes, without requiring the presence of any preformed structure such as connective tissue sheets so as to demonstrate the existence of the unique band. This concession in itself, nonetheless, indicates that the dissector intrudes into the wall along random pathways by lacerating the underlying syncytial structure, following a carefully worked out plan which does no more than display the preconceived notion for myocardial structure.

With this possibility in mind, therefore, we recently set ourselves to unravel the ventricular myocardium, but using freshly excised, and as yet not denatured, porcine hearts. The procedure does not differ from that used by Torrent-Guasp [33] on boiled hearts, except that it needs some more strength in pulling apart the fibres, and in some areas, such as around the ventricular base, it needs incisions to sever collageneous structures (Fig. 3). Our dissection is more pertinent for the cardiac surgeon. If the arrangement purported to exist by the followers of Torrent-Guasp is a reality, then to be of value to the cardiac surgeon, it must be recognisable in the living heart, and not just in the heart prepared by prior boiling in water.

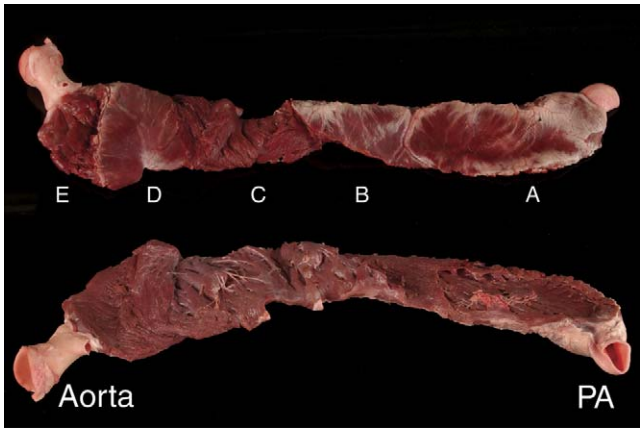

Fig. 3. Two aspects of a freshly excised and unravelled myocardial band of a pig, segmented according to the suggestion of Torrent-Guasp [33]. A: right ventricular free wall, B: inferior, posterior and superior aspects of the left ventricular base, C: descending segment, D: left ventricular apical region, E: ascendent segment of the left ventricular apical loop which reaches from the apex to the aortic root and which contains essential parts of the septum. A and B form the basal loop, C–E form the apical loop.

As can be seen, the band produced by our dissection is remarkably similar to the entity displayed by Torrent-Guasp [33]. When we then boiled our previously non-denatured specimens, however, they adopted bizarre patterns (Fig. 4), with the different segments reflecting the overall three-dimensional arrangement of the myocytes making up the ventricular walls, and with each part deforming to markedly variable extent (Table 1).

We have assumed that, under the action of boiling, each contractile element shrinks along its long axis. We infer, therefore, that the peculiar reshaping of each segment is an indicator of its intricate initial internal structure. Thus, we also infer that the amount of shortening is independent of the number of parallel fibres. When fibres are intermingled, since they are aligned in disparate directions, then the global deformation of a given segment is the function of the resulting prevailing tension in all fibres acting synchronously, but in different directions.

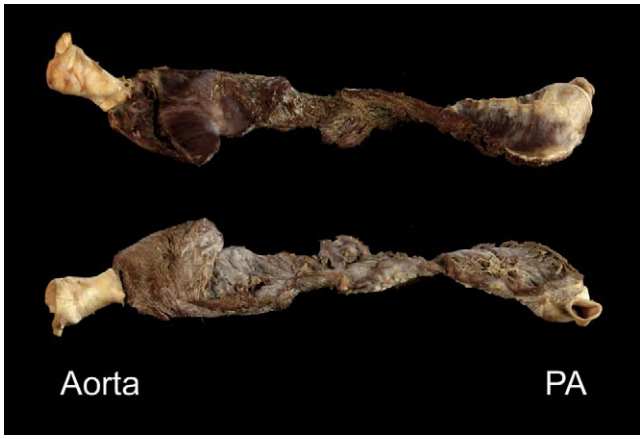

Fig. 4. The band-structure after having been denatured by boiling for 2 h. Note the marked torsion of segment B.

Table 1
Shrinkage of the components of the ventricular strip subsequent to denaturation by boiling (figures given in percentage of original length)

Segment	E	D	C	B[a]	A	Entire band
Axial length	27.3	33.3	36.8	53.3	40.5	Total: 38.6
Baso-apical length	28.6	23.8	43.75	78.6	17.65	Mean: 36

[a] Markedly contorted.

So as to demonstrate these specific alignments, we exposed the main course of the myocardial aggregates within the band by peeling off strands in a quasi-layered sequence [10,11,18]. The 'grain' on the resulting surfaces, representing the overall alignment of the long axis of the myocardial aggregates, was then digitised using a three-dimensional magnetic field digitising tablet (3-Draw Digitizer System, 3 SD 005 Polhemus, Cochester VTO 5446, USA). We started from the subepicardium, which was freed from its epicardial coating. Each new surface displayed by peeling was digitised stepwise. Sequential layers of peeling exposed progressively more uneven surfaces, with the strands being increasingly inclined towards the endocardium in the deeper parts of the walls. We show the digitised data from one porcine ventricular strip in Fig. 3. The most prominent feature is the layered rotation of the fibres from the epicardium to the endocardium upon a radial axis. This feature is in accord with established wisdom for the arrangement of the ventricular mass [29,13,18–21,26,27], but totally alien to the concept of the 'unique myocardial band' [6,7,33].

4.3. Ventricular dilation

According to Torrent-Guasp et al. [6,7,33], active lengthening of the left ventricle in late systole is the consequence of late activation and contraction of the ascending segment of the left ventricular apical loop. It concerns segment E (Figs. 3–5) which markedly shrinks in both dimensions of the plane upon denaturation by heat (Table 1). This action is supposed to erect the ventricular wall like a snake preparing to strike. If this is, indeed, the case, then the erecting musculature needs a fulcrum. Torrent-Guasp presumed that this fulcrum of support was provided by the residual intracavitary volume of blood. But how can this intracavitary volume serve as a fulcrum, which is loaded by pressure, whilst itself being augmented by the force generated by the same process? Furthermore, the assumption that the spiral myocardial tract is the main promoter of ventricular dilation does not explain ventricular dilation as seen in the failing or cardiomyopathic heart.

Perhaps recognising these potential deficiencies in his concepts, in his latter writings, Torrent-Guasp with his colleagues returned to a hypothesis of alleged aberrant fibres, to which was assigned the function of dilator of the basal ring [6,7]. The authors, however, neglected to specify the mechanisms for this enigmatic process. We can only speculate that they might have envisaged some fictional free gliding of the subepicardial myocardium on the deeper layers, presuming identified 'aberrant fibres' to be capable of turning the ventricular base in the fashion of the collar of a pullover (Fig. 6). We know, however, that the subepicardial myocardial layers are continuous with the subendocardial

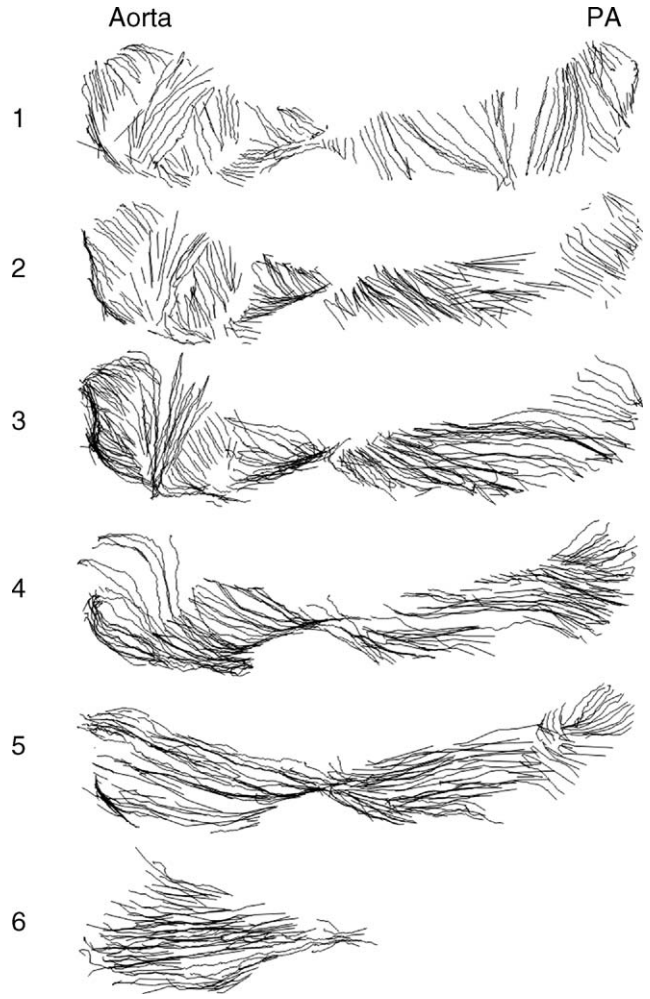

Fig. 5. The freshly excised porcine heart has been unravelled to produce a myocardial sheet (top), which was then denatured and analysed by peeling and digitising in a quasi-layered sequence (1—6). The course of fibre strands was established by following the visible 'grain'. Note the turn of the fibres upon a radial axis, therefore, all layers being aligned in variable angles relative to the long axis of the unravelled myocardial sheet. The fibres in the intact heart are continuous in the base and the apex with the thin layer of subepicardial, or so-called aberrant fibres, which are not displayed. This links giving the impression of a rope-like structure are systematically destroyed by the artefactual preparation of the myocardial sheet. Peeling off the right ventricular free wall proved to be particularly difficult because of the dense oblique intruding netting of the layers.

ones, the two joining at the ventricular base. If they worked together, therefore, they would likely pull the base downwards rather than outwards.

Alternatively, the proponents of the continuous band might have assumed that the supposed 'aberrant fibres' behave like a spring-board, which is bent inward during ventricular emptying, and which has the potential to erect late in systole or during diastole (Fig. 6). It is certainly true that, during ventricular ejection, the subepicardial layers are bent around the bulging ventricular surface. When contracting, however, they generate a centripetal force which sustains ventricular emptying, rather than contracting to widen the basal ring. It is hard, if not impossible, therefore, to defend the notion that the subepicardial fibres have a dilatory rather than a constricting function.

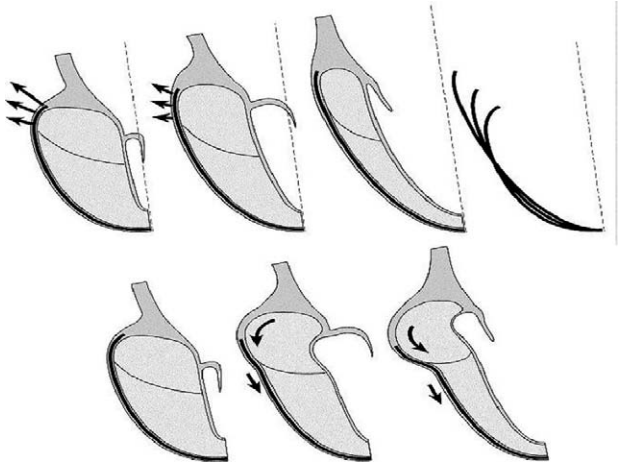

Fig. 6. The fictional action of the subepicardial layers, said to be 'aberrant fibres', as they act on the base of the left ventricle. Torrent-Guasp probably assumed that the subepicardial fibres behave like a springboard, which is bent during systole upon the convexity of the ventricle, and which actively erects while contracting pulling open the base (top sequence from left to right). Alternatively, he might have supposed that the subepicardial fibres, while gliding downward, turn the basal circular component of the muscle (the Triebwerkzeug) outward like the collar of a pullover (bottom sequence).

5. Clinical results challenging the concept of the unique myocardial band

The advent of ventricular reduction surgery [1], in which essential parts of the purported band-like structure are removed, has now provided further evidence, in our opinion, that the concept itself is untenable. By making analogy to the function of the band as a pulley, Torrent-Guasp et al. [6,7] had proposed that plication or resection of the width of the right ventricle would augment the stress in the left ventricular wall, and hence cause the dilated left ventricle to shrink (Fig. 7). If a pulley is to be efficient, however, then friction between its parts must be minimal. In the setting of the ventricular mass, friction between the components is maximized, since all adjacent segments of the proposed rope model are unified within the spatially netted matrix. Furthermore, although the supporters of the myocardial band have doubted the efficacy of reduction in ventricular radius as proposed by Batista [1], we know that the intervention is not only well tolerated, but also remarkably efficacious when performed with care and precision [37—40]. These experiences also show unequivocally that plication of the right ventricle has neither acute nor late effects on the diameter of the left ventricle.

In a more recent study, Suma et al. [5] presented data obtained using a modification of the surgical technique to reduce ventricular radius, which sought to follow the recommendations of Torrent-Guasp [33]. The aim was to preserve the purported apical loop. Thus, the left ventricular free wall was resected towards the base, purporting to leave intact the presumed apical spiralling muscle loop [1,4,6,7,33]. At all events, even this theory is itself less than perfect, since it seems to us that the chosen resection would divide both the descending and ascending segments of the apical loop described by Torrent-Guasp (Fig. 8), should such a loop exist?

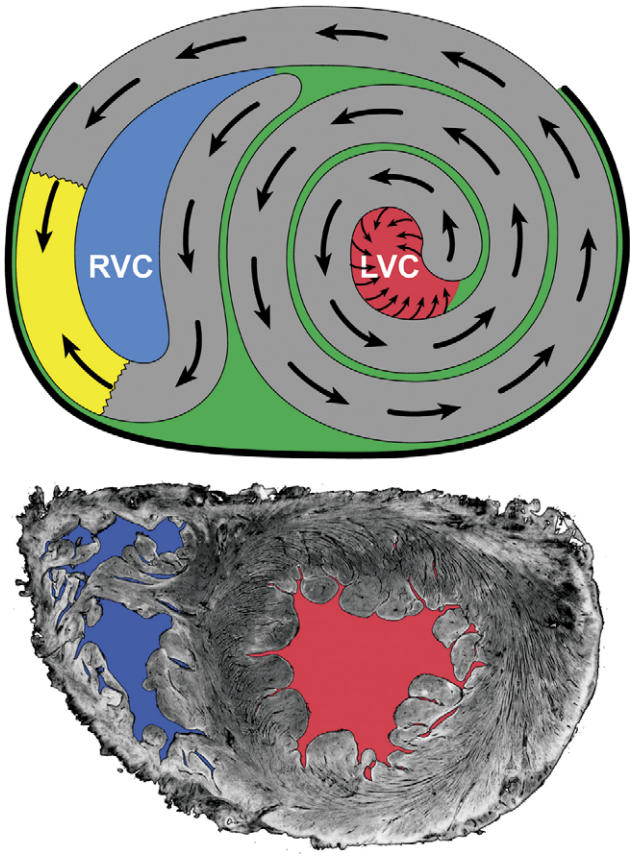

Fig. 7. In the upper cartoon, the green line represents the fictional cleavage plane, never demonstrated histologically, which was supposed to serve as a hypothetical gliding plane to permit the necessary freedom of motion of a rope in a pulley. Torrent-Guasp had assumed that, by resecting one segment (yellow) from the right ventricular wall, the resulting increment in tension would be transmitted all along the band. Based upon this misassumption, he presumed that a dilated left ventricle would start to shrink. As demonstrated in the lower cross-section through the walls of both ventricles, there is no evidence supporting the existence of such a 'cleavage plane'. The assumed freedom of motion of the 'rope in the pulley' is nothing but fiction because all segments of the alleged rope, in reality, are unified within the overall spatially netted ventricular mesh. RVC: right ventricular cavity (blue); LVC: left ventricular cavity (red); alleged cleavage plane (green); resected segment from the right ventricular free wall (yellow).

Interestingly, the stroke-volume of the Japanese patients undergoing surgery remained reduced, and over one-fifth needed postoperative support by intra-aortic balloon pumping, or even an assist device. Their long-term survival was inferior to that achieved by Konertz et al. [39,40,47], or by Schäfers et al. [42]. The functional improvement observed could also be explained on the basis of repair of the mitral valve, needed in virtually all the patients. The minimal changes in ventricular function, with an increase in ejection fraction from 20 to only 30%, reflects the minimal reduction achieved in left ventricular diastolic radius, from 80.9 ± 8.9 mm to 70.9 ± 8.6 mm. This might also be interpreted as a critical drawback of the proposed apex-preserving technique. In our experience, the size of the reconstructed left ventricle in itself would remain an indication for further radius reduction surgery [1,37,39,40,42,47]. It is gratifying, nonetheless, to note that Suma et al. [5] have adopted our preoperative routine [37] in assessing mural thickening as a guideline to identify potential disorders in regional wall motion.

6. Antagonism determines ventricular dynamics

Within the overall vectorial field of forces engendered by the myocardial mesh, it is those forces acting more or less parallel to the ventricular surfaces which contribute to ventricular emptying. Any forces acting at an angle of inclination relative to the surface planes which exceeds 45° will mostly produce ventricular dilation, since they will oppose the systolic thickening of the ventricular walls. By taking the long axis of the myocardial aggregates as a surrogate of the force vectors generated, we have been able to quantitate the proportions if dilating fibres. Such discrete populations of fibres intruding at angles of more than 45° were found in one-third of all histological sections cut using circular knives [9]. The mass of contractile units contained in these strands, however, is small. Furthermore, although the myocardial netting couples the subendocardial to the subepicardial fibres (Fig. 9), the angle of intrusion of the netting in the normal heart does not reach 45°. The netting of the myocardial meshwork alone, therefore, is unlikely to explain the source of those dilating forces, which drive end-systolic ventricular filling.

By measuring contractile forces within the ventricular walls using needle force probes on several thousand animals [32,43], and since in several hundred human patients, we have found reproducibly that, in up to four-fifths of impalements in the hearts of animals, there is a so-called 'unloading' type of signal, which means that the developed force decreased during the period of ejection while the ventricle gets smaller. This is in accord with predictions derived from the law of Laplace. In contrast, in the remaining one-fifth of all impalements, we measured a so-called 'auxotonic' signal. This means that, contrary to expectations, the developed force increases with ventricular emptying while the wall gets thicker.

In further studies in porcine and canine hearts in which we correlated our sites of impalement with histology, we found the auxotonic signal to be engendered by fibres which are inclined by less than 45° with respect to the epicardial surface lining, meaning that the predominant force vector should still be tangential. We suspected, therefore, that connective tissue would be needed to steepen the direction of intrusion of the contractile forces. It cannot be coincidence, therefore, that we measured a significantly higher incidence of auxotonic force signals, up to four-fifths, in patients suffering from long-term ischaemic heart disease when compared to our laboratory animals. In a population of native Brazilians undergoing surgery for acute rheumatic mitral valvular disease, in contrast, the figures were not different from our laboratory results.

We conclude, therefore, that connective tissue is essentially involved in intercepting the transmission of forces from the strictly tangential to the obliquely intruding direction. Thus, it could be that the myocardium makes use of some obliquely intruding bridging produced by connective tissue so as to function in antagonistic fashion, with the prevailing myocardial tangential structures acting to produce

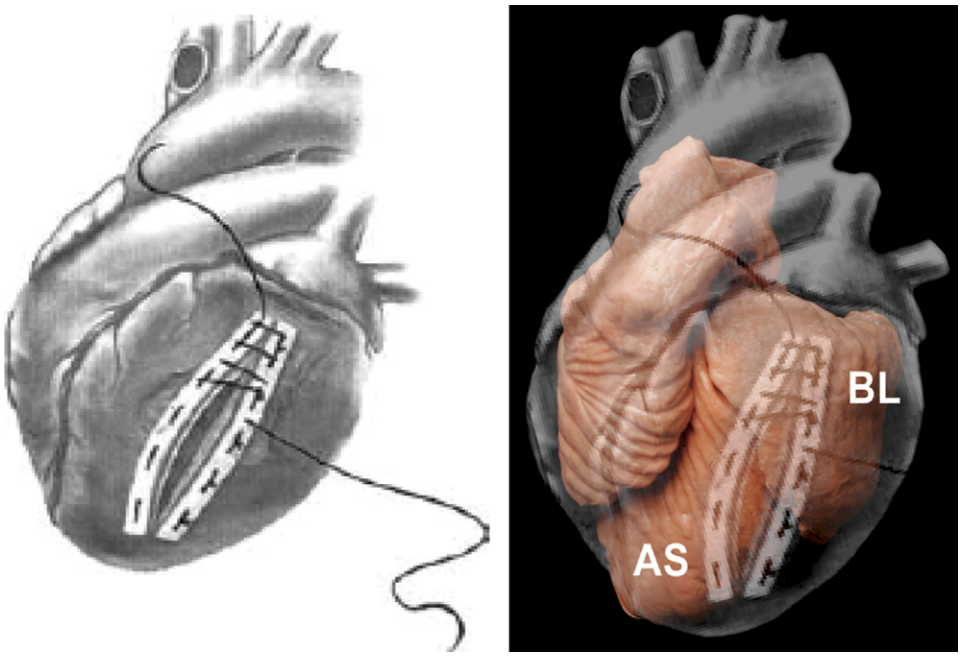

Fig. 8. Synopsis of the site of the incision proposed by Suma et al. compared with the band model of Torrent-Guasp. The proposed incision would divide the ascending segment (AS) of the apical loop, its subjacent descending segment (not visible), and the basal loop (BL), if these components truly existed, which of course they do not.

constriction, while the combined obliquely intruding trajectories counteract systolic wall thickening, and thereby control the inward displacement of the inner myocardial layers relative to the epicardium. The activation of both the tangential and the intruding myocardial fibres, nonetheless, occurs more or less synchronously. The less the contractile system is allowed to shorten, the longer is its activation. While tangential fibres shorten during ventricular ejection, the obliquely intruding structures are restrained in shortening, because mural thickness increases during systole. According to Jewell and Wilkie [44], and Brutsaert et al. [34—36], the duration of the active state increases the more the fibre is hindered in its shortening. We measured the auxotonic activity in the intruding alignment to persist up to 140 ms longer than that in the prevailing tangential muscle mass [32]. This confirms our notion that, at the end of ventricular emptying, a measurable amount of antagonistic forces persists, potentially sustaining ventricular dilation.

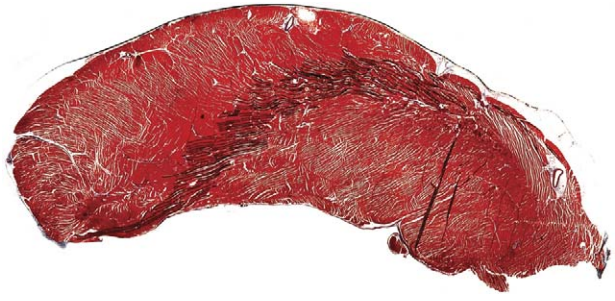

Fig. 9. Cross-section through the left ventricular base obtained by using a semicircular twin-knife. The upper edge is the epicardium and the lower edge is the endocardium. The right end is taken from near the septum. The left end originates from between obtuse margin and inferior ventricular groove. Note the labelled array of axially sectioned fibres which extends from the epicardium to the endocardium. The myocardial aggregates within the identified tract are densely interwoven, thus representing a contractile continuum. Note also that the subepicardial and subendocardial tails of the array are thin, while in the midportion the fibres are grouped in thicker layers. The reason is that angles of rotation of the fibres in the radial axis are much greater near the bordering layers than in the midportion.

7. Path morphology and disturbed ventricular function

Disarray of the myocardial aggregates is a well-recognised part of myocardial pathology [22]. Therefore, aberrant forces need to be implemented into the clinical and theoretical evaluation of ventricular dynamics. In the hypertrophied heart, the obliquity of the intruding netting is progressively erected to higher angles of inclination so that dilating forces increase. As a result, the tangential fibres need to cope with an augmenting intrinsic load, which sustains their own hypertrophy, thus sustaining a vicious circle. In the setting of myocardial fibrosis, the coupling between the epicardium and the endocardium will increase in rigidity [41,45,46]. This again, we presume, will augment the intrinsic antagonism and sustain myocardial hypertrophy. In both settings, a point might be reached from which dilating forces will increase to the extent that the diseased ventricle eventually will dilate on the basis of its intrinsic structure.

8. Conclusion

In keeping with the basic philosophy underpinning the thoughts of Torrent-Guasp [33], we also recognise the erectile properties of the myocardium that permit ventricular shaping, diastolic unfolding, and ventricular dilation when the heart is diseased. Unlike Torrent-Guasp et al.

[6,7,33], however, we do not assign this erectile property to a unique myocardial band. Indeed, we are not aware of any supporting evidence from independent anatomists, nor of any validating histologic studies, that endorse the concept of a continuous myocardial band that extends from the aorta to the pulmonary trunk. On the contrary, we believe the observed changes reflect the three-dimensional netted mesh to be found throughout the ventricular mass. The aggregates of myocardium are predominantly tangential, yet throughout the wall populations of myocytes are to be found that intrude at angles up to 35° relative to the epicardium. Because of this mixed composition, each part of the ventricular wall possesses the properties of stiffening by self-bracing, self-erecting, self-shaping and self-limiting in shortening distances. In this respect, therefore, the ventricular myocardium resembles the tongue in terms of both its structure and function. The very arrangement of the supporting connective tissue, which is sufficient to exclude the concept of a unique myocardial band, is likely to play a pivotal role in the function of the normal heart, but particularly in the diseased heart.

Unlike the hypothesis advanced by Torrent-Guasp, our own concept does not require sequential activation to be necessary for the ventricular walls first to move inwards and then, during the period of relaxation, to be unfolded. Rather, in compliance with established wisdom, and confirmed by our direct measurements of regional contractile forces, our concept recognises that myocardial activation occurs nearly synchronously. Part of the necessary late dilating forces are explained on the basis of persisting tension in the obliquely intruding populations of fibres, which are confined in their degree of shortening during the period of ejection.

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