Torrent Guasp’s [1] continuous ventricular myocardial band hypothesis in cardiac mechanics implies a series of associated muscular movements. These occur in the band eliciting left ventricular narrowing, shortening, twisting, lengthening, untwisting and expansion phases during the cardiac cycle.

According to Guasp [1], longitudinal diffusion of stimuli along the ventricular myocardial band explained heart performance (Figure 1, lower pannel). However, this sequential “peristaltic” activation did not correlate with some currently well-known fundamental phenomena, as clockwise and counter-clockwise twisting at the left ventricular apex and base, which are mainly responsible for its mechanical efficiency (Figure 1, upper pannel). In an attempt to explain the mechanism of muscle twisting, we publish the sequence of ventricular electrical activation by means of simultaneous three-dimensional endo-epicardial segment mapping [2].

Electrical activation is the consequence of the propagation of stimuli through the muscular structure of the heart. The cardiac mechanism of suction and ejection requires structural-functional integration capable of unraveling the different dynamic aspects arising from the propagation of excitation. This indicates that the diastolic phase is an active process of contraction producing increasing suction (due to its similarity we have called it “plunger mechanism”), which at a certain level of intra ventricular pressure opens the atrio ventricular valves producing rapid filling.

Stimulus propagation and left ventricular torsion

The integrated endo-epicardial three-dimensional mapping performed in that research supports [2], the activation model that propagates along the descending and ascending bands.

In that experience we found a stimulus trajectory different from that described by Guasp [1], but which explains the twisting phase of the heart, defined as the opposing rotational movement of the base and apex. At the point of band intersection the activation propagates from the endocardium to the epicardium (radial propagation), that is, from the descending to the ascending band.

From this point onwards, the ascending band depolarizes in two senses: towards the apex and towards the base, at the same time that the descending band completes its activation towards the apex. Thus, two essential phenomena occur:

1. As the apical loop depolarizes from band intersection in two simultaneous wave fronts (from the descending and from the ascending bands) it generates their synchronized contraction.

2. The activation of the ascending band propagates from band intersection in two opposing directions: towards the apex and towards the base. The resulting mechanical contraction will also have a divergent direction, giving origin to the apical and basal clockwise and counter-clockwise rotations, respectively.

According to Lewis and Rothschild [3], stimuli diffused from the endocardium to the epicardium through the muscle walls. Contrary to this concept, Robb and Robb [4] published in 1936 that stimuli propagation occurred longitudinally, and in 1942 [5] inquired: “How is it possible that impulse transmission occurs from the endocardial to the epicardial surface... given that the ventricular wall is composed of well differentiated bundles, separated by sheaths of connective tissue?”. Surprisingly, according to their experimental studies, Armour and Randall [6] concluded that stimuli diffusion in the left ventricular anterior wall was generated from the endocardium to the epicardium. This local event in the left ventricular anterior wall contrasts with previous concepts and with the remaining
Thus, a twisting mechanism similar to “wringing a towel” would be by allowing opposing forces on its longitudinal axis, generating the ascending band plays an essential role in ventricular twisting along the ventricular myocardial band, radial propagation towards (radial propagation). Although the electrical conduction progresses because the electrical stimulation propagates towards the descending band (axial propagation) and simultaneously to the ascending band is feasible due to the twisting movement. This action is achieved on the onset of ejection during an interval lasting 20% of the systolic phase. The pressure generated to eject the highest amount of blood at the beginning of diastole, acts as a tight tutor keeping the apex immobile. The ascending band, rigid in systole and at the descending in systole and ascending in diastole. This is explained fact that the apex remains fixed, is due to the movement of the base, produced longitudinally, as the ring contracts before the apex. The muscle mass where the electrical activity of subepicardial muscle bundles takes place before those in the subendocardium. However, this discrepancy of the impulse transmission theory through the ventricular myocardial band was not resolved until our research shed light on its understanding, with patent relevant considerations for cardiac mechanics. In 1980, Guasp [7] expressed: “The subendocardial layers contracted by the descending segment come into activity before the subepicardial ones, which are components of the ascending segment” and in 1998 [8] he reaffirmed “the descending segment and then the ascending segment successively enter into activity”. Towards 2001, Buckberg et al. [9] ratified the hypothesis that excitation spreads uni directionally along the ventricular myocardial band. Our research modifies these concepts since stimulus propagation is simultaneously axial and radial [10].

The ventricular narrowing phase (isovolumic systole) at the beginning of systole is produced by the contraction of the basal loop right and left segments. The overlapping shortening phase is due to the descent of the base, at the same time as twisting occurs, which is produced longitudinally, as the ring contracts before the apex. The fact that the apex remains fixed, is due to the movement of the base, descending in systole and ascending in diastole. This is explained better because the ascending band, rigid in systole and at the beginning of diastole, acts as a tight tutor keeping the apex immobile. The pressure generated to eject the highest amount of blood at the onset of ejection during an interval lasting 20% of the systolic phase is feasible due to the twisting movement. This action is achieved because the electrical stimulation propagates towards the descending band (axial propagation) and simultaneously to the ascending band (radial propagation). Although the electrical conduction progresses along the ventricular myocardial band, radial propagation towards the ascending band plays an essential role in ventricular twisting by allowing opposing forces on its longitudinal axis, generating the necessary intra ventricular pressure to achieve abrupt blood ejection. Thus, a twisting mechanism similar to “wringing a towel” would be produced.

The historical term of systole and diastole did not take into account the meaning of electrical activation and contraction, but only the hemodynamic concept of ventricular ejection and filling. It is therefore necessary to find a relationship between activation and the mechanical outcome. The explanation is provided by the simultaneous axial and radial electrical conduction when it reaches band intersection, also confirmed by the spatial arrangement of fibers, with subendocardial fibers on the right side and subepicardial fibers on the left. This layout also agrees with the evolutionary loop of the circulatory system forming the two developed ventricles in birds and mammals. Torsion -different rotation between the apex and base of the heart- generates: a) high pressures, b) reduces ventricular stress, and c) homogenizes its distribution in the ventricular wall thickness.

Guasp [8] declared that “the sequence of ventricular muscle entry into activity in the different ventricular regions takes place along the band”, similarly to a peristaltic movement. Then, how could the ventricle achieve its twisting movement, since this action requires two opposing forces at the same time? Unidirectional activation does not explain twisting or the evolutionary-structural development designed to apply a force capable of ejecting the ventricular content at a speed of 300 cm/s at low energetic cost. This is understood by the simultaneous axial and radial activation we have found.

**References**