Letter to the Editor: Atrioventricular plane displacement is not the sole mechanism of atrial and ventricular refill

Per M. Arvidsson,1 Marcus Carlsson,1 Sándor J. Kovács,2 and Håkan Arheden1
1Department of Clinical Physiology, Lund University, and Lund University Hospital, Lund, Sweden; and 2Cardiovascular Biophysics Laboratory, Cardiovascular Division, Washington University School of Medicine, Saint Louis, Missouri

WE HAVE READ WITH GREAT INTEREST the recent Perspective by Arutunyan (2), titled “Atrioventricular plane displacement is the sole mechanism of atrial and ventricular refill,” in which the author revisited important, and perhaps not well-known, aspects of cardiac pumping physiology in an attempt to uncover a “universal” explanation for how cardiac filling is achieved. There is a considerable body of evidence showing that atrioventricular (AV) plane displacement (AVPD) is not the sole mechanism of atrial and ventricular refill. The explanation proposed in the recent Perspective would therefore benefit from contextualization with the existing data. Arutunyan approaches the problem of cardiac filling from four main perspectives: 1) constant-volume pumping, 2) the importance of momentum conservation, 3) atrial contraction, and 4) pressure gradients. We will address these accordingly.

Constant-volume pumping. The concept of constant-volume pumping has interested readers of this journal since its first edition in 1898 (24). Arutunyan first concludes that “ventricular contractions cause circulation of the blood by pumping blood into arteries and simultaneously pulling it from large veins. Ejection and pulling momentums of the blood act as counterpartables, negate each other, and provide spatial stability to the heart during contraction.” In other words, the heart is described as a constant-volume, self-reciprocating pump that balances output with filling. As the ventricles contract, the AV plane moves longitudinally toward the apex, as first described by Leonardo da Vinci in the 15th century (31). Longitudinal pumping is mediated through AVPD and is quantified as the product of AVPD and epicardial short-axis area (23). Given that both the epicardial apex (residing within the spatially fixed pericardial sac) and the back of the atria (anchored in the mediastinum by pulmonary veins) of the heart remain stationary, ventricular contraction shortens the long-axis dimension of the chamber by displacing the valve planes toward the apex and by necessity must lead to a reciprocal enlargement of the atria by aspiration of extracardiac blood. Perfectly balanced inflows and outflows would result in a perfectly constant-volume heart, as first postulated by Hamilton and Rompf (16) in 1932, an appealing concept since no work is wasted moving extracardiac tissues. This view, however, is only an approximation in light of the existing data.

Initial investigations by Hoffman and Ritman (18) supported the idea, finding an average total atrial volume variation (THVV) of 2.7% in dogs. The authors state, “the combined atrial filling appears to approximate the volume ejected by the combined right and left ventricles” (18). Later studies have investigated THVV in humans using magnetic resonance imaging and have found numbers of 5-11% (4, 8). Total heart volume diminishes during systole and recovers during diastole, indicating that venous return to the heart is not temporally matched to ventricular ejection (26, 29, 32). Furthermore, THVV was found to increase and AVPD decreased during moderate intensity supine exercise (30). For perfect constant-volume pumping, atrial filling must be matched to ventricular ejection, but the atrial reservoir only houses 40–60% of the ventricular stroke volumes (19, 20). The remaining blood, the conduit fraction, is drawn from the veins through the atria during the early rapid filling phase, seen in pulmonary venous flow profiles as the D wave whose volume corresponds to the total heart volume recovery during early diastole (5). The D wave gives rise to the “crescent effect” as seen on cardiac MRI (32), and its existence proves that the ventricular stroke volume is not fully compensated by the pulmonary vein S wave; if venous return to the heart was entirely balanced and driven by AVPD, there would be no D wave. On average, 70% of atrial filling occurs during systole, again incompatible with perfectly constant-volume pumping (29). Furthermore, healthy volunteers display a 2- to 3-mm cardiac center of volume variation, indicating that the heart shifts position over the cardiac cycle (10). And, finally, the existence of cardiac ballistography, i.e., measurement of total body motion due to cardiac pumping, demonstrates that the ejection and filling momentums of the heart do not fully cancel each other out (28). Therefore, the heart is not a perfectly balanced, self-reciprocating pump, nor is it a perfect constant-volume pump. Rather, the contents of the pericardial sac can be more accurately described as a near-constant volume pump that changes by ~8% over the cardiac cycle.

The importance of momentum conservation. Arutunyan next concludes that “venous return to the chambers of the heart is a momentum-driven process that is powered by ventricular contraction” and elucidates that “once forcefully accelerated, the columns of blood in the veins will inertially continue to flow toward the heart until the momentums of the columns are negated by a mechanical or higher pressure barrier.”

Momentum has been suggested to contribute to ventricular function in various ways. Noble (25) demonstrated in 1968 that momentum contributes to ventricular ejection in dogs, and Kilner et al. (21) postulated that momentum conservation in organized, swirling blood flow within the heart might contribute to optimize cardiac function. This would benefit diastolic filling, with the greatest potential benefit during exercise when diastole is markedly shortened. In atrial blood, kinetic energy (KE), a measure closely connected to momentum, is determined in part by AVPD during systole, but a negligible amount of KE “spills over” from systole to diastole in the left atrium [Arvidsson et al. (3), Figs. 3 and 6A]. Data from the same article support that for the right heart, blood momentum may benefit from contextualization with the existing data.
contribute significantly to ventricular filling, as KE was preserved in rotational flow patterns of right atrial blood [Arvidsson et al. (3), Fig. 11]. Momentum contribution to cardiac function therefore remains an interesting concept with some support in data.

Arutunyan notes, “During ventricular contraction, blood, accelerated in the venous trees, finds room first in refilling atria... The momentums of accelerated blood columns then open the AV valves at the end of isovolumic relaxation, and blood rushes into the ventricles.” During early diastolic filling, however, KE increases in both atria and ventricles, which means that blood is being accelerated (3). This acceleration is caused by ventricular elastic recoil, which generates suction, as shown in the left ventricle (LV) in dogs by Brecher (7), in turtles by Kraner and Ogden (22), and in the right ventricle by Sabbah et al. (27). Diastolic recoil is driven by the myocyte protein titin, a molecular, linear, bidirectional spring that provides a restoring force after being compressed (17), and by elastic elements in the extracellular matrix (15). Ventricular suction is, to the best of our knowledge, presently the only mechanistic explanatory model that predicts suction and accounts for the recovery of the total heart volume by the pulmonary vein D wave (3, 26, 32). The mitral valve then opens because of the pressure decrease in the ventricle, not because of inertial effects of the inflowing blood, as evidenced by the chamber expanding faster than it can fill (dP/dV < 0) at mitral valve opening, inscribing a negative pressure gradient (LV pressure < left atrial pressure) from ventricle to atrium at the onset of early filling (E-wave acceleration) (12).

The role of atrial contraction. Arutunyan discusses how atrial contraction influences cardiac performance and concludes that “atrial contractions play an important role in reshaping ventricles and veins for each new contraction.” The first part of that statement is uncontroversial; it is well known not only that atrial contraction “tops off” the ventricular blood volume but also that the stretching of ventricular myocytes primes the LV for more efficient ejection through the Frank-Starling mechanism.

The reshaping of veins, on the other hand, is a more challenging concept. Arutunyan expounds, “Along with the pressure gradient-driven slow blood flow, the small back pressure wave of atrial contraction helps to open the veins squeezed from the previous contraction and prepare them for a new AVPD-driven process as a result of back pressure wave of atrial contraction and AVPD, veins passively ‘behave’ as stretchable blood storages operating during the cardiac cycle.” While the attribution of positive effects to the atrial wave reversal volume (AWRV) is innovative, there is no current evidence supporting this theory. During atrial contraction, the orifices of the pulmonary veins act like sphincters (6), thereby minimizing the AWRV. The AWRV is known to be larger in the right atrium due to larger vessel orifices and accounts for ~11% of THV (11). We suggest that venous backflow is counterintuitive from a functional perspective. Backflow would increase THV, cause a pendular movement of the blood and hence waste energy, and decrease the capacity to accommodate venous return.

Furthermore, Arutunyan states that “During the contraction, atrial push of blood into ventricles occur via the pull-up of the ventricular edges. ... During this process, in essence, the contracting edges of atria slide back over the blood.” Such an encompassing move would transfer blood from the atrium into the ventricle without accelerating any blood. However, studies on intracardiac KE and the acceleration feature of the Doppler A wave demonstrate that encompassing of blood is only a partial explanation. KE manifesting as transmitral flow increases during atrial contraction (Doppler A wave), indicating that external work is being performed on the blood. This manifests as transient fluid acceleration, the Doppler A wave, rather than the blood remaining stationary in space and only being encompassed by tissue sliding over it (3, 9, 13, 14). Thus atrial contraction acts both by sliding past the blood and accelerating it.

Venous pressure and AVPD. Arutunyan writes, “For example, 5–7 mm pressure difference between venules and veins provides a slow flow of blood toward the heart. But a subtle pressure difference between the large veins and the heart chambers, as qualitative estimates show, is not sufficient for filling the ventricle within a diastole.” This hypothesis can be rejected on the basis of previous in vivo experiments, which demonstrated sufficient LV filling in dogs occurring with a peak pressure gradient from left atrium to LV of 2.8 ± 0.3 mmHg (12). Furthermore, Arutunyan remarks, “the systolic pressure gradient powers blood circulation up to the large veins close to the heart, but the beat-to-beat transfer of blood to atria and ventricle occurs via a forceful, AVPD-driven suction, pull-up process that is powered by ventricular systole.” Pulsatile flow is present in the veins of the legs and contains waveforms related to both respiration and heart frequency (1), suggesting that venous flow is affected by both venous pressure and AVPD even in distal parts of the vasculature.

In conclusion, AVPD is clearly an important and a definite major contributor to cardiac filling. Substantial data from the literature, however, suggest that the heart fills through a combination of AV plane reciprocity, diastolic recoil, venous pressure, and momentum conservation with significant differences between the systemic and pulmonary circulations. Together, these mechanisms make the heart a near-constant volume pump, but the notion of a universal explanation for cardiac filling based solely on AVPD lacks support in available data.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

P.M.A. drafted manuscript; P.M.A., C.M., S.J.K., and H.A. edited and revised manuscript; P.M.A., C.M., S.J.K., and H.A. approved final version of manuscript.

REFERENCES

5. Bowman AW, Kovács SJ. Left atrial conduit volume is generated by deviation from the constant-volume state of the left heart: a combined