Mechanical events and the pressure–volume relationships

Emrys Kirkman

Abstract

Depolarization of cardiac muscle fibres spreads from fibre to fibre throughout the myocardium. In a single fibre, contraction starts just after depolarization and lasts until just after repolarization is complete. The atria contract, completing the filling of the ventricles and thus enhancing their action. In the absence of effective atrial contraction (e.g. atrial fibrillation) cardiac output is decreased on average by 15%. During diastole, when cardiac muscle is relaxed, blood returns to the heart and passes through the atrioventricular (AV) valves into the ventricles. The semilunar valves, between the ventricles and the arteries, are closed as arterial pressure exceeds ventricular pressure. Under normal circumstances, 70% of ventricular filling occurs by late diastole.

Keywords Extrinsic regulation; Frank–Starling mechanism; pressure–volume loops

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Learning objectives

After reading this article, you should be able to:

- describe the mechanical phases of the cardiac cycle and relate them to the electrical events and the heart sounds
- explain the relationship between pressure and flow in the heart
- explain the intrinsic and extrinsic mechanisms regulating the force of myocardial contraction

The depolarization of cardiac muscle fibres spreads from fibre to fibre throughout the myocardium. In a single fibre, contraction starts just after depolarization and lasts until just after repolarization is complete. The atria contract, completing the filling of the ventricles and thus enhancing their action. In the absence of effective atrial contraction (e.g. atrial fibrillation) cardiac output is decreased on average by 15%.

During diastole, when cardiac muscle is relaxed, blood returns to the heart and passes through the atrioventricular (AV) valves into the ventricles. The semilunar valves, between the ventricles and the arteries, are closed as arterial pressure exceeds ventricular pressure (Figure 1). Under normal circumstances, 70% of ventricular filling occurs by late diastole.

Immediately after the P wave of the ECG, the atrial muscle fibres contract, giving atrial systole. This helps to push a final amount of blood towards the ventricles. Although there are no valves between the atria and the veins, only small amounts of blood regurgitate into the veins because atrial contraction tends to narrow the orifices between the atria and the superior and inferior venae cavae and the pulmonary veins. In addition, the inertia of blood flowing towards the heart tends to keep the blood within it. The small regurgitation of blood that does occur into the veins gives rise to the 'a' wave of the venous pulse (Figure 1). Thereafter, the action potentials sweep through the ventricles (giving rise to the QRS complex) causing ventricular contraction and ventricular systole. During the first phase of ventricular systole, the overall muscle fibre length is not shortened. This is because, as the filaments slide, tension is generated in the elastic elements of the walls of the ventricles, which raises pressure in the ventricles above that in the atria. This causes closure of the AV valves (and generation of the first heart sound). However, ventricular pressure is initially below arterial pressure and therefore the semilunar valves remained closed. Thereafter, pressure increases rapidly within the ventricles and, as there is no change in volume, this is termed the isovolumetric phase of ventricular systole (Figure 1). As ventricular pressure continues to rise it eventually exceeds arterial pressure (at about 10.8 kPa (80 mm Hg) in the left and 1.3 kPa (10 mm Hg) in the right ventricle) and blood is ejected rapidly into the arteries so that there is a large and rapid decrease in ventricular volume, accelerating blood flow into the arteries and increasing arterial pressure. This period of rapid ejection accounts for about the first third of ejection time and culminates in the attainment of the peak arterial and ventricular pressures, about 16.0 kPa (120 mm Hg) and 3.3 kPa (25 mm Hg), respectively, in the left ventricle/aorta and right ventricle/pulmonary artery (Figure 1), though there is considerable normal variation between and within individuals. Thereafter, there is a further period of reduced ejection as blood continues to be ejected into the arteries. However, during this phase, blood flow decelerates as potential energy stored in the elastic wall of the arteries reverses the pressure gradient and arterial pressure exceeds ventricular pressure (Figure 1). Despite this reversed pressure gradient, flow continues into the arteries because of its momentum. However, pressure in the arteries falls in this phase because the run-off of blood into the tissues exceeds the amount of blood entering the arteries from the ventricles.

In a normal heart, immediately before ventricular systole, the ventricles contain about 130 ml blood (end-diastolic ventricular volume, EDV). During the entire ventricular ejection phase, about 70–90 ml of blood is ejected from each ventricle (stroke volume, SV), leaving about 50 ml of blood in the ventricles (endsystolic ventricular volume). Ejection fraction (SV/EDV) is thus about 65%. The rapid increase in arterial pressure causes the arteries in some places to impact with the wall of adjacent veins. This, together with a pushing of the tricuspid valves towards the atria in early systole, contributes to the second venous pressure wave of the cardiac cycle: the c wave (Figure 1).

About 50 ms after the end of the myocardial action potential the fibres begin to relax (during and immediately after the T wave of the ECG); this is ventricular diastole. The rapid reduction

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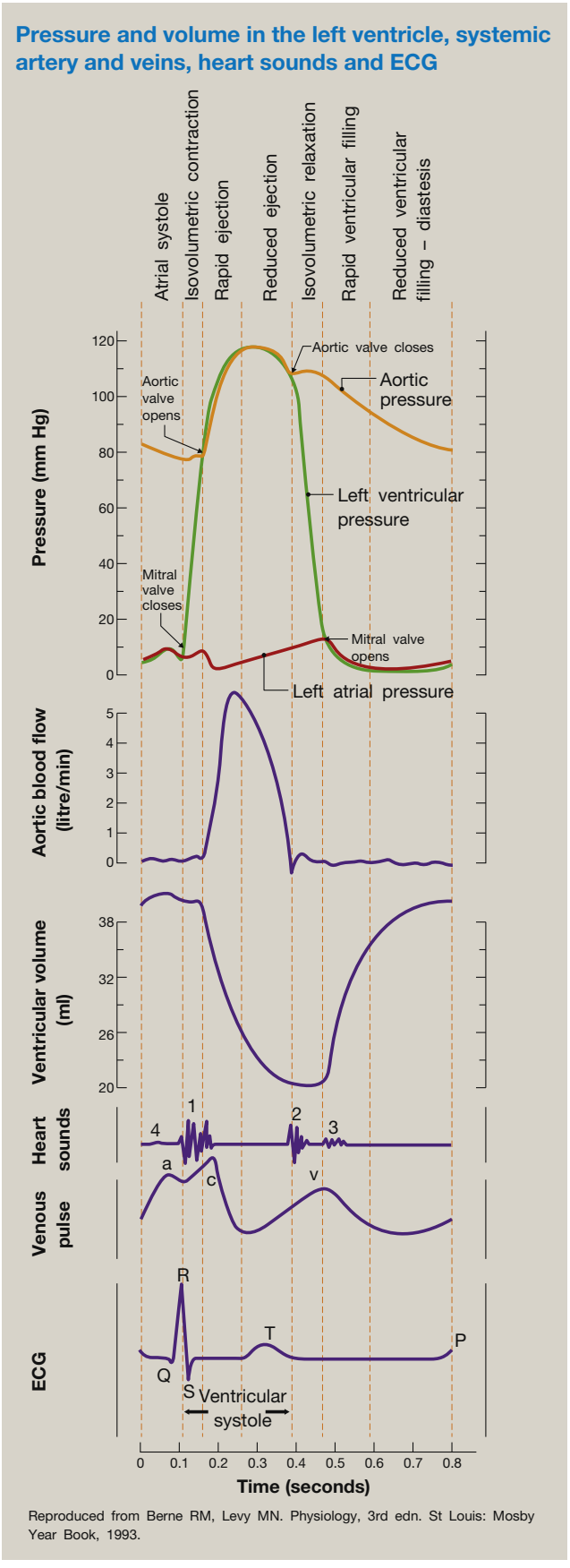

Figure 1

in ventricular pressure results in it falling quickly below the arterial pressure, and the threatened regurgitation of blood from the arteries towards the ventricles causes the semilunar valves to snap shut, giving rise to the second heart sound and the dicrotic notch, evident on the arterial pressure waveform. Pressure within the arteries is now kept higher than ventricular pressure because of the elastic recoil of elements of the arterial wall, which were stretched during systole.

During the first phase of ventricular diastole both semilunar and AV valves are closed. Therefore, the rapidly falling pressure is not accompanied by a change in volume and this phase is called isovolumetric relaxation. Thereafter, ventricular pressure falls below atrial pressure and the AV valves open. Initially, blood flow into the ventricles is rapid, giving a sharp rise in intraventricular volume (Figure 1), and a fall in venous pressure (which had previously been increasing as blood was unable to flow from the atria through the closed interventricular valves). This sharp fall in venous pressure produces the third venous pressure pulse of the cardiac cycle: the v wave (Figure 1).

Following the rapid filling phase, blood continues to flow into the ventricles during the remainder of diastole, but now at a reduced rate. This phase is termed diastesis, at the end of which, ventricular filling is about 70% complete, before the whole cycle is initiated again by atrial systole immediately after the next P wave of the ECG.

It should be evident from this description that atrial contraction is not essential for ventricular filling, which occurs even in atrial fibrillation. At lower heart rates, atrial contraction is even less important because ventricular filling is essentially complete by the end of diastesis. However, as heart rate increases there is a much more marked reduction in diastolic than in systolic time. Thus, atrial systole becomes increasingly important for ventricular filling during tachycardia.

Regulation of cardiac contraction

The two main mechanisms important for regulating the force of cardiac contraction and stroke volume are those that are intrinsic to cardiac muscle and those that are mediated by nervous and hormonal control of the heart.

Intrinsic mechanisms: the Frank–Starling mechanism

A relationship exists between the maximal force a muscle fibre can produce and its length immediately before contraction (preload). This was first described for cardiac muscle by Frank in 1895. He showed that over a limited range of preloads, increasing preload can increase force of contraction. This idea was refined by Starling in a series of studies published in 1914, which showed that an increase in right atrial pressure led to increased force of ventricular contraction and thus stroke volume. This can be represented graphically (Figure 2) as a relationship between end-diastolic volume (which dictates the length of the ventricular muscle fibres just before systole) and an index of cardiac muscle force of contraction: in practice this is taken as either stroke volume or stroke work.

Normal cardiac function is represented by a point on the upward slope of this relationship (Figure 2). Thus, if venous return and end-diastolic volume increase (increased preload), the heart responds by producing a more forceful contraction to eject the excess blood.

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