THE CIRCULATORY PHYSIOLOGY OF HELIX POMATIA

II. THE ISOLATED HEART

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(Received 30 January 1973)

INTRODUCTION

This work was undertaken to study the mechanical factors affecting the function of the isolated heart of *Helix pomatia* in order to help understand the variations in heart activity observed in intact animals.

Biedermann (1884), working upon the isolated heart of *Helix*, observed that the frequency of beat increased with distension of the heart. Biering (1929) found that in the isolated *Helix* heart the frequency and output varied directly with the venous pressure. Schwartzkopff (1954) confirmed this and also found that the frequency and output varied inversely with the arterial pressure and peripheral resistance. Civil & Thompson (1972) found that the isolated heart of *Helix* would only pump fluid effectively when surrounded by a sealed chamber of fixed volume (artificial pericardium). They found that the heart output increased as the venous pressure rose and that reducing the volume of fluid in the artificial pericardium so that the heart increased in volume resulted in an increase in heart output.

MATERIALS AND METHODS

Isolated hearts were prepared by ligaturing Polythene cannulae (1 mm bore tapering to 0.5 mm bore) into the aorta and pulmonary vein near the heart. In the first series of experiments the tip of the aortic cannula passed through the first aortic valve into the ventricle, whilst in the second series the cannula was placed in the anterior (cephalic) aorta so as not to interfere with the action of this valve, and the posterior (visceral) aorta was tied off (fig. 1 of Sommerville, 1973a).

Each cannula was connected to a vertical glass tube containing Ringer solution in which floated a hollow perspex cylinder supporting a glass mast. A marker at the top of each mast recorded the vertical movements of the cylinder on a lightly smoked kymograph drum. The sides of the column and floats were treated with silicone. Calibration experiments showed that when the heart pumped through 1 ml of fluid the float connected to the ventricle rose by 30.3 mm and the float connected to the auricle fell by 14.1 mm. In the second series of experiments the columns were connected to 600 ml reservoirs (Fig. 1). These slightly reduced the immediate response of the floats to fluid displacement but the pressure differential was maintained reasonably constant despite the pumping activity of the heart.

The heart preparations were perfused with Hedon-Fleig Ringer solution to which a small quantity of snail blood was added. In the second series of experiments it was
found necessary to add a minimal quantity of 5% snail blood in order to obtain regular heart contractions. The experiments were carried out within 12 h of preparation.

RESULTS

Each contraction of the ventricle caused the arterial float to move upwards by 1–2 mm, representing a stroke volume of 0.03–0.07 ml.

In the first series of experiments in which the first aortic valve (Sommerville, 1973a) was not included in the preparation it was noted that about four out of five hearts would not begin to beat until 0.1 ml of snail blood had been introduced into the perfusion fluid near the venous cannula. In the second series of experiments where the first aortic valve was present there was about 1 l of circulating fluid in the system and no preparation would beat rhythmically unless a minimal concentration of 5% snail blood was present. The rate of beat increased as more blood was added up to a concentration of 20%, but beyond this no further increase in heart rate was observed.

The perfused hearts would beat rhythmically for up to 3 days but all experiments were carried out within 12 h of preparation.

The arterial and venous pressures were measured, taking the level of the fluid surrounding the heart as zero; they ranged from 0 to 10 cm H₂O. In the absence of the first aortic valve fluid was passed through the heart only when there was a positive pressure gradient from vein to artery of 4–5 cm H₂O, whereas with a functional first aortic valve the heart was competent when there was a negative pressure gradient of up to 8 cm H₂O.
In these experiments, carried out before the importance of the first aortic valve was appreciated and using preparations without this valve, it was found that:

1. At a constant venous pressure the frequency of beat varied directly and the amplitude of beat inversely with the arterial pressure.

2. At a constant arterial pressure the frequency of beat varied inversely and the amplitude of beat varied directly with the venous pressure.

3. At a constant venous and arterial pressure the frequency of beat varied inversely and the amplitude directly with the depth of the fluid surrounding the heart.

In these experiments, where the first aortic valve was functional, it was found that:

1. At a constant venous pressure, variation in the arterial pressure did not affect the frequency of beat as much as in the first experiments, and if any change occurred it was in the reverse direction, i.e. the frequency decreased as the arterial pressure rose. Some preparations showed no change in frequency. There was never a significant change in amplitude of beat.

2. At a constant arterial pressure the frequency and amplitude of beat varied directly with the venous pressure.

3. The heart output increased as the arterial pressure fell or the venous pressure rose.

These results are summarized in the graphs shown in Figs. 2–4.

If the first aortic valve was damaged during preparation and failed to function, the results of varying the venous and arterial pressures were exactly as in the first series of experiments.
Fig. 3. The variation in heart rate with venous pressure at a constant arterial pressure. O—O, First aortic valve present; arterial pressure 3.7 cm H₂O; correlation coefficient 0.97; significance, $P < 0.01$. ×—×, First aortic valve absent; arterial pressure 6.5 cm H₂O; correlation coefficient 0.99; significance, $P < 0.01$.

Fig. 4. The variation in heart output with the ratio of arterial to venous pressure. A/V, correlation coefficient 0.838; significance, $P = 0.01$.

**DISCUSSION**

The failure of the competent isolated heart to beat rhythmically unless 5% of snail blood was present in the perfusing fluid is in agreement with the finding of Jullien *et al.* (1959). Schwartzkopff (1954) obtained continuous rhythmical beating using only an inorganic perfusion fluid but the present work suggests that this is possible only if a small volume of recirculating perfusion fluid is used when sufficient of some essential factor has been carried over with, or manufactured by the preparation. Hogben (1925) claimed that a mineral Ringer solution would support rhythmical
activity in the isolated Helix heart so long as the ratio of Ca\textsuperscript{2+}:K\textsuperscript{+} was at least 3:2. Only Schwartzkopff used a Ringer conforming to this ionic ratio so it is possible that the organic factor enables the heart to compensate for an inadequate mineral composition of the perfusing solution. However, Civil & Thompson (1972) used a Ringer with a Ca\textsuperscript{2+}:K\textsuperscript{+} ratio greater than 3:2 and found that the initial beat rate of 10/min had fallen to 2-3/min after 3 h and, at the best, 1/min after 2 days. In the present experiments negligible slowing occurred during the first 24 h and in some preparations the initial level of activity was only slightly reduced over 2-3 days. It appears that an organic constituent of snail blood is essential for optimal heart function.

The first series of isolated heart experiments showed that without the first aortic valve the heart failed to circulate fluid unless the venous pressure exceeded the arterial pressure. In life such a pressure relationship could only be transitory. In the second series of experiments the isolated heart preparation included the first aortic valve and was identical with that of Schwartzkopff (1954). The results corroborate his findings regarding the effect of pressure on frequency and heart output. Fluid was still circulated efficiently when the arterial pressure was 5-7 cm H\textsubscript{2}O above the venous pressure. When the arterial pressure was raised higher than this fluid began to flow back from the aorta into the ventricle at each systole, probably as a result of distension of the aorta causing the first aortic valve to become incompetent. In the intact animal the second aortic valve (Sommerville, 1973a) would probably limit the arterial pressure near the heart to that of the visceral rather than the cephalopedal haemocoel.

The results of the two series of experiments also differed with regard to the influence of pressure on frequency, and this may be explained by considering the interaction of the effects of heart distension and resistance to flow. Distension of the heart causes increased frequency of beat (Biederman, 1884; Biering, 1929; Türk-Meiningen, 1934; Jullien et al. 1953) and increased resistance to emptying causes decreased frequency of beat (Schwartzkopff, 1954). If the arterial pressure is raised in the absence of an aortic valve the ventricle becomes distended and this tends to increase its rate of beat, whereas with an intact valve there is no backflow and therefore no distension while the increased resistance to emptying results in a decreased rate of beat. In the experiments on the effect of varying the venous pressure the arterial pressure was invariably higher than the venous pressure. This meant that in the first series of experiments, i.e. without an aortic valve, the ventricular blood was at a higher pressure than the auricular blood. In the second series of experiments, however, the ventricular diastolic pressure was lower than the auricular systolic pressure, due to the action of the first aortic valve, and so the auricle was able to empty into the ventricle at each beat. In the first series of experiments it was observed that increased venous pressure resulted in a decreased heart rate but in the second series an increased heart rate resulted. Whilst the increased venous pressure distends the auricle, tending to increase the frequency in both instances, the decreased heart rate in the first series indicates that increased resistance to auricular contraction was the overriding factor determining rate of beat. These results suggest the presence of a pacemaker controlling heart rate situated in the auricle.

Schwartzkopff covered a wider range of pressures than those of the present experi-
ments. He found that at zero arterial pressure the frequency and output increased up to a venous pressure of 20 cm, above which the frequency remained constant while the output continued to rise. Measurements made on the intact animal (Sommerville, 1973a) show that the venous pressure is rarely higher than 10 cm H₂O above atmospheric so that a pressure gradient from vein to artery in excess of 10 cm H₂O is outside the physiological range of the heart. The pressure gradient across the heart is normally in the reverse direction.

Civil & Thompson (1972) were unable to obtain a sustained regular heart-beat in the absence of an artificial pericardial chamber unless there was an 8 cm head of pressure on the venous side of their preparation. This limitation was not observed in the present experiments, and the discrepancy may be due to the difference in arterial pressures (zero in theirs and 1-10 cm H₂O in these), or more probably in the different compositions of the Ringer solutions used.

SUMMARY

1. In isolated heart preparations of Helix pomatia perfused with Hedon Fleig solution, a minimum of 5% Helix blood was necessary for sustained rhythmical beating.

2. In the absence of the first aortic valve the heart was unable to pump fluid against a negative pressure gradient, the frequency of beat was directly proportional and the amplitude of beat inversely proportional to the arterial pressure while the frequency was inversely proportional to the venous pressure.

3. When the first aortic valve was included in the preparation the heart pumped efficiently and the frequency response was reversed in each case.

I wish to thank Professor A. Graham for his advice, encouragement and for the facilities of the University of Reading, where most of this work was carried out; Dr H. Dawson and Mr C. Purvis for their advice and gift of manometers; Dr R. H. Nisbet and Professor R. J. Linden for their help; my husband, Dr A. Chadwick, for his great help with the work and the preparation of this paper; and Professor J. M. Dodd and the Department of Pure and Applied Zoology, University of Leeds, where this work was finished.

REFERENCES


Circulatory physiology of H. pomatia. II


