RESEARCH COMMUNICATIONS

ELECTRICAL IMPULSE CONDUCTION IN PULMONARY VEINS

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It has been documented that the cardiac musculature of many mammalia, including man, extends from the heart along the pulmonary veins to the lungs, and, at least in some rodentia, as far as the small venules, and that smooth muscle is not present (1–3). However, these reports are of structural studies only, and in the one publication where a speculation is made concerning the spread of the contractile impulse, it is based on the assumption that the contraction has to assist the flow of blood, that is, pass from the lung to the heart (2). A study of the ontogenetic development of this musculature by two of the present authors (Challice and Chohan) prompted some preliminary experiments to ascertain whether or not the passage of a wave of excitation along this musculature might be detectable, and if so, in which direction it traveled.

It is well accepted that in embryonic development of the mammalian heart the governing pacemaker migrates from ventricle to atrium and finally to the sinoatrial node (4). No evidence suggesting that this pacemaker might arise from a site on the pulmonary veins in the lungs has been produced, and it is difficult to envisage how this might happen. It therefore appears that there is an anomaly. If the primary pacemaker is indeed located in the sinoatrial node, it is difficult to see how this extension of the cardiac musculature along the pulmonary vein can carry an electrical impulse other than in a direction *opposite* to that of normal blood flow. If this occurred, it would presumably produce a retrograde peristaltic contraction along the pulmonary veins. Should this prove to be the case, it could have interesting implications concerning the blood flow through the pulmonary veins and into the heart, and possibly, within the pulmonary capillaries. It was with this in mind that some preliminary experiments on the conduction of the electrical impulse in the pulmonary veins were undertaken.

Electrophysiological recordings were made from the pulmonary veins *in situ* on mice

following the simplest surgical procedures which allowed access to that part of the circulatory system. Adult mice were killed by cervical dislocation and the rib cage was quickly opened. The pericardial sac was removed to allow access to the pulmonary veins in the region where they enter the left atrium. These veins were followed distally as they collect from the posterior-dorsal surface of the lungs. The heart and thoracic area were continuously washed with aerated Locke's solution (pH 7.4, 37°C). Under these conditions it was possible to maintain heart function for over an hour in the absence of ventilation of the lungs.

Two unipolar suction electrodes were applied to the exterior walls of the vessels (or, extrapulmonary veins, [2]) using as great a separation as permitted by the anatomy. The ground lead of each electrode was positioned as far from the heart as possible, to minimize those potentials transmitted electrotonically from this source. Signals were amplified (band pass of amplifier 0.3 Hz to 1 kHz), displayed on a dual beam storage oscilloscope, and recorded on magnetic tape.

Electrical activity travelling away from the heart along the pulmonary vein has been recorded in each of seven mice. In the composite trace shown in Fig. 1, the potentials recorded proximal to the heart were used to trigger the oscilloscope trace. The potentials recorded several mm distally on the same vessel occurred at a fixed latency, but were not completely constant in amplitude. These potentials were propagated at a velocity of approximately 40 cm/s;[1] a velocity also characteristic of all preparations. Fig. 2 is a record of groups of five consecutive sweeps recorded at successive 10-min intervals from the same animal. It is seen that the latency between spikes (reciprocal of velocity) after the first 10 min interval increased by 1.5 ms and after the second interval by a further 1 ms. This slowing of conduction with time was typical of all the experiments, but variations greater than these were not seen. Once a potential was generated in the pulmonary vein it was observed to be conducted along the vessel without failure; however, the coupling between atrial and venous potentials was not so consistent. In fresh preparations, propagation from heart to pulmonary vein was found to be 1:1, but this coupling became less secure as a preparation deteriorated as shown in Fig. 3. These tracings were taken over a 4 min period as the coupling became weaker; 2 min elapsing between each of the three sets of traces. The small positive going potentials on the upper trace of each pair of traces represents the electrotonically conducted atrial depolarization recorded by the proximal electrode. When an atrial action potential produced a venous action potential this was conducted over the length of the vessel without fail; however, with deterioration fewer action potentials were conducted to the veins. When the oscilloscope was triggered from the small atrial action potential (Fig. 4) the causal relationship between atrial and venous spikes became clearly indicated by the fixed latency between these events. The five venous spikes on these traces were completely superimposed on one another.

As a control the pulmonary vein was compressed by forceps at the point of entry

[1] Average velocity from seven animals, 42.8 $cm \cdot s^{-1}$; estimated accuracy of measurements ± 10%; standard deviation 6.5 $cm \cdot s^{-1}$.

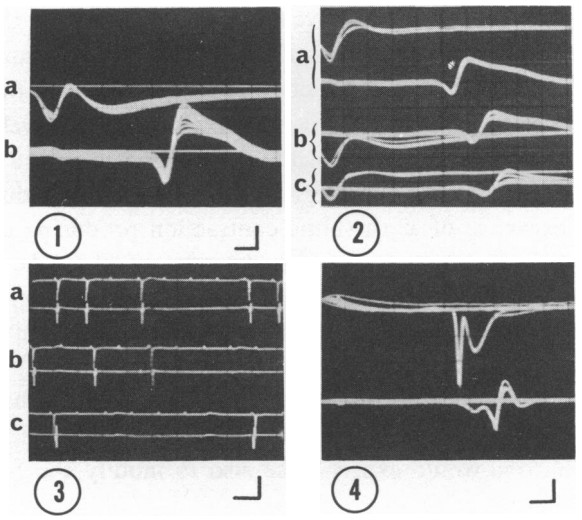

FIGURE 1 Illustration of retrograde conduction along the pulmonary vein as recorded by unipolar suction electrodes applied to the outside of the vessel. The upper spikes (*a*) recorded close to the heart were also used to trigger the oscilloscope sweeps. The lower spikes (*b*) were recorded 4.2 mm distally to the first. 20 consecutive sweeps stored on the oscilloscope are shown.

FIGURE 2 Activity from the same animal and with the same electrode placement recorded (*a*) 10 min, (*b*) 20 min, and (*c*) 30 min after the records in Fig. 1. Each pair of traces is of five consecutive sweeps.

FIGURE 3 Three pairs of traces of pulmonary action potentials recorded at 2-min intervals during which time the coupling between atrial and venous potentials became weaker. The small positive going potential represents the electrotonically conducted atrial depolarization recorded by the proximal electrode located adjacent to the heart.

FIGURE 4 Illustration of the fixed latency between atrial and venous potentials. The oscilloscope traces were triggered from the small atrial potentials. 12 traces were stored with venous spikes occurring only five times.

FIGURES 1, 3, Calibrations: *Vertical.* 2 mV in Figs. 1*a*, 3; 1 mV in Fig. 1*b*; 0.5 mV in Fig. 4. *Horizontal.* 2 ms in Fig. 1; 200 ms in Fig. 3; 5 ms in Fig. 4.

to the heart. This compression did not disrupt the integrity of the vessel and blood flow into the heart continued after release; electrical conduction along the vein, however, was totally absent following this intervention. As a further control, recordings were made from the dorsal aorta of the mouse in several animals. The walls of the aorta do not contain cardiac musculature, but only smooth muscle. In each case, spikes were recorded representing weak signals conducted electrotonically from the heart, which became progressively weaker the further from the heart they were recorded, and which occurred simultaneously at each of the two electrodes. Propagated action potentials were never found.

These simple experiments indicate, under the experimental conditions described, the existence of retrograde conduction of an electrical impulse along the pulmonary veins

at least to the points where the veins leave the lungs. Presumably these spikes represent myoid conduction through the muscle tissue. No summation nor facilitation appears to be involved in this 1:1 conduction. If the validity of the observations is accepted, it would seem likely that this depolarization would travel to the extent of the cardiac musculature along the veins within the lungs, but such a suggestion represents an extrapolation of the present observations. Retrograde conduction along these veins suggests the existence of a peristaltic contraction producing a pumping action (albeit possibly only a weak one) against the direction of blood flow. It would presumably supply a degree of haemodynamic matching to the pumping action of the heart, but it also seems possible that it could be part of a mechanism for preventing evacuations of pulmonary capillaries, or "pulmonary sinusoids" (5) during the heart's pumping action. In addition, malfunction of such a system might be expected to produce respiratory disorders. Since there appear to be cholinergic nerves associated with these veins (6), one would expect these also to modify the functioning of the musculature.

Further work is in progress involving the development of a more complex experimental system, but in view of the hitherto apparently unreported observation of retrograde conduction in these veins it was thought desirable to make these preliminary results available.

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