THE PULSATILE NATURE OF THE RELEASE OF POTASSIUM FROM HEART MUSCLE DURING THE SYSTOLE*

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My colleagues, James M. O'Brien and Irene Bay, and I have produced direct evidence that the potassium release of cardiac contraction or activity is truly pulsatile. A turtle heart is heavily labeled *in vivo* with intraperitoneal K^{42}. The isotope must be prepared in the intense neutron flux of the Low Intensity Testing Reactor at Oak Ridge. The excised heart is perfused (FIGURE 1) and the outflowing perfusate is caught on a filter-paper strip moving past on a timed kymograph. The strip is cut into rectangular samples that are serially collected at known times. The electrocardiogram and a myogram are recorded simultaneously. The potassium release of activity may thus be related in time to the electrical events. A second radioactivity is added as a volume indicator in the perfusion fluid. This may be I^{131}-albumin or inorganic phosphate tagged with P^{32}, and it is counted after the potassium K^{42} has been allowed to decay through its short half life. An arithmetical division of the K^{42} count by the volume indicator count on each paper segment then gives us the concentration of potassium K^{42} for that particular sample.

FIGURE 2 is a typical curve for one of the two hearts on which runs have been successful in the last four years. The ECG waves are above, and the concentration waves for K^{42} are below — our so-called "effluogram" record. The first K^{42} wave is for a systole elicited by electrical stimulation at the A-V junction, and the second wave is for an idioventricular beat. Thus, we see that the wave of release is real and not the result of electrolysis due to the stimulating current. Note that there are about seven samples represented in the upstroke of the wave and about forty in the entire wave. The samples measured may each contain as little as 0.003 ml. of perfusate. There is no correction for travel time on this chart.

Note that the perfusion irrigation is not by way of the ventricular cavity, but only by way of the coronary vessels (FIGURE 3). Cannulation for the inflow is into the single coronary artery. Occasionally two coronary arteries arise independently from the innominate artery and thus spoil an experiment in which we have invested more than a hundred dollars' worth of isotopes. Unfortunately the outflowing venous coronary vessels do not collect into a single vessel for cannulation, but open by several vents into the neighborhood of the sinus venosus. The collective outflow from these veins is allowed to flow down the outside of a solid glass stylus, penlike in form. The stylus is anchored by a ligature about the A-V junction. The trimmed right auricle and the sinus venosus drape about this stylus like the cloth of an umbrella. Resting flow is very steady (lower curve of FIGURE 5).

FIGURE 4 shows the general layout of the apparatus.

FIGURE 5 shows our most successful run, in a serial recording of six systoles. The gradual fall in base line, whatever its cause, is real. It is not due to the attenuation of the tagging within the heart.

The outflowing K^{42} naturally suffers a travel time and a scatter phenomenon as it travels from each capillary, a variable distance, to the tip of the stylus.
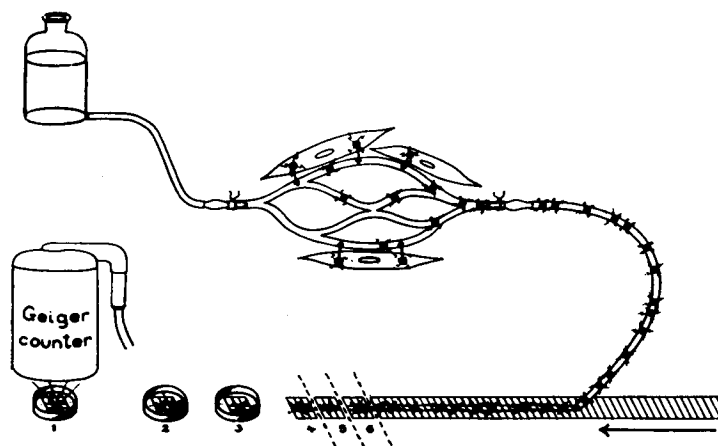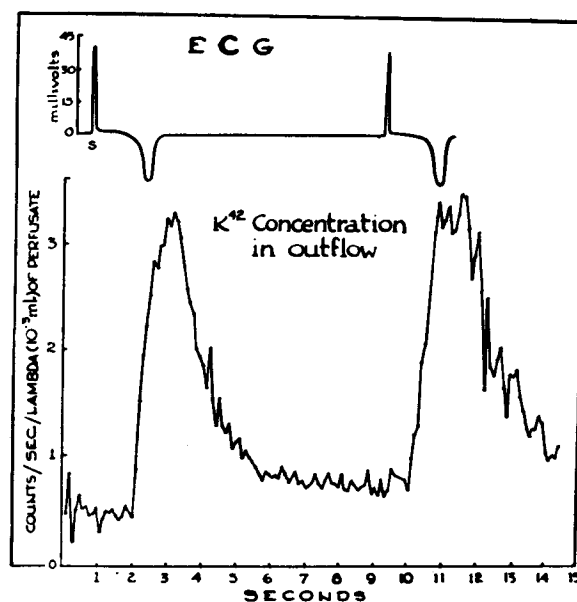

FIGURE 1. Principle of effluography.

FIGURE 2. The effluogram.

FIGURE 6 shows how we have attempted to calibrate for this.   It is a model of the coronary circulation made with strings.   Each string travels the full length of the coronary bed, artery to vein, but traverses only one capillary loop.   In the upper figure the arches represent capillary loops.   Thus, the strings enter the loops from the artery on the left and leave the loops to travel

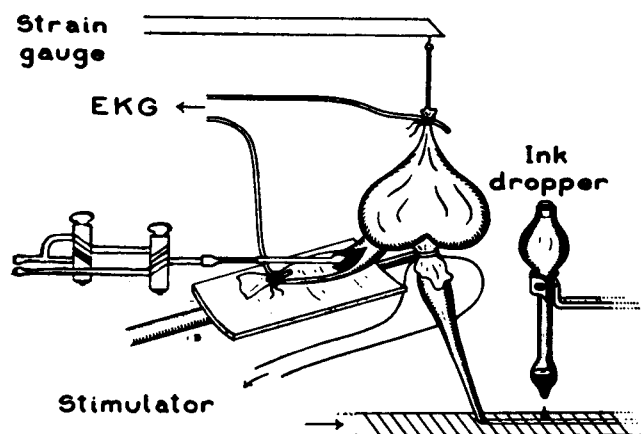FIGURE 3.   Coronary cannula and stylus.

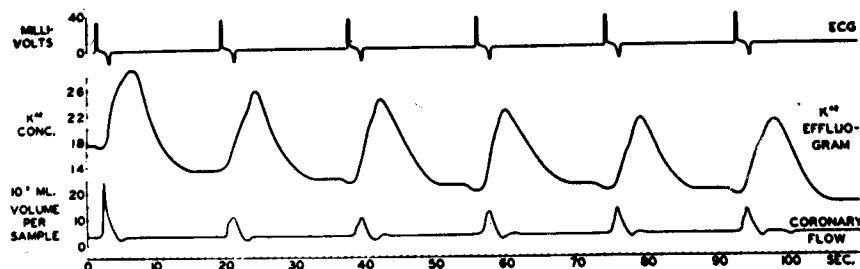FIGURE 4.   Heart mounted over kymograph.

FIGURE 5.   Effluograms.   Six consecutive systoles electrically stimulated.   The lower recording is of volume collected per sample or per 0.096 second.

to the vein on the right.   On each end of the model the strings are tied tightly together so that they may be slipped collectively back and forth through the model to represent flow.   The scatter of K^{42} is illustrated by staining the strings at the peak of each arch or capillary loop.   Then, as the strings are drawn collectively to the right, these stained segments take on the pattern illustrated at the right.

A graphical representation of this scatter is shown above at the right.   It is plotted by imagining the vein or bundle of strings to be cut into segments. The number of stained elements in each segment is then plotted vertically to construct the plot shown above.   Note that this is analogous to plotting concentration in the outflow against accumulated volume.
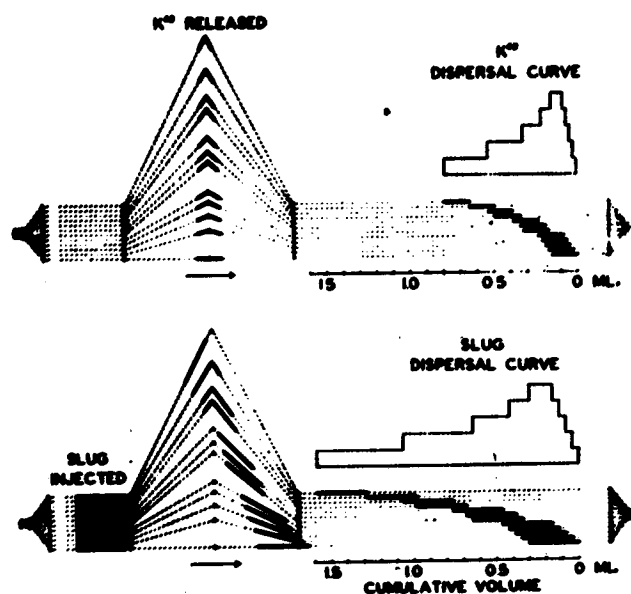

FIGURE 6.   String model of the mechanism of vascular dispersal.   The upper curve represents the dispersal of K^{42} released into capillaries.   The lower curve represents the dispersal of the injected slug.
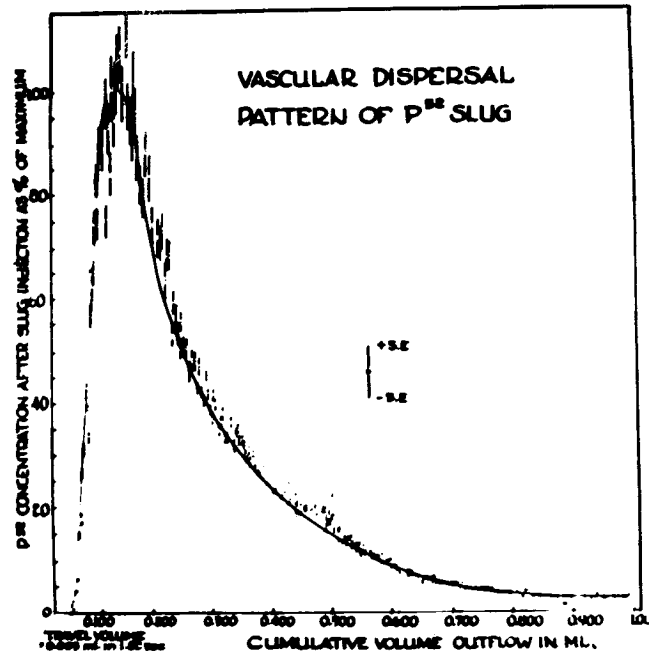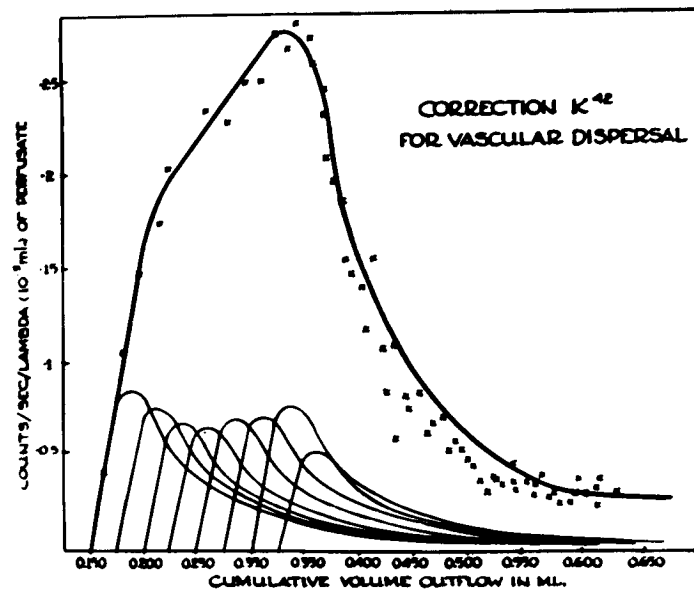
FIGURE 7. Actual slug dispersal curve.

FIGURE 8. Volume effluogram (above) showing the graphical summation of pattern-release curves (below).

It might be thought that changes in flow would distort the shape of this scatter pattern. If the strings are jerked back and forth to mimic variable flow rate, however, it will be seen that this pattern is retained. In actual plotting, this pattern will be obtained only if concentration is plotted against accumulated volumè. If, instead, the concentration is plotted against serial time, flow changes will indeed distort the pattern. We have thus resorted to the stratagem of plotting all concentrations against accumulated volume and have thus caught this uniformity of pattern.

Since it is impossible to construct this ideal scatter pattern, we mimic it by constructing the pattern for a parcel of a third radioactivity injected suddenly into the coronary artery. This will produce the pattern modeled in the lower diagram in FIGURE 6. Note that, since the travel distances are roughly double, this pattern will be twice as wide as the ideal pattern for the release of potassium mid-length of each capillary loop.

Such a pattern is shown in FIGURE 7, as constructed from the pattern set by an actual parcel after injection into the coronary artery. This injection is accomplished by catching a third indicator radioactivity in the plug of a stopcock (left side of FIGURE 4). A sudden turn of the stopcock at the end of a series of regular runs then allows us to construct the pattern curve as shown in FIGURE 7.

In FIGURE 8 we imagine what series of pattern curves, representing successive releases in time of K^{42} into each of the capillaries, would, when added, give the over-all curve that is drawn through actual experimental points. This is accomplished by a graphical differentiation in which one pattern curve is subtracted from the over-all curve, a second pattern curve is substracted from the residue of points, and so on until all the area of the over-all curve has been used up. Thus, in FIGURE 8 the total area of the over-all curve is the sum of the areas of all the individual pattern curves shown below.

Since this is a plot of concentration vertically against accumulated volume horizontally, the area of each of these pattern curves represents a mass of released K^{42}. The area of each pattern curve is compressed into a rectangle, the width of which is equal to the accumulated volume between two successive upswings of pattern curves. Later, this same area is compressed into a time
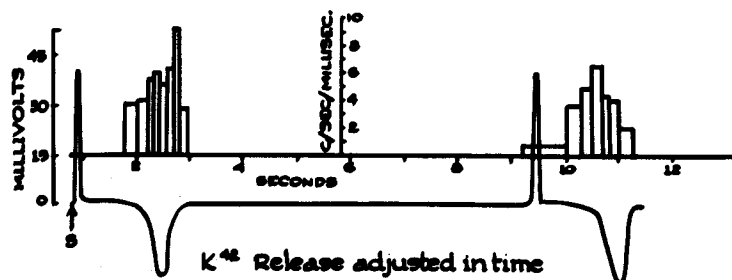

FIGURE 9. Chemocardiogram. The rectangles are K^{42} release parcels extrapolated backward in time for vascular travel and dispersal. The altitudes of each rectangle are the rate of release for the interval.

interval covered by this same collection period and is fitted against the corresponding electrocardiogram, as shown in FIGURE 9.

In summary, our firm conclusion is that the release of potassium is indeed pulsatile. A less firm impression is that the release begins during what Weidmann calls the "plateau" and continues with special rapidity during the quick phase of the repolarization wave, as recorded with intracellular electrodes.