Muscle-to-Cardiac Timing for Aortomyoplasty

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ABSTRACT

Aortomyoplasty is a new surgical treatment for heart failure in which the latissimus dorsi muscle (LDM) is wrapped around the aorta and electrically activated to provide diastolic counterpulsation. We hypothesized that muscle-to-cardiac timing significantly influences the effectiveness of counterpulsation by aortomyoplasty. In dogs (n=9, 20–25 kg), the LDM was isolated, leaving the neurovascular pedicle intact, instrumented with stimulus electrodes, and wrapped around the descending thoracic aorta. Left ventricular and aortic pressures were measured and limb-lead ECG provided R-wave detection. LDM stimulation was initiated at the dicrotic notch and stimulus duration was systematically increased until contraction was prolonged into the subsequent cardiac systole. For each stimulus duration, endocardial-viability ratio (EVR) was calculated. The stimulus train maximizing EVR was identified and compared with stimulus trains of longer and shorter duration. The optimal train duration produced significantly greater increases in EVR than stimulus trains of sub-optimal duration. These data suggest that muscle-to-cardiac timing is important in aortomyoplasty and using timing other than optimal timing diminishes effectiveness.

I. INTRODUCTION

Aortomyoplasty provides hemodynamic benefits similar to the intra-aortic balloon pump (IABP). In intra-aortic balloon pumping, a balloon-equipped catheter is positioned in the descending thoracic aorta, inflated at the beginning of diastole, and deflated before the ensuing systole to provide diastolic counterpulsation [1]. The IABP has proven valuable in treating cardiogenic shock, but it cannot be positioned long term as would be required to treat chronic heart failure.

Like the IABP, counterpulsion-to-cardiac timing is important in aortomyoplasty. While the timing of the IABP has been thoroughly investigated, skeletal muscle is not a mechanical balloon—the dynamics of skeletal muscle contraction and relaxation are different from intra-aortic balloon inflation and deflation. We measured the effects of varied muscle-to-cardiac timing sequences during acute aortomyoplasty on pressure-based indices of diastolic counterpulsation.

II. METHODS

Anesthesia was induced and maintained using pentobarbital sodium (25 mg/kg, IV) and the dogs (n=9, 20–25 kg) were intubated. Limb-lead ECG was monitored. The left latissimus dorsi muscle (LDM) was mobilized at the origin, while the point of insertion was left intact. The thoracodorsal nerve was instrumented with pacing electrodes. Approximately 4 cm of the second rib was resected and the LDM was transposed into the left chest through the bed of the resected rib. A left thoracotomy was performed through the fifth interspace and a 10-cm length of the descending thoracic aorta was exposed from the level of the subclavian artery. Three to four pairs of intercostal arteries were ligated as needed. The muscle was then wrapped around the descending thoracic aorta using a technique previously described [2] (Fig. 1). Solid-state pressure transducers were placed into the left ventricle at the apex, and into the aortic arch via the right carotid artery. A physiologic recorder provided R-wave triggering from a limb-lead ECG.

Data were acquired over 7–10 second intervals while the muscle was stimulated (pulse width 210 µs, intraburst stimulus frequency 50 Hz) on every other heartbeat (1:2 mode). The LDM was allowed to rest for at least one minute between data runs. The stimulus R-wave delay was adjusted so that the increase in aortic pressure (due to muscle contraction) coincided with the dicrotic notch. Once the R-wave was set, the muscle was stimulated with trains containing an increasing number of stimulus pulses, thus sequentially increasing the duration of muscle contraction from one data run to the next. Stimulus durations were increased until peak left-ventricular pressure in the beat following muscle contraction increased. This procedure generated a set of data as a function of stimulus duration, from too short, to the optimum, and then finally too long.

For each heart beat in the data run, endocardial viability ratio (EVR) (a clinical indicator of the supply-to-demand ratio for the heart), was calculated as the aortic diastolic pressure-time integral divided by the left ventricular systolic pressure-time integral [3], and reported normalized to the unstimulated beats. Trains were then sorted into timing groups spanning 10% of the R-R interval. Data were then organized with respect to the train duration producing the maximum increase in EVR—designated as the optimal train. This resulted in a data set of five stimulus timings per dog—optimum train duration and the optimum train duration plus and minus 10% and 20% of the R-R interval. Data were compared by one-way repeated measures ANOVA. When this indicated significance, paired t-tests compared optimum timing with non-optimum timing. Differences were considered significant at p < 0.05. Data are reported as mean ± SD.

III. RESULTS

Figure 2 shows sample left-ventricular and aortic pressure, together with the ECG, taken during aortomyoplasty counterpulsation. The muscle was stimulated...
following every second R-wave (1:2 mode), on the first and third beats in this record. The closely spaced spikes on the ECG are noise due to the muscle stimulation pulses. During counterpulsation beats, the increase in aortic pressure due to muscle contraction began at the dicrotic notch and was maintained until the muscle relaxed after termination of the pulse train. Peak left-ventricular pressure in the beat following stimulation was decreased suggesting muscle contraction reduced left ventricular afterload.

Figure 3 shows that endocardial-viability ratio is roughly a parabolic function of train endpoint. The optimum stimulus train produced a maximum EVR that was significantly greater than trains ending earlier (-20%, -10%) or later (+10%, +20%) in the cardiac cycle.

IV. DISCUSSION

When evaluating the affects of aortomyoplasty as a treatment for chronic heart failure, it is important to understand and properly adjust muscle-to-cardiac timing. It is well recognized that timing of balloon inflation and deflation is critical in intra-aortic balloon pumping. Similarly, timing of muscle contraction and relaxation is important in aortomyoplasty. Investigators studying aortomyoplasty, while understanding the importance of timing, have not explored the affects of muscle-to-cardiac timing.

Our results indicate that muscle-to-cardiac timing influences the effectiveness of counterpulsation during aortomyoplasty and optimum muscle-to-cardiac timing has significantly greater hemodynamic effects than sub-optimum muscle-to-cardiac timing. Trains that were too short reduced the beneficial effect of counterpulsation on endocardial-viability to a lesser extent than trains ending later in the cardiac cycle. Setting of muscle-to-cardiac timing should carefully considered when using counterpulsation by aortomyoplasty to ensure maximum cardiovascular benefit.

V. REFERENCES

