Acute progressive resistance exercise alters intracardiac pressure in ponies

KH McKeever1,*, SE Alway2, JW Farris3, KW Hinchliff4 and JA Lombardo5

1Equine Science Center, Department of Animal Science, Rutgers – The State University of New Jersey, University, New Brunswick, NJ 08901, USA
2Division of Exercise Physiology, School of Medicine, West Virginia University, Morgantown, WV 26506, USA
3Department of Physical Therapy, Arkansas State University, State University, AR 72467, USA
4Equine Exercise Physiology Laboratory, College of Veterinary Medicine, Ohio State University, Columbus, OH 43210, USA
5Department of Family Medicine, Ohio State University, Columbus, OH 43210, USA
*Corresponding author: mckeever@aesop.rutgers.edu


Abstract
Five male ponies (3–6 years, 208 ± 11 kg) were used to examine the haemodynamic effects of resistance exercise. The experiment consisted of a 30 min catheterization period and a 15 min equilibration period followed by the test. Initially, the ponies stood quietly on a treadmill for collection of 5 min of baseline data. They then walked at 1.9 m s−1 for 1 min sets while carrying 0 kg, 67 kg or 134 kg of weight loaded on the saddle area. Weights were applied in a randomized order using a chain hoist and sling. The ponies rested unloaded for 2 min between sets and all cardiovascular parameters returned to baseline before beginning the next set. Haemodynamic measurements (heart rate (HR), carotid artery systolic (SP), diastolic (DP), pulse (PP) and mean pressure (MAP), right atrial pressure (RAP), right ventricular pressure (RVP) and right ventricular maximum rate of pressure rise (dP/dtmax)) were recorded continuously and data collected during the last 18 s of each set were averaged and analysed. HR increased (P < 0.05) with unloaded exercise (201%) and with increases in load (252 and 271% at 67 and 134 kg, respectively). RAP increased (P < 0.05) with exercise, with differences (P < 0.05) between the response to the 0 kg (66%), 67 kg (121%) and 134 kg (138%) loads. RVP increased (P < 0.05) incrementally with load during exercise (58, 110 and 136%, respectively), with differences (P < 0.05) between 0 versus 67 kg and 67 versus 134 kg. Right ventricular dP/dtmax increased (P < 0.05) incrementally and showed differences (P < 0.05) between loads (131, 191 and 252% at 67 and 134 kg, respectively). There were increases (P < 0.05) in MAP (20%), SP (20%) and DP (27%) with exercise, but no difference (P > 0.05) between the response to 67 versus 134 kg load (43 and 51%; 40 and 46%; 49 and 62, respectively). Pulse pressure did not change (P > 0.05) from baseline values. These data suggest that resistance exercise in the form of weight carried increases intraventricular pressure through a significant increase in both cardiac pre-load and after-load.

Keywords: heart; equine; weight-lifting; blood pressure; haemodynamics

Introduction
Eight weeks of progressive resistance training, in the form of progressive sets of weight carriage, results in a significant hypertrophy of the forelimb muscles of ponies1. That investigation was conducted to establish the pony as a better animal model to use in studies of the response to progressive resistance exercise. Carotid artery pressure measurements made during one workout session performed in the trial demonstrated that resistance exercise appeared to cause a substantial increase in mean arterial pressure that paralleled the amount of weight carried1. That observation was consistent with human studies in which peripheral arterial blood pressure measurements demonstrated an increase in arterial blood pressure during weight-lifting2-6. These documented increases in peripheral
arterial pressure during resistance exercise have led to speculation that, over time, repeated increases in cardiac after-load may contribute to the development of what has been termed ‘weight-lifter’s heart’. There are studies that have reported differences in oxygen uptake in horses due to exercise on the incline (a form of resistance exercise) as opposed to on a level treadmill. Unfortunately, no published study to date has measured intracardiac pressure during progressive resistance exercise. Therefore, the purpose of the present study was to test the hypothesis that acute progressive resistance exercise would result in acute increases in intracardiac pressure.

Materials and methods

Subjects
Five male ponies ranging in age from 3 to 6 years with body weight from 179 to 241 kg were used for this study. The ponies had their left carotid arteries surgically relocated to a subcutaneous position no less than 2 months prior to the experiment. The ponies were accustomed to the laboratory and walking on the treadmill and were taught to carry weights draped over a saddle pad placed on their backs. This experiment was performed in accordance with the Guiding Principles in the Care and Use of Animals of the American Physiological Society and was approved by the Institutional Review Boards for the Care and Use of Animals of Rutgers University and the Ohio State University.

General experimental design
The general protocol for the experiment consisted of a 30 min catheterization period and a 15 min equilibration period, where the ponies stood quiet on the treadmill, followed by a 5 min baseline data collection period. Each pony was catheterized prior to the study with a catheter introducer (7.5 F; Argon Medical Corp., Athens, TX) percutaneously placed in the right jugular vein and a fluid-filled catheter (17 gauge; Deseret, Inc., Sandy, UT) placed into the carotid artery. Catheterizations were performed under sterile conditions using lidocaine anaesthesia.

Exercise test
During the test, the ponies walked on an equine treadmill (Sato I, Uppsala, Sweden) at a speed of 1.9 m s⁻¹ (level grade) for 1 min sets while carrying either 0 kg, 67 kg or 134 kg of weight loaded on the saddle area. Absolute values of the weights used in the experiment were a consequence of the fixed size of the lead sheets, much like the fixed weights used in weight-lifting. However, there was some variation in the weights of the ponies and thus on a relative basis the weights used represented 32% and 64% of the average body weight of the ponies. Weights were applied in a randomized order using a chain hoist and sling (Fig. 1). A 2 min warm-up walk on the treadmill and 2 min rest preceded the weight carriage phase of the trials. The ponies rested unloaded for 2 min between treatment sets. During this unloaded period all cardiovascular parameters returned to baseline with no cumulative or carryover effect.

Haemodynamic measurements
Haemodynamic measurements included heart rate (HR), carotid arterial systolic (SP), diastolic (DP), pulse (PP) and mean pressure (MAP), right atrial pressure (RAP), and right ventricular pressure (RVP) and maximum rate of pressure rise (dP/dt max) . Right atrial and right ventricular pressure were measured using a dual-sensor micromanometer catheter that was custom-fabricated for use in horses (Micro-tip; Millar Instruments, Houston, TX). The catheter was inserted into the heart through the catheter introducer placed into the left jugular vein. Carotid artery pressure was measured via the fluid-filled catheter attached to a pressure transducer (Gould, Inc., Cleveland, OH). Analogue-to-digital conversion of the data from the haemodynamic recorder was performed using a Datalogger-32 system (Buxco, Inc., Stamford, CT). This system calculated heart rate and right ventricular dP/dt max from the analogue ventricular pressure signal . Catheter position was verified before and after the experiment via the pressure waveform recorded on a haemodynamic recorder (VR12 Hemodynamic Recorder; PPG Biomedical Instruments, Pittsburgh, PA). The transducers were calibrated at the level of measurement (i.e. the heart or the carotid depending on the transducer) just prior to the study using a mercury manometer. Haemodynamic data were measured continuously.

Fig. 1 Equine treadmill with chain hoist apparatus used to lower and remove weights from the saddle area of the ponies.
and data collected during the last 18 s of each set were averaged and analysed.

**Statistical analysis**
A one-way analysis of variance for repeated measures was used to examine for significant main effects. The Student–Newman–Keuls test was used, when appropriate, to examine for differences between treatment means. The null hypothesis was rejected at $P < 0.05$.

**Results**

Weight carriage was associated with significant changes in heart rate, and atrial, arterial and ventricular pressures (Figs 2–5). HR (Fig. 2) increased substantially ($P < 0.05$) with unloaded exercise (201%) and increased further with increases in load (252 and 271% at 67 and 134 kg, respectively). RAP (Fig. 2) increased progressively ($P < 0.05$) with exercise (66, 121 and 138%), with differences ($P < 0.05$) between the response to the 0, 67 and 134 kg loads. Similarly, RVP (Fig. 3) increased ($P < 0.05$) incrementally (58, 110 and 136%) during exercise with differences ($P < 0.05$) between 0 versus 67 kg, 67 versus 134 kg and 0 versus 134 kg loads. Right ventricular $dP/dt_{\text{max}}$ (Fig. 3), an indirect measure of contractility, increased ($P < 0.05$) incrementally (131, 191 and 252%) with differences ($P < 0.05$) between 0 versus 67 kg and 67 versus 134 kg. There were increases ($P < 0.05$) in SP (20, 40 and 46%), DP (27, 49 and 62%) and MAP (20, 43 and 51%) (Figs 4 and 5) with exercise, with a significant difference between 0 kg versus load, but no difference ($P > 0.05$) between the response to 67 versus 134 kg load. PP (Fig. 4) showed non-significant increases (25, 27 and 17%) above baseline values.

**Discussion**

The present study is the first published experiment that we are aware of which attempts to measure intracardiac pressure during progressive resistance exercise in an animal model. The major finding was that resistance exercise caused a greater increase in all haemodynamic parameters (HR, RAP, RVP, $dP/dt_{\text{max}}$, SP, DP and MAP) measured except PP when compared with unloaded exercise. Other published experiments have measured arterial pressure$^{2–6,15}$ and some have used echocardiography$^{7,16,17}$ to document changes in ventricular wall thickness and chamber size following long-term progressive resistance exercise training. Furthermore, there are studies that have reported differences in
oxygen uptake in horses due to exercise on the incline (a form of resistance exercise) as opposed to on a level treadmill\textsuperscript{12}. One could speculate that there could be differences in the blood pressure response to work on the hill \textit{versus} the flat. However, while that paper demonstrated an increase in the metabolic cost working up a hill, cardiac pressures were not measured\textsuperscript{12}. The authors are unaware of any published studies that have documented directly measured changes in intracardiac pressure during acute progressive resistance exercise where the intent was to measure cardiovascular function in an animal model and using a regimen of exercise documented to be useful to simulate progressive resistance exercise performed by humans\textsuperscript{3}.

\textbf{Changes in arterial pressure – after-load effects}

Arterial pulse pressure is the only variable measured in the present study that was not affected by weight carriage. Most studies of horses and ponies have documented moderate increases in mean arterial pressure and arterial pulse pressure with increases in exercise intensity (running on a treadmill, not weight carriage)\textsuperscript{13,14,18,19}. These moderate increases in mean arterial pressure are similar to those seen in humans performing dynamic exercise\textsuperscript{10,20}. Rowell\textsuperscript{20} has speculated that a feed forward mechanism moves the set point for the defence of arterial pressure to higher levels during exercise. He suggests that this is a response to increases in dynamic exercise in which there are increases in cardiac output and decreases in total peripheral vascular resistance\textsuperscript{10,18}. During dynamic exercise, vascular conductance increases with increasing work intensity and pulse pressure usually widens\textsuperscript{18–20}. Pulse pressure increases primarily through an increase in arterial systolic pressure that is coupled with a disproportionately smaller increase (or even a slight decrease) in arterial diastolic pressure. However, the increase in mean arterial pressure during weight-lifting appears to involve increases in diastolic pressure that are more proportional with the concomitant increases in systolic pressure\textsuperscript{15}. Proportional increases in both systolic and diastolic pressure would lead to an increase in mean arterial pressure without an increase in pulse pressure. One could speculate that the lack of an increase in pulse pressure observed in the present study could have been due to the very low walking speed (1.9 m s\textsuperscript{−1}) used. However, increases in diastolic pressure paralleled increases in systolic pressure and it is most likely that, as in humans\textsuperscript{6,15}, the lack of a rise in pulse pressure with weight carriage was possibly due to the effect of increased peripheral vascular resistance.

\begin{figure}[h]
\centering
\includegraphics[width=0.4\textwidth]{fig4}
\caption{Effect of progressive resistance exercise on carotid arterial systolic pressure (SP) and carotid arterial diastolic pressure (DP) in ponies. Values are means with standard error shown by vertical bars ($n = 5$). Mean values with unlike superscript letters are significantly different ($P < 0.05$).}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=0.4\textwidth]{fig5}
\caption{Effect of progressive resistance exercise on carotid arterial pulse pressure (PP) and carotid mean arterial pressure (MAP) in ponies. Values are means with standard error shown by vertical bars ($n = 5$). Mean values with unlike superscript letters are significantly different ($P < 0.05$).}
\end{figure}
Changes in right atrial pressure – pre-load effects
The weight carriage-induced increase in right atrial pressure seen in the horses of the present study is indicative of greater pre-load of the heart independent of the increase in venous pressure induced by splenic reserve mobilization. This conclusion is supported by previously reported data from horses demonstrating that submaximal exercise, like the 2 min warm-up and 2 min recovery period used in the present study, would have been sufficient to result in the mobilization and accommodation of the fluid load caused by splenic contraction. While not reported here, we observed a change in RAP during the warm-up consistent with splenic mobilization and accommodation. Re-sequestration of the mobilized splenic red cell reserve takes more than 30 min; thus, each of the weight carriage bouts would have been performed during a period independent of the systemic effects of splenic mobilization. Further evidence for the lack of an effect of splenic mobilization on the right atrium is that the order of the weight carriage was randomized and the effect on RAP was still detected. Had it been an effect of exercise, but not weight carriage, then one would not have seen an effect of weight carriage that was dose-dependent. Therefore, this increase in pre-load during weight carriage was not due to splenic reserve mobilization and most likely was due to a subsequent increase in venous return due to muscle pumping action coupled with a component due to increases in intrathoracic pressure.

Changes in right ventricular pressure
Substantial increases in right ventricular pressure were also seen with weight carriage in the present study. On one hand, an increase in ventricular pressure during dynamic exercise is consistent with other studies of exercising horses. However, the magnitude of the increase in ventricular pressure was substantially greater during weight carriage and, more importantly, directly associated with the amount of weight carried. Therefore, the subsequent increases in ventricular pressure were most likely due to the effects of the progressive resistance work, not just due to unloaded dynamic exercise.

The observed increase in mean arterial pressure, and especially diastolic pressure, suggests that the concomitant increase in intraventricular pressure was due in part to an increase in cardiac after-load. An increase in ventricular pressure, coupled with increases in cardiac pre-load and after-load, would likely cause the heart to perform a substantial amount of isometric work. Chronic isometric work is thought to be one of the leading factors in the development of so-called ‘weight-lifter’s heart’. However, the intensity and frequency of isometric cardiac contraction needed to cause an increase in ventricular wall thickness are not known.

While the present experiment was not designed to examine the long-term adaptive changes in cardiac muscle architecture associated with weight-lifting, it does provide new insight into the question of whether there are substantial changes in the intracardiac forces needed to induce such long-term, and potentially detrimental, changes in heart muscle function. Similarly, studies utilizing echocardiography have reported increases in ventricular dimensions associated with resistance exercise training in humans. These observations have led to the development of an opinion in the medical literature suggesting that repeated increases in cardiac after-load caused by elevated arterial blood pressure cause the heart muscle to undergo isometric work, and that long-term exposure to excessive isometric cardiac work may be one factor contributing to the concentric cardiac hypertrophy reported for some weight-lifters. While this hypothesis sounds attractive, it is only associative in its scope, leading some researchers to speculate that elevated peripheral arterial pressure may not be the sole factor responsible for hypertensive cardiac hypertrophy. Many factors can affect arterial pressure during exercise; therefore, without direct confirmation that resistance exercise induced increases in intracardiac pressure, the link between the rise in peripheral arterial pressure and concentric cardiac hypertrophy is only speculation.

Conclusions
In summary, the present study is the first published experiment that we are aware of which documents that progressive resistance exercise increases intracardiac pressure. It was concluded that resistance exercise, in the form of weight carried, increases ventricular pressure and ventricular maximum rate of pressure rise through a significant increase in both cardiac pre-load and after-load. These observations suggest an additional stress on the heart that may be of interest to those prescribing resistance exercise for patients involved in cardiac rehabilitation programmes. The data also provide potential insight into the stimulus for induction of the architectural remodelling that occurs in concentric cardiac hypertrophy. Speculation that repeated increases in intracardiac pressure associated with resistance training may stimulate cardiac remodelling in quadruped species are supported by recently published data that demonstrated changes in cardiac morphology in the hearts of sled dogs performing exercise with a high-resistance component. Intracardiac pressures were not measured in that study; however, the authors observed considerable ventricular hypertrophy over the course of 2700 miles of training. They reported that the ventricular
hypertrophy in the dogs was not consistent with left ventricular overload secondary to volume load.\textsuperscript{17} Taken together, data from studies of weight-lifters,\textsuperscript{7-16} cats,\textsuperscript{7} highly resistance-trained dogs\textsuperscript{17} and the present study suggest that effects of repeated elevations in intracardiac pressure during long-term resistance training may indeed be a strong enough stimulus to induce changes in cardiac morphology. Future studies using the equine model that can be directed at understanding the long-term effects of progressive resistance exercise training on cardiac physiology have the potential to answer questions of importance to both human and equine athletes. Such is the case because horses perform many activities, such as driving, that have a large resistance component.

However, data from the present study also suggest that a contribution to the increase in ventricular pressure may also have come from the combined effects of increases in cardiac pre-load as well as increases in contractility.\textsuperscript{10,20} This conclusion is supported by the observed increases in right atrial pressure, mean arterial pressure and right ventricular maximum rate of pressure rise during loaded work. The latter, however, is only a gross index of contractility and is a parameter with limitations due to its dependence on factors such as heart rate, pre-load and after-load, which also appear to be altered by exercise with weight.\textsuperscript{10,20} Even so, the large magnitude of the significant change in maximum rate of pressure rise suggests a substantial alteration in force of cardiac contraction during exertion.\textsuperscript{14}

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