

**Differential Cardiovascular Responses to Acute Exercise in Adults with Cerebral Palsy**

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**ABSTRACT**

Cerebral palsy (CP) is a non-progressive and permanent neurological disorder leading to musculoskeletal dysfunction and immobility. A major clinical problem with CP is early development of cardiovascular diseases with increased rates of mortality. Due to the inevitability of motor dysfunction adults with CP can develop health risk factors, such as obesity and hypertension, at a higher rate compared to the general population. To date, the physiological basis for CP has not been established; how cardiovascular dynamics, such as heart rate (HR), blood pressure (BP), and blood flow (BF), are controlled in individuals with CP has never been identified. **PURPOSE:** To determine differential cardiovascular responses to acute dynamic exercise in adults with CP. **METHODS:** Total of eighteen adults with and without CP participated in the study. HR from ECG, beat-to-beat arterial BP from Finapres and brachial BP, and respiration via pneumobelt were continuously measured before, during and after 2 minutes of dynamic handgrip exercise at 35% and 50% of maximal voluntary contraction. In addition, diameter, blood velocity, and flow of the brachial artery were measured using Doppler ultrasound on the contracting arm throughout the experiment. **RESULTS:** At rest, both control and CP groups had similar resting HR ( $60.7 \pm 1.9$  control and  $63.0 \pm 7.5$  CP, bpm). While resting respiratory rate was lower in CP group compared to the control, resting mean arterial pressure (MAP) and brachial blood flow tended to be higher in CP ( $p=0.08$ ). MAP and HR were significantly increased to exercise from rest in both groups with no group differences (MAP,  $\Delta 9.3 \pm 2.2$  control and  $\Delta 11.2 \pm 3.3$  CP, mmHg; HR,  $\Delta 8.4 \pm 1.5$  control and  $\Delta 12.1 \pm 4.1$  CP, bpm). Respiratory rate was significantly increased to exercise from rest in only the CP group. The delta changes of blood flow from rest to exercise was slightly smaller in the CP group. **CONCLUSIONS:** While HR and MAP increased to exercise from rest in similar fashion in both groups, increase in BF to exercise was blunted in adults with CP. Our preliminary data suggest that there are differential neural control mechanisms to regulate BP in the CP population. Other mechanisms, possibly vascular contribution from non-contracting limbs or chemoreceptor activity, may contribute to BP response during exercise in the CP population.

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**EXPANDED ABSTRACT**

**INTRODUCTION**

Cerebral palsy (CP) is a non-progressive and permanent neurological disorder leading to musculoskeletal dysfunction and immobility (1, 2). A major clinical problem with CP is early development of chronic diseases with increased rates of mortality (3, 4). Previous studies have identified that health conditions, such as health-related risk factors (e.g., obesity, hypertension, dyslipidemia) that are known to increase chances of developing chronic cardio-metabolic disease (e.g., type 2 diabetes, cardiovascular disease), are concurrent with individuals with CP at significantly higher rate compared to populations without CP (4, 5, 6). Such health-related problems further burden the CP population and their caretakers; thus, it is worthy to study physiological basis to gain a better understanding of the state of CP.

Individuals with CP experience higher rates of cardiovascular issues starting early in adulthood and worsening with age (6, 7). Cardiovascular function appears to be compromised in the general population with cardiovascular and metabolic diseases; vascular dysfunction is an indicative of atherosclerotic cardiovascular disease risk (8). Although it lacks enough scientific evidence, the vascular health of children and adolescents with CP seems to match that of the typically developing peers (9). However, recent evidence suggests that endothelial function driven from the flow mediated dilation was significantly lower in the adult CP population compared to controls (10). Individuals with CP exhibit different pathophysiology stemming from unique neurological disturbances. However, the early aging effect in the adults with CP may be one of the contributing factors to facilitate the cardiovascular dysfunction.



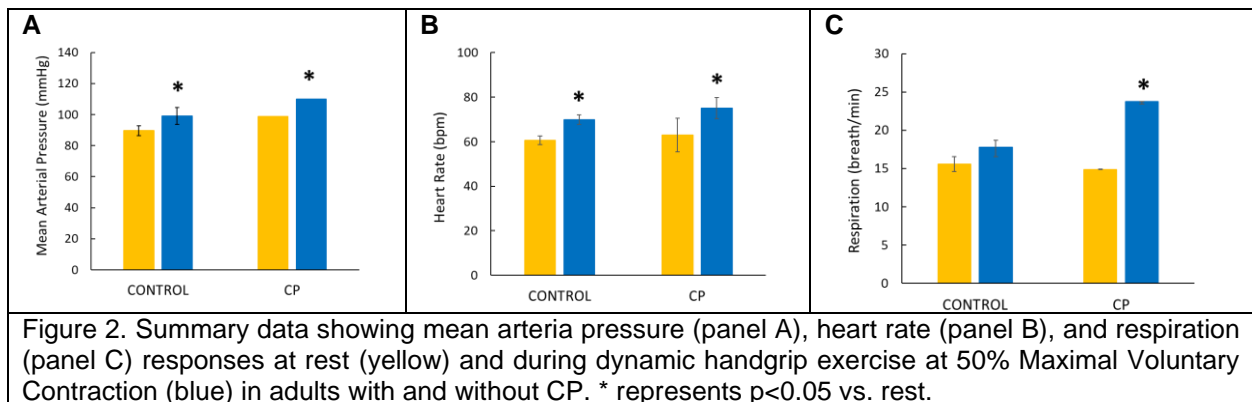
respiration via pneumobelt were continuously measured before, during and after 2 minutes of dynamic handgrip exercise at 35% and 50% of maximal voluntary contraction. Experimental setting is shown in Figure 1A. In addition, diameter, blood velocity, and flow of the brachial artery were measured using Doppler ultrasound (flow images shown in Figure 1B) on the contracting arm throughout the experiment.

In addition, diameter, blood flow velocity, and flow of the brachial artery were measured using Doppler ultrasound (flow images shown in Figure 1B), and a force transducer setting for a rhythmic handgrip exercise (Figure 1C) on the contracting arm throughout the experiment.

## RESULTS

At rest, both control and CP groups had similar resting HR ( $60.7 \pm 1.9$  control and  $63.0 \pm 7.5$  CP, bpm, Figure 2A). While resting respiratory rate was lower in CP group compared to the control, resting mean arterial pressure (MAP) and brachial blood flow (BF) tended to be higher in CP ( $p=0.08$ ).

MAP and HR were significantly increased to exercise from rest in both groups with no group differences (MAP,  $\Delta 9.3 \pm 2.2$  control and  $\Delta 11.2 \pm 3.3$  CP, mmHg; HR,  $\Delta 8.4 \pm 1.5$  control and  $\Delta 12.1 \pm 4.1$  CP, bpm, Figure 2A and 2B). Respiratory rate was significantly increased to exercise from rest in only the CP group (Figure 2C).



The diameters of the brachial artery were similar at baseline and during exercise when compared to control (Figure 3A). The changes in brachial diameter resting to exercise are minimal in both groups. As noted, the diameter during exercise indicates the average values of diameters during muscular contraction (constricted) and relaxation (dilated) phases. As a result of both diameter and velocity changes, brachial blood flow was increased from rest to exercise in both groups in qualitatively similar fashion (Figure 3C). The delta changes of blood flow from rest to exercise was slightly smaller in the CP group.

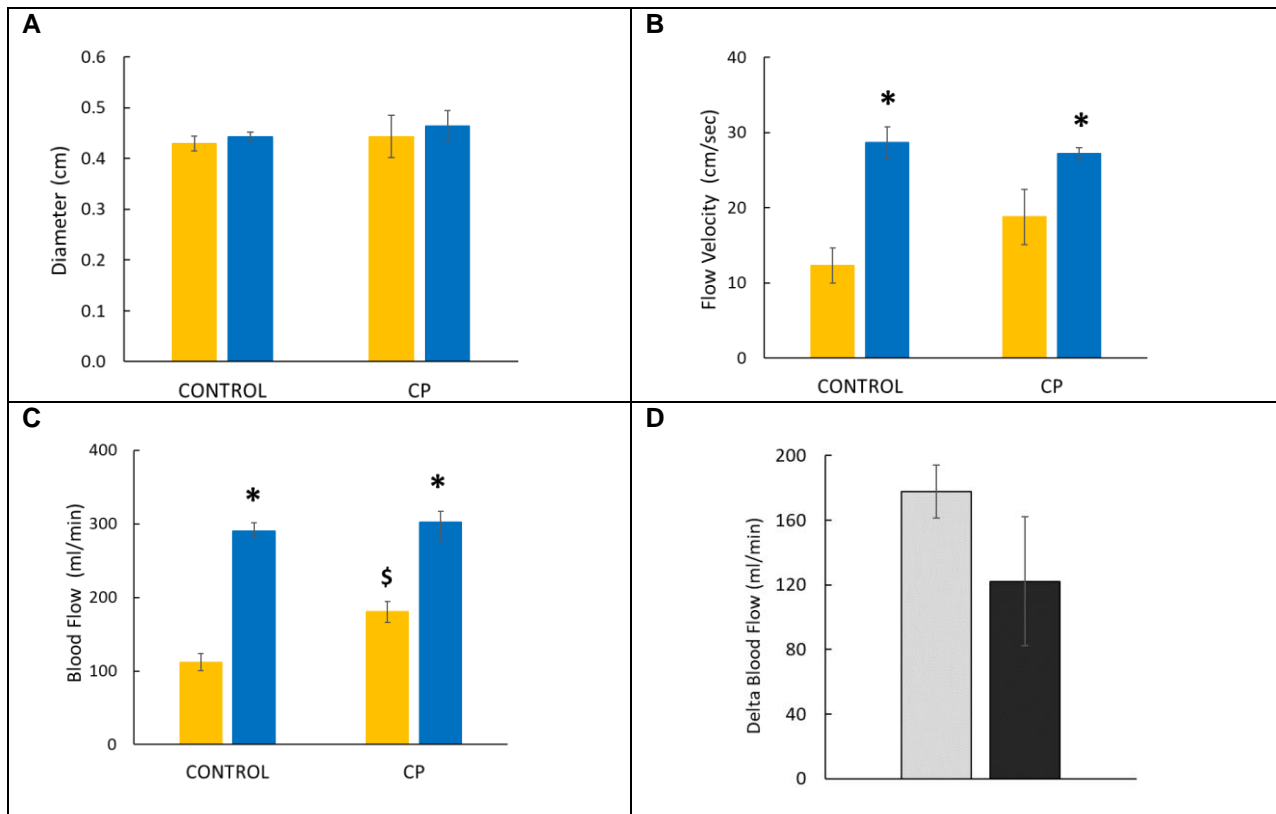


Figure 3. Summary data showing diameters (panel A), blood flow velocity (panel B), and brachial blood flow (panel C) responses at rest and during dynamic handgrip exercise at 50% Maximal Voluntary Contraction (blue) in adults with and without CP. Changes in blood flow from rest to exercise (panel D) in control (gray) and CP (black) groups. \* represents  $p < 0.05$  vs. rest. \$ represents  $p < 0.05$  vs. control.

## CONCLUSION

While HR and MAP increased to exercise from rest in similar fashion, increase in BF to exercise was blunted in adults with CP. Interestingly, resting BF was higher in adults with CP compared to controls which made us speculate that CP participants may have a higher demand of flow to resting skeletal muscles due to general spasticity which all our CP participants have as a main characteristic of CP. Nonetheless, BF response to exercise appears to be blunted in CP groups which may indicate impaired vascular function. Increase in MAP to exercise is a result from higher cardiac output (a product of HR and stroke volume) and total peripheral resistance. Similar increase in MAP to exercise may be due to both higher HR (Figure 2A, 2B) with differential changes in BF (attenuated vasodilation) to contracting muscles (Figure 3C and 3D) in the CP population.

To conclude, our preliminary data suggest that there are differential neural control mechanisms to regulate BP in the CP population. Other mechanisms, possibly vascular contribution from non-contracting limbs or chemoreceptor activity, may contribute to BP response during exercise in the CP population.

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