

## Research article

# IMMEDIATE RE-HYDRATION POST-EXERCISE IS NOT COINCIDENT WITH RAISED MEAN ARTERIAL PRESSURE OVER A 30-MINUTE OBSERVATION PERIOD

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### ABSTRACT

This investigation assessed the effects of immediate or delayed re-hydration post-exercise, on mean arterial blood pressure (MAP) and on blood plasma volume (PV) expansion post-exercise. It was hypothesised that fluid ingestion would raise MAP and attenuate PV expansion. On two occasions separated by seven days, eight males (age  $20.4 \pm 1.7$  years, mass  $79 \pm 5$  kg [means  $\pm$  SD];  $VO_{2\max}$   $48 \pm 11$  mL·kg<sup>-1</sup>·minute<sup>-1</sup>, [mean  $\pm$  SE]) cycled in the heat (35°C, 50% relative humidity) at a power output associated with 50%  $VO_{2\max}$ , until 1.0kg body mass was lost. 1L water was given either immediately thereafter, or two hours post-exercise by random assignment. On both occasions, MAP was calculated every five minutes for a period of 30-minutes post-exercise, and change in PV was calculated 24-hours post-exercise. Repeated measures ANOVA for MAP results suggested a low probability of a treatment effect ( $p = 0.655$ ), a high probability of a time effect ( $p = 0.006$ ), and a moderately high probability of a time x treatment interaction ( $p = 0.076$ ); MAP tended to be lower when fluid had been consumed. PV expansions 24-hours post-exercise were not significant changes with respect to zero, and were not significantly different by treatment condition. In conclusion: (a) The exercise was not sufficient to elicit significant PV expansions; thus, we were unable to determine the effects of the timing of post-exercise re-hydration on PV expansion. (b) The hypothesis regarding MAP in response to drinking was not supported, rather there was a 92% probability that the inverse affect occurs.

**KEY WORDS:** Dehydration, re-hydration, blood pressure, plasma volume.

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### INTRODUCTION

Twenty-four hours following intense exercise, blood plasma volume (PV) has been shown to increase by  $\sim 7 \pm \sim 1\%$  (Gillen et al., 1991; Haskell et al., 1997; Nagashima et al., 1999). The increased resting PV appears to be a compensatory response to the exercise, resulting in increased maximal stroke volume and consequently increased maximal cardiac output (Hopper et al., 1998; Kanstrup and Ekblom,

1982; Krip et al., 1997). Increased maximal cardiac output in turn improves  $VO_{2\max}$  (Coyle et al., 1990). These adaptations are important, as cardiovascular stress during subsequent exercise at any given intensity is ameliorated (Fellmann, 1992; Hopper et al., 1998; Krip et al., 1997; Maw et al., 1996; Mitchell et al., 2000; Watt et al., 1999).

Previous research (Hayes et al., 2000) has suggested that a period of central-venous hypotension post-exercise may be critical in eliciting PV expansion. This is so, as translocation of albumin

from the lymph to the plasma pool appears to be the primary mediator of PV expansion (Gillen et al., 1991; Nagashima et al., 1999). In order for albumin to be moved from the lymph to the plasma, the lymphatic outflow pressure must exceed the central venous blood pressure (CVP) (Wu and Mack, 2001). Mean arterial pressure (MAP) is related to CVP via the Frank-Starling Law of the Heart, therefore MAP has previously been deemed an acceptable marker to non-invasively infer trends in CVP (Hayes et al., 2000; Nagashima et al., 1999; Wu and Mack, 2001). Other researchers (Endo et al., 2001) have reported a transient increase in MAP, immediately following the ingestion of water. These findings provide for the current hypothesis that immediate re-hydration post-exercise may, while ameliorating dehydration, adversely affect CVP with respect to the processes leading to expansion in PV.

### **Statement of the problem and hypotheses**

PV expansion 24-hours post-exercise is likely to be related to post-exercise CVP/MAP (Gillen et al., 1991; Hayes et al., 2000; Nagashima et al., 1999; Wu and Mack, 2001). Drinking immediately post-exercise may, while rapidly ameliorating dehydration (Mitchell et al., 2000), actually attenuate the post-exercise hypotension (Endo et al., 2001) required for maximal PV expansion (Hayes et al., 2000; Wu & Mack, 2001). Therefore the purpose of the present study was to assess MAP for a period of 30-minutes post-exercise, and to calculate PV expansion 24-hours post-exercise: a) when re-hydration fluid was given immediately, or b) withheld for two hours post-exercise. It was hypothesised that drinking immediately post-exercise would be associated with increased MAP over the following 30-minutes, and that this would be associated with lower PV expansions 24-hours post-exercise (Hayes et al., 2000; Nagashima et al., 1999; Wu and Mack, 2001).

## **METHODS**

### **Subjects**

Eight recreationally active males (age  $20.4 \pm 1.7$  years, body mass  $79 \pm 5$  kg [means  $\pm$  SD];  $VO_{2\max}$   $48 \pm 11$  mL $\cdot$ kg $^{-1}\cdot$ minute $^{-1}$ , cycling power output associated with  $VO_{2\max}$   $281 \pm 13$ W [means  $\pm$  SE]) volunteered to take part and provided written informed consent. The Waikato Institute of Technology Human Research Ethics Committee approved all procedures. Subjects underwent pre-screening for exercise contraindications, specifically a) a comprehensive written medical history questionnaire, b) resting blood pressure in excess of 145 / 90 mmHg, and c) resting heart rate in excess of 100 beats $\cdot$ minute $^{-1}$ .

### **Experimental design overview**

Prior to the first experimental session (7-14 days), subjects had their individual  $VO_{2\max}$  determined using a standard incremental cycle ergometer (Monark 818e or 828e, Sweden) protocol. Exercise was subsequently conducted in an environmental chamber (35°C, 50% relative humidity) on the cycle ergometer with power output set to 50% of that associated with  $VO_{2\max}$ . Exercise was continuous in 15-minute blocks separated by short rests while nude weighing occurred, until 1kg body mass ( $1.3 \pm 0.1\%$  [mean  $\pm$  SE] body mass) was lost. In one trial, subjects consumed 1L water immediately following the exercise, while on the other occasion the same amount of water was consumed two hours post-exercise. Subjects completed trials in random, counterbalanced order. Immediately following exercise in both trials, subjects underwent a 30-minute period of continuous postural control (sitting on a chair in the thermo-neutral environment [20°C] with feet flat on the floor and palms hands down on the knees), during which time blood pressure measurement (systolic:diastolic, mmHg) was conducted every five minutes using an automatic arm-cuff pneumatic sphygmomanometer (Datascope Accutorr1, USA). MAP was calculated from these blood pressure parameters, according to  $MAP = (systolic\ pressure + (2 \times diastolic\ pressure)) / 3$  (Nagashima et al., 1999). Changes in PV (%) over each 24-hour period were calculated using haemoglobin (Hb) concentration (g $\cdot$ dL $^{-1}$ ), and haematocrit (Hct, %) from venous blood samples taken prior-to exercise, and again after 24-hours recovery, according to  $((Hb_{pre}/Hb_{post}) \times (100-Hct_{post} / 100-Hct_{pre}) - 1) \times 100$  (Yang et al., 1998). The validity of this change in PV (%) estimation method is discussed at length elsewhere (Gillen et al., 1991; Haskell et al., 1997; Nagashima et al., 1999; 2001; Yang et al., 1998). Urinary outputs (mL) were recorded over the 24-hour recovery period by each subject in a journal supplied by the researchers. Ambient temperature, relative humidity, relative exercise intensity / cadence, body posture, diet / hydration (24-hours prior to and post-exercise), and recovery procedures (24-hours post-exercise) were controlled during both trials (see detailed procedures to follow).

### **Pre experimental protocols**

#### **Determination of $VO_{2\max}$**

Subjects reported to the laboratory 7-14 days prior to beginning the experimental protocol, for the determination of cycle  $VO_{2\max}$ . After a five minute self-directed warm up, subjects were connected to the open circuit metabolic gas analysis equipment (Vmax 29 series, SensorMedics, USA).  $VO_2$ , and

$\text{VCO}_2$  ( $\text{mL}\cdot\text{kg}^{-1}\cdot\text{minute}^{-1}$ ) were monitored breath-by-breath, and smoothed by 30-second average during the exercise protocol. The test was conducted using a constant cadence of 60 revolutions $\cdot\text{minute}^{-1}$ , and began at a power output of 100W. Power output was increased by 25W every minute, and  $\text{VO}_2$  max was deemed to have been achieved when (a) additional loading elicited no further increase in  $\text{VO}_2$ , and/or (b) respiratory exchange ratio exceeded 1.05, and/or (c) volitional exhaustion or inability to maintain 60 revolutions $\cdot\text{minute}^{-1}$  was accompanied by (b).

### ***Pre-control and blood sampling***

Subjects were asked during the entire experimental period to refrain from consuming any nutritional supplement, ergogenic aid (or proposed ergogenic aid), alcohol, or other recreational drug. Further, subjects were asked to refrain from caffeine in food and drink for 48-hours prior to, and during the 24-hours of testing. Finally, subjects were asked to maintain their normal sporting and exercise activities, except to refrain from exercise for 24-hours prior to testing, and during the 24-hours post testing. Compliance was assessed via each subject completing a nutritional and training diary, which was supplied by the researchers. Prior to and during each experimental 24-hour period, dietary and hydration control occurred. During this time all subjects consumed the same foods. Specifically, energy intakes were  $8350 \pm 203.6$  kJ (mean  $\pm$  SD),  $3412 \pm 127.3$  mg  $\text{Na}^+$ , (breakfast 05:00; lunch 13:00; dinner 20:00), 1.5 L water [five equal aliquots, taken at 09:00; 13:00; 17:00; 21:00; 05:00]).

Following the first 24-hour period of dietary and hydration control subjects reported to the laboratory at the appointed time of day (06:00), which was held constant throughout the testing period. After subjects' journals were checked for compliance, subjects were cleared to proceed with testing. Subjects first voided, and nude body mass was recorded in a private room. Subjects then underwent postural control (as described previously) for a continuous period of 20-minutes. This postural control was to allow for stabilisation of PV (Kargotich et al., 1998). Next, resting MAP was determined as described previously. Following this, a 5mL sample of blood was collected from a superficial vein in the antecubital fossa of either arm. Samples were immediately refrigerated at 3°C, and analysed within 120-minutes (see below).

## **Experimental protocol**

### ***Exercise protocol***

Following blood sampling, subjects entered the environmental chamber (35°C, 50% relative humidity), and began cycling at a power output

equal to 50% of the power output associated with individual  $\text{VO}_2$  max ( $141 \pm 7\text{W}$  [mean  $\pm$  SE]). Eating and drinking during the experimental sessions was not allowed. Every 15-minutes, subjects moved to a private room, towelled themselves as dry as possible, and repeated nude weighing. When 1kg was lost, exercise ceased and subjects returned immediately to postural control in the thermo-neutral environment. Mass actually lost ranged between 0.95 and 1.05kg. Subjects all reached the target weight loss within 90-minutes of exercise.

Subjects (in the immediate re-hydration trial only) were then given 1L of tap water without delay, which they consumed immediately and in full. Following this, MAP was determined every five minutes under continuous postural control as above. Subjects in the delayed re-hydration trial were issued with their bolus of water and instructed to consume it in full at the appropriate time (2-hours post-exercise cessation). Finally, before departure subjects were asked to refrain from adopting a supine posture for at least 2-hours (Mack et al., 1998; Nagashima et al, 1999), to refrain from water immersion for at least two-hours (Hinghofer-Szalskay et al., 1987), and to refrain from exercise until they next reported to the laboratory 24-hours later (06:00) for repeated nude weighing, postural control, and blood sampling as above.

### ***Blood sampling and analysis***

Blood (5mL antecubital venous sample) was collected on each occasion using a standard 'complete blood count (CBC) lithium / heparin vacutainer', and a 20-gauge hypodermic needle. Haematocrit was determined in triplicate using capillary tubes filled from each sample vacutainer after they were inverted ten times to homogenise the blood. Capillary tubes were then centrifuged at 3600 revolutions $\cdot\text{minute}^{-1}$ , for a period of five-minutes. A ruler marked with 0.5 mm increments was used to determine the percentage of packed red cells after centrifuging, according to (*packed cell length / total sample length*)  $\times 100$ . Haemoglobin concentration in the samples was determined in duplicate following homogenisation, using a Co-oximeter (Ciba-Corning, 860 series, USA).

### ***Statistics, reliability of plasma volume change (%)***

All changes (%) and absolute values are reported as mean  $\pm$  SE. Changes in PV and body mass, as well as absolute urinary outputs (mL) were subjected to *t*-tests for dependant paired means. Alpha was set at ( $p < 0.05$ ). MAP values were submitted to ANOVA with repeated measures analysis (treatment, time, and treatment  $\times$  time). Pre-experimental repeated Hb and Hct determinations were used to determine the reliability of change in PV method as described by

**Table 1.** Mean arterial pressure at rest, and at 5-minute intervals post-exercise.

Condition	Subject	Mean Arterial Pressure (mmHg)						
		rest	5-min post-ex	10-min post-ex	15-min post-ex	20-min post-ex	25-min post-ex	30-min post-ex
Immediate Re-hydration	A	100	101	97	99	114	90	90
	B	93	95	92	83	93	90	90
	C	103	106	100	97	95	106	107
	D	101	93	92	95	93	110	90
	E	86	92	92	83	75	78	80
	F	90	110	90	81	97	92	96
	G	83	100	77	90	86	95	83
	H	87	87	83	78	87	77	100
<i>Mean (±SD)</i>		93 (8)	98 (8)	90 (7)	88 (9)	93 (11)	92 (12)	92 (9)
Delayed Re-hydration	A	92	116	106	108	110	102	105
	B	96	99	107	93	87	106	93
	C	95	99	92	90	89	91	97
	D	79	99	90	105	92	99	94
	E	83	105	83	99	77	85	85
	F	87	100	76	98	83	97	83
	G	77	93	88	89	89	82	89
	H	89	99	99	89	91	96	83
<i>Mean (±SD)</i>		87 (7)	101 (7)	93 (11)	96 (7)	90 (10)	95 (8)	91 (8)

Treatment (condition);  $F = 0.217$ ,  $p < 0.655$ ; Time;  $F = 3.524$ ,  $p < 0.006$ ; Treatment x Time (interaction);  $F = 2.078$ ,  $p < 0.076$

Dill and Costill (1974). Subjects supplied blood samples at rest following controls as described above; on three occasions separated by seven days. This analysis indicated that subjects PV routinely changed from reading to reading (without any intervention) by (on average)  $0\% \pm 2.96\%$  (i.e. mean  $\pm$  SE).

## RESULTS

Subjects experienced an identical  $1.27 \pm 0.06\%$  dehydration (% body mass) in both trials. Subjects' body masses at 24-hours post-exercise were also identical ( $99.7 \pm 0.3\%$  of baseline). Whole body re-hydration was therefore not different between trials. 24-hour urinary outputs were  $1488 \pm 132\text{mL}$  after immediate re-hydration, and  $1353 \pm 94\text{mL}$  after delayed re-hydration. These values were not statistically different. The change in PV (%) 24-hours after the immediate re-hydration protocol was  $1.57 \pm 2.48\%$ , while the change in PV (%) 24-hours after delayed re-hydration was  $0.82 \pm 0.88\%$ . Neither of these mean PV expansions were significant increases over zero, nor significantly different between trials.

Mean arterial pressure at rest, and at 5-minute intervals post-exercise (absolute values) is presented

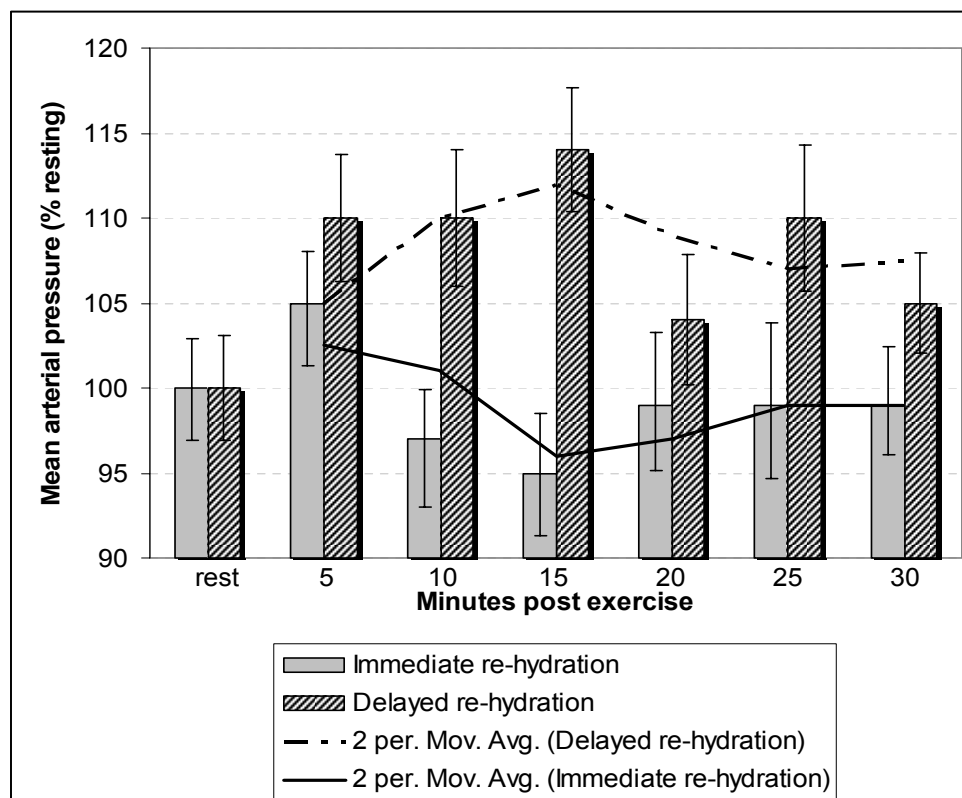
in Table 1. Mean arterial pressure (% resting) is presented in Figure 1.

## DISCUSSION

### MAP responses

The important finding of this study was that drinking immediately post-exercise (at the intensity and dehydration level here assessed), did not result in raised MAP over 30-minutes post-exercise; as compared to withholding oral fluids for two hours, as expected. Rather, there was a tendency ( $p = 0.07$ ) toward the reverse. The practical utility of this finding is that drinking immediately following exercise is not apparently contraindicated for those who wish to induce PV expansion 24-hours subsequent, assuming the theoretical basis alluded to (Wu and Mack, 2001) for PV expansion in the period of several hours post-exercise, holds over a 24-hour period. However, further research is required in order to clarify the relationship between post-exercise MAP and PV expansion over 24-hours post-exercise, as changes in PV reported here were not significant.

The finding that MAP tended lower, rather than higher as hypothesised, in the immediate re-hydration trial cannot be easily explained by differential influences of confounding variables



**Figure 1.** Mean arterial pressure (% resting value  $\pm$  SE,) after an identical exercise / dehydration protocol completed twice in random counterbalanced order. Re-hydration was given immediately post-exercise, or not until 2-hours post-exercise, i.e. 90-minutes after blood pressure measurements had ceased.

discussed elsewhere by others (Anderson et al., 1998; Blackman et al., 2000; Carter et al., 1998; Convertino, 2003; Crandall et al., 1999a; 1999b; Fellmann, 1992; Ferrari et al., 2001; Flamm et al., 1990; Graça et al., 2002; Hinghofer-Szalskay et al., 1987; Johansen et al., 1998; Krier et al., 1998; Lundvall and Lindgren, 1998; Nishida et al., 1988; Nose et al., 1988; Nagashima et al., 1999; Nagashima et al., 2001; Planitzer et al., 2001; Sandler et al., 1984; Stewart et al., 2002; Wu and Mack, 2001), as comprehensive control procedures have been adopted. Ambient temperature, relative humidity, relative exercise intensity / cadence, body posture, diet and hydration volume and composition (24-hours prior to and post-exercise), and recovery procedures (24-hours post-exercise) were controlled during both trials as previously discussed. Future research is required to elucidate the mechanism of this phenomenon, therefore.

### **PV expansions**

The exercise regimen used during the current research lead to insignificant PV expansions (with respect to zero) 24-hours later. The current researchers proceeded under the parsimonious assumption that fluid loss from the vasculature was likely to be the primary causal stimuli for reduced MAP post-exercise. Indeed, percentage dehydration

levels (by body mass lost) were similar to those induced by other research designs using shorter, intermittent and higher relative intensity exercise protocols (Gillen et al., 1991; Haskell et al., 1997; Kay et al., 2004; Kay et al., 2005; Nagashima et al., 2001). Paradoxically, those researchers were invariably able to demonstrate significant PV expansions 24-hours later. Indeed Kay et al. (2005) were able (subsequent to the collection of this data) to demonstrate that percentage dehydration is not likely to be the primary causal mediator.

Rather, those authors (Kay et al., 2004; 2005) proposed relative exercise intensity may be the primary causal mediator. To elucidate, the current research shows that cycle exercise at 50%  $VO_{2max}$  for up to 90-minutes elicited insignificant PV expansions of  $\sim 1.2\%$  (pooled results). Further, PV expansions elicited by exercise at  $\sim 75\%$   $VO_{2max}$ , were  $\sim 3.7\%$  (Kay et al., 2004; Kay et al., 2005), somewhat lower than PV expansions noted by other researchers ( $\sim 7.3\%$ ) when intensity was 85%  $VO_{2max}$  (Gillen et al., 1991; Haskell et al., 1997; Nagashima et al., 2001). The likely underlying mechanism of increased PV expansion reported after higher intensity protocols (Gillen et al., 1991; Haskell et al., 1997; Kay et al., 2004; Kay et al., 2005; Nagashima et al., 2001) may be altered Starling forces (Haskell et al., 1997), and the likely effects of nitric oxide on

MAP post-exercise (Blackman et al., 2000; Ferrari et al., 2001; Planitzer et al., 2001). Further research is required in order to test this new hypothesis, and clarify this suggested relationship between exercise intensity, nitric oxide, MAP, and PV expansion.

## CONCLUSIONS

It was originally hypothesised that drinking immediately post-exercise would raise MAP, and lower the magnitude PV expansion 24-hours post-exercise. Under the conditions imposed here, the first hypothesis was not supported, and the second cannot be commented upon. Immediate fluid ingestion was associated with somewhat lower MAP values post-exercise and very slightly higher PV expansions 24-hours post-exercise (review results for exact magnitudes and probabilities) when compared to delayed fluid ingestion. The relatively low (~1.2%) PV expansions noted here make the statistical interpretation of any differences between conditions with respect to PV changes impossible, due to the inherent precision (~2% typical error from biological and technical sources combined) of the PV expansion calculation adopted.

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##### **Degrees**

MSc (SpExSci), BSpExSci (Hons)

##### **Research interests**

Metabolism, muscular fatigue, cardiovascular function, sport specificity of training and fitness assessment.

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##### **Brendan J. O'BRIEN**

##### **Employment**

Lecturer, School of Human Movement and Sport Sciences, University of Ballarat, Ballarat, Australia.

##### **Degree**

PhD

##### **Research interests**

Metabolism, muscular fatigue, cardiovascular function.

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##### **Nicholas D. GILL**

##### **Employment**

Senior lecturer, School of Sport and Exercise Science, Waikato Inst. of technology, Hamilton, New Zealand.

##### **Degree**

PhD

##### **Research interests**

Metabolism, muscular fatigue, cardiovascular function, strength / power / conditioning, Rugby Union, sport specificity of training and fitness assessment.

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**KEY POINTS**

- Post exercise hypotension is perhaps the most important mediator of plasma volume expansion post exercise
- It was hypothesised that drinking water immediately post exercise would attenuate post exercise hypotension by rapidly ameliorating dehydration
- We found that not only was our hypothesis incorrect, but rather a 92% probability exists that the inverse is true, i.e. drinking water in fact leads to lowered blood pressure, as compared to not drinking.

**✉ Bartholomew Kay**

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