

# NTproBNP, Lactate, and Hemodynamic Responses to Multiple vs One-Repetition Max Strength Tests

Jill Nustad, DSC, ACSM-CEP<sup>1</sup>, Casey Sailer, BS, ACSM-EP-C<sup>1</sup>, Kelsey Ekstedt, BS, ACSM-EP-C<sup>1</sup>, Aaron Brydl, BS<sup>1</sup>, Eric Drake, BS, ACSM-EP-C<sup>1</sup>, Jason Shaul, BS, ACSM-EP-C<sup>1</sup>, Taylor Chavez, BS<sup>1</sup>, Moran Saghiv, PhD<sup>1</sup>

## ABSTRACT

**Background:** Cardiac stress biomarker N-Terminal pro-brain natriuretic peptide (NTproBNP), blood lactate, and hemodynamic responses were compared between one-repetition max (1RM) and multiple-repetition max (MRM) strength assessments in overweight and obese conditions.

**Methods:** Twenty sedentary males, ages 19–28 years, were divided into overweight ( $n = 11$ ) and obese ( $n = 9$ ) groups. Subjects performed MRM and 1RM using a chest press machine. Heart rate (HR), blood pressure, blood lactate, and NTproBNP were measured at baseline, immediately post, and 15-min post strength assessment. Power output was also calculated.

**Results:** Significant increases in all variables except NTproBNP were observed from baseline to immediate post ( $p < 0.05$ ) in both 1RM and MRM. Significant differences in HR were found between 1RM and MRM immediate post (1RM = 122 bpm, MRM = 147 bpm;  $p = 0.001$ ), and blood lactate immediate post (1RM =  $3.0 \text{ mmol} \cdot \text{L}^{-1}$ , MRM =  $5.7 \text{ mmol} \cdot \text{L}^{-1}$ ;  $p = 0.001$ ) and 15-min post (1RM =  $1.9 \text{ mmol} \cdot \text{L}^{-1}$ , MRM =  $4.0 \text{ mmol} \cdot \text{L}^{-1}$ ;  $p = 0.001$ ). BMI interactions were found with systolic blood pressure for 1RM ( $p = 0.014$ ) and MRM ( $p = 0.015$ ), and diastolic blood pressure for 1RM ( $p = 0.047$ ) and MRM ( $p = 0.006$ ), where obese individuals had higher blood pressure responses compared to overweight. Power output was greater in 1RM vs MRM and greater in the obese compared to overweight.

**Conclusion:** NTproBNP remained at safe levels for MRM and 1RM tests in both groups. Findings suggest MRM may induce greater hemodynamic and metabolic stress compared to 1RM, and obesity may provoke greater blood pressure responses to maximal resistance exercise. *Journal of Clinical Exercise Physiology*. 2017;6(4):61–70.

**Keywords:** blood pressure, heart rate, brain natriuretic peptide, strength assessment, obesity

## INTRODUCTION

Cardiac stress biomarker responses may offer new information to the safety of strength assessments in overweight and obese conditions.

The benefits of resistance exercise are well established; thus, resistance exercise is recommended as part of a

routine exercise program for most populations (1–3). In obese populations, resistance exercise has been shown to offset deleterious effects of obesity sarcopenia, including improvement of insulin resistance (4,5), reduced inflammatory markers (6), and reduced functional impairment (7). Additionally, muscular strength has been shown to be a

<sup>1</sup>Exercise Physiology Department, University of Mary, Bismarck, North Dakota

Address for correspondence: Jill Nustad, DSC, ACSM-C, Exercise Physiology Department, University of Mary, 7500 University Drive, Bismarck, ND 58504; Cell: (701) 400-8424, Work: (701) 355-8149, Fax: (701) 255-7687; email: jnustad@mary.edu.

Conflicts of Interest: No conflicts of interest.

Source of Funding: The University of Mary, Department of Exercise Physiology, funded this research in full, with no additional involvement or funding from any outside agency.

Copyright © 2017 Clinical Exercise Physiology Association

negative predictor of disability and mortality (8–10); hence, it is important to include strength training as an essential component of exercise routines for overweight and obese individuals. Likewise, assessment of strength provides clinical exercise physiologists and other rehabilitation professionals with information for the development of safe and effective individualized exercise programs, educating participants about their functional health/fitness levels, evaluation of progress, and motivation by establishing reasonable and attainable goals (1).

The one repetition maximum (1RM) strength assessment is used to measure the maximal amount of weight a person can lift or move through full range, in one repetition. A multiple repetition maximum (MRM) strength assessment measures the maximal amount of weight a person can lift in a given number of repetitions, where the last repetition maximizes volitional fatigue. The MRM is generally recommended to assess strength and base exercise prescriptions for clinical and special populations (1–3), although the evidence for this practice is an enigma. Gjølvaag and colleagues (11) suggested these recommendations may stem from reports that MacDougall et al. published in 1985, where exaggerated blood pressure responses were noted in high load/low repetitions (12). These results were explained by a combination of factors, including mechanical compression, pressor reflex, and increased thoracic pressure from brief moments of Valsalva maneuver (12). These findings, however, were the results of five healthy young males who were experienced body builders, and the data actually demonstrated higher mean pressure responses in MRM compared to 1RM. They also reported more exaggerated responses when exercising relatively larger muscle groups (leg vs arm and double vs single leg) in the same individuals (12), whereas systolic and diastolic BP responses to 8–12 repetition maximum were found to be independent of cross-sectional muscle size among different individuals (13).

More recently, Gjølvaag et al. (9) reported heart rate, blood pressure, rating of perceived exercise (RPE), and blood lactate responses increase with resistance training as a function of time and not only load, and found hemodynamic responses, lactate, and RPE in men and women with revascularized coronary artery disease (CAD) to be greater in a 15RM compared to 4RM (11). These findings, together with MacDougall's data (12) suggest a MRM approach might actually be a more stressful, and potentially dangerous, strength assessment approach compared to a high load/low repetition approach such as the 1RM. NTproBNP, a 76-amino acid fragment of brain natriuretic peptide (BNP; 14), is a biomarker of cardiac stress (diastolic pressure and wall stress in particular) commonly used for diagnosis and prognosis of heart failure (15,16). This biomarker has been shown to elevate from resting values as a result of endurance and strenuous shorter-term exercise (17,18), with higher resting values observed in untrained compared to trained individuals (18,19). While NTproBNP is predominantly secreted by the heart and commonly associated with heart failure, it also is secreted by neurons of the brain and has

been associated with psychological stress as well as genetic predispositions (20,21). Considering inconsistencies in the literature pertaining to safety and efficacy of 1RM and MRM strength assessments, it is of interest to explore NTproBNP responses to such strength assessments and relationships with lactate, hemodynamic (blood pressure and heart rate), and mechanical work outcomes. To the best of the authors' knowledge, NTproBNP has never been studied (acute or persisting responses) as an outcome measure of 1RM and MRM strength assessments, which could add to the information regarding safety with such assessments. Additionally, hemodynamics, lactate responses, and mechanical output relationships to NTproBNP concentrations have not been reported in obese and overweight populations in response to the mentioned strength assessments. Thus, the purpose of this study was: (a) to compare NTproBNP, hemodynamics, blood lactate responses, and mechanical output of 1RM vs 8–10 MRM in obese and overweight groups, and (b) to identify the magnitude of the responses and potential dangers associated. We hypothesized that the 8–10 MRM strength assessment would have greater NTproBNP, hemodynamic, and blood lactate responses (yet not reach dangerous levels), and that 1RM might be a less stressful strength assessment from a cardiovascular perspective but will likely induce greater mechanical load.

## METHODS

This study was approved by the University of Mary Institutional Review Board, University of Mary, Bismarck, ND.

### Participants

Twenty-two individuals volunteered for participation in this study. One was dismissed for having a normal body mass index and one was not able to complete both strength assessment sessions. Therefore, 20 untrained, low-to-moderate risk for cardiovascular disease (3), overweight ( $n = 11$ ) and obese ( $n = 9$ ) male subjects ages 20–28 years completed the study. *Overweight* was defined as body mass index (BMI) of 25–29.9  $\text{kg} \cdot \text{m}^{-2}$  and *obesity* as BMI of 30–40  $\text{kg} \cdot \text{m}^{-2}$ . *Untrained* was defined as not having exercised for a minimum of three times a week for 30 min in the last three months (3). Subjects were also excluded if they had known cardiac, pulmonary, or metabolic disease, hypertension or medicated for hypertension, had existing musculoskeletal injury, took prescription or over-the-counter medication/supplements that affect blood pressure or heart rate, smoked or chewed tobacco products, or were under perceived unusual psychological or emotional stress. All subjects signed an informed consent form, completed a health history questionnaire, and underwent risk stratification (2) and an interview session to assure understanding of the study protocol, accuracy of health history information, and adherence to pre-test instructions.

### Study Design

A randomized mixed design with repeated measures was used to compare NTproBNP, heart rate, blood pressure, and

blood lactate responses to MRM and 1RM strength assessments, and explore possible differences between obese and overweight conditions. Subjects completed a familiarization session, baseline, and two strength assessment sessions that were randomly determined such that half the subjects completed the 1RM first and the other half completed the MRM first. Outcome measures were obtained at baseline, immediate post and 15-min post 1RM and MRM. Power output was also compared between groups and conditions.

Assessments were completed at the same time in the morning and subjects were interviewed each morning to assure pre-test instructions were followed (small simple breakfast 2 h before testing, hydrate with approximately two cups of water that morning, avoid alcohol, tobacco, coffee or other caffeinated beverage, avoid exercise, alert researchers if suffering from any ailments of unusual stress). At baseline and strength assessment sessions, subjects sat for 5 min at rest prior to any measurements. Outcome variables were measured at baseline, immediate post and 15-min post 1RM and MRM, in the following order each time: heart rate (HR), blood pressure (BP), blood lactate (LA), and a 5-mL blood sample for NTproBNP. Baseline values were cross-referenced with pre-strength assessment measures for 1RM and MRM to account for potential psychological influence on outcome variables provoked by the anticipation of the strength assessment or blood draw.

### 1RM and MRM Strength Assessment Protocols

The 1RM and MRM strength assessments were conducted using a Life Fitness Chest Press exercise machine (model PSCPSE), and performed in randomized order with 48 h to 1 week between each bout. Subjects completed a general warm-up that consisted of 10–12 repetitions at a low sub-maximal weight followed by resting for 3–5 min, then incrementally increasing the resistance to reach maximal effort within about four trials (2). For both 1RM and MRM protocols, subjects were instructed on proper form (maintaining 5 points of contact: head, shoulders, low back, buttocks, and feet) and breathing mechanics, and allowed 3–5 min of recovery between trials. For 1RM, the weight was increased until the participant successfully moved through the full range of motion for no more than one repetition to volitional fatigue using proper form. The MRM was conducted using a metronome set at 60 bpm; the participant was allowed one second for the concentric phase and one second for the eccentric phase. The weight was adjusted until the participant completed 8–10 repetitions through full range of motion to volitional fatigue using proper form. Eight to 10 repetitions were chosen for MRM based upon reported accuracy to predict 1-RM (22) and recommendations for assessing strength in clinical populations (2,3). Borg's Rating of Perceived Exertion (RPE; scale from 0–10, with 10 being maximal exertion) was also obtained, where the goal was to ensure subjects reached volitional maximum fatigue, rating the trial 9 or 10 on the 10-point RPE scale (23). All subjects received verbal encouragement for both strength assessments.

## Outcome Measurements and Instrumentation

### *N-Terminal Pro-Brain Natriuretic Peptide*

NTproBNP ( $\text{pg} \cdot \text{mL}^{-1}$ ) was extracted from a 5-mL blood sample drawn at the antecubital fossa (cubital vein) while the subject was seated, by a certified phlebotomist following universal precautions for bloodborne pathogens. Venous blood collected in ethylenediaminetetraacetic acid (EDTA) tubes was refrigerated and then centrifuged at 1200 rpm for 10 min at 23 °C within 5 h from the blood draw. Blood plasma was then pipetted from the EDTA tubes; the rest of the EDTA tube's content was thrown away and destroyed. The plasma extract was kept in micro-tubes and stored at  $-4$  °C until further analysis by means of enzyme-linked immunosorbent assay (ELISA). A RayBio® Human proBNP ELISA Kit (catalog #: ELH-proBNP) was used to determine NTproBNP, following the instructions of RayBio®. The limits of detection as provided by the manufacturer were 5  $\text{pg} \cdot \text{mL}^{-1}$  for NTproBNP. The NTproBNP ELISA method is mainly characterized by the absence of cross-reactivity with circulating NTproBNP, allowing for specific determination of human NTproBNP.

Plasma was loaded into three wells for every sample taken (baseline, immediate post, and 15-min post, where immediate post was obtained from 30 sec to 5 min after completing the assessment). The average of the results of the three wells was calculated and is presented as the representing value of NTproBNP for that sample. Regarding reproducibility, the intra-assay CV% was <10%, and the inter-assay CV% was <12%.

### *Blood Pressure*

Systolic (SBP) and diastolic (DBP) blood pressure (mmHg) were measured via manual auscultation using a calibrated automatic Welch Allen DDS6 Trigger Aneroid Sphygmomanometer, and recorded for baseline, pre-exercise, immediate post 1RM and MRM, and 15-min post 1RM and MRM. Placement of the cuff was standardized to the left side of the upper arm at heart level. Appropriate cuff size (adult large or adult regular) was determined using designated markings on the cuff, and placement of the stethoscope was at the antecubital space over the brachial artery. During all measurements, the participants were instructed to relax the arm and allow the technician to support arm weight at the level of the heart. The cuff was inflated to approximately 20 mmHg above the first Korotkoff sound, with pressure slowly released at a rate of 2–5 mmHg per second (2). SBP was recorded as the first Korotkoff sound heard and DPB at the last sound heard before the disappearance of Korotkoff sounds.

### *Heart Rate*

HR (bpm) was measured noninvasively via a Polar F1 Heart Rate Monitor, with the transmitter strapped directly to the chest and the associated Polar Heart Rate watch held in close proximity.



### Blood Lactate

Blood lactate ( $\text{mmol}\cdot\text{L}^{-1}$ ) was obtained via fingertip point of care (POC) lactate meter (NOVA Biomedical Lactate Plus analyzer). The fingertip was cleaned with an alcohol swab and punctured with a 28-gauge Dynarex Sensilance Safety Lancet. The first expression of blood was wiped with a sterile gauze and the second expression of blood taken for the measure.

### Power Output

Power ( $\text{Watts} = \text{Force} \times \text{Distance} / \text{Time}$ ) was calculated for 1RM and MRM using measured distance (m) each subject moved the weight (kg) stack per one-second repetition.

### Statistical Analyses

SPSS 23 for Windows was used to analyze the data, comparing responses to 1RM vs MRM treatments and between overweight and obese conditions. Comparisons between treatments were conducted via paired *t*-tests. One-way repeated measures ANOVA and the Tukey post hoc test were used to compare between group conditions (overweight vs. obese). Significance was set at  $p < 0.05$ . Results are presented as means  $\pm$  standard deviation, T and F values when appropriate.

## RESULTS

All subjects ( $n = 20$ ) completed both 1RM and MRM treatments without sustaining any injury and without exaggerated, potentially dangerous responses in the outcome variables. Mean number of sets/trials to achieve the 1RM vs MRM were significantly different ( $3.7 \pm 1.22$  and  $2.75 \pm 1.02$ , respectively;  $p = 0.026$ ), where it took an average of one additional set/trial to complete the 1RM. Subject characteristics for overweight ( $n = 11$ ) and obese ( $n = 9$ ) conditions are presented in Table 1, as means  $\pm$  standard deviation. Blood draws were not obtainable for three subjects immediate post, and five subjects 15-min post treatment. Therefore NT-proBNP within subjects' data analysis subject numbers were 17 and 15, respectively, whereas  $n = 20$  for all other outcome variables.

### 1RM vs MRM Treatments

NTproBNP responses were not significantly different for 1RM and MRM treatment groups immediate post or 15-min post, respectively ( $50.3 \text{ pg}\cdot\text{mL}^{-1} \pm 1.81$  vs  $46.3 \text{ pg}\cdot\text{mL}^{-1} \pm 1.56$ ,  $t(16) = 0.720$ ,  $p = 0.482$  and  $45.7 \text{ pg}\cdot\text{mL}^{-1} \pm 0.99$  vs  $47.3 \text{ pg}\cdot\text{mL}^{-1} \pm 1.58$ ,  $t(14) = -0.331$ ,  $p = 0.745$ , respectively).

Heart rate response was significantly higher in MRM compared to 1RM immediate post (MRM =  $147 \text{ bpm} \pm 12.49$  vs 1RM =  $122 \text{ bpm} \pm 18.44$ ,  $t(19) = -5.857$ ,  $p < 0.0001$ ) and was not significantly different 15-min post (MRM =  $85 \text{ bpm} \pm 10.58$  vs. 1RM =  $82 \text{ bpm} \pm 11.2$ ,  $t(19) = -1.555$ ,  $p = 0.136$ ).

Blood lactate was significantly higher in MRM compared to 1RM immediate post (MRM =  $5.7 \text{ mmol}\cdot\text{L}^{-1} \pm 2.03$  vs 1RM =  $3.0 \text{ mmol}\cdot\text{L}^{-1} \pm 1.67$ ;  $t(19) = -4.803$ ,  $p < 0.0001$ ) and 15-min

post (MRM =  $4.1 \text{ mmol}\cdot\text{L}^{-1} \pm 1.53$  vs 1RM =  $1.9 \text{ mmol}\cdot\text{L}^{-1} \pm 0.56$ ;  $t(19) = -6.408$ ,  $p < 0.0001$ ).

Systolic and diastolic blood pressure responses were not significantly different among MRM and 1RM treatments immediate post or 15-min post (SBP:  $142 \text{ mmHg} \pm 16.32$  vs  $144 \text{ mmHg} \pm 16.55$ ,  $t(19) = -0.545$ ,  $p = 0.592$  and  $124 \text{ mmHg} \pm 10.56$  vs  $125 \text{ mmHg} \pm 11.72$ ,  $t(19) = -0.612$ ,  $p = 0.548$ , respectively; DBP:  $67 \text{ mmHg} \pm 10.16$  vs.  $67 \text{ mmHg} \pm 11.80$ ,  $t(19) = 0.037$ ,  $p = 0.971$ ; and  $73 \text{ mmHg} \pm 9.28$  vs  $72 \text{ mmHg} \pm 10.25$ ,  $t(19) = 0.805$ ,  $p = 0.431$ , respectively).

A significant moderate, positive correlation was found between power and 15-min post NTproBNP responses for both 1RM and MRM, respectively ( $r = 0.552$ ,  $p = 0.022$ ;  $r = 0.529$ ,  $p = 0.029$ ).

### Overweight ( $n = 11$ ) vs Obese ( $n = 9$ ) Conditions

A significant time (baseline to immediate post to 15-min post)  $\times$  BMI category interaction was found for systolic blood pressure for both 1RM ( $F(1) = 7.334$ ,  $p = 0.014$ ) and MRM ( $F(1) = 7.296$ ,  $p = 0.015$ ) (Figure 1), and diastolic blood pressure for both 1RM ( $F(1) = 4.559$ ,  $p = 0.047$ ) and MRM ( $F(1) = 9.911$ ,  $df = 1$ ,  $p = 0.006$ ) (Figure 2), where obese individuals had higher blood pressure responses compared to overweight for both 1RM and MRM (Figures 1 and 2). A time  $\times$  BMI interaction did not occur for NTproBNP (Figure 3), HR (Figure 4), or LA (Figure 5).

## DISCUSSION

NTproBNP responses to 1RM and MRM have not been reported previously to the best of our knowledge, providing new insight and clinical assessment regarding cardiac stress concerns with strength tests. NTproBNP responses in the present study did not differ between treatment groups (1RM vs MRM) or conditions (overweight vs obese). A significant positive correlation between NTproBNP 15-min post and power output was found, which supports an expected mild cardiac stress with both maximal strength assessments, yet there was no significant difference between rest, immediate post, and 15-min post NTproBNP. Efforts were made to control for psychological stress influences by conducting a familiarization session in the testing environment and comparing familiarization day HR, BP, and LA resting values to baseline data on assessment days; however, cortisol levels were not measured, and there may have been some psychological anxiety when the blood draw was taken. It is possible that immediate post and 15-min post NTproBNP responses may have been blunted under the influence of potentially increased cortisol. Amir and colleagues reported an inverse relationship between NTproBNP response and cortisol during high psychological stress in young college students, summarizing that mental stress entails an interface between the hypothalamic-pituitary-axis and peripheral natriuretic peptide system (20). Normal values for NTproBNP in males  $<45$  years of age are  $10\text{--}51 \text{ pg}\cdot\text{mL}^{-1}$  (24). According to New York Heart Association (NYHA) functional classification, in people  $<50$  years old, heart failure is unlikely with NTproBNP values  $<300 \text{ pg}\cdot\text{mL}^{-1}$  and heart failure is very

**TABLE 1.** Subject characteristics between conditions (overweight vs obese) and treatments (one-repetition max [1RM] vs multiple-repetition max [MRM] strength assessment).

Variables	Overweight (n = 11)	Obese (n = 9)
Age (years)	22.5 ± 1.8	22.9 ± 2.8
Height (cm)	176.7 ± 4.8	177.2 ± 3.0
Weight (kg·m <sup>-2</sup> )	86.0 ± 5.9*	113.1 ± 14.9*
BMI (kg·m <sup>-2</sup> )	27.6 ± 1.4*	36.0 ± 4.0*
1RM (kg)	109.3 ± 28.2*†	122.7 ± 21.0*
MRM (kg)	73.4 ± 17.6*†	83.1 ± 11.6*
Power 1RM (Watts)	355.8 ± 84.6*†	474.8 ± 126.5*
Power MRM (Watts)	239 ± 56.0*†	320.0 ± 72.3*
Rest HR 1RM (bpm)	77 ± 7.59	81 ± 8.92
IP HR 1RM (bpm)	120 ± 22.28†	125 ± 13.22
15-min post HR 1RM (bpm)	79 ± 9.39†	86 ± 12.45
Rest HR MRM (bpm)	77 ± 7.60	81 ± 9.92
IP HR MRM (bpm)	150 ± 11.12†	144 ± 13.72
15-min post HR MRM (bpm)	85 ± 11.13†	86 ± 10.51
Rest SBP 1RM (mmHg)	119 ± 4.20*	128 ± 8.15*
IP SBP 1RM (mmHg)	137 ± 11.46*	150 ± 19.36*
15-min post SBP 1RM (mmHg)	120 ± 3.32*	130 ± 13.69*
Rest SBP MRM (mmHg)	119 ± 4.20*	128 ± 8.15*
IP SBP MRM (mmHg)	138 ± 10.99*	152 ± 19.44*
15-min post SBP MRM (mmHg)	121 ± 8.58*	131 ± 12.72*
Rest DBP 1RM (mmHg)	70 ± 7.95*	76 ± 8.59*
IP DBP 1RM (mmHg)	63 ± 10.25*	70 ± 9.49*
15-min post DBP 1RM (mmHg)	69 ± 8.55*	78 ± 8.12*
Rest DBP MRM (mmHg)	70 ± 7.95*	76 ± 8.59*
IP DBP MRM (mmHg)	61 ± 10.07*	70 ± 9.49*
15-min post DBP MRM (mmHg)	66 ± 9.47*	78 ± 8.12*
Rest LA 1RM (mmol·L <sup>-1</sup> )	1.44 ± 0.54	1.47 ± 0.40
IP LA 1RM (mmol·L <sup>-1</sup> )	2.63 ± 0.91†	3.48 ± 2.27
15-min post LA 1RM (mmol·L <sup>-1</sup> )	1.97 ± 0.69†	1.87 ± 0.38
Rest LA MRM (mmol·L <sup>-1</sup> )	1.44 ± 0.54	1.47 ± 0.40
IP LA MRM (mmol·L <sup>-1</sup> )	5.05 ± 1.53†	6.42 ± 2.39
15-min post LA MRM (mmol·L <sup>-1</sup> )	4.02 ± 0.91†	4.28 ± 2.12
Rest NTproBNP 1RM (pg·mL <sup>-1</sup> )	53.97 ± 16.70	45.33 ± 17.20
IP NTproBNP 1RM (pg·mL <sup>-1</sup> )	47.17 ± 8.22	53.00 ± 24.03
15-min post NTproBNP 1RM (pg·mL <sup>-1</sup> )	42.41 ± 7.51	48.79 ± 10.65
Rest NTproBNP MRM (pg·mL <sup>-1</sup> )	53.97 ± 16.70	45.33 ± 17.20
IP NTproBNP MRM (pg·mL <sup>-1</sup> )	48.41 ± 20.47	43.96 ± 8.01
15-min post NTproBNP MRM (pg·mL <sup>-1</sup> )	50.74 ± 17.34	42.21 ± 11.22

Mean ± Standard Deviation; \* =  $p < 0.05$ , significant difference between conditions (overweight vs. obese); † =  $p < 0.05$ , significant difference between treatments (1RM vs MRM); IP = immediate post; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; LA = blood lactate; NTproBNP = N-Terminal Pro-Brain Natriuretic Peptide.

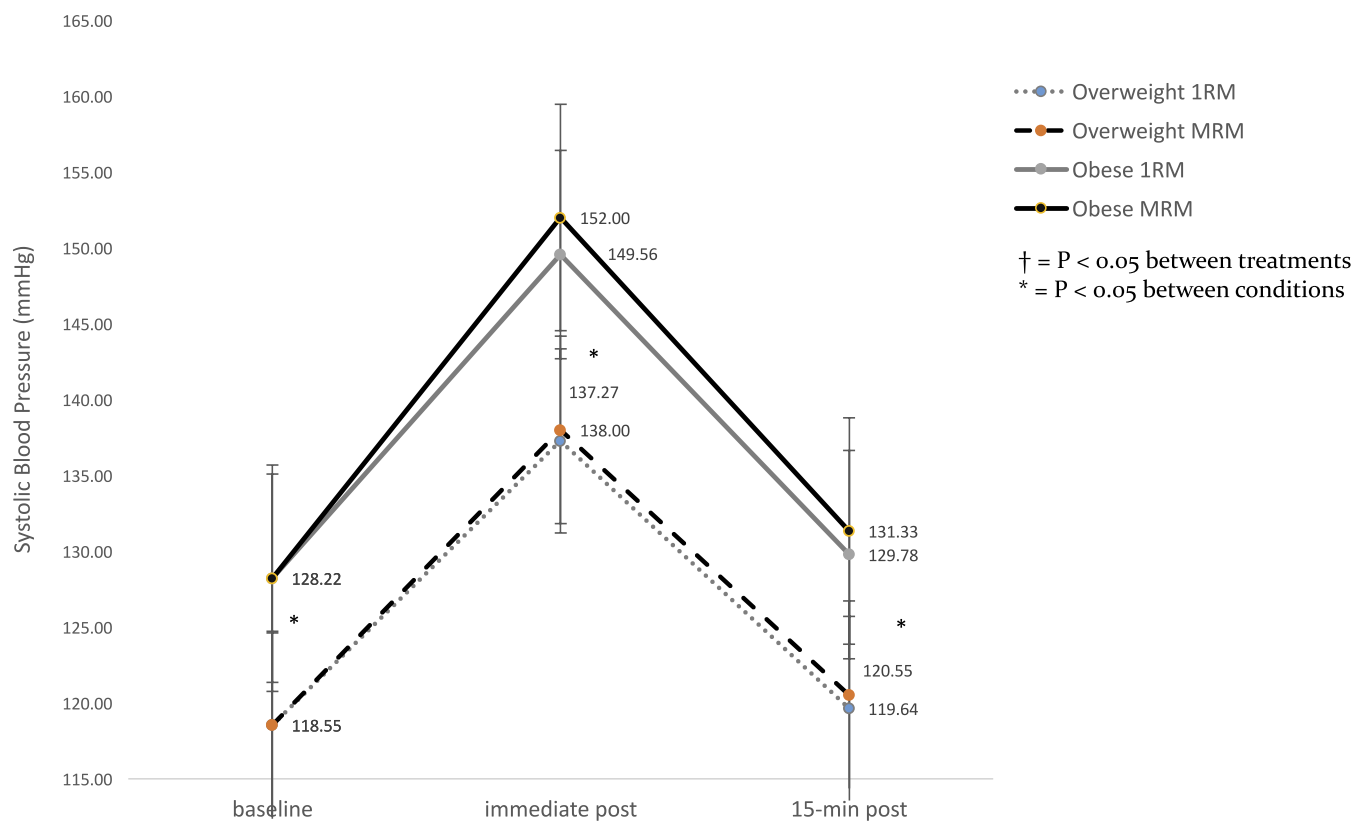


FIGURE 1. Systolic blood pressure responses to 1RM and MRM in obese vs overweight males.

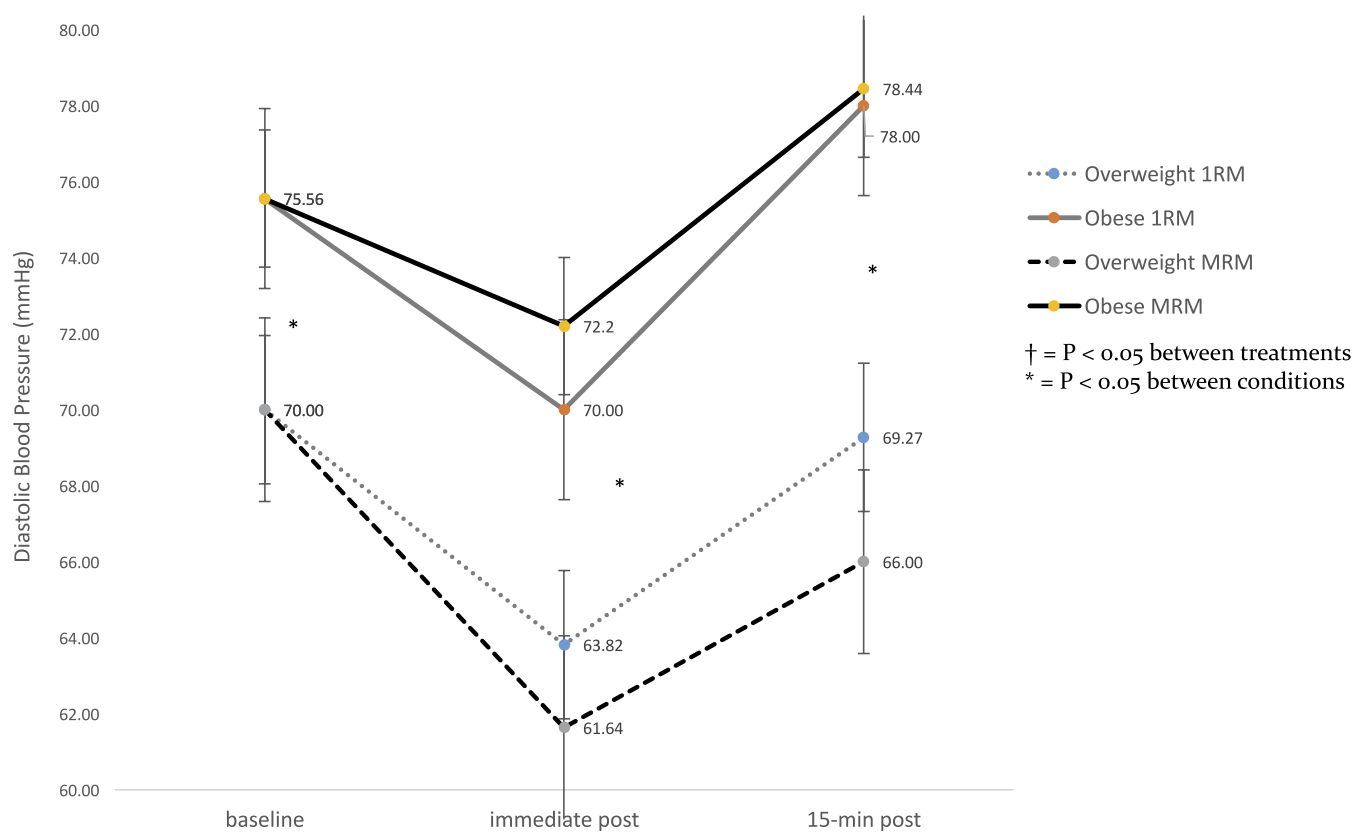


FIGURE 2. Diastolic blood pressure responses to 1RM and MRM in obese vs overweight males.

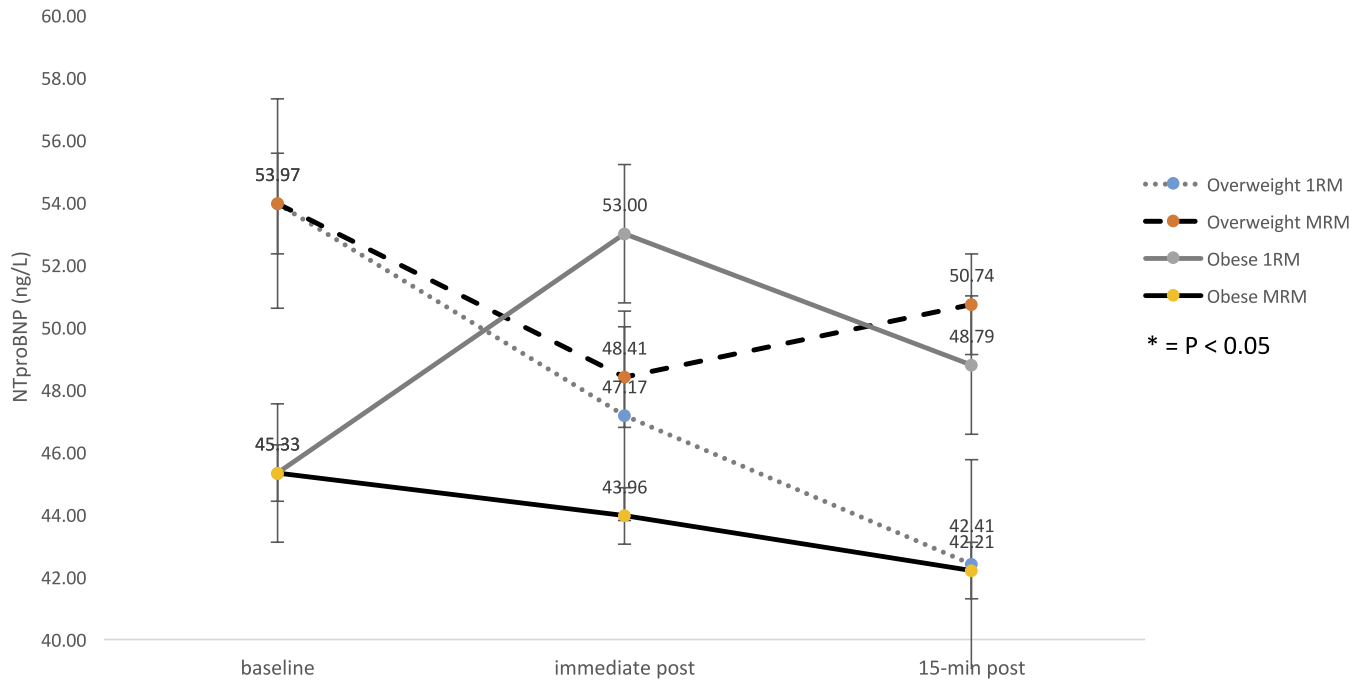


FIGURE 3. N-Terminal pro-brain natriuretic peptide responses to 1RM and MRM in obese vs overweight males.

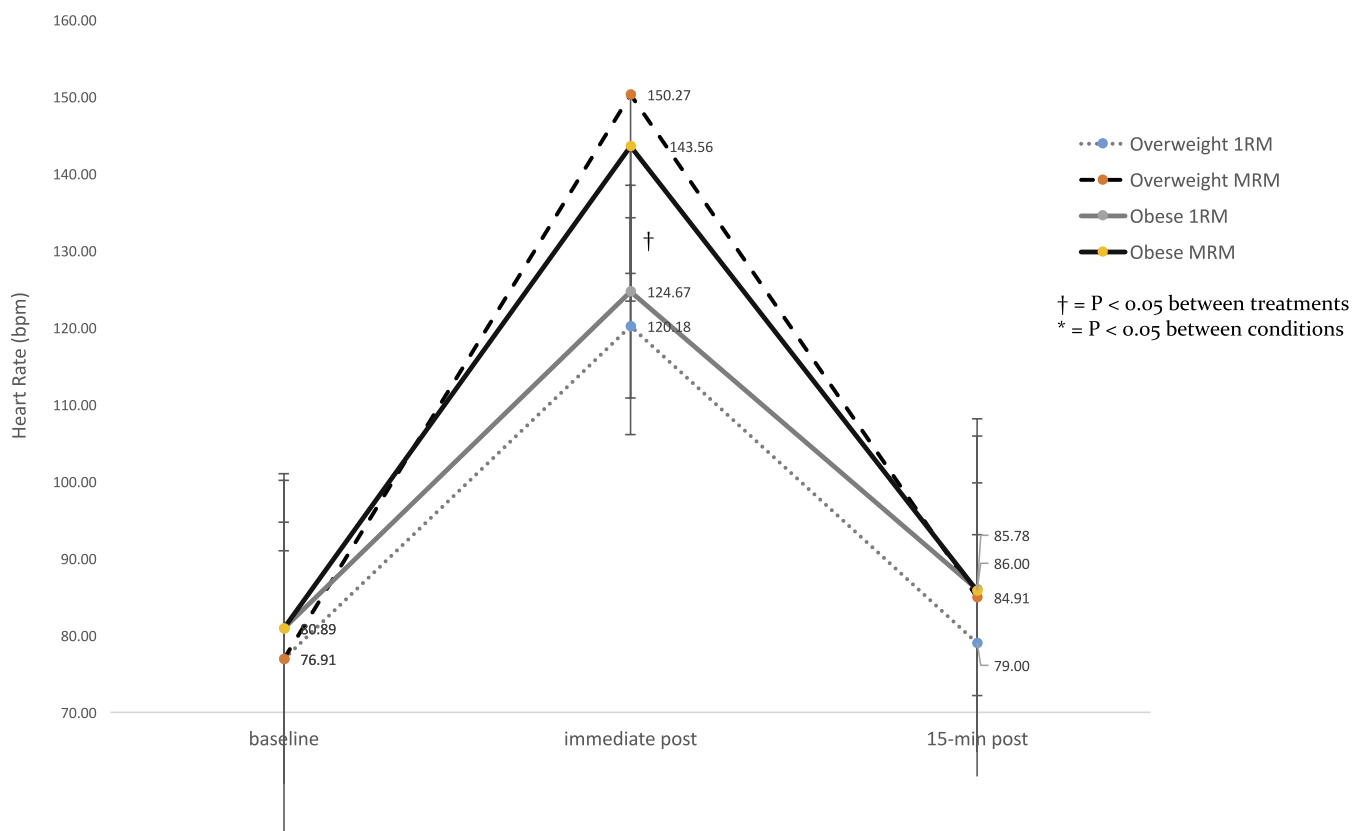


FIGURE 4. Heart rate responses to 1RM and MRM in obese vs overweight males.

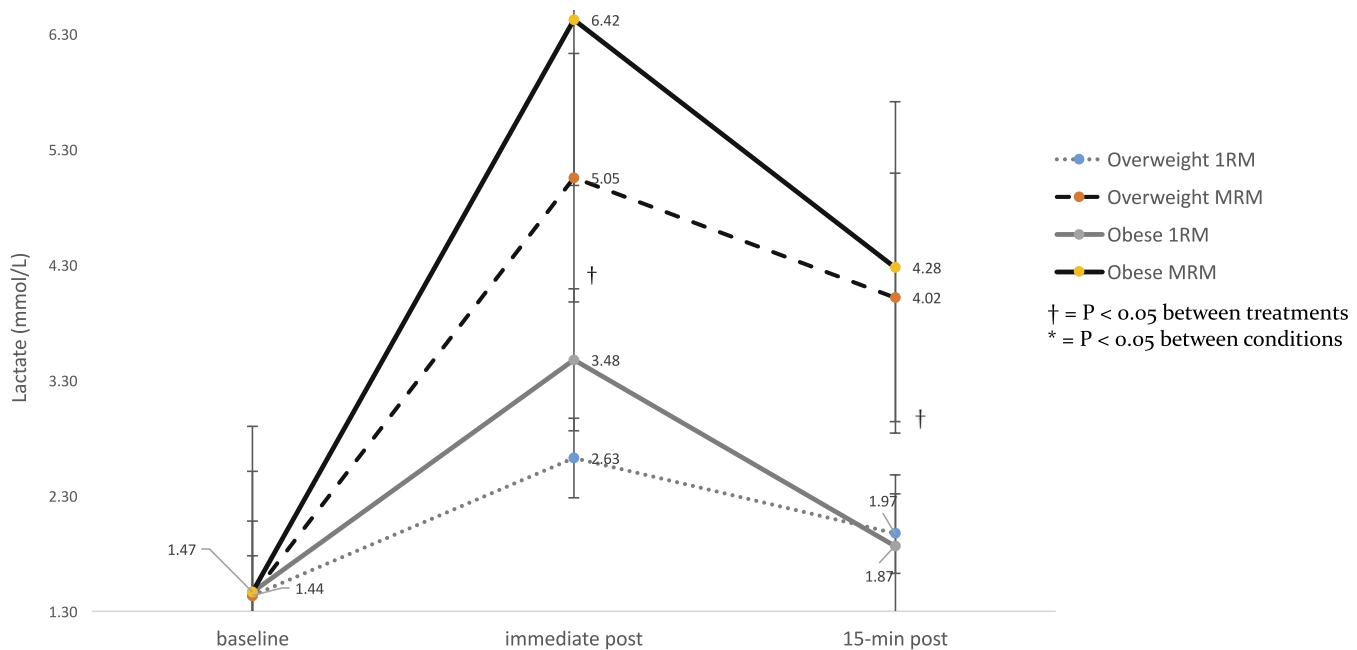


FIGURE 5. Blood lactate responses to 1RM and MRM in obese vs overweight males.

likely at values  $>450$   $\text{pg} \cdot \text{mL}^{-1}$  (24). The present study involved untrained overweight and obese males who were young and without symptoms or known disease. Baseline NTproBNP values were on the high end of normal reference values in this population— $53.97 \pm 16.70$  and  $45.33 \pm 17.20$   $\text{pg} \cdot \text{mL}^{-1}$ —consistent with evidence showing untrained controls have higher NTproBNP values compared to aerobic and resistance trained individuals (15). NTproBNP responses to stress experienced from 1RM vs MRM may likely be different in clinical and female populations, as baseline NTproBNP values have been shown to be higher in those with cardiac disease, renal disease, elderly, and females (23,25). NTproBNP changes induced by 1RM and MRM strength tests remain to be seen in cardiac patients, comorbid conditions, older overweight and obese individuals, and females. However, hemodynamic and metabolic responses to strength assessments have been reported previously.

Heart rate and blood lactate responses in the present study suggest MRM creates greater cardiovascular and metabolic stress compared to 1RM, which contradicts standard recommendations that purport MRM as a recommended strength assessment for clinical populations compared to 1RM (1–3,26). However, our findings are consistent with a growing body of literature that supports making changes to these recommendations, where the evidence points to duration of resistance exercise resulting in greater cardiovascular stress compared to load (11,27,28). Metabolic demands are influenced by efforts to maintain contraction forces. In the present study, contraction forces in the 1RM were about 2 sec compared to 18 to 20 sec in the MRM. Subjects completed more sets to achieve 1RM vs MRM, but the time under tension was considerably greater for MRM—true for MRM assessments in general, supporting the idea that duration under stress may be of greater concern for clinical

populations. Gjøvaag et al. compared a 4RM to that of 15RM leg extension exercise in patients with coronary artery disease (men and women, mean age  $64.2 \pm 7.3$  years), finding significantly ( $p < 0.001$ ) higher heart rate, blood lactate, systolic and diastolic blood pressure responses to the 15RM (11). Ratings of perceived exertion were also significantly higher for 15RM (11). Subjects in the present study reported anecdotally that the MRM was more taxing, but performed to a 9 or 10 RPE for both MRM and 1RM. Lovell and colleagues studied hemodynamic responses to strength testing in elderly men ( $73.9 \pm 2.9$  years) without known disease, reporting significantly ( $p < 0.05$ ) higher heart rate, systolic and diastolic blood pressures during 15 repetitions at 50% of 1RM compared to 1RM of inclined squat exercise (28). Likewise, Lomotte et al. found heart rate and systolic blood pressure to be higher ( $p < 0.001$ ) in lower intensity/higher repetitions compared to higher intensity/low repetitions of leg extension exercise in cardiac patients (27). Comparatively, our study used upper body exercise (chest press), with similar results in heart rate responses, but not blood pressure.

Blood pressure results in this study suggest greater concern for potential abnormal responses in people who are obese compared to overweight, for both 1RM and MRM strength assessments. However, the current study did not find a significant difference between 1RM and MRM systolic or diastolic blood pressure responses. This was an unexpected result, considering HR responses were considerably different and when comparing to other researchers' findings, where systolic and diastolic blood pressures were higher in lower intensity/higher repetitions compared to higher intensity/low repetitions (11,27,28). However, it has been shown that the magnitude of the hemodynamic response is positively related to the size of muscle mass involved during strength testing, where larger muscle groups impose



greater intramuscular mechanical compression (29). The present study looked at responses to chest press exercise, where other studies used lower body (leg extension and squat), larger muscle group exercises. Use of manual auscultation to determine blood pressure has been shown to result in lower blood pressure measures compared to techniques such as Finapres (28) and more invasive catheterization of the brachial artery (13). Other researchers have reasoned that MRM creates a greater cardiac stress via mechanisms involving central drive to the muscle as well as probable recruitment of additional musculature to maintain contraction force (12). The recruitment of smaller muscle groups required to complete a chest press—in comparison to larger, lower body muscle groups—would reasonably induce lower blood pressure responses. Lovell et al. found a greater post-exercise hypotensive response in elderly men following lower load/higher repetition resistance exercise as well, implying a greater cardiovascular stress from lower load/higher repetitions (28). We did not observe a post-exercise hypotensive response for either treatment or condition in the present study, which might be an indication of age differences in the recovery process imposed via arterial compliance properties (30). Additionally, use of BMI as criteria for overweight and obese in the present study theoretically could have misclassified individuals with larger than usual skeletal muscle mass and mass to fat ratio, thus overlooking a potential positive influence of higher muscle mass on lactate and blood pressure and warranting body composition assessment in future studies. Past participation in athletics and weight lifting was not controlled for, thus potential longer-term effects of past extensive training were not considered.

Shaw and colleagues addressed the issue of musculoskeletal injury risk due to 1RM in elderly men and women with different levels of weight-lifting experience, finding 1RM to be relatively safe with 2/83 subjects incurring injury (97.6% were injury free; 31). The two who did experience injury had no previous weight-lifting experience; one sustained a back injury and the other fractured a rib (31). The present study notes that mechanical load, force production, and power output were greater for 1RM compared to MRM (Table 1). However, no musculoskeletal injuries occurred for either treatment (1RM vs MRM) or condition (overweight vs obese). Subjects in the present study were young and the

majority had some weight-lifting experience but were not currently training. The present study also used a Life Fitness Chest Press machine and researchers monitored form and five points of contact closely.

Limitations of the present study included subjects' self-report on adherence to pre-participation guidelines and use of manual auscultation rather than more invasive, direct, or continuous technology. Saghi and colleagues (32) found that manual auscultation (indirect) and intra-arterial catheter (direct) methods of obtaining blood pressure at peak exercise was weakly to moderately correlated ( $r = 0.40$  and  $r = 0.58$ , respectively). Blood samples were not corrected for plasma volume changes. Obesity and overweight categories for future studies might be better identified by body composition rather than BMI. Additionally, the familiarization session did not include a blood draw and thus did not control for psychological nervousness/anxiety associated with a blood draw or fear of needles.

In conclusion, NTproBNP responses for overweight and obese groups were similar, at the upper range of normal reference levels for young males and considered safe in terms of cardiac stress. Heart rate and blood lactate responses were significantly higher in the MRM, suggesting MRM may provoke higher cardiovascular and metabolic demand compared to 1RM. Obesity may induce higher blood pressure responses in both 1RM and MRM, although initial blood pressure levels and size of the muscle mass involved in strength testing should be considered in regard to pressor responses and the role of mechanical compression. Further research is needed to assess NTproBNP responses to 1RM and MRM using larger muscle groups, and for elderly, female, and clinical populations, as strength testing may produce greater cardiovascular risk under these conditions. Recommendations for using MRM as a preferred strength assessment for clinical populations should be reconsidered, with a preference for using 1RM strength assessment or high load/low repetition assessments rather than MRM low load/higher repetitions. Susceptibility for joint injury should be evaluated and considered in the decision to conduct strength tests.

**Acknowledgments:** The authors would like to thank Dr. Christine Fleischer for allowing us to use the University of Mary Biology Laboratory and for providing assistance and expertise in use of the ELISA technology.

## REFERENCES

1. American College of Sports Medicine. Position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *MSSE*. 2011;1334–59.
2. American College of Sports Medicine. *ACSM Guidelines for Exercise Testing and Prescription*, 9th ed. Baltimore, MD: Wolters Kluwer/Lippincott Williams & Wilkins; 2014: 27, 60.
3. American Association of Cardiovascular and Pulmonary Rehabilitation. *Guidelines for Cardiac Rehabilitation and Secondary Prevention Programs*. 5th ed. Champaign, IL: Human Kinetics; 2013.
4. Goisser S, Kemmler W, Porzel S, Volkert D, Sieber CC, Bollheimer LC, Freiburger E. Sarcopenic obesity and complex interventions with nutrition and exercise in community-dwelling older persons – a narrative review. *Clin Interv Aging*. 2015;10:1267–82.
5. Nikseresht M, Azarbayjani MA, Ebrahim K. Effects of nonlinear resistance and aerobic interval training on cytokines and insulin resistance in sedentary men who are obese. *J Strength Cond Res*. 2014;28(9):2560–8.

6. Markofski MM, Flynn MG, Carrillo AE, Armstrong CLH, Campbell WW, Sedlock DA. Peptide in the general community; determinants and detection of left ventricular dysfunction. *JACC*. 2006;47(2):345–53.
7. Germain CM, Batsis JA, Vasquez E, McQuoid DR. Muscle strength, physical activity, and functional limitations in older adults with central obesity. *J Aging Res*. 2016;2016:8387324.
8. Rantanen T. Muscle strength, disability and mortality. *Scand J Med Science Sports*. 2003;13(1):3–8.
9. Stenholm S, Mehta NK, Elo IT, Heliovaara, Koskinen S, Aromaa A. Obesity and muscle strength as long-term determinants of all-cause mortality – a 33-year follow-up of the Mini-Finland Health Examination Survey. *Int J Obes*. 2014;38:1126–32.
10. Volakliska KA, Halle M, Meisinger C. Muscular strength as a strong predictor of mortality: a narrative review. *Eur J Intern Med*. 2015;26(5):303–10.
11. Gjøvaag TF, Mirtaheri P, Simon K, Berdal G, Tüchel I, Westlie T, Bruusgaard KA, Nilsson BB, Hisdal J. Hemodynamic responses to resistance exercise in patients with coronary artery disease. *MSSE*. 2015;48(4):581–8.
12. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol*. 1985;58(3):785–90.
13. MacDougall JD, McKelvie S, Moroz DE, Sale DG, McCartney N, Buick F. Factors affecting blood pressure during heavy weight lifting and static contractions. *J Appl Physiol*. 1992;73(4):1590–7.
14. Weber M, Hamm C. Role of B-Type Natriuretic Peptide (BNP) and NT-ProBNP in Clinical Routine. *Heart*. 2006;92:843–9.
15. Smart NA, Steele M. Systematic review of the effect of aerobic and resistance exercise training on systematic brain natriuretic peptide (BNP) and N-terminal BNP expression in heart failure patients. *Int J Cardiol*. 2010;140(3):260–5.
16. Januzzi JL, van Kimmenade R, Lainchbury J. NT-proBNP testing for diagnosis and short-term prognosis in acute destabilized heart failure: an international pooled analysis of 1,256 patients. The International Collaborative of NT-proBNP Study. *Eur Heart J*. 2006;27:330–7.
17. Ohba H, Takada H, Musha H, Nagashima J, Mori N, Awaya T, Omiya K, Murayama M. Effects of prolonged strenuous exercise on plasma levels of atrial natriuretic peptide and brain natriuretic peptide in healthy men. *Am Heart J*. 2001;141(5):751–8.
18. Banfi G, D'Eril GM, Lippi G. N-terminal proB-type natriuretic peptide (NT-proBNP) concentrations in elite rugby players at rest and after active and passive recovery following strenuous training sessions. *Clin Chem Lab Med*. 2008;46(2):247–9.
19. Bentzen H, Pedersen RS, Nyvad O, Pedersen EB. Influence of training habits on exercise-induced changes in plasma atrial and brain natriuretic peptide and urinary excretion of aquaporin-2 in healthy man. *Scand J Clin Lab Invest*. 2002;62:541–52.
20. Amir O, Sagiv M, Eynon N, Yamin C, Rogowski O, Gerzy Y, Amir RE. The response of circulating brain natriuretic peptide to academic stress in college students. *Stress*. 2010;13(1):83–90.
21. McGrath MF, de Bold AJ. Determinants of natriuretic peptide gene expression. *Peptides*. 2005;26(6):933–43.
22. Reynolds JM, Gordon TJ, Robergs RA. Prediction of one repetition maximum strength from multiple repetition maximum testing and anthropometry. *J Strength Cond Res*. 2006;20(3):584–92.
23. Hiscock DJ, Dawson B, Peeling P. Perceived exertion responses to changing resistance training programming variables. *J Strength Cond Res*. 2015;29(6):1564–9.
24. Mayo Clinic. Mayo Medical Laboratories. Interpretive handbook. Test 84291: NT-pro B-type natriuretic peptide (BNP), serum clinical information. Available from: [http://www.mayomedicallaboratories.com/interpretive-guide/?alpha=N&unit\\_code=84291](http://www.mayomedicallaboratories.com/interpretive-guide/?alpha=N&unit_code=84291) Accessed 09/17/2016.
25. Weber M, Hamm C. Role of B-type natriuretic peptide (BNP) and NT-ProBNP in clinical routine. *Heart*. 2006;92:843–9.
26. Pollock ML, Franklin BA, Balady GJ, Chaitman BL, Fleg JL, Fletcher B, Limacher M, Piña IL, Stein RA, Williams M, Bazzarre T. Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescription. An advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association; Position paper endorsed by the American College of Sports Medicine. *Circulation*. 2000;101:828–33.
27. Lamotte M, Neset G, van de Borne P. The effect of different intensity modalities of resistance training on beat-to-beat blood pressure in cardiac patients. *Eur J Cardiovasc Prev Rehabil*. 2005;12(1):12–7.
28. Lovell DI, Cuneo R, Gass GC. The blood pressure response of older men to maximum and sub-maximum strength testing. *J Sci Med Sport*. 2011;14:254–8.
29. Markofski MM, Flynn MG, Carrillo AE, Armstrong CLH, Campbell WW, Sedlock DA. Peptide in the general community; determinants and detection of left ventricular dysfunction. *JACC*. 2006;47(2):345–53.
30. Brito LC, Queiroz AC, Forjaz CL. Influence of population and exercise protocol characteristics on hemodynamic determinants of post aerobic exercise hypotension. *Braz J Med Biol Res*. 2014;47(8):626–36.
31. Shaw CE, McCully KK, Posner JD. Injuries during the one repetition maximum assessment in the elderly. *J Cardiopulm Rehabil*. 1995;15:283–7.
32. Sagiv M, Goldhammer E, Sagiv M, Ben Sira D, Hanson P. Comparison of direct vs. indirect blood pressure measurements on treadmill and bicycle in hypertensive responders. *Clin Exp Pharmacol*. 2016;6:215.