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Acute Effects of Exercise Mode on Arterial Stiffness and Cardiac Autonomic Function in Healthy Young Adults

Dissertação elaborada com vista à obtenção do Grau de Mestre em
Exercício e Saúde

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Abbreviations

ACSM – American College of Sports Medicine

AD – Arterial distensibility

AEE – Activity energy expenditure

AIx – Augmentation index

AIx75 – Heart rate corrected augmentation index

AS – Arterial stiffness

bBP – Brachial blood pressure

bDBP – Brachial diastolic blood pressure

BP – Blood pressure

bPP – Brachial pulse pressure

BRS – Baroreflex sensitivity

bSBP – Brachial systolic blood pressure

CAD – Coronary artery disease

cBP – Central blood pressure

cDBP – Central diastolic blood pressure

cd-PWV – Carotid-to-distal pulse wave velocity or lower limb pulse wave velocity

cf-PWV – Carotid-to-femoral pulse wave velocity or aortic pulse wave velocity

CON – Control, or no exercise session

CRF – Cardiorespiratory fitness

cr-PWV – Carotid-to-radial pulse wave velocity or upper limb pulse wave velocity

cSBP – Central systolic blood pressure

CV – Cardiovascular

dP/dt-max – Maximal rate of rise of left ventricular pressure

DPTI – Diastolic pressure–time index

DPTI/SPTI – Ratio between the diastolic and systolic pressure–time indexes

ECG – Electrocardiogram

EE – Energy expenditure

ε – Peterson's pressure-strain elastic modulus

GT – Global training

HF – High-frequency power band

HR – Heart rate

HR Max−HR Min – The average difference between the highest and lowest heart rates during each respiratory cycle

HRR – Heart rate recovery

HRV – Heart rate variability

IBI – Interbeat interval

LF – Low-frequency power band

LF/HF – Ratio between low- and high- frequency power bands

Ln – Natural logarithm

LVMDP – Left ventricular mean diastolic blood pressure

MAP – Mean arterial pressure

Peak VO_2 – Peak oxygen uptake

pNN50 – Percentage of adjacent NN intervals that differ from each other by more than 50ms

PNS – Parasympathetic nervous system

PUMP – Pump power

PP – Pulse pressure

PWA – Pulse wave analysis

PWV – Pulse wave velocity

PWV-β – Pulse wave velocity beta

REE – Resting energy expenditure

RMSSD – Root mean square of successive differences

RR intervals – R-wave to R-wave intervals

SDNN – Standard deviation of NN intervals

SEVR – Subendocardial viability ratio

SNS – Sympathetic nervous system

SPTI – Systolic pressure–time index,

TEE – Total energy expenditure

VCO_2 – Carbon dioxide production

VO_2 – Oxygen consumption

β – Beta stiffness

Abstract

Purpose: To analyse the time-course of post-exercise cardiovagal modulation and local and regional changes on indices of arterial stiffness (AS) to three group fitness classes (GFC), in healthy young adults.

Methods: 12 participants attended separated sessions of either BIKE, Pump Power (PUMP), Global Training (GT), or no exercise. In each session, participants initially undertook a supine rest followed by one of the sessions in a randomized order. Testing after each session took place at 10-, 20-, and 30-min, including assessments of regional and local indices of AS, cardiovagal modulation and baroreflex sensitivity (BRS).

Results: No post-exercise changes were found in aortic and upper-limb pulse wave velocity (PWV). Local carotid indices remained unchanged following GT but increased immediately following (10-min) BIKE and PUMP. Lower-limb PWV, Ln-RMSSD, Ln-HF, BRS and BEI were immediately decreased following the three group fitness classes.

Conclusions: These findings suggest that 1) exercise modality influences indices of local AS while having no effect on the remaining assessments, and 2) evidence derived from laboratorial experiments on the acute effects of exercise on indices of arterial stiffness seem to have ecological validity, but this may not be the case for cardiovagal modulation and BRS responses.

Keywords: Acute exercise · Fitness classes · Arterial Stiffness · Cardiovagal Modulation · Pulse Wave Velocity · Heart Rate Variability · Baroreflex Sensitivity · Cardiovascular Health · Cardiorespiratory Fitness · Ecological Validity

Resumo

Objetivo: Analisar a evolução temporal da modulação cardiovagal pós-exercício e as mudanças locais e regionais nos índices de rigidez arterial (RA), após 3 aulas de grupo, em jovens adultos saudáveis.

Métodos: 12 participantes realizaram sessões de BIKE, Pump Power (PUMP), Global Training (GT) ou nenhum exercício. Em cada sessão, os participantes permaneceram em repouso seguido por uma das sessões. As avaliações após cada sessão ocorreram aos 10, 20 e 30 minutos, incluindo avaliações regionais e locais de RA, modulação cardiovagal e sensibilidade barorreflexa (SBR).

Resultados: Não foram encontradas quaisquer alterações pós-exercicio na velocidade de onda de pulso (VOP) da aorta e dos membros superiores. Índices de RA local permaneceram inalterados após GT, porém aumentaram imediatamente após (10-min) BIKE e PUMP. A VOP dos membros-inferiores, Ln-RMSSD, Ln-HF, SBR e BEI diminuíram imediatamente após as três aulas de grupo.

Conclusões: Estes resultados sugerem que 1) a modalidade de exercício influencia os índices de RA local, não tendo qualquer efeito nas restantes avaliações, e 2) estudos realizados em contexto laboratorial sobre efeitos agudos do exercício nos índices de rigidez arterial parecem ter validade ecológica, mas este não parece ser o caso para a modulação cardiovagal e SBR.

Palavras-chave: Exercício Agudo · Aulas De Grupo · Rigidez Arterial · Modulação Cardiovagal · Velocidade Da Onda De Pulso · Variabilidade Da Frequência Cardíaca · Sensibilidade Barorreflexa · Saúde Cardiovascular · Aptidão Cardiorrespiratória · Validade Ecológica

Introduction

Large artery distensibility is physiologically important for cardiovascular (CV) efficiency. Distensible large arteries reduce impedance to systolic ejection and cardiac work, slows pulse wave velocity (PWV) so that the return of reflected pressure waves is delayed until after aortic valve closure, and favours coronary perfusion during diastole (Naka, 2003). Conversely, stiffening of the central arteries results in an elevation in systolic blood pressure (SBP) and a lowering of diastolic blood pressure (DBP) which, in turn, increases left ventricular afterload and alters coronary artery perfusion (Hamilton, Lockhart, Quinn, & McVeigh, 2007). These changes may result in left ventricular hypertrophy (Girerd, 1991) and increased fatigue of arterial wall tissues (Blacher, 1999), all of which substantially increase the risk of CV events and all-cause mortality (McEniery, 2006; Vlachopoulos, Aznaouridis, & Stefanadis, 2010).

Identified as a risk factor for future morbidity and fatal CV events, 'Arterial Stiffness' (AS) has been studied and accessed using many methods, such as, localized assessment of blood vessel mechanics, pulse pressure (PP), pulse waveform analysis and/or PWV (London, 2002). The recent expert consensus document on AS describes carotid-femoral pulse wave velocity (cf-PWV) as the 'gold standard' measurement of AS (Laurent et al., 2006). The measurement of arterial stiffness via carotid-femoral PWV has been reported as highly predictive of the future risk of CV events (Mattace-Raso et al., 2006) with an 1 m/s increase in cf-PWV associated with a 10% increase in CV event risk (Vlachopoulos et al., 2010). In fact, increased AS denotes true damage of arterial wall integrity and alteration of its function, whereas other risk factors such as blood pressure (BP), glycaemia and/or lipid levels vary and may not be representative enough of the cumulative effects of CV risk factors on the arterial system (Arnett, 1994; London, 2002; Obeid, Ouedraogo, & Hallab, 2017).

The sympathetic nervous system (SNS) is a key regulator of BP, and abnormal activity in this system is related to CV risk (Charkoudian, 2009). Specifically, indices of cardiac autonomic function such as heart rate recovery (HRR) and heart rate variability (HRV) are associated with brachial blood pressure (bBP) and aortic blood pressure (D. A. Fei, R.; Arrowood, J. A.; Kraft, K. A., 2005; van Ittersum et al., 2004). When measured after a maximal exercise effort, HRR and HRV are considered powerful independent predictors of mortality in healthy subjects and in various clinical populations (M. Buchheit & Gindre, 2006; Cole, 1999; Prince J. Kannankeril, 2004).

In addition to their ability to predict CV event risk, AS and indices of cardiac autonomic function are predictive of the success of different therapies (D. B. Aronson, A. J. , 2001; Mahmud & Feely, 2008), such as, CV medications including beta-blockers that have been reported to reduce aortic pulse wave velocity (cf-PWV) and wave reflection

(Mahmud & Feely, 2008), most likely due to reductions in SNS activity on the heart (Harvey et al., 2017; van Ittersum et al., 2004). Moreover, exercise training is associated with reductions in sympathetic activity (Cornelissen & Fagard, 2005; Roveda et al., 2003) and this effect may represent an important mechanism by which exercise contributes to long term CV health (J. R. Carter & Ray, 2015). In this way, a single bout of exercise has been shown to reduce both AS (i.e., cf-PWV) (Heffernan, Collier, Kelly, Jae, & Fernhall, 2007; B. A. B. Kingwell, K. L.; Cameron, J. D.; Jennings, G. L.; Dart, A. M., 1997)) and wave reflection (i.e., AIx and reflection magnitude) (Millen, Woodiwiss, & Norton, 2016a; Munir et al., 2008)), and significantly impact on indices of cardiac autonomic function (de Paula et al., 2019; Heffernan, Collier, et al., 2007; Kliszczewicz, 2016; Teixeira, Ritti-Dias, Tinucci, Mion Junior, & Forjaz, 2011).

The emerging area of “recovery physiology” allows the expansion of the use of AS beyond its predictive capacity for risk of CV events (Blacher, 1999; Charkoudian, 2009; Girerd, 1991; Hamilton et al., 2007; McEniery, 2006; Vlachopoulos et al., 2010), bringing it closer to predicting the success of different therapies (D. A. Fei, R.; Arrowood, J. A.; Kraft, K. A., 2005; van Ittersum et al., 2004), and in the optimization of the exercise prescription for health and physical performance (M. G. Buchheit, C., 2006). For example, not only some adaptations to training seem to result from the sum of the acute effects of the exercise sessions, as in the case of BP reduction (Thompson, 2001), but the induced changes by exercise can be influenced by the exercise mode, with several studies reporting disparate results (Saz-Lara et al., 2021). HRR and HRV vary depending on whether exercise is practiced on a treadmill or on a cycle ergometer, since the physiological stress appears to be higher on the treadmill (Abrantes, Sampaio, Reis, Sousa, & Duarte, 2012; Esco, Flatt, & Williford, 2017). Regarding AS and HRV, only a limited number of studies directly compared the acute changes induced by different modes of dynamic exercise (Leicht, Sinclair, & Spinks, 2008; Pierce, Doma, Raiff, Golledge, & Leicht, 2018). However, this evidence comes from studies that used exercises (aerobic and resistance) rather than activities, to simulate typical sessions according to current guidelines for improving and maintaining CV health (D. Riebe, et al., 2018). An obvious disadvantage of this option is its reduced ecological validity.

The participation in group fitness classes is an increasingly common method by which subjects may fulfil the criteria set down in the American College of Sports Medicine (ACSM) recommendations for enhancing and/or maintaining CV fitness (D. Riebe, et al., 2018), and to induce favourable changes in the CV risk profile. These can appropriately function to enhance measures of cardiorespiratory fitness (CRF) when performed for at least 3 days/week for 30 minutes and induce favourable changes in CV risk profile (Blacher, 1999; Girerd, 1991; Hamilton et al., 2007; McEniery, 2006; Naka, 2003;

Vlachopoulos et al., 2010). The popularity of group fitness classes with the general public has been demonstrated for more than two decades and is most likely due to the social and non-competitive class environment (Eys, Bruner, & Martin, 2019; Graupensperger et al., 2019). A wide range of group fitness classes are now available in gyms and health clubs, incorporating a variety of exercise modalities (e.g.: stationary cycling, step classes, group resistance exercise, pilates and aerobic dance, etc.), using a combination of music and instructor choreographed routines, designed to accommodate different levels of physical fitness. However, despite the high rates of participation in these classes, no available evidence exists to describe and/or compare the alterations of arteries' mechanical properties and the metabolic costs of different group classes. Additionally, the extent to which participation in such classes may help identify periods of significant concern eventually caused by transient changes in AS immediately following exercise, benefiting or not arterial function.

The present master thesis includes: 1) an overview on the essentials of the CV system, targeting the Heart and the Vascular systems; 2) a review on the AS and cardiovagal modulation responsivity to acute aerobic and resistance exercise; 3) a methodology section where the measurement of the two main outcomes, namely AS, and HRV, are explained; 4) two interventional studies that are delineated along with their methodology section, where the recruitment process, study design, intervention conditions, assessment instruments and protocols are introduced, and their results, along with, its integrative discussions, limitations, as well as conclusions; and 5) the final conclusions and practical applications elucidated for disclosure purposes as well as the future research directions.

Essentials of the CV System

The CV system is a complex organ system that functions with multiple other physiologic systems in an integrated way coupling every physiological system (Levick, 2013). The heart, the vasculature and the blood are the 3 interconnected components that together provide a basic function of the CV system, which in a closed-circuit system, its primary duty is to supply body cells with nutrients and eliminate the waste products.

Concerning the heart, the myocardium provides the necessary contractile force to distribute blood to the various organs. The measure of the heart's ability to eject blood to support the needs of the body on a per minute basis is the cardiac output (Q) (Vincent, 2008). It can be determined by the product of heart rate (HR) (number of beats per minute) and stroke volume (amount ejected per beat) and under normal resting conditions is approximately 5 L/min (Arena, Myers, & Guazzi, 2008; Rivera-Brown & Frontera, 2012). However, HR and Q must vary in response to the needs and body requirements under varying conditions. The HR is primarily affected by the autonomic nervous system stimulation, comprising two primary branches, the sympathetic and parasympathetic which increase (positive chronotropic effect) and decrease (negative chronotropic effect) HR, respectively (R. Gordan, J. K. Gwathmey, & L.-H. Xie, 2015).

The blood vessels are not just passive "tubes" made merely to carry liquid around, like pipes within the walls of a home (Aitkin, 1838). They are actually active, dynamic organs, capable of contracting and expanding as they deliver oxygen and nutrients to cells throughout the body and carry away waste products, doing their part in maintaining that all important BP. The vessels constantly alter their diameter to change blood flow to meet its requirements (Tennant, 1990). Also, vessel's wall participates in the inflammatory response and in blood clotting by releasing chemical mediators (Pober & Sessa, 2014).

The cardiac autonomic activity and the vascular function are two interconnected CV concerns that are associated with the risk of adverse cardiac events. The regulation of vascular wall contractility and tension is an essential role of the autonomic nervous system (Sheng & Zhu, 2018). Particularly, the SNS is crucial for the vascular function (Bruno et al., 2012). The sympathetic and parasympathetic nerves work together to balance the functions of autonomic effector organs and the neurotransmitters released from the varicosities in the autonomic system can regulate the vascular tone (degree of

constriction experienced by a blood vessel relative to its maximally dilated state) (Sheng & Zhu, 2018).

Electrical Activity of the Heart

Conduction System of the Heart

Unlike skeletal muscles, which must be stimulated by the nervous system, the heart generates its own electrical stimulation and to facilitate an organized conduction, it has a specialized conduction system. The autonomic nervous system can make the heartbeats go faster or slower but cannot generate them (Mohan, Boukens, & Christoffels, 2017; D. S. Park & Fishman, 2017). The impulses start from a small group of specialized cardiomyocytes called the pacemaker cells, part of the cardiac conduction system, that can contract and become specialized for initiating and conducting action potentials (R. H. Anderson, Yanni, Boyett, Chandler, & Dobrzynski, 2009; Kennedy et al., 2016). The Sinoatrial (SA) node is the primary pacemaker of the heart, located at the junction of the superior vena cava with the right atrium. It initiates all heartbeats and controls HR once its cells can depolarize by themselves (R. H. Anderson et al., 2009; Mohan et al., 2017). The pacemaker cells of the SA Node fire spontaneously generating action potentials that spread through the contractile cardiomyocytes of the atria (DiFrancesco, 1993). The myocytes are connected by gap junctions that form channels (Dhein, 1998) that allow ions to flow from one cell to another and enable electrical coupling of neighbouring cells. An action potential in one cell triggers another action potential in its neighbour and the signals propagate rapidly. Through the internodal tracks (R. H. Anderson, Ho, S. Y., 1998), the impulses reach the Atrioventricular (AV) node, located on the other side of the right atrium, near the AV valve (R. H. Anderson et al., 2009), slow down a little to allow the atria to contract, then follow the conduction pathway and spread through the ventricular myocytes. The AV node serves as the electrical gateway to the ventricles (Jansen, Quinn, & Rose, 2018). It delays the passage of electrical impulses to the ventricles (Boullin & Morgan, 2005) and this delay, of approximately 0.12 seconds, is to ensure that the atria have ejected all the blood into the ventricles before this contract (Kennedy et al., 2016). The AV node receives signals from the SA node and passes them onto the atrioventricular bundle, located between two ventricles – AV bundle or bundle of His – this bundle is then divided into right and left bundle branches which conduct the impulses toward the apex of the heart (R. H. Anderson et al., 2009). The signals are then passed onto Purkinje fibers, turning upward, and spreading throughout the ventricular myocardium. This transmission is very rapid (2-3 m/s) in order to conduct the cardiac potential throughout the ventricles and ensure that

the muscle contractions are in the correct order and blood is sufficiently ejected (Ideker, 2009).

Control of Heart Rate and Blood Pressure

The HR is controlled by autonomic nervous system innervation which comprises the parasympathetic and sympathetic systems richly innervating the heart (Kimura, Ieda, & Fukuda, 2012). The vagus nerve is the longest of the autonomic nervous system, providing parasympathetic innervation to the heart, lungs, and digestive tract. It controls involuntary body processes, such as heartbeat, breathing, swallowing, BP, taste, circulation, and digestion (Garamendi-Ruiz & Gomez-Esteban, 2019; H. Yuan & Silberstein, 2016). The sympathetic system, specifically its fibers richly innervate the ventricular muscle, beyond the distribution to other parts of the heart, including the SA and AV nodes (R. Gordan, J. K. Gwathmey, & L. H. Xie, 2015). In healthy hearts, tonic discharges by both parasympathetic and sympathetic systems influences the SA node (M. Kollai, Jokkel, G., Bonyhay, I., Tomcsanyi, J., Naszlady, A., 1994). The release of norepinephrine results from sympathetic stimulation which also increases the rate of conduction throughout the heart and the force of contraction (Lymperopoulos, Rengo, & Koch, 2013; D. Y. Zhang & Anderson, 2014), while parasympathetic stimulation causes the release of acetylcholine. The firing of the vagus nerve causes a release of acetylcholine which in turn causes a change in the membrane potential of autorhythmic cardiac cells (such as the SA node), resulting in a decreased HR (Brack, Winter, & Ng, 2013). These increases and decreases in HR can be caused by reciprocal changes in sympathetic and parasympathetic stimulation (M. Kollai, Koizumi, K., 1979). Notwithstanding, these autonomic divisions can act alone without a reciprocal action (McCorry, 2007). It also should be taking into consideration, as such an important aspect, the differential speed at which autonomic systems stimulation controls HR. As an example, during exercise onset, a rapid increase in HR is required and caused by vagal withdrawal (Falcone, Buzzi, Klersy, & Schwartz, 2005; Robinson, 1966). Moreover, the speed at which HR returns to baseline at the cessation of exercise, depends on the amount of vagal reactivation and residual sympathetic stimulation (Arai, 1989; Cunha, Midgley, Goncalves, Soares, & Farinatti, 2015; Gladwell, Sandercock, & Birch, 2010). Accordingly, the HRR usually takes longer than the initial increase in HR (Freeman, Dewey, Hadley, Myers, & Froelicher, 2006). Also, HR is influence by respiration. It decreases during expiration and increases during inspiration. Primarily, this influence occurs through the vagal activity which is probably caused by differential rates of removal of acetylcholine versus norepinephrine (R. Gordan et al., 2015). Therefore, the respiratory variations - respiratory sinus arrhythmia (RSA) - are primarily a function of

vagal activity, as well, evidenced in well-trained endurance athletes who have large respiratory variations in HR with enhanced vagal tone (J. B. Carter, Banister, E. W., Blaber, A. P., 2003; Sacknoff, 1994).

The autonomic control of BP occurs on a beat-to-beat basis and over seconds to minutes. Elevations or falls in BP cause a proportionally greater or lesser deformation of the arterial walls, which are encoded by baroceptors to a greater or lesser frequency of action potential firing. The neural regulation of the CV system, controlled by the arterial mechanoreceptors, operates as a reflex arc. The arterial baroreflex responds to short-term adjustments in stretch of mechanosensitive afferents, the baroreceptors, in key areas of the circulation (carotid sinus, aortic arch, pulmonary great vessels, and atria/ventricles) (Ferguson, Abboud, & Mark, 1985). These stretch-sensitive baroreceptors send afferent information to central nuclei involved in efferent control of sympathetic and parasympathetic (vagal) neural output. The potential changes in arterial pressure are minimized by transient changes in BP which, therefore, result in reciprocal changes in sympathetic and vagal activity (Hart & Charkoudian, 2014; Sanders, 1989). The acute baroreflex corrections in response to exercise is essential to the normal maintenance of arterial pressure (Kaufman, 2012). The renin-angiotensin system is best known by the long-term control of the BP and sodium homeostasis. These actions are coordinated through integrated actions in the kidney, CV system, and the central nervous system. This is partly achieved by changes in HR, such as, a decrease in HR induces an increase in BP and vice-versa (Crisafulli, Marongiu, & Ogoh, 2015).

Heart Rate Variability

The measurement of HR as a single number is an average of several beats. There is a natural variability in the spacing between the heart beats or interbeat interval (IBI), a phenomenon called HRV. It refers to the beat-to-beat variability of the R-wave to R-wave intervals (RR intervals) (Cygankiewicz & Zareba, 2013; Electrophysiology, 1996). Simply the HRV is a measurement of the balance between the parasympathetic and sympathetic nervous systems (Berntson, 1997). These two systems make up the autonomic nervous system comprising the parasympathetic and sympathetic nerves (R. Gordan et al., 2015). The vagus nerve, a major component of the parasympathetic nervous system (PNS), can be considered as a superhighway information gathering and relaying messages from the internal and external sense organs to the brain where subconscious decisions are made whether to supress or activate the sympathetic response. Activation of the SNS causes an instant increased heart (Dong, 2016) and respiratory rate concomitant with the production of norepinephrine, increased diaphoresis, and decreased interval between heart beats (R. Gordan et al., 2015). The

parasympathetic system engages processes such as the production of saliva and peristalsis and uses acetylcholine to slower heartbeat and increase its intervals. When in balance, the sympathetic and parasympathetic systems therefore produce high HRV through larger intervals between heartbeats (Ernst, 2017). The more variable the HR is, the faster the response to environment changes. HRV is used as a health marker and its analysis provides insight regarding autonomic control of the heart (Joyce & Barrett, 2019). It is common to evaluate the HRV response to a stressor, such as exercise, to evaluate CV autonomic control (Perini & Veicsteinas, 2003). High HRV is an indication of CV health and fitness (Young & Benton, 2018), calm and positive emotions (Mather & Thayer, 2018), and the ability to handle stress (Kim, Cheon, Bai, Lee, & Koo, 2018). Low HRV is related to inflammation, chronic stress, chronic pain, depression, cancer, and low emotional flexibility (Cygankiewicz & Zareba, 2013; Weber et al., 2010).

HRV can be calculated via ultra-short-term (<5 min), short-term (~5 min) or long-term recordings (24-h) of the electrocardiogram (ECG). The 24h HRV recordings are considered the 'gold standard' for clinical HRV assessment and achieve greater predictive power than short-term measurements (Bigger, 1989; D. C. Fei, X.; Malik, M.; Camm, A., 1996; R. E. Kleiger, Stein, Phyllis K., Bigger, J. Thomas, 2005; Nolan, 1998). The short-term recordings are generated by two distinct but overlapping sources. The first source is the complex and dynamic relationship between the sympathetic and parasympathetic branches, and the second one includes the regulatory mechanisms that control HR via RSA, in other words, the respiration-driven speeding and slowing of the heart via the vagus nerve (Fumihiko, 2004), the baroreceptor reflex (negative-feedback control of BP), and rhythmic changes in vascular tone (Ben-Tal, Shamailov, & Paton, 2014).

Vascular System and Hemodynamics

The human vascular system is a vast array of blood vessels (Pugsley, 2000). The structure of the blood vessel largely determines the functions of the different vascular beds (Tennant, 1990). The blood flow is determined by a series of physical properties and forces and by physiological mechanisms that control the diameter of arteries and arterioles, ensuring that organs receive a blood supply that matches metabolic needs of the tissue (Roddie, 1963).

Structure of Blood Vessels

Blood vessels are not homogenous tubes, and the size and structure can vary in different parts of the same vessel, depending on their location in the body and the functions they serve. Most blood vessels share a similar structure comprising 3 key layers (the intima, media, and adventitia), named 'tunicas', surrounding the lumen, that actually hold the blood (Pugsley, 2000). The innermost section is the tunica intima which is often primarily composed of a basement membrane and a single layer of endothelial cells. Surrounding the tunica intima is the middle layer, the tunica media, located between the adventia and the intima and mostly composed of smooth muscle cells installed in a matrix of collagen, elastin, and various glycoproteins (Mazurek et al., 2017; Pugsley, 2000). The tunica media plays a key role in blood flow and BP since the smaller the diameter of the blood vessel, the harder it is for blood to move through it (Pugsley, 2000). Finally, the outermost layer of blood vessels is the adventitia or tunica externa which contains nerve innervation and is composed of loosely woven collagen fibrils that protect and reinforce the vessel (Mazurek et al., 2017; Pugsley, 2000). In arteries these layers function together to sense and respond to acute changes in BP via dilation or constriction and respond to chronic changes by undergoing growth and/or remodelling (Lyle & Raaz, 2017). A relatively large amount of smooth muscle is required in the smaller arteries and arterioles for these to contract and regulate organ blood flow and arterial BP (Tykocki, Boerman, & Jackson, 2017). In contrast, elastic arteries, such as the aorta, are built to passively expand as blood is PUMPed into the vessel because they contain layers of smooth muscle cells interwoven with a large amount of elastin (Pugsley, 2000).

CV risk factors and the incidence of CV disease has been associated with artery wall thickness (Burke et al., 1995). Thus, the measurement of the combined thickness of intima and media layers of the common carotid artery, the carotid intima-media thickness (IMT) is used as a surrogate for subclinical CV disease and as a variable predictive of CV events (Bots, Hoes, Koudstaal, Hofman, & Grobbee, 1997; Burke et al., 1995; Chambless et al., 1997; Lorenz, Schaefer, Steinmetz, & Sitzer, 2010; Rosvall, Janzon, Berglund, Engström, & Hedblad, 2005; Salonen & Salonen, 1991). Increased carotid IMT

might occur in an earlier phase of the atherosclerotic process and can be used to detect an accelerated disease process and subclinical disease (myocardial infarction, stroke, peripheral arterial disease). The detection of subclinical atherosclerosis allows the prevention to a devastating CV event (Cobble & Bale, 2010; Zureik et al., 2000). Thus, increases in the thickness of the intima and media of the carotid artery are directly associated with an increased risk of myocardial infarction and stroke in older adults without a history of CV disease (Polak & O'Leary, 2016). In addition to carotid IMT, assessment of carotid extra-media thickness (EMT), a reproducible non-invasive measure of elastic artery adventitial thickness assessed by ultrasonography (Salonen & Salonen, 1991; M. R. Skilton et al., 2011; Michael R. Skilton et al., 2009), may provide a more complete indication of the structural modification of the vasculature associated with CV risk factors than that obtained by the measurement of carotid IMT alone (Michael R. Skilton et al., 2009). Carotid EMT is correlated with both carotid IMT and AS from the same arterial segment and thus associated with CV risk factors (Cai, Sullivan, et al., 2016; Michael R. Skilton et al., 2009; M. R. Skilton et al., 2012). In adults at risk of coronary artery disease (CAD), carotid EMT, unlike carotid IMT measured from the same arterial segment, was not significantly associated with CV events (Cai, Magnussen, et al., 2016).

Mechanical Properties of Large Arteries

The viscoelastic properties of the aorta and the large arteries permit them to have not only a passive function of transfer oxygenated blood from the heart to the periphery, but also a central hemodynamic function whereby the elastic nature of the vessel wall provides a significant biomechanical buffering capacity, as they are able to buffer left ventricular stroke volume (Belz, 1995). In fact, there is an alternation between the systole, in which a given amount of blood is forcibly pushed into the arterial system, and diastole, in which ventricular filling occurs in each cardiac cycle. Herewith, the large arteries have the task of damping the pulsatile output of the left ventricle and of changing the rhythmic, intermittent, and discontinuous activity of the cardiac PUMP into a continuous one (Belz, 1995). The change from intermittent flow (on leaving the heart) to continuous flow (in the organs and tissues) occurs thanks to the viscoelastic properties of the aorta and large arteries. With preserved viscoelastic properties, therefore, only a fraction of the stroke volume during systolic phase is sent directly to the periphery, while most of it is stored in the system of large elastic arteries (Paolo Salvi, 2016).

The stability, resilience, and compliance of the vessel wall are dependent on the relative contribution of its 2 prominent scaffolding extracellular matrix proteins: collagen (Ricard-Blum, 2011) and elastin (Cocciolone et al., 2018), which function to provide

structural integrity and elasticity (Silver, Horvath, & Foran, 2001). Thus, the relationship between these two principal components of the arterial wall contribute to the viscoelastic properties of the aorta and large arteries (Viscoelastic properties of the aorta = Elastin fibers/Collagen fibers). The dysregulation of this balanced relationship leads to overproduction of abnormal collagen and diminished quantities of normal elastin, which origins marked parietal stiffness and reduced vascular distensibility in the aorta and large arteries (Zieman, Melenovsky, & Kass, 2005). Histological examination of the intima of stiffened vessels reveals abnormal and disarrayed endothelial cells, increased collagen, frayed and broken elastin molecules (Lakatta, 2003). Thereby, some clinical conditions, mechanisms, and effects such as aging, hypertension, inflammation, and metabolic alterations can alter the anatomical, structural, and functional properties of large arteries, affecting their mechanical properties (Lyle & Raaz, 2017). Under these conditions, during the systolic ejection time there is a decrease in the amount of the stroke volume that is stored by the aorta, while most of it is ‘‘pushed’’ directly towards the periphery of the vascular system. Consequently, the propulsive effect of the aorta, in diastole, is reduced. So, an alteration in the mechanical properties of large arteries causes three important consequences on BP, an increase in SBP values, a decrease in diastolic pressure, and an increase in PP. These changes may result in left ventricular hypertrophy (Girerd, 1991) and increased fatigue of arterial wall tissues (Blacher, 1999), all of which substantially increase the risk of CV events and all-cause mortality (McEniery, 2006; Vlachopoulos et al., 2010). Moreover, studies have shown that the control of SBP values is of much importance in CV prevention (Basile, 2003; Ettehad et al., 2016).

Arterial distensibility (AD) and PWV are commonly used to characterize AS, as the clinical assessment of AS relies on non-invasive measurements of local distensibility or regional PWV (Cai, Magnussen, et al., 2016; Groenink, de Roos, Mulder, Spaan, & van der Wall, 1998; Laffon et al., 2005; McDonald, 1968; Mohiaddin, Firmin, & Longmore, 1993; Vulliémoz, Stergiopulos, & Meuli, 2002).

Arterial Distensibility

Large artery distensibility is physiologically important for CV efficiency. AD is a measure of arterial ability to expand during systole and is defined as the relative change in the cross-sectional area of the artery (strain) divided by the local PP (Dogui et al., 2011), which is inversely related to AS. Distensibility is known as the value of arterial compliance in respect to the artery's original diameter (Paolo Salvi, 2012). Arterial compliance is defined as the change in artery diameter (or section) in absolute values for a given arterial length at a particular pressure level (Paolo Salvi, 2012). A decrease of AD (increased artery wall stiffness) seems to have a several adverse consequences

for the CV system, being a common pathologic mechanism for many factors that lead to the occurrence and progression of the vascular changes associated with CV disease (Godia et al., 2007). However, AD cannot only undergo a reduction but also an increase (Giannattasio et al., 1997; Stella et al., 1998). It also occurs under more physiological circumstances, i.e. as a result of exercise training. B. A. Kingwell, Berry, Cameron, Jennings, and Dart (1997) have shown overall arterial compliance to be increased in athletes as compared to sedentary subjects. Regarding the radial artery studies show that: (1) distensibility is greater in the arm involved in a heavy physical exercise (baseball players) compared with the contralateral arm (Giannattasio et al., 2001) and (2) markedly reduced following prolonged absence of exercise because of arm fracture and cast enclosure, with a recovery after cast removal and physical rehabilitation (Giannattasio et al., 1998).

Several methods for quantifying AD in large arteries have been proposed. AD as the change in vessel diameter divided by PP normalized for the initial diastolic diameter can be measured in a relatively easy fashion in the common carotid artery and the abdominal aorta above the bifurcation of the renal arteries (Godia et al., 2007). The mechanical properties of arteries can be measured starting from the volume/pressure ratio of an isolated arterial segment. From a general point of view, the mechanical properties of a blood vessel are not linear; that is to say, they depend on the pressure they are subjected to and at which they are measured, whether regional or local measures (Zieff et al., 2019). Therefore, different parameters are used for assessing the mechanical properties of large arteries and for supplying indications about their viscoelastic properties, such as, distensibility coefficient, beta stiffness (β), and Peterson's pressure-strain elastic modulus (ε). Of these parameters, the carotid artery distensibility coefficient, which indicates the intrinsic local stiffness of the carotid artery, may be determined using ultrasound and magnetic resonance imaging, and also significantly predict future total CV events, CV mortality and all-cause mortality (C. Yuan, Wang, & Ying, 2016). Distensibility coefficient is the relative change in the cross-sectional area/diameter during the cardiac cycle for a stroke change in BP, and it is inversely correlated with AS. β is the logarithm of the ratio between SBP and DBP (SBP/DBP) and the relative change in arterial diameter (McVeigh, Hamilton, & Morgan, 2002). However, there are some limitations regarding assessment methods. Changes in vessel diameter cannot be related to PP changes at the same or a nearby vessel site, making quantification of the stimulus to arterial wall distension somewhat inaccurate. In the carotid artery this can only be avoided in part by obtaining the BP waveform through a tonometer positioned on the skin above the vessel because even modest variations in

the pressure applied by the tonometer alter the shape and thus the reproducibility of the BP waveform (Giannattasio & Mancia, 2002).

Aortic Pulse Wave Velocity

PWV represents the speed of a pressure wave propagating down a blood vessel, is most commonly measured as the time it takes a pulse wave to travel from the carotid to the femoral arteries divided by the distance multiplied by 0.8 and is consequently a marker of aortic stiffness (Sehestedt & H. Olsen, 2015). The pulse wave is transmitted through the arterial vessels, and its speed is inversely related to the viscoelastic properties of the wall itself. Therefore, the PWV differs within the arterial tree, once it is much slower in a central artery, such as the aorta, than the PWV in a peripheral artery. The more distensible or less stiff, the slower velocity. Hence, central arteries are typically distensible rather than peripheral arteries that are stiffer (less compliant), likely due to the difference in arterial wall composition. As people age, this difference tends to disappear and can be probably related with the increase in the AS in older subjects (O'Rourke, 2007). cf-PWV is considered the clinical gold standard gold-standard method for AS assessing and is used by most studies in the field (Laurent et al., 2006). Assessment requires the use of high-fidelity transducers to record the pressure of pulse waves in two sites. In this way, a proximal transducer is placed on the carotid artery, and a distal transducer on the femoral artery and the PWV along the aorta is measured. Therefore, aortic PWV reflects the viscoelastic properties of the aorta and is consider as an independent predictor of CV mortality (Duman et al., 2015). Increased PWV owing to arterial stiffening resembles the basis for further indices of aortic stiffness (Cecelja & Chowienczyk, 2012).

Factors Affecting Arterial Distensibility and Pulse Wave Velocity

Structural alterations and transient functional changes in the arterial wall affect PWV and subsequently AD (Cavalcante, Lima, Redheuil, & Al-Mallah, 2011; Cohn, Quyyumi, Hollenberg, & Jamerson, 2004). The aging process (Safar, O'Rourke, & Frohlich, 2015), hypertension (Safar et al., 2015) and metabolic diseases (Maloberti et al., 2020) are considered structural alterations occurring due to changes in the elastin fiber/collagen fiber ratio in the arterial wall (Cocciolone et al., 2018). The aging process causes histological alterations in the arterial wall, and the degeneration of elastin fibers is accompanied by a boost in collagen fibers (Safar et al., 2015). Arterial hypertension is characterized by a steady increase in BP values causing structural alterations that remain even when normal BP values are restored after effective antihypertensive treatment. In this condition, the arterial wall tends to increase biosynthesis of collagen fibers to face increasing pressures in the vascular lumen, and further changes of the

elastin/collagen ratio are found (Safar et al., 2015). Structural alterations have been described even in metabolic diseases such as diabetes, kidney failure, liver failure, alterations in calcium metabolism (Maloberti et al., 2020).

Regarding functional factors causing transitory alterations in AD, these are: 1) mean arterial pressure (MAP), affecting PWV, even if only in part (Franklin, Khan, Wong, Larson, & Levy, 1999; Zheng & Murray, 2011); 2) smooth muscle cell tone, in relation to adrenergic activity (Failla et al., 1999) and 3) left ventricular ejection fraction, which can be assessed by finding the left ventricular ejection time (Giannattasio et al., 2002; Giannattasio et al., 1995). SBP and PP may nevertheless simultaneously increase, adversely modulating two important and possibly independent CV risk factors and DBP may undergo a reduction (if the arterial stiffening is particularly marked) (Zheng & Murray, 2011). Sympathetic tone and a loss of vagal tone exerts a marked tonic restraint on AD (Failla et al., 1999). When in reduced AD, an increase in left ventricular afterload occurs even when BP does not change (Giannattasio et al., 2002; Giannattasio et al., 1995).

Pulse Wave Velocity in Other Arterial Segments

PWV can also be assessed in other arterial districts, such as, radial, femoral and/or tibial arteries. Carotid–radial or upper-limb pulse wave velocity (cr-PWV) assesses the mechanical properties of the upper limb arteries. In this case, the distal transducer records pulse wave in the radial artery. Carotid-distal or lower-limb pulse wave velocity (cd-PWV) assesses the viscoelastic properties of the arterial system in the lower limb and this method can supply useful information about the functional state of the peripheral blood circulation. The proximal transducer records the pulse wave in the carotid artery, while the distal transducer records the pulse wave in the dorsalis pedis or the posterior tibial artery. Most likely, cr-PWV reflects the functional condition of the arterial tree, which is closely related to activation of the sympathetic system (Genovesi et al., 2020). However, studies targeted at comparison between different arterial districts have confirmed that only cf-PWV has independent prognostic significance for CV disease (Willum-Hansen et al., 2006).

Central Arterial Blood Pressure

The BP in the ascending aorta, on leaving the left ventricle, is commonly termed ‘Central arterial blood pressure’. This phenomenon is very important as it affects the hemodynamic relationship between the heart and the aorta, both in systole and in diastole. In systolic phase, central arterial pressure defines the work that the left ventricle must perform to maintain adequate stroke volume, while in the diastolic phase, affects

the regular blood flow to ventricular myocardium and maintains adequate subendocardial perfusion (Paolo Salvi, 2012). The assessment of central arterial pressure values is important because there is a difference between peripheral and central blood pressure (cBP) values (Giannattasio, 2003). Along the arterial tree, the difference between central and peripheral DBP values is insignificant, whereas peripheral SBP values (in the radial, brachial, and femoral arteries) are higher than the ones measured in the ascending aorta. The difference between BP in the aorta and in the brachial artery is, on average, about 15 mmHg, but differences can be recorded up to 40–50 mmHg in young adults (P. Salvi, Grillo, & Parati, 2015).

Reflected Waves, Central & Peripheral Blood Pressure

The arterial system behaves like a hydrodynamic system characterized by the existence of reflected waves, where the wave is generated by the activity of an intermittent “PUMP” (the heart) that travels down a “pipe” (aorta, arteries, arterioles, etc.). Reflected waves are generated at reflection sites (arterial bifurcations, atherosclerotic plaques, and terminal arterioles) and travel towards the centre of the system. As the forward pulse wave encounters these sites or other changes in arterial anatomy, some portion of that wave is reflected back up the arterial tree, then merging with the next forward wave, forming a more peaked systolic portion of the generated pulse wave. Consequently, the amplitude of the resultant wave will be defined by the sum of the amplitude of the forward wave and the backward wave (Paolo Salvi, 2012). A pressure waveform always depends on the temporal relationship between the encounter and superimposition of the forward and backward pressure waves.

In the ascending aorta, a central artery, its encounter with the forward wave occurs at the end of the systolic phase, and the superimposition of the two waves lasts the whole diastolic phase, when the viscoelastic properties of large arteries are undamaged. Consequently, the central pressure waveform shows the following consequences: 1) the peak SBP is not affected by the backward waves, and the backward waves do not change SBP values; and 2) backward waves affect diastolic time, so that the pulse waveform, in diastolic phase, will appear full and convex. So, when in normal physiological conditions, reflected waves play a positive role and have advantageous effects as they maintain high BP values during the diastolic phase, and supply good coronary blood flow, with no increase in left-ventricular afterload (Paolo Salvi, 2012).

In the peripheral arteries such as the femoral, brachial, radial arteries, etc., the superimposition of the forward pressure wave and backward waves is particularly important once it starts very early during the systolic phase. Consequently, the pressure

peak will be strongly affected by backward waves, and the reflected component will strongly affect SBP values, so the SBP in the peripheral arteries is mainly defined by the presence of wave reflection.

Factors Affecting Central Blood Pressure

The viscoelastic properties of the aorta and the large arteries are one of the main factors affecting cBP. An alteration in the mechanical properties of large arteries causes an increase in SBP, a decrease in DBP, and, consequently, an increase in PP (PP = SBP - DBP).

Arterial Stiffness

At the periphery of the arterial system, the difference between diastolic and systolic BP becomes more marked, owing to the early superimposition of the reflected wave. Stiffness occurs in central and conduit vessels while sparing more the peripheral arteries (Benetos, Laurent, Hoeks, Boutouyrie, & Safar, 1993; Gillessen, Gillessen, Sieberth, Hanrath, & Heintz, 1995), which means that the dissemination throughout the vascular tree isn´t uniform (Bassiouny, Zarins, Kadowaki, & Glagov, 1994; Beattie, Xu, Vito, Glagov, & Whang, 1998). Herewith, the generated and travelling pulse waves from the heart to the periphery are typically reflected at sites of impedance mismatch. Under increased AS conditions, early backward waves cause further increase in SBP values and in PP, and therefore in afterload, creating difficulties for the PUMP function of the heart and leaving the diastolic phase increasingly depleted, also causing problems for coronary blood flow. Therefore, the decreased ability of an artery to expand and recoil during cardiac contraction and relaxation (reduced compliance) at the central vasculature alters arterial pressure and flow dynamics (Glasser et al., 1998). Compliant arteries generate slower PWV (Glasser et al., 1998) and the reflected waves return in diastole, augmenting central diastolic blood pressure (cDBP). In contrast, stiffer arteries and high PWV cause the earlier arrive of reflected waves and augment central systolic blood pressure (cSBP) (Mitchell, 2009; Wang et al., 2010).

Central Blood Pressure and Cardiovascular Risk

Buckberg Index: Subendocardial Viability Ratio

Assessment of CV risk, particularly in CAD risk, includes the use of the central pressure waveform. Subendocardial viability ratio (SEVR), also well known as the ratio between the diastolic pressure–time index (DPTI) and the systolic pressure–time index (SPTI) (DPTI:SPTI ratio) (Buckberg, Fixler, Archie, & Hoffman, 1972) or Buckberg Index is an example. The SEVR represents the ratio between myocardial oxygen demand and supply and can be defined, noninvasively, based on central pulse wave analysis (PWA).

The myocardial oxygen demand mainly depends on HR, ejection pressure, and myocardial contractility. SPTI is the area under the left ventricular (or aortic) pressure curve in systole from the onset of ventricular systole to the dicrotic notch, and is, perhaps, the most reliable parameter representing the afterload, i.e. the arterial pressure against which the left ventricle must contract to guarantee effective Q. The area between the aortic and left ventricular pressure curves in diastole is the DPTI, which indicates potential subendocardial blood supply. DPTI is obtained by subtracting the left ventricular mean diastolic blood pressure (LVMDP) from the area, in diastole, of the aortic pressure curve. LVMDP can be, substantially and successfully, replaced by an estimate of the left ventricular (or atrial) end-diastolic pressure, which is more easily assessed, noninvasively, by echocardiography. To sum up, the SEVR describes the relationship between supply and demand, i.e., between the myocardial blood supply and myocardial oxygen requirement.

Methods and Assessments

How to measure Arterial Stiffness?

Non-invasive measurement of AS entails measurement of surrogate parameters that are intrinsically associated with stiffness. This involves three main methodologies: 1) pulse transit time, 2) analysis of the arterial pressure pulse and its wave contour, and 3) direct stiffness estimation using measurements of diameter and distending pressure (Pannier, Avolio, Hoeks, Mancia, & Takazawa, 2002). A variety of validated devices and methods are currently used to quantify arterial mechanical properties. The device that provide information about more than one aspect of the circulation can thus be classified as devices that: quantify pulse transit time; analyse the pressure pulse waveform; or provide direct estimation of vessel stiffness by the simultaneous assessment of arterial diameter and a corresponding distending pressure (Hamilton et al., 2007). Such devices usually measure one of three possible indices of AS: systemic stiffness (i.e. a measure of the stiffness of the entire circulation); regional or segmental stiffness (i.e. a measure of the stiffness of a segment of the arterial tree); or local stiffness (i.e. a measure of the stiffness in a small section of one blood vessel under study).

Systemic Arterial Stiffness

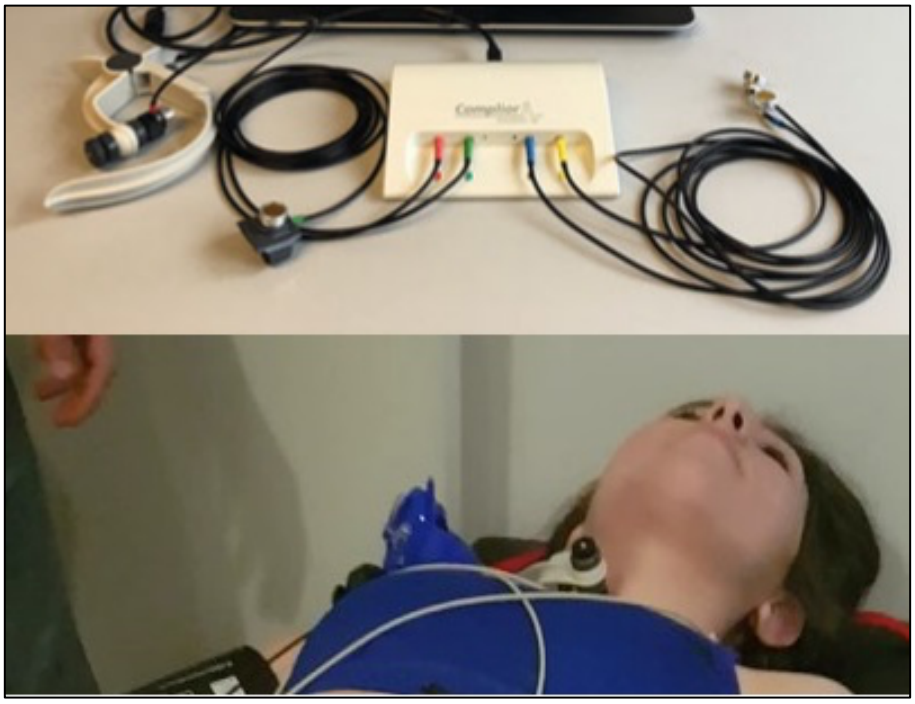
Systemic AS affects the global buffering properties of the arterial system and may reflect the overall opposition of large arteries to the pulsatile effects of ventricular ejection. The measurement is based on numerous theoretical approximations. In general, it requires the direct measurement of a single peripheral, and often distal, parameter: the pressure curve (Pannier et al., 2002).

Regional or Segmental Stiffness

Regional AS is measured at arterial sites of major physiologic importance such as the carotid where the arterial buffering function is principally expressed, or a particular limb such as the arm (a major site of BP recording) (Pannier et al., 2002). Most of the measurement methods are based on the principle of PWV recording. The assessment involves measurement of two quantities: transit time of the arterial pulse along the analysed arterial segment, and distance on the skin between both recording sites. The cf-PWV is recognized as the ‘gold standard’ measurement of AS according to the expert consensus document on AS (Laurent et al., 2006).

Figure 1

Complior Analyse and Assessment of Regional AS



Nevertheless, a velocity is determined by distance/time. Therefore, PWV is calculated using the same formula, measuring the distance between two arterial segments divided by the time taken for the pulse wave to travel that distance – pulse transit time. Perhaps the most important methodological confounder of PWV measurements is calculation of the wave travel distance. The travel distance is typically estimated from surface measurements between the recording sites. These measurements should be as accurate as possible because small errors in distance measurement may translate into much larger errors in the calculated PWV, so the measurement method and vascular territory should be clearly stated (Townsend et al., 2015). The distance is usually acquired using a tape measure over the body surface, although a recent expert consensus document on the measurement of aortic stiffness advised, as a new standard, the usage of 80% of the direct tape measure distance (Van Bortel et al., 2012) The time can be performed by measuring the interval between points on a pressure or flow waveform, or be obtained between a defined point on an ECG and a defined point on the pulse waveform and compared between the different sites (Parati, 2006). There are a variety of available devices on the market to measure PWV. Some measure pulse waveforms in multiple sites using various transducers (tonometers, ultrasound probes, oscillometers, mechanoreceptors) and there are two different methods to carry out the assessment: 1) when two or more transducers are simultaneously used, or 2) combining electrocardiographic tracing with the use of just

one transducer (P. Salvi et al., 2015). The Complior 2.0, (Alam Medical; Saint Quentin Fallavier, France) gives an automated measurement of PWV (Roland Asmar et al., 1995) for one or two arterial segments simultaneously (Kosch et al., 2001) with dedicated mechanotransducers. For the transit time calculation during 15 sec under vision control, only adequate pressure waves are used, which are selected by an in-built quality control processing.

Local Stiffness

Local determination of AS involves measurement of cross-sectional AD. The Moens– Korteweg equation (1878) and later modified by Bramwell and Hill (Bramwell, 1997) describes the main determinants of PWV and establishes the correlation between the distensibility of a vessel to the velocity of the pulse wave traveling in that vessel, based on the calculation of the speed of transmission of transverse elastic waves. For the corresponding arterial segment, the assumption is that the segment is a cylindrical tube. That means the combination of diameter measurement and simultaneously or within a few minutes, local BP recording. Systems are based on a vascular echo tracking device using the Doppler shift principle or on echo imaging (Pannier et al., 2002). One of the first instruments used for recording the diameter change curve was the Wall Track System (WTS; Pie Medical, Maastricht). This device assesses arterial diameter change, during a cardiac cycle, using a radiofrequency signal integrated with ultrasound scanning. An echo-Doppler test of the carotid axis is carried out and the anterior and posterior walls of the artery are analysed. At the end of the analysis procedure, the curves representing the systo-diastolic motion of the anterior and posterior wall are displayed, and under these, the arterial diameter change curve, resulting from the sum of the two previous ones, is displayed as well (Hoeks et al., 1997). This methodology allows the cross-sectional measurement or AD through quantification of changes in arterial diameter, assessing the local stiffness in carotid artery (van der Heijden-Spek et al., 2000).

How to measure Cardiovagal Modulation?

Evaluation of the autonomic nervous system has been described and applied in scientific studies or clinical practice (Hilz & Dutsch, 2006; Jaradeh & Prieto, 2003; Junqueira, 2008; Klein, 2008; Lambert, 2007; Low, 2003; Zygmunt & Stanczyk, 2010). In addition, it has become an essential tool in scientific research (Yu et al., 2018).

The analysis of HRV and the assessment of the BP response to physiological stimuli are non-invasive measures of CV parasympathetic function and CV sympathetic function, respectively. HRV has been used as a non-invasive method to evaluate cardiac

autonomic regulation by the parasympathetic and sympathetic divisions of the autonomic nervous system.

The automated devices to assess autonomic function have become widely available (Illigens & Gibbons, 2019). Finapres® Nova, (Ohmeda, Louisville, Colorado, USA) device was the first to introduce automated technology for the measurement of HRV from the quantification of the fluctuations in RR intervals derived from beat-to-beat ECG intervals (Imholz, Wieling, van Montfrans, & Wesseling, 1998). Finapres® Nova is in accordance with the ANSI (American National Standards Institute) recommendations for the accuracy of automated non-invasive devices.

Heart Rate Variability

HRV has been considered a useful non-invasively method to monitor cardiovagal modulation (Foster et al., 2001; Lewis & Short, 2010; K. Martinmäki & H. Rusko, 2008) by quantifying the fluctuations in RR intervals (D. C. Fei, X.; Malik, M.; Camm, A., 1996). RR intervals are derived from beat-to-beat ECG intervals using the Finapres 5 ECG lead module (Finapres® Nova, Ohmeda, Louisville, Colorado, USA).

ECG-based evaluation of HRV may be analysed by different methods, including time- and frequency-domain analyses (Electrophysiology, 1996; Federico Lombardi 2001; Huikuri, Mäkikallio, & Perkiömäki, 2003; Stein, Domitrovich, Huikuri, & Kleiger, 2005). The time-domain indices of HRV quantify the amount of variability during monitoring periods that may range from ~2 min to 24 h in measurements of the IBI, which is the time period between successive heartbeats, and includes the standard deviation of normal-to-normal RR intervals with filtered artefacts and ectopic beats (SDNN), the square root of the mean squared differences of successive normal RR intervals (RMSSD), the percentage of adjacent NN intervals that differ from each other by more than 50 ms (pNN50), the average difference between the highest and lowest heart rates during each respiratory cycle (HR Max − HR Min), between others. The frequency-domain analyses corresponds to the evaluation of the spectral power of the RR variability in relation to very-low-frequency (VLF), low-frequency (LF; 0.04–0.15 Hz) and high-frequency (HF; 0.15–0.40 Hz) spectra (Shaffer & Ginsberg, 2017).

Included in the time-domain indices, the SDNN values predicts both morbidity and mortality and is considered the "gold standard" for medical stratification of cardiac risk, although it is more accurate when calculated over 24 h period than during the shorter periods monitored during biofeedback sessions (Electrophysiology, 1996). SDNN is influenced by the activity of both SNS and PNS (Umetani, 1998). Based on a 24 h recordings, patients are classified as unhealthy with SDNN values are below 50 ms, and as healthy when values are above 100 ms (R. Kleiger, Miller, J., Bigger, J., Moss, A.,

1987). The RMSSD is used as the primary measure to estimate the vagally mediated changes reflected in HRV (Shaffer, McCraty, & Zerr, 2014) and 24h measurements of RMSSD are strongly correlated with HF power and pNN50 (Bigger, 1989). Although this correlation between RMSSD and HF power, the influence of respiration rate on this index is still uncertain (Aysin, 2006; Penttilä, 2001). In epilepsy, lower RMSSD values are correlated with higher scores on a risk inventory of sudden unexplained death (DeGiorgio et al., 2010). Correspondingly, the pNN50 is correlated with the RMSSD and HF power and closely correlated with parasympathetic system activity (Umetani, 1998). HR max – HR min reflects RSA by being specially sensitive to the effects of respiration rate, independent of vagus nerve traffic and at least a 2-min sample is required to calculate it (Shaffer & Ginsberg, 2017). Within the non-linear measurements, SD1 reflects short-term HRV and is identical to RMSSD (Ciccone et al., 2017). It measures short-term HRV in ms and correlates with baroreflex sensitivity (BRS), which is the change in IBI duration per unit change in BP, and HF power (Shaffer & Ginsberg, 2017).

The parasympathetic influence on the SA node mediates the HF oscillations (Berntson, 1997; Electrophysiology, 1996). Moreover, the greater the influence of vagal activity on the SA node, the greater the HF power. On other way, the sympathetic modulations on the SA node or the combination of vagal and sympathetic influences mark the LF oscillations (A. Malliani, 1994; A. Malliani, Pagani, Lombardi, & Cerutti, 1991). However, the LF power interpretation is mostly controversial so instead of interpreting it, the commonly evaluated measure for the sympathovagal balance is the LF/HF ratio. An increase in this ratio signifies a higher sympathetic influence (Shaffer & Ginsberg, 2017), although it may be more appropriate to think about the ratio as an index of sympathovagal dominance. At the isometric handgrip exercise is commonly to evaluate the HRV response. During this exercise, the HF power is reduced which indicates a vagal withdrawal whereas during recovery there is a vagal reactivation, and by contrast a greater sympathetic dominance demonstrated by the increase in LF/HF ratio that then returns to baseline during recovery indicating sympathetic withdrawal (Farah et al., 2020).

The Reactivity Hypothesis

Despite homeostasis being a typical aspect of the basic human physiology, human's regulatory system has the capacity to adapt to build impressive transitory changes in order to react to different stressors (Zanstra & Johnston, 2011). In acute response to those physiological and psychological stresses, the regulatory systems trigger a complex chain of reactions that lead to temporary increases in HR and BP. These reactions are suggested to happen as result of a reduced vagal tone associated to the PNS (Huang, Webb, Zourdos, & Acevedo, 2013; Spalding, 2000) or due to an increment in afferent sympathetic neuromuscular activation (Huang et al., 2013).

The "reactivity hypothesis" states that subjects showing exaggerated CV reactivity to stress are at higher risk of developing hypertension, atherosclerosis, and CAD (Krantz, 1984; Treiber, 2003), so adverse CV events may be predicted from CV responses to a stressor, such as exercise (Michael, Graham, & Davis, 2017). The regulatory systems, when in response to physical stressors have shown to allow the increases in the secretion of adrenocortical and adrenomedullary hormones such as norepinephrine (NE) and epinephrine (EPI), and these increases are likely responsible for the concomitant increases in HR and BP (Huang et al., 2013). The marked increase in HR that accompanies exercise is due in part to a reduction in vagal tone. HRR is commonly defined as the decrease of HR after cessation of exercise. HRR immediately after exercise is a function of vagal reactivation. Once, a generalized decrease in vagal activity is known to be a risk factor for death (Thayer & Lane, 2007), a delayed decrease in the HR during the first minute after graded exercise is an important predictor of all-cause mortality and death associated with CAD (Cole, 1999). Moreover, a recent study evidenced that decreased HRR at 10 seconds after cessation of exercise is a superior predictor of outcome compared with HRR at later time intervals (van de Vegte, van der Harst, & Verweij, 2018).

Monitoring these cardiac autonomic responses is likely to support a valuable understanding of the autonomic stress reactivity, which seems to be in accordance with the "reactivity hypothesis". For example, aerobic exercise is associated with the attenuation of the CV response during psychological stress and recovery (Dienstbier, 1989; Spalding, Lyon, Steel, & Hatfield, 2004). Additionally, aerobic exercise training has been suggested to defend against reactive oxygen species (ROS) induced lipid peroxidation and to decrease the occurrence of ROS-associated diseases such as CV diseases and Alzheimer disease (Linke, Erbs S Fau - Hambrecht, & Hambrecht; Mattson & Wan, 2005; Seo, Heo, Ko, & Kwak). Some studies have shown that exercise training can enhance the adaptation of oxidative stress by increasing antioxidant defence's (Pingitore et al., 2015; Sallam & Laher, 2016; Watson, 2005). Even though these

physiological responses are the same in reaction to a stressor of any form, the type of reaction experienced, and the volume of reactivity has been shown to be influenced by numerous factors, including inter-individuality (high reactor/low reactor), anthropometric elements, level of cardiorespiratory endurance, own perception of management over the situation and the possible connection between stressors (Huang et al., 2013). In addition, the experienced effects induced by physical stressors, such as physical exercise, are dependent on the type of exercise (Michael, Graham, et al., 2017). However, using the acute exercise model can be advantageous as it allows for an efficient manipulation of exercise variables (i.e. mode, intensity, duration, etc.) and permits greater experimental control of confounding variables (Padilla, Harris, & Wallace, 2007). Predicting the effects of chronic exercise (accumulation of single bouts of exercise over time) can be feasible through the effects of a single bout of exercise (acute exercise), as is the case in several variables (Thompson, 2001). Thus, mechanisms of the exercise (acute or chronic) response can be investigated through the acute exercise model.

Arterial Stiffness During Recovery

It is well established that large artery stiffening increases with age, even in healthy subjects (Vaitkevicius et al., 1993) but can be diminished and less pronounced in those who engage in a program of physical exercise (Cameron & Dart, 1994; Tanaka et al., 2000). Although, the physiological mechanisms by which physical exercise benefits AS have not been completely established, some previous systematic reviews and meta-analyses have analysed the effects of acute exercise on AS regarding different types of exercise, different populations (Motohiko Miyachi, 2012; Pierce, Doma, & Leicht, 2018; Sardeli, Gáspari, & Chacon-Mikahil, 2018; Saz-Lara et al., 2021), and assessed arterial segments (upper and lower limbs) (Mutter, Cooke, Saleh, Gomez, & Daskalopoulou, 2016). Thus, AS during recovery reveals distinct responses whether it is a single bout of aerobic and/or resistance exercise originated from distinctive CV (i.e., BP, HR) and non-cardiovascular (i.e., inflammatory products) processes.

Acute Aerobic Exercise

Studies that examined cf-PWV after an acute aerobic exercise intervention reported conflicting findings. As indicated in B. A. Kingwell et al. (1997), cf-PWV, an outcome measure of AS, was reduced 30 minutes following acute aerobic exercise in both central and peripheral arteries, in healthy young men. In contrast, a cross-over study showed that two types of aerobic exercise, continuous moderate intensity aerobic exercise and high intensity interval aerobic exercise did not affect the cf-PWV relative to the baseline measurements and had different effects on the aortic and peripheral AS

(Siasos et al., 2016), whereas other study stated that cf-PWV, might be further decreased even after 60 minutes of recovery with prolonged exercise (Kobayashi, Hatakeyama, Hashimoto, & Okamoto, 2017). These divergent findings possibly result from the timing of measurement. However, other conflicting findings can be possibly explained from differences between studied arterial segments (Mutter et al., 2016) intensity and type of exercise (Kobayashi et al., 2020), studied population (Mattace Raso et al., 2010) and measurement devices (Milan et al., 2019). The CV mechanisms as well as methodological factors of exercise modulate PWV and may be the explanation for the significant changes in cf-PWV following aerobic exercise. This concurs to the findings by Mutter et al. (2016), reporting that the effects of acute aerobic exercise on AS is dependent on the timing of the measurements post-exercise. Moreover, a differential response to acute aerobic exercise in the lower versus upper/aortic arterial segments in healthy adult subjects was revealed in this systematic review that should be taken into consideration, suggesting a decreased AS induced in lower limbs, proximal to the primary working muscles (Mutter et al., 2016). Regarding other populations, in middle-aged sedentary men, 3 months of aerobic training enhanced carotid artery compliance to levels observed in similarly aged endurance-trained men (Tanaka et al., 2000). A randomized, cross-over study in subjects with systolic hypertension, found that moderate aerobic exercise had no impact on large artery compliance, an inverse outcome of AS (Ferrier et al., 2001). In 2018, a meta-analysis quantified the effect of acute exercise mode on AS and wave reflection measures including cf-PWV, augmentation index (AIx), and HR corrected AIx (AIx75) and reported that acute aerobic exercise did not modify the cf-PWV (Pierce, Doma, & Leicht, 2018). Nevertheless, a 2021 meta-analysis (Saz-Lara et al., 2021) aimed to assess the acute effect of exercise interventions on AS in healthy adults and have shown that the peripheral PWV values present the highest acute effect from exercise, showing a significant decrease immediately after exercise, lasting 24 h. Furthermore, this last study reports that aerobic exercise seems to show a greater acute effect on PWV reduction, observed between 30 and 59 min after the exercise session. Currently, aerobic exercise is considered the most effective type of exercise to reduce AS, since the most studies that examined cf-PWV after aerobic exercise intervention reported a decrease in PWV at the aortic and peripheral levels in the first hour after exercise (Kobayashi et al., 2017; J. Sugawara, Komine, Miyazawa, Imai, & Ogoh, 2015).

Acute Resistance Exercise

In resistance exercise, the protocol used, specifically the number of exercises and exercises employed, the number of repetitions, the number of sets, and the total

work (volume) performed are components of dynamic resistance exercise which largely determines CV responses in this extent (M. L. Pollock et al., 2000). Regarding this type of exercise, most studies have shown that the AS increases immediately after the end of the exercise (Fahs, Heffernan, & Fernhall, 2009; Kingsley, Mayo, Tai, & Fennell, 2016; Yoon et al., 2010). In terms of acute effects, García-Mateo et al. (García-Mateo, García-de-Alcaraz, Rodríguez-Peréz, & Alcaraz-Ibáñez, 2020) reported that lower intensities of resistance exercise imply greater changes showing higher AS (Nico Nitzsche, 2016; Okamoto, Min, & Sakamaki-Sunaga, 2014) compared to higher intensities (lower AS) (Augustine, Tarzia, Kasprowicz, & Heffernan, 2014; Kingsley, Tai, Mayo, Glasgow, & Marshall, 2017; Lefferts, Hughes, & Heffernan, 2015; Nico Nitzsche, 2016). Although, as Kingsley et al. (2017) predicted, the results obtained in different studies are not uniform. Several studies have shown both an acute increase of aortic AS, respectively after resistance exercise (Collier et al., 2010; DeVan et al., 2005; Fahs et al., 2009; Heffernan, Collier, et al., 2007; Yoon et al., 2010). However, there is a relative trend towards increases in AS occurring at lower intensities (Nico Nitzsche, 2016; Okamoto et al., 2014; Yoon et al., 2010). Otherwise, results from a recent meta-analysis evidenced aortic AS is increased following acute resistance exercise (Sardeli et al., 2018).

The accountable mechanisms for the increase in aortic artery stiffness remain unknown although during a resistance exercise bout, the high BP has been speculated to alter the load bearing from more compliant elastin fibers to the stiffer collagen fibers (Heffernan, Fahs, et al., 2009; O'Rourke & Safar, 2005). Some studies rationales state that an high-intensity resistance exercise independently increases aortic stiffness since it can invoke the valsalva maneuver (Heffernan, Jae, Edwards, Kelly, & Fernhall, 2007), being related to the increase in thoracic pressure, which produces both an increase in BP and an outside-in force on the artery. Concerning, peripheral artery stiffness a study from Heffernan et al. (Heffernan, Rossow, et al., 2006) reports that AS decreases only in the exercised leg, and this decrease in peripheral artery stiffness is thought to be due to an increase in peripheral artery vasodilation in response to exercise.

Cardiovagal Modulation During Recovery

The examination of HRV in response to acute exercise yields valuable insight into autonomic CV modulation and possible underlying risk for disease (Kingsley & Figueroa, 2016). HRV upon exercise cessation, demonstrate a time-dependent recovery and eventual return to pre-exercise levels (Stanley, Peake, & Buchheit, 2013). In the initial minute following exercise is commonly observed a rapid (though incomplete) recovery (Al Haddad, Laursen, Chollet, Ahmaidi, & Buchheit, 2011; M. Buchheit et al., 2009; M.

Buchheit, Laursen, & Ahmaidi, 2007), while complete recovery may take up to 48 h following some bouts of exercise (Hautala et al., 2001).

An acute bout of aerobic exercise can be modified by three primary factors constituting the exercise dose: intensity, duration, and modality, as stated by the ACSM (M. L. Pollock, 1998). Although few studies have examined post-exercise HRV following different modalities of “aerobic” exercise. Cunha, Midgley, Goncalves, et al. (2015) investigated the immediate recovery period (5 min) following incremental exercise of three modalities: walking, cycling, and running, reporting that HRV recovery was more rapid following exercise involving a smaller muscle mass or energy expenditure (EE) (i.e., cycling > walking > running) and therefore concluding that muscle mass and/or EE are determinants of post-exercise parasympathetic reactivation.

Acute resistance exercise has shown to decrease cardiac parasympathetic modulation more than aerobic exercise in young healthy adults suggesting an increased risk for CV dysfunction after resistance exercise (Marasingha-Arachchige, Rubio-Arias, Alcaraz, & Chung, 2020). Acute resistance exercise appears to decrease parasympathetic activity regardless of age. An acute bout of upper-, lower- or whole-body resistance exercise may cause a prolonged decrease in vagal modulation in young healthy adults (Kingsley & Figueroa, 2016). Previous studies have reported that acute resistance exercise increases the cardiac sympathetic modulation while decreasing the cardiac parasympathetic modulation (Kingsley et al., 2019; Monteiro, 2018; Rezk, Marrache, Tinucci, Mion, & Forjaz, 2006).

Some studies suggest that resistance training can change the HRV indices in the population of healthy young adults, and this is due to increased vagal activity or preventing the high sympathetic activity, provided by the practice of this type of training. Only some of the studied indices indicated an improvement in cardiac autonomic modulation which may be related to the population studied, once a young healthy adult population, in theory, present no autonomic dysfunction and has an intact autonomic nervous system (Heffernan, Jae, et al., 2009).

Ecological Validity

Over the last few decades, sport scientists have frequently discussed how to overcome some of the limitations of laboratory-based experiments. Research has typically been conducted in laboratory or clinical settings, with evidence methodologically delineated with exercises (aerobic and resistance) rather than activities, to simulate typical sessions according to current guidelines for improving and maintaining CV health (D. Riebe, et al., 2018). However, it remains unknown whether the evidence from laboratory research settings can be translated to real-world events. How can we ensure that laboratory-to-

field results are accurately translated? Many researchers have called for more 'ecologically valid' data that is more closely related to and generalizable to the 'real-world.' The term 'Ecological Validity' reflects a study's ability to generalize its findings to real world setting. Due to the involvement of the researcher in manipulating and controlling variables, a study can lack ecological validity and findings may not be easily generalized to the real-life settings. Resistance training is one such example, with laboratorial experiments indicating that the energy expenditure during a resistance exercise session (consecutive multiple-set or circuit) ranges from 64 to 534 kcal (Binzen, Swan, & Manore, 2001; Hunter, Wetzstein, Fields, Brown, & Bamman, 2000; Melby, Scholl, Edwards, & Bullough, 1993; Thornton & Potteiger, 2002), whereas the energy expenditure during a 60-minute Bodypump fitness class appears to be only around 250 kcal in young healthy men and women (Berthiaume, Lalande-Gauthier, Chrone, & Karelis, 2015). There is some agreement that research conducted outside of the laboratory will better represent the researched topics in real-world public health context and provide precise field-related answers, resulting in enhanced ecological validity. Therefore, research assembling of real-world public health contexts is required to assess whether the current data have good ecological validity and can be extrapolated to the real-world environment.

Purpose

Despite the high rates of participation in gyms and health clubs available group fitness classes, no evidence exists to describe and/or compare the post-exercise cardiovagal modulation and AS alterations to different fitness classes. The aim of the present work is to compare the time-course of post-exercise cardiovagal modulation and changes in local and regional indices of AS to different commercially available group fitness classes (BIKE, PUMP Power (PUMP) and Global Training (GT)), as prescribed for health (D. Riebe, et al., 2018).

Acute Effects of Exercise Mode on Arterial Stiffness in Healthy Young Adults

Abstract

Participation in group fitness classes is an increasingly common method by which subjects may fulfil the criteria set down in the ACSM guidelines for enhancing and maintaining CV fitness. However, little is known on the acute effects of different group fitness classes on indices of AS. The aim of the present study was to compare the acute changes in local and regional indices of AS to different fitness classes (BIKE, PUMP and GT). Twelve participants (7 male and 5 females, mean±SE = 24.7±1.0 years) attended 4, separated, group fitness classes of BIKE, PUMP, GT, or no exercise, Control (CON). Testing took place during 20 minutes before and after each group fitness class at 10-, 20- and 30-min. Resting and post exercise measures included SBP and DBP followed by 1) regional assessments of PWV of the aortic, upper-limb, and lower-limb segments, and 2) assessments of carotid AS indices. No change was found for aortic and upper-limb PWV. Lower-limb PWV was significantly decreased 10-min following the three group fitness classes (p=0.030). β (p=0.001; η2=0.377), ε (p<0.001; η2=0.462), and pulse wave velocity beta (PWV-β) (p<0.001; η2=0.457) remained unchanged following GT but increased immediately following (10-min) BIKE (dβ=0.9; dε=9.3 kPa; dPWV-β=0.4 m/s) and PUMP (dβ=0.9; dε=10.3 kPa; dPWV-β=0.5 m/s) group fitness classes. In conclusion, regional AS appears to be decreased solely in the lower segments whereas no changes were observed in the aortic and upper segments, following the three fitness classes. Local AS indices increase immediately following the acute aerobic (BIKE) and resistance (PUMP) fitness classes, but not following combined fitness class (GT). These findings suggest that the effect of commercially available fitness classes on indices of AS were dependent on the mode of exercise, the anatomical segment assessed, and on the timing of the measurement.

Keywords: Acute exercise · Fitness classes · Arterial stiffness

Introduction

Increased AS is highly predictive of future morbidity and fatal CV events (Ecobici & Voiculescu, 2017). A 1 m/s increase in cf-PWV is associated with a 10% increase in CV event risk (Vlachopoulos et al., 2010). In fact, increased AS denotes true damage of arterial wall integrity and alteration of its function, whereas other risk factors such as BP, glycaemia and/or lipid levels vary and may not be representative enough of the cumulative effects of CV risk factors on the arterial system (Arnett, 1994; London, 2002; Obeid et al., 2017).

In addition to their ability to predict CV event risk, AS is predictive of the success of different therapies (D. Aronson & Burger, 2001; Mahmud & Feely, 2008). CV medications including beta-blockers have been reported to reduce aortic PWV and wave reflection (Mahmud & Feely, 2008). Additionally, a single bout of aerobic exercise has been shown to reduce both AS (i.e., cf-PWV) (Heffernan, Collier, et al., 2007; B. A. Kingwell et al., 1997)) and wave reflection (i.e., AIx and reflection magnitude) (Millen, Woodiwiss, & Norton, 2016b; Munir et al., 2008)) but these seem to be influenced by the exercise intensity (Cortez-Cooper et al., 2005; Hoonjan et al., 2011; Kobayashi, Hashimoto, Hatakeyama, & Okamoto, 2018; Kobayashi et al., 2020) and mode (Ashor, Lara, Siervo, Celis-Morales, & Mathers, 2014; Pierce, Doma, & Leicht, 2018). However, this evidence is derived by only a limited number of studies (Leicht et al., 2008; Pierce, Doma, Raiff, et al., 2018) that focused on exercises rather on group fitness classes, with an obvious disadvantage resting on their reduced ecological validity (S. G. Dasilva, Guidetti, L., Buzzachera, Cosme F., Elsangedy, Hassan M., Krinski, K., De Campos, W., Goss, Fredric L., Baldari, C., 2011; Unick, Michael, & Jakicic, 2012).

Participation in group fitness classes is an increasingly common method by which subjects may fulfil the criteria set down in the ACSM guidelines for enhancing and maintaining CV fitness (D. Riebe, et al., 2018). These can appropriately function to enhance measures of CRF when performed for at least 3 days/week for 30 minutes and induce favourable changes in CV risk profile (Blacher, 1999; Girerd, 1991; Hamilton et al., 2007; McEniery, 2006; Naka, 2003; Vlachopoulos et al., 2010). The popularity of group fitness classes with the general public has been demonstrated for more than two decades and is most likely due to the social and non-competitive class environment. A broad range of group fitness classes are now available in both commercial and community fitness center that incorporate a wide range of exercise modalities (e.g., stationary cycling, step classes, group resistance exercise, pilates and aerobic dance) using a combination of music and instructor choreographed routines and designed to accommodate varying fitness levels. However, despite the high participation rates of group fitness classes, little is known on the acute effects of different group fitness classes on indices of AS; therefore, the aim of the present study was to compare the changes in local and regional indices of AS to three different group fitness classes (BIKE, PUMP and GT), as prescribed for health (D. Riebe, et al., 2018) and commercially available.

Methods

Participants

Twelve apparently healthy adults (7 males and 5 females) were recruited to participate in this study. Participants were aged 21 to 34 years old and were outwardly active as assessed by the International Physical Activity Questionnaire (IPAQ) with some experience in both aerobic and resistance exercise (~3–4 times/week, >3 months). All participants were healthy or perceived to be healthy based on the sport's medical examination or the preparticipation screening process, Physical Activity Readiness Questionnaire for Everyone (PARQ+). Exclusion criteria included any form of CV disease, more than one CV disease risk factor (Swain, American College of Sports, & American College of Sports, 2014), resting hypertension (SBP >140 mmHg, DBP > 90 mmHg), any prescription medication use, being an athlete and currently smoking. Prior to the first evaluation day, all participants provided written, informed consent in accordance with approval by the Ethical Committee of Faculty of Human Kinetics – University of Lisbon - 2019.

Study Design

The study was constructed as a randomized, cross-over, repeated-measures intervention. Participants attended 4, separated, intervention sessions consisting of an initial rest, a group fitness class of either BIKE, PUMP, GT, or no exercise, CON, and a recovery period. A minimum of 72 h between intervention sessions was accomplished. Body composition and CRF for each participant were evaluated prior to the 4 separated sessions. In each session, participants initially undertook 20-min of supine rest on a cushioned examination table with resting energy expenditure (REE) measured by indirect calorimetry (K5, Cosmed, Rome, Italy), followed by regional assessments of PWV and PWA of the carotid, brachial and femoral and distal arteries on the right side of the body using applanation tonometry (Complior 2.0, Alam Medical; Saint Quentin Fallavier, France), and local carotid AS indices analysis using an ultrasound (Arietta V60, Hitachi Aloka Medical Ltd, Mitaka-shi, Tokyo, Japan). In the time following, participants performed a 45-minute group fitness class of either BIKE, PUMP or GT, while activity energy expenditure (AEE) was continuously measured by indirect calorimetry (K5, Cosmed, Rome, Italy). These group fitness classes were characterized by distinct metabolic demands, representing the typical classes provided by gyms and health clubs to improve or maintain CV health. In CON, the participants remained quietly seated for 45-min, maintaining a good posture. Upon completion of each group fitness class,

participants returned immediately to the examination table and recovered in the supine position for 30-min while local and regional stiffness indices are re-evaluated at 10-, 20- and 30-min during recovery and compared to those at rest. Participants were blinded to the order of the experimental interventions until arrival at the laboratory. All sessions were conducted in the morning with each participant performing sessions at the same time of the day to minimize any potential diurnal variation. Participants were also instructed not to ingest any food or drink (except water) 4h before the sessions, and to avoid alcohol, caffeine, and exercise for at least 24h preceding each session.

Group Fitness Classes

The BIKE group fitness class consisted of a rhythmic indoor cycling class, with fluctuations in intensity set to changes in position, music rhythm, cadence, and revolutions per minute (Battista et al., 2008). Participants were advised to strictly adhere to the verbal cues from the instructor when told to adjust cycling cadence and resistance. PUMP intended to provide a comprehensive total-body weight-training program aiming to improve strength, muscular endurance, and general fitness. The class was choreographed to music and involved participants performed a combination of barbells, body-weight exercises, and free-weight plates. Participants selected weights based on the target muscle group for the specific song or track and their own personal goals. The GT class encompassed both aerobic and resistance components, combining athletic movements like running, lunging, and jumping with strength exercises such as barbells, body-weight exercises, and free-weight plates. All classes were 45 minutes long and contained 10-12 tracks or songs, each lasting from 4–7 minutes. They were led by the same fitness instructor, and all started with an opening warm-up, followed by eight tracks, each targeting specific muscle groups, finishing with a static stretch cool down. The choreography of the music plays an important role because it may modify the participant's motivation and the intensity of the exercise (Maher, Gottschall, & Conroy, 2015).

Measurements

Body Composition

Height and body weight were measured to the nearest 0.1 cm and nearest 0.1 kg, respectively, on a scale with an attached stadiometer (model 770, Seca; Hamburg, Deutschland). Body composition was also measured with a seca mBCA 515 using four pairs of electrodes (eight electrodes in total) positioned at each hand and foot. The 8-electrode technique enables segmental impedance measurement of the arms and legs.

By this means, impedance was measured with a current of 100 µA at frequencies between 1 and 1 000 kHz.

Cardiorespiratory Fitness

Maximal aerobic capacity was assessed using a cycle ergometry protocol. Each participant performed a ramp incremental cycle ergometer test to exhaustion on a calibrated electronically braked cycle ergometer (Monark 839 E, Ergomedic; Monark, Vansbro, Sweden) at a pedal cadence of 70 to 75 rev/min. The seat was adjusted so that the participant's legs could be at near full extension during each pedal revolution.

Inspired and expired gases were continuously analyzed, breath-by-breath, through by indirect calorimetry using a portable gas analyzer (K5, Cosmed, Rome, Italy), which was previously validated (Perez-Suarez et al., 2018). Before each test, the oxygen (O_2) and carbon dioxide (CO_2) analyzers were calibrated using ambient air and standard calibration gases of known concentration (16.7% O_2 and 5.7% CO_2). The calibration of the turbine flowmeter of the K5 was performed using a 3-L syringe (Quinton Instruments, Seattle, Wash., USA) according to the manufacturer's instructions. HR was continuously monitored by a chest strap monitor (Garmin, US). Data were evaluated in 10-second averages, and peak VO_2 defined as the highest 10-second value attained in the last minute of effort provided when 2 of the following criteria are met: (1) Attaining ~90% of age-predicted maximal HR; (2) Plateau in VO_2 with an increase in workload (<2.0 mL/kg/min); (3) Rating of perceived exertion ≥ 18 (6-20) and; (4) Respiratory exchange ratio ≥ 1.1; (5) subjective judgment by the observer that the participant could no longer continue, even after encouragement.

Energy Expenditure

For the resting EE subjects were instructed to report to the laboratory 60 min prior to each session and after 2-3 hour fast. After 10 min of quiet, seated rest in a dimly lit room, resting EE was measured for 15 min through the mentioned indirect calorimetry method at an environmental temperature and humidity of ±22ºC and 40–50%, respectively. Outputs of oxygen uptake, carbon dioxide production, respiratory exchange ratio, and ventilation were collected and averaged over 1-min intervals for data analysis. The first 5 min of data collection were discarded and the mean of a 5-min steady state interval between the 5^{th} and the 15^{th} min with respiratory exchange ratio between 0.7 and 1.0 was used to calculate resting EE. Steady state was defined as a 5-min period with ≤10% coefficient of variation for oxygen uptake and carbon dioxide production (Compher, Frankenfield, Keim, & Roth-Yousey, 2006). The mean oxygen uptake and carbon dioxide production of 5-min steady states were used in the equation of Weir (1949) and the period with the lowest resting EE was considered.

Oxygen uptake was measured throughout the session using the system described above, for total EE measurement. The evaluator marked the exact beginning of each phase to be distinguishable in offline analysis. The k5 is a compact device, easy to attach without constricting the patient's movements. The k5 weighs 475g and was not expected to significantly affect the energy demands of the participants, and none of them had any negative remarks on the system's weight or on their mobility and vision during the session.

Activity EE was calculated as the difference between total EE and the sum of resting EE with 0.1*total EE (assuming the thermic effect of food is ~10% of total EE) (Hills, Mokhtar, & Byrne, 2014). These estimates were summed throughout the entire duration of the session. Resting EE was also used to calculate the intensities in METs.

Aortic and Regional (Limb) Arterial Stiffness

AS as measured by PWV was obtained in all measurement rounds. Pressure waveforms from the carotid, femoral, radial, and distal arteries were captured in tandem with applanation tonometry (Complior 2.0, Alam Medical; Saint Quentin Fallavier, France). The distance between pulse sites were directly measured and entered into the Complior Analyse software. Sensors were positioned with specific holders (carotid and radial arteries) and held manually (femoral and distal posterial tibial arteries). When 10 carotid pulse waveforms of sufficient quality were obtained, simultaneous carotid and femoral, radial, and distal posterial tibial pressure curves were recorded for pulse waveforms. The time delay (aortic transit time) between two pulse waveforms was then automatically calculated. Right bBP was also measured and entered into the Complior Analyse software, and then signal acquisition was launched. Values obtained by the generation of the pressure waveforms at the assessed pulses, carotid to femoral (cf) artery, carotid to radial (cr) artery and carotid to distal posterial tibial (cd) artery were taken as indices of aortic, upper, and lower limb AS, respectively.

Carotid Arterial Stiffness Indices

The right common carotid artery was scanned with a Arietta V60 ultrasound machine (Hitachi Aloka Medical Ltd, Mitaka-shi, Tokyo, Japan) using a 7.5-MHz linear array probe incorporating a 5-MHz Doppler transducer in all measurement rounds. In longitudinal view, the probe was manipulated so that the intima of the artery was imaged clearly from both the anterior and posterior walls, and a single scan line was aligned perpendicularly to the vessel walls at a site 20 mm proximal to the carotid bulb. On-screen cursors were then placed on the anterior and posterior intima-media borders to enable tracking of both walls. The corresponding displacement waveforms and diameter curve were thus calculated using high-resolution online wall tracking ("E-track"

technology), with a sampling rate of 1 kHz. Arterial pressure waveforms were obtained automatically in real time by calibrating peak and bottom values with systolic and diastolic BP measured with sphygmomanometry, as previously validated (M. Sugawara, Niki, Furuhata, Ohnishi, & Suzuki, 2000). A pulse wave Doppler ultrasound beam was aligned to the vessel walls at the site of acquisition of the diameter waveform, to simultaneously acquire velocity data (Swampillai, Rakebrandt, Morris, Jones, & Fraser, 2006). Arterial diameter and velocity were recorded continuously for 20 s. After completing the acquisition, all data were displayed and any individual beats with noisy or unrepresentative waveforms were rejected; all other beats (typically about 20) were selected and signal-averaged to give single waveforms of diameter and velocity (Rakebrandt et al., 2009).

Common carotid diameter values and diameter-derived pressure data were used to calculate established indices of local AS, such as ε and β index, according to published algorithms (O'Rourke, Staessen, Vlachopoulos, Duprez, & Plante, 2002), in all measurement rounds.

$$\varepsilon = [(SBP - DBP)/(Ds - Dd)] \times Dd,$$

and

$$\beta = ln(SBP/DBP)/(Ds - Dd)/Dd,$$

where SBP and DBP are systolic and diastolic pressure, respectively, and Ds and Dd are the maximum and minimum arterial diameters measured by wall tracking of the intima-media borders of the carotid artery.

Central Blood Pressure Indices

Resting bSBP and resting bDBP were measured in the supine position using an automated oscillometric cuff (HEM-907 XL; Omron Corporation, Japan). Brachial BP was taken in duplicate and the average of the two values was recorded and used for subsequent analysis. PP was calculated as SBP – DBP. cSBP was assessed by applanation tonometry (Complior 2.0, Alam Medical; Saint Quentin Fallavier, France) in all measurement rounds, from right carotid traces acquired during the cf-PWV assessment. The waveforms were then averaged, and the mean values were extracted from a 15-s window of acquisition. The carotid waveforms are calibrated from MAP and brachial systolic blood pressure (bSBP), measured immediately before the acquisition. The Buckberg index and its included parameters, systolic and diastolic pressure-time indexes, were also extracted from the Complior Analysis Software.

Statistical Analysis

All data are reported as mean ± standard error. Based upon a medium effect size of 0.98 derived from published changes in cf-PWV within-between modes of exercise (Heffernan, Collier, et al., 2007), an a priori power analysis suggested that a number of 8 participants were required to detect significant differences between sessions (1-β = 80%) and a statistical significance was established as $p < .05$. Normality of distribution for variables was assessed qualitatively using histograms and Q-Q plots as well as quantitatively using the Shapiro-Wilk test, kurtosis, and skewness summary statistics. When distribution was found to be skewed, log transformations were performed to conform to normality. Comparisons between sessions (BIKE, PUMP, GT, and CON) over time (rest vs. post-intervention time points) for local and regional changes in AS, PWV and PWA variables were examined via two-way (session × time), repeated measures analysis of variance (ANOVA). Adjustments were performed when interactions with possible confounders (sex, brachial arterial pressure, mean AEE) were significant. Differences in physiological demands between sessions were analyzed via one-way repeated measures ANOVA. Post-hoc comparisons were performed via Bonferroni's tests. Intra and inter-day relative reliability was assessed with a two-way absolute agreement mixed model intraclass correlation coefficient (ICC (2,1)) computed with IBM SPSS Statistics Software (Version 25), using four static repeated measures. The ICC was interpreted as: poor [< 0.50], moderate [0.50, 0.75], good [0.75, 0.90], and excellent [> 0.90] (Koo & Li, 2016).

Results

Participant's demographic and CRF characteristics were as follows (mean ± SE): age 24.7±1.0 years, body mass index 23.0±0.5 kg, body fat 19.5±1.8%, resting HR 62.1±2.4 bpm, HRmax 195.3±1.0 bpm and VO_2max 27.3±1.6 ml/kg/min.

Regarding the group fitness classes, significant differences were observed for Activity and Total EE (kcal), VO_2max (%), and time spent in moderate and vigorous intensities (min) with values significantly greater for BIKE compared to PUMP and GT fitness classes (**Table 1**).

Table 1

Energy expenditure and intensity achieved during the 3 group fitness classes

	BIKE	PUMP	GT
Resting Energy Expenditure (Kcal)	50.9±2.0	49.8±2.5	52.2±2.2
Activity Energy Expenditure (Kcal)	337.9±30.0	193.5±13.2*	214.5±15.6*

Total Energy Expenditure (Kcal)	432.0±32.9	270.3±16.0*	296.4±17.6*†
HR (bpm)	143.0±3.3	130.0±5.6	127.6±5.3
HR Reserve (%)	67.5±3.2	58.3±3.9*	55.6±3.8*†
Activity METs	8.7±0.9	5.4±0.3*	5.8±0.4*
VO_2Max (%)	67.4±3.2	44.2±2.5*	48.9±3.2*†
Moderate Intensity (min)	11.8±2.5	33.4±3.3*	25.4±2.2*
Vigorous Intensity (min)	32.0±2.1	11.4±3.1*	16.8±2.1*

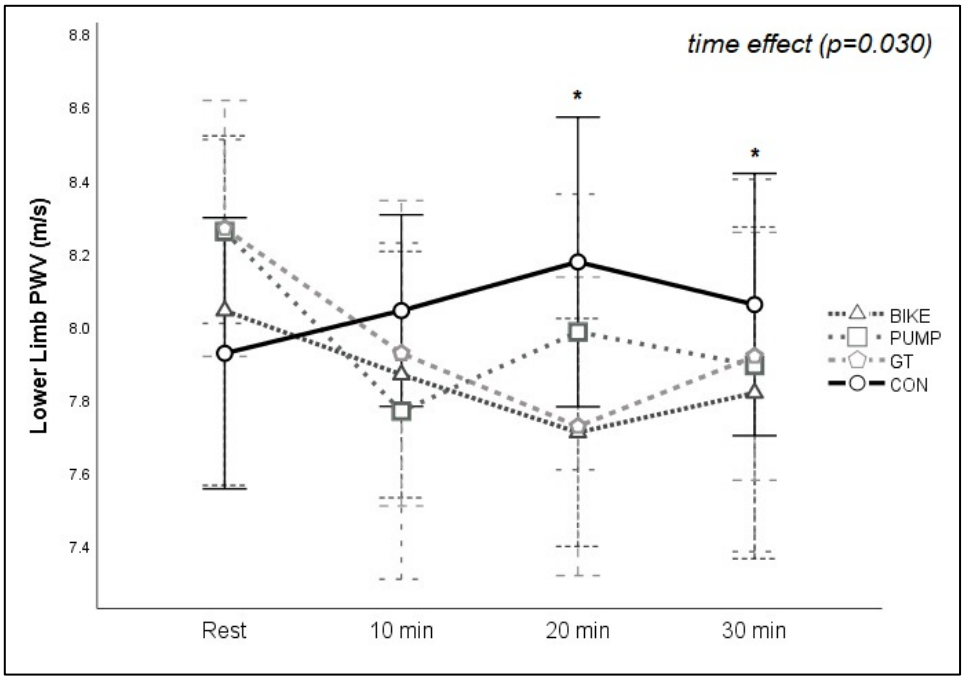
Data are expressed as mean ± SE. *indicates significant difference from BIKE; †indicates significant difference from PUMP

Aortic and Regional (Limb) Arterial Stiffness

Intra- and inter-day reliability was assessed for cf-PWV (ICC: 0.85; 95% CI: 0.64 to 0.95 and ICC: 0.59; 95% CI: -0.00 to 0.87), cr-PWV (ICC: 0.81; 95% CI: 0.56 to 0.94 and ICC: 0.64; 95% CI: 0.13 to 0.89) and cd-PWV (ICC: 0.63; 95% CI: 0.08 to 0.88 and ICC: 0.47; 95% CI: -0.26 to 0.83). No significant differences were identified for cf-PWV and cr-PWV, although a main time effect was found in cd-PWV (p=0.030; η2=0.235) in which an immediate (10min) decrease following the three group fitness classes was observed (d_{BIKE}=-0.3 m/s; d_{PUMP}=-0.5 m/s; d_{GT}=-0.4 m/s) (**Figure 2**). Values did not return to baseline within the analysed 30 minutes of recovery.

Figure 2

Time-course Changes of PWV in the Lower Segments



Data are expressed as mean ± SE. Legend: *indicates significant difference from Post 10

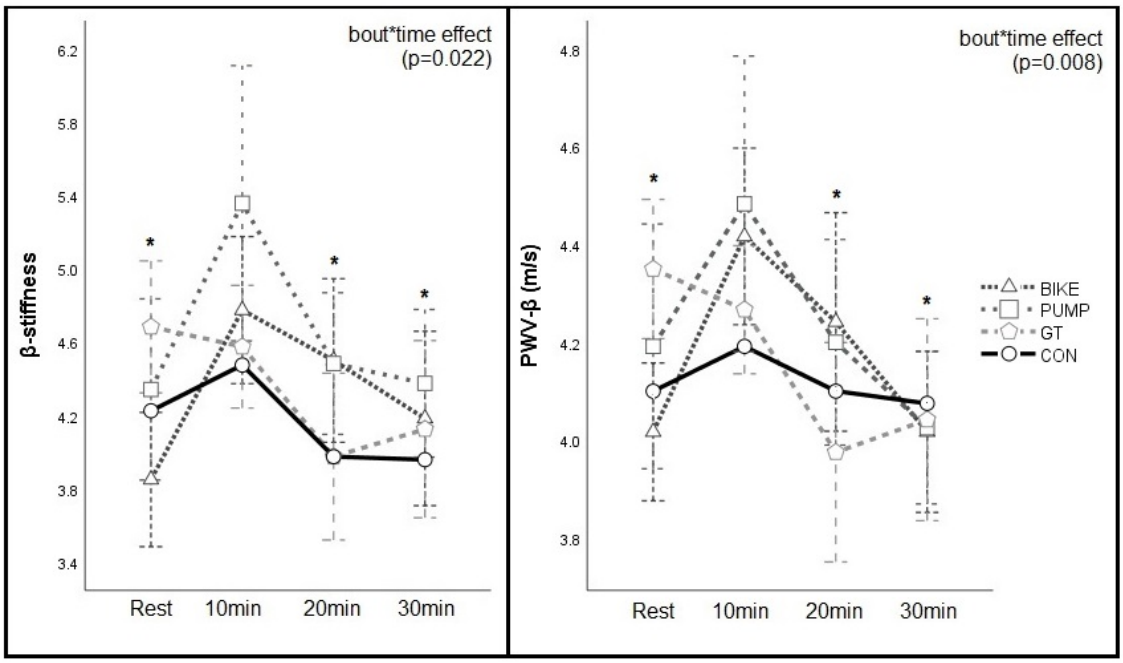
Carotid Arterial Stiffness Indices

Intra-day and inter-day reliability were assessed for β (ICC: 0.67; 95% CI: 0.24 to 0.89 and ICC: 0.65; 95% CI: 0.22 to 0.88), ε (ICC: 0.86; 95% CI: 0.66 to 0.95 and ICC: 0.73; 95% CI: 0.37 to 0.91) and PWV beta (ICC: 0.89; 95% CI: 0.74 to 0.96 and ICC: 0.55; 95% CI: 0.01 to 0.85).

A bout-by-time interaction was detected for β (p=0.001; η2=0.377), ε (p<0.001; η2=0.462), and PWV-β (p<0.001; η2=0.457) due to a significant increase immediately following BIKE (d_{β}=0.9; d_{ε}=9.3 kPa; $d_{PWV\text{-}\beta}$=0.4 m/s) and PP (d_{β}=0.9; d_{ε}=10.3 kPa; $d_{PWV\text{-}\beta}$=0.5 m/s) group fitness classes compared to GT. As seen in **Figure 3**, these values returned to baseline 30 min into recovery. Significant interactions were found with brachial arterial pressure (p=0.047; η2=0.711) and mean AEE (p=0.034; η2=0.642), but not with sex (p=0.054; η2=0.595). Increases observed immediately following the BIKE group fitness class remained significant when adjusted for brachial arterial pressure, but not when adjusted for mean AEE.

Figure 3

Time-course Changes of Carotid Stiffness Indices



Data are expressed as mean ± SE. Legend: *indicates significant difference from 10 min post exercise

Central Blood Pressure Indices

bSBP (p<0.001; η2=0.458), brachial pulse pressure (bPP) (p=0.001; η2=0.397) and maximal rate of rise of left ventricular pressure (dP/dt-max) (p=0.042; η2=0.218) were immediately increased after the effort performed during the PUMP class, reaching

a peak at 10 minutes (d_{bSBP}=5.2 mmHg; d_{bPP}=7.0 mmHg; $d_{dP/dt\text{-}max}$=190.0), and returning to baseline values after 20 min into recovery **(Table 2)**.

Table 2

Acute Effects of Group Fitness Classes on Central Blood Pressure Indices

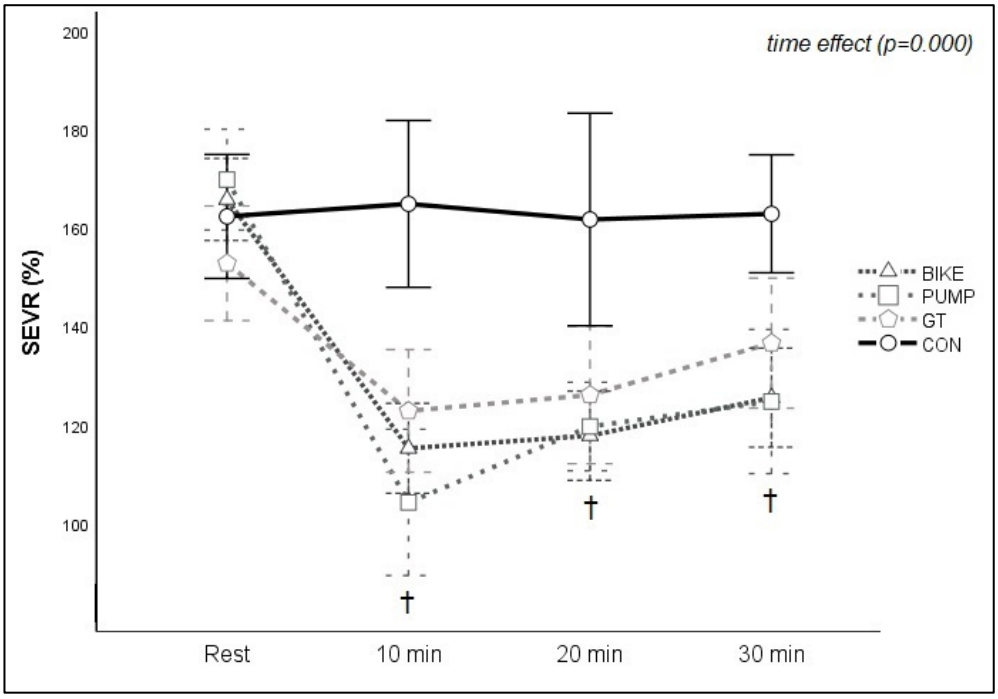
		Rest	10min	20min	30min	Main effect of time (p-value; partial eta square)
bSBP						(*p*<0.001; η2=0.458)
(mmHg)	**BIKE**	113.1±2.6	113.5±3.2	110.5±3.2	109.8±2.3	-
	PUMP	111.8±2.6	116.0±2.7	110.8±2.2*	109.3±2.2*	10>20,30
	GT	114.6±3.5	114.9±3.5	111.2±3.6	111.0±3.0	-
	CON	111.3±2.9	110.8±2.4	113.2±2.2	111.1±2.8	-
bPP						(*p*=0.001; η2=0.397)
(mmHg)	**BIKE**	46.3±3.4	49.3±3.7	47.0±3.6	47.3±3.7	-
	PUMP	47.1±3.1*	55.2±3.0	48.1±2.7*	50.0±2.7	Rest<10>20
	GT	50.3±3.5	51.7±3.4	48.7±3.2	47.5±3.2	-
	CON	47.3±3.7	48.6±2.6	45.9±3.3	45.0±2.8	-
dP/dt_max						(*p*=0.042; η2=0.218)
	BIKE	766.7±92.5	763.3±53.3	728.3±50.8	681.7±61.8	-
	PUMP	720.0±51.7*	910.0±63.0	762.5±63.1	820.8±71.5*	Rest<10>30
	GT	824.2±114.3	858.3±68.7	764.2±62.1	782.5±68.2	-
	CON	762.5±80.7	767.5±70.7	758.3±75.1	733.3±72.8	-

Data are expressed as mean ± SE.*indicates significantly different from 10 min post; bSBP – brachial systolic blood pressure; bPP – brachial pulse pressures; dP/dt-max – maximal rate of rise of left ventricular pressure.

Significant main time effects were found in SEVR (p<0.001; η2=0.895), an index of availability and myocardial oxygen demand, with values decreased at 10 minutes post group fitness classes (d_{SEVR}=-50.5) followed by an increase, nearly returning to baseline values. As seen in **Figure 4**, the SEVR remained low even after 30 minutes of recovery. No significant interactions with sex were found for cBP indices. When adjusted for EE, there were no significant effects identified for these indices.

Figure 4

Time-course Changes of Subendocardial Viability Ratio



Data are expressed as mean ± SE. Legend: † indicates significant difference from Rest.

Discussion

To the best of our knowledge, this was the first study to directly compare the effects of three different fitness classes on indices of local and regional AS and PWA. We found that the effect of commercially available fitness classes on indices of AS was dependent on the mode of exercise, the anatomical segment assessed, and on the timing of the measurement. β, Ep and PWV-β increased immediately (<10 min) following aerobic (BIKE) and resistance (PUMP) exercise sessions, whereas regional cd-PWV, but not cf-PWV or cr-PWV, decreased immediately following aerobic, resistance or combined exercise sessions. Values returned to baseline 30 minutes into recovery.

Reliability

The analysis of inter-day reliability in this study design is crucial to distinguish changes caused by the stimulus applied from those of daily variation or measurement error. High inter-day reliability has been reported for the cf-PWV and cd-PWV (ICC: 0.920 and 0.913, respectively; $P < 0.001$ for both), and moderate inter-day reliability has been reported for cr-PWV (ICC: 0.598, $P = 0.03$) (Miyatani et al., 2012). Although our study presents a good inter-day reliability for cf-PWV and cr-PWV (ICC: 0.59; 95% CI: -0.00 to 0.87 and ICC: 0.64; 95% CI: 0.13 to 0.89), inter-day reliability of cd-PWV was poor (ICC: 0.47; 95% CI: -0.26 to 0.83). As resting PWV values were similar to those reported by others, although using different methods and segments (R. Asmar et al., 1995; Avolio et

al., 1983; Bertovic et al., 1999; B. A. B. Kingwell, K. L.; Cameron, J. D.; Jennings, G. L.; Dart, A. M., 1997), differences might be due to the number of measures included to estimate reliability. In fact, when inter-day reliability was assessed with the best of two measurements as in others, we obtained comparable inter-day reliability values of the cf-PWV and cd-PWV (ICC values: 0.945 and 0.950 respectively; $p< 0.001$ for both) and better inter-day reliability for cr-PWV (ICC: 0.986; $p<0.001$).

Arterial Stiffness

cf-PWV has been reported to be increased immediately following (2-10 min) acute aerobic exercise (Campbell, Fisher, Sharman, McDonnell, & Frenneaux, 2011; R. J. Doonan, Mutter, Egiziano, Gomez, & Daskalopoulou, 2013; Robert J. Doonan et al., 2011; Hull et al., 2011), although divergent results were also published (Gkaliagkousi, Gavriilaki, Nikolaidou, Triantafyllou, & Douma, 2014; Milatz, Ketelhut, Ketelhut, & Ketelhut, 2015; Munir et al., 2008). Given that the characteristics of the study population were similar (healthy adults aged between 18-65 years) and that the PWV measurements were taken in the same segment (carotid to femoral arteries), the conflicting findings could be attributed to: 1) the intensity of the cycle ergometer exercise protocol, and/or 2) the different time points in which measurements were taken after exercise cessation (2-10 min). Regional AS is dependent on the anatomical segment being assessed following acute aerobic exercise (Mutter et al., 2016). Several studies in healthy young cohorts reported a significant decrease in peripheral AS measured in the lower limb segments (proximal to the primary working muscles in most instances) immediately following (0–10 min) acute moderate intensity (B. A. Kingwell et al., 1997), high-intensity (Melo et al., 2016; Naka, 2003; Ranadive et al., 2012), accumulated (Kobayashi, Hatakeyama, Hashimoto, & Okamoto, 2018), and other modalities of aerobic exercise (Rakobowchuk, Stuckey, Millar, Gurr, & Macdonald, 2009; Tordi, Mourot, Colin, & Regnard, 2010). Our results demonstrate a decrease in lower limb PWV, within the first 10 min following the three fitness classes. It is likely that post-exercise lower limb AS reflects two concomitant vessel characteristics: vessel dilation due to increased shear stress, which theoretically reduces AS in the lower limbs via an increased vessel diameter, and a decrease in vasomotor tone (Wagenseil & Mecham, 2009). After acute aerobic exercise, there is a decrease in muscle sympathetic nerve function (Halliwill, Taylor, & Eckberg, 1996). Carotid and aortic baroreceptors transmit afferent signals to the brainstem in response to stretching, which serve to suppress sympathetic activity to the periphery (Joyner, 2000), resulting in vessel vasodilation and related changes in AS. Vasodilation has previously been shown to occur alongside sympathetic stimulation, so it is conceivable that reductions in AS occur concurrently with

sympatho-excitation (Reed et al., 2000). Aiming to clarify the possible mechanisms here inherent, a complementary analysis of indices of autonomic nervous system should be further addressed.

AS can also be locally assessed (Segers, Rietzschel Ernst, & Chirinos Julio, 2020). Hu et al. (2013) showed that carotid ε and β were increased in healthy young subjects following an acute bout of aerobic exercise. This finding was later confirmed by Liu et al. (2015) following a series of four 30-second bouts of cycling exercise in basketball players (Liu, Yuan, Qin, & Hou, 2015). Consistent with these findings, carotid β, ε, and PWV- β in the present study remained increased immediately following (10 min) the aerobic fitness class (BIKE). The discrepant findings between regional and local AS immediately following the acute aerobic fitness class (BIKE) highlight the significant structural differences between the central dampening and peripheral conduit arteries, such as wall thickness, percentage of muscular and elastic components, magnitude of distensibility, and sympathetic control dependence (Laurent et al., 2006; McGrath et al., 2005; O'Rourke & Hashimoto, 2007; Tennant, 1990). While both the carotid artery and the abdominal aorta are elastic arteries, the carotid artery has a more abdominal aorta-like ultrastructure than the ascending aorta. Evidence suggests that increased stiffness in the common carotid artery compared to the abdominal aorta at a young age indicates a difference in arterial wall mechanics in different vascular beds (Länne, Hansen, Mangell, & Sonesson, 1994). Moreover, the abdominal aorta appears to dilate more compared to the common carotid artery in adults (27% vs 17%, respectively) (Länne et al., 1994). In addition, the elastin/collagen ratio, which progressively decreases towards the periphery, defines the elasticity of the broad arteries (Nichols & Singh, 2002), which changes the tone of the peripheral arteries, allowing them to change the pace at which the pressure wave moves along their length (Gkaliagkousi & Douma, 2009).

Unlike aerobic exercises, the effects of muscle strengthening activities on AS remain controversial. Some studies suggest that aortic AS increases immediately following resistance exercise (Grigoriadis et al., 2020; Kingsley et al., 2016; Kingsley et al., 2017; Lefferts et al., 2015; Mak & Lai, 2015; Nico Nitzsche, 2016; Yoon et al., 2010), although this is not a universal finding (Heffernan, Rossow, et al., 2006; Okamoto et al., 2014; Thiebaud et al., 2016). Our findings are in line with those by Heffernan, Rossow, et al. (2006) and Thiebaud et al. (2016) as aortic AS remained unchanged immediately following the resistance fitness class (PUMP). The discrepancies between the acute effects of resistance exercise on aortic AS may be due to the participants characteristics and/or variations in exercise intensity (Lefferts et al., 2015; Okamoto et al., 2014), total exercise volume (Nico Nitzsche, 2016), or shorter rest periods between sets (Yoon et al., 2010). Shorter rest periods can prevent BP from returning to baseline, resulting in a

greater increase in BP during subsequent exercise and thereby amplifying aortic stiffness responses post-exercise (Lim et al., 2015). Following the resistance fitness class (PUMP), carotid β, ε, and PWV beta, were immediately increased. To our knowledge, the acute effects of resistance exercise on indices of local AS have not been studied. However, five studies (Kawano, Tanaka, & Miyachi, 2006; M. Miyachi et al., 2004; Okamoto, Masuhara, & Ikuta, 2006, 2009a, 2009b) suggested that the chronic (8 weeks - 4 months) effects of resistance training in young subjects (n=115) were associated with an 14.3% (95% CI 8.5% to 20.1%; I2, 71%; heterogeneity, p<0.001) increase in β index compared to controls. Therefore, high-quality studies on the effects of acute resistance exercise on local indices of AS are needed. Although the mechanisms underlying the increase in local AS after resistance exercise remains unknown, there are some possible explanations (Naka, 2003; Sharman et al., 2005). BP has a direct effect on AS and there is a recognized pressure-dependence of local AS measures (Zieff et al., 2019). In the present study, the SBP and PP significantly increased following the resistance fitness class (PUMP), exhibiting the same tendency as indices of local stiffness. Studies have shown that acute rises in BP during high-intensity resistance exercise induce structural and functional changes in the arterial and ventricular walls, as well as persistent changes in resting BP (MacDougall, Tuxen, Sale, Moroz, & Sutton, 1985). With a rise in distension pressure, there is a change in the type of fibers that sustain vessel-wall stress: load bearing is transposed from more compliant elastin fibers to stiffer collagen fibers, passively increasing local AS (Rossow, Fahs, et al., 2010; Rossow, Yan, et al., 2010). Moreover, elevated arterial pressure alone shifts the pressure load burden within the artery wall from elastin to collagen, directly increasing AS (Smulyan, Mookherjee, & Safar, 2016).

Although combined exercise training is the recommended exercise modality for maintaining muscular strength and endurance, and flexibility of the major muscle groups (Michael L. Pollock et al., 1998), the acute effects of this exercise modality on local and regional AS remain scarce. A recent abstract presented in the ACSM Medicine & Science in Sports & Exercise evaluated the acute effects of an integrated concurrent exercise on BP and AS suggesting similar acute CV responses result from integrated concurrent exercise compared to aerobic and resistance exercise (Spicer et al., 2019). Nevertheless, evidence from short-term effects (1 to 24 weeks) suggest that cf-PWV remains unchanged (ES=-0.09, CI: -0.32 to 0.13, P=0.40) (Cortez-Cooper et al., 2008; Guimarães et al., 2010; Kawano et al., 2006; Stewart et al., 2005).

In general, our results bring ecological validity to the study of the acute effects of exercise (aerobic, resistance and/or combined) (B.C., 2013; S. G. Dasilva et al., 2011), since they have so far focused on exercises rather on fitness classes, to represent typical

sessions in accordance with current guidelines for improvement and maintenance of CV health (D. Riebe, Ehrman, Liguori, Magal, & American College of Sports Medicine, 2018).

Pulse Wave Analysis

PWA is a highly reproducible and simple to use technique that, when combined with ECG-gated PWV assessment, provides useful information about AS (Wilkinson, Cockcroft, & Webb, 1998). The use of PWA to assess AS can allow for a more accurate risk assessment and care of those who are most in need (Wilkinson et al., 1998). However, research on the after-effects of different exercise modes (i.e., aerobic vs. resistance exercise) on PWA and AS are scarce and produced unconclusive findings (Pierce, Doma, & Leicht, 2018; Pierce, Doma, Raiff, et al., 2018). Accumulated evidence acknowledges that exercise training can help to reduce AS, reducing left ventricular afterload during systole, increasing diastolic myocardial perfusion, (Dischl et al., 2011) and reducing the risk of CV events (Heffernan, Jae, Echols, Lepine, & Fernhall, 2007; Munir et al., 2008). In this respect, SEVR, a measure of myocardial perfusion, has been gaining interest as it has been shown to be independently associated with CV events (Tsiachris et al., 2012). The small number of studies that addressed cBP indices on PWA suggest a decreased myocardial perfusion following an acute bout of aerobic exercise (Robert J. Doonan et al., 2011; Radhakrishnan, Swaminathan, Pereira, Henderson, & Brodie, 2017), and following acute resistance exercise for at least 10-20 min (Kingsley et al., 2017; Parks, Marshall, Tai, & Kingsley, 2020). The decreases in SEVR in the present study were likely associated with an increased EE during each fitness class, by the influence of augmented HR (Yahui et al., 2017). The elevated HR throughout an incremental exercise causes the myocardium to have a high energy demand. During recovery, Q will decrease, and SEVR will adjust as a result of the reduced diastolic time caused by the elevated HR and high energy demands (Yahui et al., 2017). The myocardial perfusion occurs primarily during diastole because compression of the vasculature limits coronary blood flow during systole (Fokkema et al., 2005).

Limitations

It is possible that only monitoring post-intervention AS responses every 10 minutes for the first 30 minutes resulted in a failure to detect subtle changes or those that occurred outside of this initial timeframe (e.g., >30min – 72 h). Furthermore, due to space constraints of measures, we were unable to capture measures immediately, i.e. 0–5 minutes following exercise. All the participants were recreationally active, so these healthy responses can vary from those seen in older, sedentary, and/or clinical populations, which needs to be further investigated. Finally, the menstrual cycle variation

in adult female subjects was not regulated. While it has been stated that the elastic properties of central arteries do not fluctuate significantly with the phases of the menstrual cycle (Robb et al., 2009; Willekes, Hoogland, Keizer, Hoeks, & Reneman, 1997; Williams et al., 2001), this is not recognized as a universal finding (Hayashi et al., 2006). Moreover, previous studies have found sex variations in both resting cf-PWV and after acute aerobic exercise (Baldo et al., 2018; R. J. Doonan et al., 2013; Lane et al., 2013; Nieman, Dew, & Krasen, 2013) with menstrual cycle phase potentially affecting cf-PWV values (M & Ta, 2014). In our sample, both male and female participants had identical PWV values at rest and at recovery ($p>0.05$; data not shown), implying that there were no sex variations in the arterial response to acute aerobic, resistance, and combined fitness classes. Previous reports suggesting that men and women have similar arterial responses to acute perturbations (Heffernan, Jae, Edwards, et al., 2007; Heffernan, Rossow, et al., 2006; Ranadive et al., 2012) support our findings.

Conclusions

The present study demonstrated that the effect of commercially available fitness classes on indices of AS were dependent on the anatomical segment assessed, mode of exercise, and on the timing of the measurement. We found that 1) local AS immediately increased following acute aerobic and acute resistance fitness classes but not following acute combined fitness class, with values reaching its peak at 10min post-exercise, returning to baseline 30 min into recovery, and 2) regional AS immediately decreased following the three acute group fitness classes in the lower segments, but not in the upper and aortic segments.

Acute Effects of Exercise Mode on Cardiac Autonomic Function in Healthy Young Adults

Abstract

Participation in group fitness classes is an increasingly common method by which subjects may fulfil the criteria set down in the ACSM guidelines for enhancing and maintaining CV fitness. However, little is known on the acute effects of different group fitness classes on cardiovagal modulation and BRS. The aim of the present study was to acutely compare the time-course changes in cardiovagal modulation and BRS to different fitness classes (BIKE, PUMP and GT). Twelve participants (7 male and 5 females, mean (SE) = 25 (4) years) attended 4, separated, group fitness classes of BIKE, PUMP, GT, or no exercise, CON. Testing took place during 20 minutes before and after each group fitness class at 10-, 20- and 30-min. In each session, participants initially undertook 20-min of supine rest on a cushioned examination table with REE measured by indirect calorimetry (K5, Cosmed, Rome, Italy), followed by the assessment of HRV indices and BRS. Resting and post exercise measures included BP followed by assessment of HRV indices and BRS. A significant decrease was found for Ln-RMSSD and Ln-HF following BIKE (dLn-HF=-1.9 ms^2; dLnRMSSD=-0.9 ms), PUMP (dLn-HF=-1.7 ms^2; dLnRMSSD=-0.9 ms) and GT (dLn-HF=-1.5 ms^2; dLnRMSSD=-0.7 ms). LF/HF ratio was only significantly increased following GT (d=1.81). BRS was reduced during the entire recovery period following the three group fitness classes. Baroreflex Effectiveness Index significantly decrease immediately following the three group fitness classes (d_{BIKE}=-15.6 %; d_{PUMP}=-13.6 %; d_{GT}=-16.4 %), with values returning to baseline following BIKE and PUMP but not GT. In conclusion, cardiovagal modulation indices respond similarly to different exercise modalities as commercially available in health clubs, although sympathovagal balance appears to be altered following combined exercise. These findings suggest that previous laboratorial findings seem to have low ecological validity, in a way that findings cannot be easily generalized to fitness class settings.

Keywords: Acute exercise · Fitness classes · Cardiovagal Modulation

Introduction

HRV and BRS are recognized indexes of cardiovagal modulation, in research, as well as applied settings of health, disease and sports performance (Billman, 2011; Dong, 2016; "Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology," 1996; La Rovere, Pinna, & Raczak, 2008).

Even though HRV was primarily used to predict sudden cardiac death and diabetic neuropathies in measuring disease development (Khandoker, Jelinek, & Palaniswami, 2009; Tereshchenko et al., 2012), there has been an increasing interest in monitoring HRV during exercise, particularly during post-exercise recovery, demonstrating HRV's use in exercise training. Furthermore, HRV has been used to efficiently change the day-to-day prescription of exercise training in moderately fit participants, resulting in larger improvements in CRF (Kiviniemi et al., 2010; Kiviniemi, Hautala, Kinnunen, & Tulppo, 2007).

HRV and BRS have been studied following acute aerobic (Kaikkonen, Rusko, & Martinmäki, 2008; Michael, Jay, Graham, & Davis, 2017; Michael, Jay, Halaki, Graham, & Davis, 2016; Niemelä et al., 2008), resistance/strength exercise (Cunha, Midgley, Soares, & Farinatti, 2015; Michael et al., 2016; S. Park, Rink, & Wallace, 2008), or aerobic vs resistance (Heffernan, Collier, et al., 2007; Heffernan, Kelly, Collier, & Fernhall, 2006) in healthy subjects. Following these data, modality and intensity have been suggested as major determinants of post-exercise changes in cardiovagal modulation. A more rapid recovery of RMSSD (Cunha, Midgley, Goncalves, et al., 2015), and larger reductions in BRS within 60-min of recovery (Cunha, Midgley, Soares, et al., 2015) were found following the exercise modality involving a smaller muscle mass or EE (i.e., cycling > walking > running). Increased exercise intensity was associated with delayed recovery of temporal and spectral HRV measurements, notably RMSSD and HF, respectively (Kaikkonen et al., 2008; Michael, Jay, et al., 2017; Michael et al., 2016; Niemelä et al., 2008; Niewiadomski, Gąsiorowska, Krauss, Mróz, & Cybulski, 2007; Seiler, Haugen, & Kuffel, 2007).

Overall, these studies have been performed in controlled exercise conditions, focusing on exercises, rather than fitness classes, neglecting the representation of typical sessions in accordance with current guidelines for improvement and maintenance of CV health (D. Riebe, et al., 2018). An obvious disadvantage rests on their reduced ecological validity (S. G. Dasilva, Guidetti, L., Buzzachera, Cosme F., Elsangedy, Hassan M., Krinski, K., De Campos, W., Goss, Fredric L., Baldari, C., 2011; Unick et al., 2012). The rising popularity and vast participation in group fitness classes have allowed subjects not only to fulfil with the ACSM guidelines for enhancing and maintaining CRF (D. Riebe, et al., 2018) but also to experience a rewarding social and non-competitive class environment. A broad range of group fitness classes are now available in both commercial and community fitness center that incorporate a wide range of exercise modalities (e.g., stationary cycling, step classes, group resistance exercise, pilates, and aerobic dance) using a combination of music and instructor choreographed routines and designed to accommodate varying fitness levels. However, no available data exists that

describes and/or compares the cardiovagal modulation following different group fitness classes. Therefore, the aim of the present study was to compare the time-course of post-exercise cardiovagal modulation to different fitness classes (BIKE, PUMP and GT), as prescribed for health and commercially available.

Methods

Participants

Twelve apparently healthy adults (7 males and 5 females) were recruited to participate in this study. Participants were aged 21 to 34 years old and were outwardly active as assessed by the International Physical Activity Questionnaire (IPAQ) with some experience in both aerobic and resistance exercise (~3–4 times/week, >3 months). All participants were healthy or perceived to be healthy based on the sport's medical examination or the preparticipation screening process, Physical Activity Readiness Questionnaire for Everyone (PARQ+). Exclusion criteria included any form of CV disease, more than one CV disease risk factor (Swain et al., 2014), resting hypertension (SBP >140 mmHg, DBP > 90 mmHg), any prescription medication use, being an athlete and currently smoking. Prior to the first evaluation day, all participants provided written, informed consent in accordance with approval by the Ethical Committee of Faculty of Human Kinetics – University of Lisbon (2019).

Study Design

The study was constructed as a randomized, cross-over, repeated-measures intervention. Participants attended 4, separate, intervention sessions consisting of an initial rest, a group fitness class of BIKE, PUMP, GT, or no exercise, CON, and a recovery period. A minimum of 72 h between intervention sessions was accomplished. Body composition and CRF for each participant were evaluated prior to the 4 separated sessions. In each session, participants initially undertook 20-min of supine rest on a cushioned examination table with REE measured by indirect calorimetry (K5, Cosmed, Rome, Italy), followed by the assessment of HRV indices and BRS. Group fitness classes consisted of a 45-minute of either BIKE, PUMP, or GT. In CON, the participants remained quietly seated for 45-min, maintaining a good posture. Upon completion of each group fitness class, participants returned immediately to the examination table and recovered in the supine position for 30-min while HRV and BRS indices were re-evaluated at 10-, 20- and 30-min during recovery and compared to those at rest. Participants were blinded to the order of the experimental interventions until arrival at the laboratory. All sessions were conducted in the morning with each participant performing sessions at the same

time of the day to minimize any potential diurnal variation. Participants were also instructed not to ingest any food or drink (except water) 4h before the sessions, and to avoid alcohol, caffeine, and exercise for at least 24h preceding each session.

Group Fitness Classes

The BIKE group fitness class consisted of a rhythmic indoor cycling class, with fluctuations in intensity set to changes in position, music rhythm, cadence, and revolutions per minute (Battista et al., 2008). Participants were advised to strictly adhere to the verbal cues from the instructor when told to adjust cycling cadence and resistance. PUMP intended to provide a comprehensive total-body weight-training program aiming to improve strength, muscular endurance, and general fitness. The class was choreographed to music and involved participants performed a combination of barbells, body-weight exercises, and free-weight plates. Participants selected weights based on the target muscle group for the specific song or track and their own personal goals. The GT class encompassed both aerobic and resistance components, combining athletic movements like running, lunging, and jumping with strength exercises such as barbells, body-weight exercises, and free-weight plates. All classes were 45 minutes long and contained 10-12 tracks or songs, each lasting from 4–7 minutes. They were led by the same fitness instructor, and all started with an opening warm-up, followed by eight tracks, each targeting specific muscle groups, finishing with a static stretch cool down. The choreography of the music plays an important role because it may modify the participant's motivation and the intensity of the exercise (Maher et al., 2015).

Measurements

Body Composition

Height and body weight were measured to the nearest 0.1 cm and nearest 0.1 kg, respectively, on a scale with an attached stadiometer (model 770, Seca; Hamburg, Deutschland). Body composition was also measured with a seca mBCA 515 using four pairs of electrodes (eight electrodes in total) positioned at each hand and foot. The 8-electrode technique enables segmental impedance measurement of the arms and legs. By this means, impedance was measured with a current of 100 μA at frequencies between 1 and 1 000 kHz.

Cardiorespiratory Fitness

Maximal aerobic capacity was assessed using a cycle ergometry protocol. Each participant performed a ramp incremental cycle ergometer test to exhaustion on a calibrated electronically braked cycle ergometer (Monark 839 E, Ergomedic; Monark,

Vansbro, Sweden) at a pedal cadence of 70 to 75 rev/min. The seat was adjusted so that the participant's legs could be at near full extension during each pedal revolution.

Inspired and expired gases were continuously analyzed, breath-by-breath, through a portable gas analyzer (K5, Cosmed, Rome, Italy). Before each test, the oxygen (O_2) and carbon dioxide (CO_2) analyzers were calibrated using ambient air and standard calibration gases of known concentration (16.7% O_2 and 5.7% CO_2). The calibration of the turbine flowmeter of the K5 was performed using a 3-L syringe (Quinton Instruments, Seattle, Wash., USA) according to the manufacturer's instructions. HR was continuously monitored by a chest strap monitor (Garmin, US). Data were evaluated in 10-second averages, and peak VO_2 defined as the highest 10-second value attained in the last minute of effort provided when 2 of the following criteria are met: (1) Attaining ~90% of age-predicted maximal HR; (2) Plateau in VO_2 with an increase in workload (<2.0 mL/kg/min); (3) Rating of perceived exertion ≥ 18 (6-20) and; (4) Respiratory exchange ratio ≥ 1.1; (5) subjective judgment by the observer that the participant could no longer continue, even after encouragement.

Energy Expenditure

For the resting EE subjects were instructed to report to the laboratory 60 min prior to each session and after 2-3 hour fast. After 10 min of quiet, seated rest in a dimly lit room, resting EE was measured for 15 min through the mentioned indirect calorimetry method at an environmental temperature and humidity of ±22ºC and 40–50%, respectively. Outputs of oxygen uptake, carbon dioxide production, respiratory exchange ratio, and ventilation were collected and averaged over 1-min intervals for data analysis. The first 5 min of data collection were discarded and the mean of a 5-min steady state interval between the 5^{th} and the 15^{th} min with respiratory exchange ratio between 0.7 and 1.0 was used to calculate resting EE. Steady state was defined as a 5-min period with ≤10% coefficient of variation for oxygen uptake and carbon dioxide production (Compher et al., 2006). The mean oxygen uptake and carbon dioxide production of 5-min steady states were used in the equation of Weir (1949) and the period with the lowest resting EE was considered.

Oxygen uptake was measured throughout the session using the system described above, for total EE measurement. The evaluator marked the exact beginning of each phase to be distinguishable in offline analysis. The k5 is a compact device, easy to attach without constricting the patient's movements. The k5 weighs 475g and was not expected to significantly affect the energy demands of the participants, and none of them had any negative remarks on the system's weight or on their mobility and vision during the session.

Activity EE was calculated as the difference between total EE and the sum of resting EE with 0.1*total EE (assuming the thermic effect of food is ~10% of total EE) (Hills et al., 2014). These estimates were summed throughout the entire duration of the session. Resting EE was also used to calculate the intensities in METs.

Heart rate variability

RR intervals were derived from beat-to-beat ECG intervals using the Finapres 5 ECG lead module (Finapres® Nova, Ohmeda, Louisville, Colorado, USA) in all measurement rounds. All data acquisition and post-acquisition analyses were carried out in accordance with the Task Force of the European Society of Cardiology and North American Society of Pacing and Electrophysiology ("Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology," 1996).

During all repeated assessments, participants were assessed in a relaxed supine position in a calm climate-controlled environment (22-24°C). The frequency-domain HRV measures provide information primarily on vagal modulation with the low frequency power spectrum reflecting both sympathetic and parasympathetic modulation and HF as a surrogate marker of parasympathetic modulation of the RR intervals (Mendonca et al., 2010). The LF/HF ratio was used as an indicator of sympathovagal dominance (Pagani et al., 1986). The time-domain statistics used to characterize HRV were the SDNN – a measure of overall variability, and the RMSSD – a measure of vagal modulation, both in milliseconds.

Heart rate variability off-line analysis

All analyses of HRV were performed off-line using the FisioSinal software built-in Matlab (Tavares, Martins, Laranjo, & Rocha, 2011). Following RR peak detection and semi-automatic removal of signal artifacts, time-domain and spectral power analyses were conducted using 2-min time-bins (Wu, Shi, Yu, & Liu, 2020). Ectopic heartbeats were excluded from the final analysis. The time-frequency domain analysis was conducted using fast Fourier transform algorithm, one of the most commonly used approaches for HRV analysis, which allowed the estimation of LF (0.04 to 0.15 Hz) and HF bands (0.15 to 0.4 Hz) absolute and normalized powers, and as it suited best the characterization of autonomic nervous system's acute responses during the post-exercise phase (Kaisu Martinmäki & Heikki Rusko, 2008).

Intra- and inter-day relative reliability in our laboratory for linear time-domain (Ln-RMSSD; ICC: 0.80; 95% CI: 0.52 to 0.93 and ICC: 0.92; 95% CI: 0.82 to 0.98) and

spectral power analyses (Ln-HF; ICC: 0.82; 95% CI: 0.58 to 0.94 and ICC: 0.88; 95% CI: 0.72 to 0.93) are considered good to excellent.

Cardiac Baroreflex Sensitivity

The spontaneous sequence approach was used to calculate the baroreflex effectiveness index (BEI) and cardiac BRS using FisioSinal's baroreflex module (Tavares et al., 2011). The approach, in brief, revolves around nearby oscillations (ramps) in BP (>1 mmHg) and RR intervals (>4 ms). The overlap of BP ramps with concordant changes in RR was characterized as a BRS event. The average of the BRS slopes was thus designated as BRS. The total number of BRS events was divided by the total number of BP ramps seen during the 2-min time-bin to produce the baroreflex efficacy index.

Intra- and inter-day relative reliability in our laboratory for BRS is considered moderate (ICC: 0.72; 95% CI: 0.34 to 0.91 and ICC: 0.56; 95% CI: 0.29 to 0.85).

Statistical Analysis

All data are reported as mean ± standard error. Based upon an effect size of 0.98 calculated through G-power software and derived from published changes in time-frequency domain HRV within-between modes of exercise (Sandercock, Bromley, & Brodie, 2005), an a priori power analysis suggested that a number of 8 participants were required to detect significant differences between sessions (1-β = 80%) and a statistical significance was established as $p < .05$. Normality of distribution for variables was assessed qualitatively using histograms and Q-Q plots as well as quantitatively using the Shapiro-Wilk test, kurtosis, and skewness summary statistics. All distribution was found to be skewed, and log transformations were performed to conform to normality. Comparisons between sessions (BIKE, PUMP, GT, and CON) over time (rest vs. post-intervention time points) on indices of HRV and BRS were examined via two-way (session × time), repeated measures analysis of variance (ANOVA). Adjustments were performed when interactions with possible confounders (sex, MAP, mean AEE) were significant. Session differences for CRF variables, such as EE were analyzed via one-way repeated measures ANOVA. Post-hoc comparisons were performed via Bonferroni's tests. Intra and inter-day relative reliability was assessed with a two-way absolute agreement mixed model intraclass correlation coefficient (ICC (2,1)) computed with IBM SPSS Statistics Software (Version 25), using three static repeated measures. The ICC was interpreted as: poor [< 0.50], moderate [0.50, 0.75], good [0.75, 0.90], and excellent [> 0.90] (Koo & Li, 2016).

Results

Participant's demographic and CRF characteristics were as follows (mean ± SE): age 24.7±1.0 years, body mass index 23.0±0.5 kg, body fat 19.5±1.8%, resting HR 62.1±2.4 bpm, HRmax 195.3±1.0 bpm and VO_2max 27.3±1.6 ml/kg/min.

Regarding the group fitness classes, significant differences were observed for Activity and Total EE (kcal), VO_2max (%), and time spent in moderate and vigorous intensities (min) with values significantly greater for BIKE compared to PUMP and GT fitness classes **(Table 3)**.

Table 3

Energy expenditure and intensity achieved during the 3 group fitness classes

	BIKE	PUMP	GT
Resting Energy Expenditure (Kcal)	50.9±2.0	49.8±2.5	52.2±2.2
Activity Energy Expenditure (Kcal)	337.9±30.0	193.5±13.2*	214.5±15.6*
Total Energy Expenditure (Kcal)	432.0±32.9	270.3±16.0*	296.4±17.6*†
Group fitness class HR (bpm)	143.0±3.3	130.0±5.6	127.6±5.3
HR Reserve (%)	67.5±3.2	58.3±3.9*	55.6±3.8*†
Activity METs	8.7±0.9	5.4±0.3*	5.8±0.4*
VO_2Max (%)	67.4±3.2	44.2±2.5*	48.9±3.2*†
Moderate Intensity (min)	11.8±2.5	33.4±3.3*	25.4±2.2*
Vigorous Intensity (min)	32.0±2.1	11.4±3.1*	16.8±2.1*

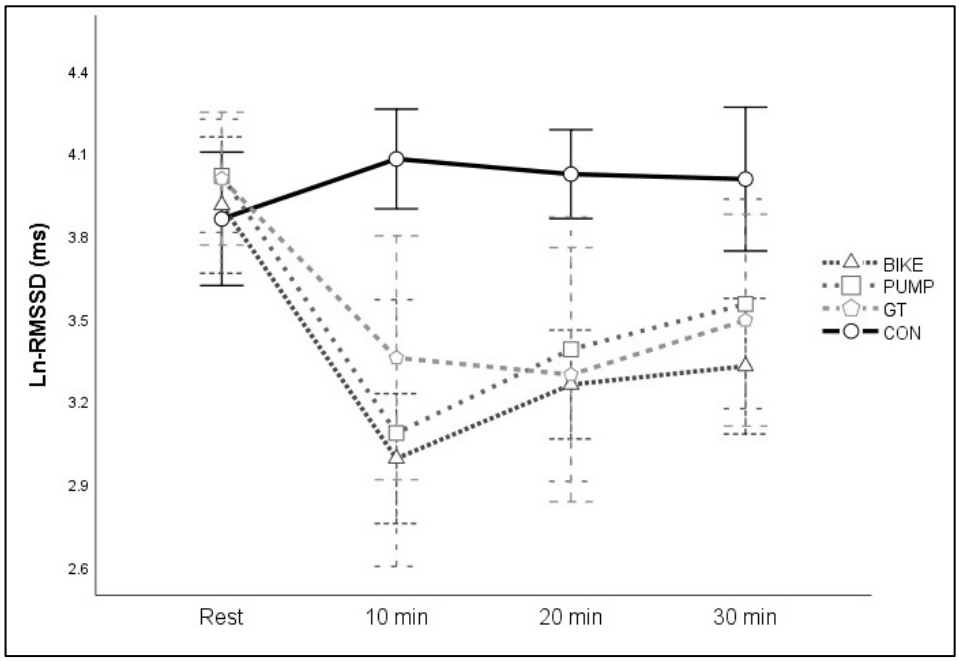
Data are expressed as mean ± SE. *indicates significant difference from BIKE; †indicates significant difference from PUMP

Heart Rate Variability

A main effect of time was observed in Ln-RMSSD *($p<0.001$; $\eta 2$*= 0.710) **(Figure 5)**, Ln-SDNN **(Table 4)**, Ln-HF **(Table 4)** and the ratio LF/HF (p<0.001; $\eta 2$=0.527) **(Figure 6)**. Ln-RMSSD and Ln-HF decreased immediately following BIKE (dLn-HF=-1.9 ms^2; dLnRMSSD=-0.9 ms), PUMP (dLn-HF=-1.7 ms^2; dLnRMSSD=-0.9 ms) and GT (dLn-HF=-1.5 ms^2; dLnRMSSD=-0.7 ms). LF/HF ratio was only significantly increased following GT (d=1.8). These indices did not return to baseline values 30 min into recovery. No significant interactions were found with mean AEE (p=0.960; $\eta 2$=0.035), or sex (p=0.860; $\eta 2$=0.085).

Figure 5

Time-course changes of Ln-RMSSD to the different exercise modalities



Data are expressed as mean ± SE

Table 4

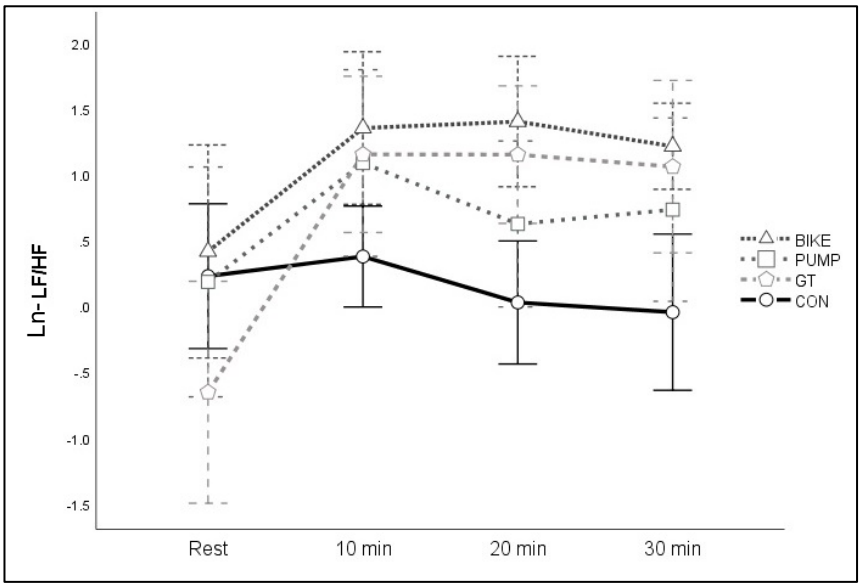
Acute Effects of Group Fitness Classes on Temporal and Spectral HRV and Cardiac Baroreflex Indices

		Rest	10min	20min	30min	Main effect of time (p value; partial eta square)
Ln-SDNN						(*p*<0.001; η2=0.483)
(ms)	**BIKE**	4.15±0.09	3.52±0.06*	3.71±0.09	3.73±0.09*	10,30<Rest
	PUMP	4.10±0.06	3.61±0.14	3.80±0.17	3.86±0.13	
	GT	4.07±0.08	3.72±0.10*	3.71±0.14	3.87±0.12	10<Rest
	CON	3.94±0.09	4.25±0.05*	4.21±0.07*	4.09±0.09	10,20>Rest
Ln-LF						n.s
(ms^2)	**BIKE**	6.99±0.37	6.00±0.17*	6.90±0.28	6.70±0.23*	
	PUMP	6.82±0.26	6.00±0.25	6.18±0.34	6.75±0.25	
	GT	6.20±0.31	6.54±0.34	6.19±0.35	6.76±0.27	
	CON	6.73±0.23	7.33±0.19	6.91±0.23	6.67±0.33	
Ln-HF						(*p<0.001; η2*= 0.598)
(ms^2)	**BIKE**	6.62±0.32†	4.69±0.25	5.54±0.25†	5.52±0.30†	10<Rest,20,30
	PUMP	6.68±0.32†	4.93±0.52	5.59±0.51†	6.06±0.43†	10<Rest,20,30
	GT	6.89±0.33	5.43±0.46*	5.08±0.48*	5.74±0.40	10,20<Rest
	CON	6.55±0.25	6.99±0.14	6.93±0.20	6.75±0.31	
BEI						(*p*=0.036; η2=0.275)
(%)	**BIKE**	63.61±18.40†	48.01±11.34	55.88±10.30†	59.67±10.01†	10<Rest,20,30
	PUMP	61.24±20.65	47.63±12.45	51.99±17.62	60.36±14.74	
	GT	64.85±12.31	48.46±15.85*	46.54±15.76*	51.46±17.34	10,20<Rest
	CON	60.79±17.06	57.10±18.50	56.01±13.70	57.56±13.47	-

Data are expressed as mean ± SE. *indicates significantly different from Rest; †indicates significantly different from 10 min post. SDNN - standard deviation of NN intervals; LF - low-frequency band; HF - high frequency band; BEI - baroreflex effectiveness index.

Figure 6

Time-course changes of Ln-LF/HF ratio to the different exercise modalities



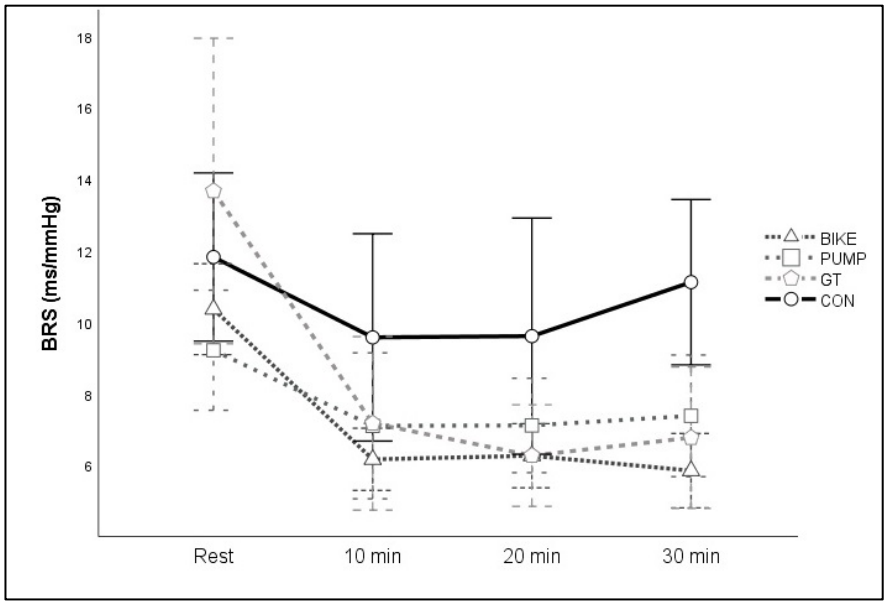
Data are expressed as mean ± SE

Baroreflex Sensitivity

Significant main effects of time were found for BRS ($p<0.001$; $\eta 2=0.497$) **(Figure 7)** and the Baroreflex Effectiveness Index (BEI) **(Table 4)**. BRS values were reduced during the entire recovery period following the three group fitness classes. Regarding BEI, a significant decrease was observed immediately following the three group fitness classes (d_{BIKE}=-15.6; d_{PUMP}=-13.6; d_{GT}=-16.4), with values returning to baseline following BIKE and PUMP but not GT.

Figure 7

Time-course changes of BRS



Data are expressed as mean ± SE

Association between resting vagal-related HRV, BRS, and AEE

AEE showed no associations with Ln-RMSSD ($r(10) = 0.81$, $p = 0.80$), Ln-HF ($r(10) = -0.22$, $p = 0.50$) and LF/HF ($r(10) = 0.10$, $p = 0.75$). BRS was positively associated with AEE ($r(45) = -0.64$, $p = 0.02$).

Discussion

To the best of our knowledge, this was the first study aimed to directly compare the acute effects of three different group fitness classes on indices of cardiovagal modulation. The main findings were that: 1) RMSSD and SDNN decreased immediately following the three group fitness classes in physically active young adults; 2) increases LF/HF ratio were only observed immediately following GT; and 3) HRV and BRS parameters did not return to baseline values 30 minutes into recovery, whereas BEI returned to baseline values after 30 minutes following BIKE and PUMP but not GT.

Post-exercise HRV and BRS responses

Our evidence does not support modality as a major determinant of post-exercise changes in cardiovagal HRV modulation in healthy young adults. The differences in EE between group fitness classes had no effects on RMSSD, contrasting with previous findings (Cunha, Midgley, Goncalves, et al., 2015). Furthermore, we found no significant associations between AEE and vagal control parameters (RMSSD or HF). Our results bring ecological validity into consideration in the study of the acute effects of exercise (B.C., 2013; S. G. Dasilva et al., 2011) on cardiovagal modulation and BRS. Research so far have made use of exercises in laboratorial settings rather on activities, to represent typical sessions in accordance with current guidelines for improvement and maintenance of CV health (D. Riebe et al., 2018). What our findings suggest is that these laboratorial findings seem to have low ecological validity, in a way that findings cannot be easily generalized to fitness class settings.

Overall, there is no effect of exercise modality on cardiovagal modulation, but it seems that the sympathovagal balance was elevated following GT. GT encompasses both aerobic and resistance components, but to our surprise, this exercise modality has not been included in studies examining the acute effects of exercise on cardiovagal modulation, although it is the recommended for maintaining muscular strength and endurance, and flexibility of the major muscle groups (Michael L. Pollock et al., 1998). Heffernan, Kelly, et al. (2006) previously reported a similar increase after both resistance (81.5% increase) and endurance (63.9% increase) exercise in LF/HF suggesting a

comparable shift towards sympathetic predominance, as in our study. However, the interpretation of spectral HRV data should be approached with extreme caution. The LF/HF ratio is based on the assumption that the SNS generates LF power while the PNS generates HF power (Shaffer & Ginsberg, 2017). Thus, the LF/HF ratio may not accurately reflect sympathovagal balance (Goldberger, 1999), and it is not universally recognized as such (Billman, 2013). It has been proposed that the sympathetic (LF power) and parasympathetic (HF power) nervous systems do not respond to stimuli in a reciprocal manner, and that the concept of a sympathetic/parasympathetic balance may not be physiologically or mathematically supported (Eckberg, 1997). Others, on the other hand, have argued that as one branch is excited, the other is inhibited, whilst there is data to support the LF/HF ratio as an indicator of this dynamic interaction (A. Malliani, 2005). Furthermore, the intructor's control of participants breathing when they experience high levels of SNS activity can engage both branches and increase RSA (Hayano & Yuda, 2019). Given the complex relation between SNS and PNS nerve activity, the ratio between LF and HF power should not be considered an index autonomic balance (Billman, 2013).

Different exercise intensities throughout the three group fitness classes did not differently affect the recovery of vagal HRV parameters, contrasting with previous findings (Kaikkonen et al., 2008; Michael et al., 2016; Niewiadomski et al., 2007; Seiler et al., 2007) that revealed greater HRV declines at higher exercise intensities (Michael et al., 2016). Thus, the duration of our group fitness classes (45 min), the different intensities achieved (BIKE: 68%; PUMP: 58%; GT: 56% of HR reserve) and the intermittent pattern of group fitness classes, may have also contributed to our divergent findings.

Our findings suggest a transiently reduction in BRS following the group fitness classes with a pronounced decline 10 min post exercise, that remained so for over the 30 minutes recovery period. This is in line with previously findings suggesting that BRS returns to baseline 60-120 min into recovery following aerobic exercise, although in an intensity-dependent manner (Niemelä et al., 2008; Reynolds, De Ste Croix, & James, 2017). BRS recovery to baseline values was slower in GT compared to BIKE and PUMP fitness classes. The physiological mechanisms for superior reduction in BRS after combined exercise compared to other activities are not clear, but several mechanisms may explain these differences (Facioli et al., 2021). Previous findings showed a relatively rapid recovery of BRS after aerobic and light resistance exercise and delayed recovery after a heavy resistance exercise (Niemelä et al., 2008; Reynolds et al., 2017). This might have been the case for GT in which heavy intensity resistance exercise was present. The delayed BRS pattern after heavy resistance exercise is controlled by a delicate

interplay between vagal outflow withdrawal and possibly increased sympathetic vasomotor tone as measured by HR (Niemelä et al., 2008).

Limitations

It is possible that only monitoring post-intervention HRV recovery every 10 minutes for the first 30 minutes resulted in a failure to detect subtle changes or those that occurred outside of this initial timeframe (e.g., >30min – 72 h) and have been enough to explore the recovery of BRS values once other studies report values recovered after 60-120 min following exercise. We did not account for RSA, which could have influenced HRV and BRS. Even though paced breathing has been proposed to reduce the impact of respiratory effects on autonomic parameters (Shaffer & Ginsberg, 2017), research has shown that it has no significant effect on postexercise HRV (Kaikkonen, Nummela, & Rusko, 2007). The menstrual cycle variation in adult female subjects was not regulated. Previous research has suggested variations in cardiac vagal activity across the menstrual cycle (Brar, Singh, & Kumar, 2015), although there are disparate results (Schmalenberger et al., 2019). Moreover, previous studies have found sex differences on indices of HRV (Geovanini et al., 2020; J. Zhang, 2007), which should have been took into consideration. Finally, our study lacked invasive measures such as catecholamine plasma concentrations and blood lactate, which could have provided additional information about sympathetic activity and skeletal muscle recovery following exercise cessation. All participants were recreationally active, so these results cannot be easily generalized to older, sedentary, and/or clinical populations.

Conclusions

The present study demonstrated that 1) cardiovagal modulation indices respond similarly to different exercise modalities as commercially available in health clubs, although 2) sympathovagal balance appears to be altered following combined exercise. Previous laboratorial findings seem to have low ecological validity, in a way that findings cannot be easily generalized to fitness class settings.

Final Conclusions

The present work sought to examine the acute changes in AS, cardiovagal modulation, and BRS in response to three commercially available group fitness classes. We found that 1) the three group fitness classes decreased carotid artery and lower limb AS, as well as cardiovagal modulation, and BRS, 2) regional AS varies depending the anatomical segment assessed, and 3) carotid artery stiffness is dependent on the mode of exercise. As a result, we concluded that exercise modality influences indices of local

AS while having no effect on parameters of regional AS, cardiovagal modulation, and BRS.

Evidence derived from laboratorial experiments on the acute effects of exercise, rather than activities, on indices of arterial stiffness seem to have ecological validity, but this may not be the case when measuring the cardiovagal modulation and BRS responses to exercise, that seems distinct from those when measuring the response to activities as they are offered in gyms.

Practical Applications

Based on our data, there are no periods of significant concern eventually caused by the transient changes on AS, cardiovagal control and BRS immediately following the three group fitness classes. In practical terms, fitness professionals should not be concerned about a possible risk of undesirable CV events in young healthy subjects after the three fitness classes. Performing any of the 3 group fitness classes might immediately result in an unfavourable CV response (increased AS), although the distinct adjustments in the recovery phase are not of concern in healthy young adults. All this together contributes to the body of research to prescribe group fitness classes in a more precise and secure manner.

Future Research Directions

Further research is warranted outside the laboratorial setting to tackle the lack of research concerning real-world public health contexts. While there is some agreement on the best way to give health-related exercise based on a huge amount of laboratory data, there appears to be a disconnect between data from laboratory and/or clinical research and data from real-world interventions. In other words, real-world treatments do not appear to be as beneficial as laboratory research predicts they should be in improving CV health. This problem is exacerbated by the scarcity of peer-reviewed research articles describing real-world research, as well as therapeutically relevant data. On this premise, a precise picture of the degree of laboratory-to-field translation is not yet possible. However, future research concerning the acute effects of different modes of exercise on CV outcomes, AS and/or cardiovagal modulation must improve upon designs that rely on crude measures inserted in the real-life settings of training and must embrace new measurements opportunities and technologies. Moreover, our results apply only to healthy young adults. Physically active older adults, as well as other populations with different CRF and CV risk profile can exhibit different post-exercise vascular and autonomic recovery patterns, which may somewhat limit the generalizability of our results.

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