Title: Blood pressure, arterial stiffness and exercise: does exercise increase the risk of acute cardiac events in older adults?

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There has been considerable popular conjecture about the appropriate volume, intensity and type of exercise required for ageing adults to sustain wellbeing and health and to promote an active lifestyle. It is widely considered that physical activity of all types is worthwhile for various reasons, including maintaining a healthy body mass, promoting positive mental health, helping neuromuscular coordination and improving body posture, while reducing the rate of decrease in bone mass and cardiovascular fitness. This has resulted in important international physical activity guidelines1 for the general public. However, it is widely known that an ageing human body is inevitably susceptible to physical decline and degeneration. There is an increase in susceptibility to myocardial infarction, stroke or other major non-communicable diseases with increasing age and arterial rupture occurs at two to three times lower pressures in patients over the age of 40 years than in younger people.2 It is also well established that high blood pressure is the greatest cause of stroke, yet the occasion when blood pressure is highest is during physical activity. This presents a conundrum as to whether physical activity per se is a help or hindrance to preserving longevity of life in ageing adults, or whether it might be appropriate to more closely consider the type of physical activity undertaken and the physiological parameters by which exercise thresholds are determined beyond that of simple frequency and volume.1,3 It seems likely that exercise guidances based on increases in blood pressure could be meaningful, as might a greater awareness of safe operating parameters for sustaining the integrity of blood vessels in ageing adults.

Hypertension is known to accelerate the structural and functional changes to the walls of blood vessels that occur with ageing, which result in decreased vascular distensibility and increased arterial stiffness. Arteries progressively stiffen in older humans and arterial walls become deranged, with thinning, splitting, fraying and fracture of the elastic laminae, while the amount of connective tissue and collagen fibres increase. Engagement in exercise has been shown to have an impact on hypertension and also on endothelial function, inflammation and sympathetic activity, all of which positively affect arterial stiffness.3,4 It therefore seems clear that exercise per se is beneficial to arterial integrity, although the frequency, intensity and type of activity performed may differentially affect the mechanical properties of blood vessels. This means that even though exercise can aid the maintenance of

healthy vessels, at the same time it imposes sustained periods of high strain above resting levels, particularly when exercise intensities are relatively high. When combined with age-related arterial weaknesses, this may lead to an increased risk of sudden rupture, particularly when individuals have advanced, yet undiagnosed, vascular disease.

Regular dynamic, moderately intense aerobic exercise has long been recommended for the prevention and treatment of high blood pressure,1 and even one to two weeks of training has been found to decrease systolic and diastolic pressures by about 10 and 7 mmHg, respectively, in people with hypertension. Conversely, arterial stiffness appears to increase in both the elastic central arteries (14.5%) and the peripheral muscular arteries (8.7%) after resistance training.6 High-intensity intermittent exercise has been proposed as a practical, time-efficient means of gaining tangible health benefits from exercise, but the impacts of this activity on the integrity of blood vessels have not yet been quantified.

Despite the benefits of both aerobic and resistance training in terms of vascular health and strength,4,5,7 acute exertion may trigger acute cardiac events in some people, particularly older adults.6,8 This is likely to be due to the progressive degeneration of arteries, combined with sustained increases in blood pressure in people with hypertension. It has been theorised that acute exertion may predispose some people to sudden coronary or cerebral events by precipitating the rupture of a vulnerable coronary artery plaque or a weakened vessel wall. When looking at the actual numbers of adverse events in exercise testing, it turns out that the occurrence of this is very low (0.04%) and that most of the adverse events occur in older adults.9 As different types of exercise increase blood pressure to greater or lesser extents, it may be prudent for new studies to experimentally explore and consider the likely increase in proposed exercise for older adults and the hypertensive profiles of individual exercisers in relation to the mechanical properties and critical pressure tolerances of blood vessels.

To be able to fully determine how exercise, blood pressure and vascular damage are related in an ageing population, it is important to take a mechanical perspective alongside the physiological mechanisms. Both long-term vascular physiological adaptations caused by hypertension associated with regular exercise and the associated short-term mechanical high-pressure situations occurring during exercise are relevant to safe exercise in terms of arterial rupture. Therefore both physiological and mechanical factors need to be taken into account when advising on age-appropriate exercise. Although any vessel will eventually break at sufficiently high pressure, the estimated rupture pressures of the aortic wall are extremely high at 400 kPa (~3000 mmHg).10 This is likely to be considerably lower in vessels containing undetected plaque and will depend heavily on the stage of degeneration and associated vulnerability. The widely cited pressure threshold for the rupture of arterial plaque is 300 kPa (~2000 mmHg),10 but plaque also contributes to arterial stiffness and the accelerated degeneration of arterial walls. Its rupture remains a primary cause of acute coronary events, accounting for more than half of all cardiovascular-induced deaths.8 In addition, specific arterial pressure tolerances vary according to size and location in the body, with smaller arteries in the neck and head susceptible to rupture at lower pressures of ~900 mmHg.

As regular exercise provides many benefits to health and the rates of adverse events are low,8,9 it would be incorrect to specify that exercise-induced increases in blood pressure present significant health risks. However, different types of exercise differentially increase blood pressure. Moderate to vigorous exercise is often the recommended intensity for older adults, which results in acute increases in systolic pressure to _180 mmHg, whereas

maximum aerobic exercise may cause temporary increases to about 250mmHg. Resistance training increases blood pressure to a greater extent (up to 480/ 350 mmHg in leg exercises) and this is likely to due to the combination of mechanical compression of the contracting muscles, the accompanying pressor reflex that occurs in static contractions and the superimposition of an increased intra-thoracic pressure caused by the Valsalva manoeuvre.11 High peak pressures are avoided in many cyclic exercises as they lead to injuries and fractures of the joints involved in exercise. The same can be said for vessels when considering a mechanical perspective.

High peak pressures might cause tissue damage and therefore the mechanical properties and actual pressures occurring in the vessels associated with exercise and with hypertension need to be taken into account. An evenly distributed pressure profile as demonstrated in endurance exercise might be more advisable for older adults to prevent ruptures than the sudden, extreme high peak pressures, occurring in high-intensity intermittent exercise.

All the quoted acute exercise-induced increases in blood pressure are less than the estimated critical pressure thresholds of healthy blood vessels. However, the mechanical degradation and critical pressure threshold of arteries has not been systematically quantified across the lifespan, with the co variants of vulnerable plaque, the presence of an undetected aneurysm, or the combination of these factors in response to maximum aerobic or resistance exercise. It would seem prudent for older adults to carefully consider the type of exercise they participate in and they should be recommended an exercise modality reflective of their stage of life and blood pressure profile. The likely health benefits from sustained exercise participation are meaningful, 1,3,4,6,8,12 but acute exercise-induced increases in blood pressure could place undue pressure on arterial walls. Therefore older adults engaging in exercise that induces high peak blood pressures may require further experimental investigation to properly evaluate the risk–benefit profile and to inform about the optimum strategies to promote safe exercise.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

References

1. Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). Eur J Prev Cardiol 2016; 23: NP1–NP96.
2. Piechna A, Lombarski L, Krajewski P, et al. Age dependence of critical pressure in the segments and branches of the circle of Willis. Neurol Neurochir Pol 2017; 51:146–148.
3. Ferreira I, Boreham CA and Stehouwer CD. The benefits of exercise for arterial stiffness. Am J Hypertens 2006; 19: 1037–1038.
4. Sandri M, Viehmann M, Adams V, et al. Chronic heart failure and aging – effects of exercise training on endothelial function and mechanisms of endothelial regeneration: Results

from the Leipzig Exercise Intervention in Chronic heart failure and Aging (LEICA) study. Eur J Prev Cardiol2016; 23: 349–358.
5. Montero D and Dı´az-Can˜ estro C. Endurance training and maximal oxygen consumption with ageing: Role of maximal cardiac output and oxygen extraction. Eur J Prev Cardiol 2016; 23: 733–743.
6. Burke AP, Farb A, Malcom GT, et al. Plaque rupture and sudden death related to exertion in men with coronary artery disease. JAMA 1999; 281: 921–926.
7. Hollings M, Mavros Y, Freeston J, et al. The effect of progressive resistance training an aerobic fitness and
strength in adults with coronary heart disease: A systematic review and meta-analysis of randomised controlled trials. Eur J Prev Cardiol 2017; 24: 1242–1259.
8. Siscovick DS, Weiss NS, Fletcher RH, et al. The incidence of primary cardiac arrest during vigorous exercise. N Engl J Med 1984; 311: 874–877.
9. Foster C. Is there risk in exercise testing of athletes? Int J Sports Physiol Perform 2017; 12: 849–852.
10. Cheng GC, Loree HM, Kamm RD, et al. Distribution of circumferential stress in ruptured and stable atherosclerotic lesions. A structural analysis with histopathological correlation. Circulation 1993; 87: 1179–1187.
11. MacDougall JD, Tuxen DSDG, Sale DG, et al. Arterial blood pressure response to heavy resistance exercise. J Appl Physiol 1985; 58: 785–790.
12. Edwards AM and Polman RC. Pacing and awareness: Brain regulation of physical activity. Sports Med 2013; 43: 1057–1064.