SA13

Introduction to the athlete's heart. What is it? What are the questions?

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SA14

Autonomic control of the athlete's circulation

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Regular endurance exercise training increases oxygen transport and utilization 15–25 % by causing structural and regulatory adaptations of the cardiovascular system. The primary adaptations include expansion of plasma volume and intrinsic and neurally mediated adjustments in the heart and vasculature. Autonomic neural activity and hormonal and intrinsic cardiac pacemaker activity constitute redundant mechanisms that contribute to the reduction of heart rate at rest and during submaximal exercise. The arterial baroreflex has greater heart rate responses due to increased vagal tone but the sympathetic arm of the baroreflex is inhibited by an increased central blood volume load on the cardiopulmonary baroreceptors resulting in a greater sympathoinhibition. Other hormonal influences increase vagal tone; these include an up-regulation of dopaminergic receptors and a down-regulation of enkephalin receptors in the heart. Aortic rheoreceptor activation by the increased stroke volume and decreased reflex signals from the trained skeletal muscles also reduce sympathetic outflow to the heart and vasculature. The mechanism by which intrinsic heart rate is lowered remains unexplained but appears to involve structural adaptation within the pacemaker cells.

SA15

Cardiac remodelling with endurance exercise

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Cross-sectional studies have demonstrated that endurance athletes have 'eccentric hypertrophy' of the heart with increased mass and volume. The functional consequence of this adaptation is a more compliant, distensible left ventricle, which operates on the steep portion of the Starling curve, facilitating a large increase in stroke volume during exercise by the Frank-Starling mechanism. However, whether this phenotype is truly in direct response to endurance training, or rather is a unique characteristic of individuals destined for athletic success in endurance sport is not clear. As part of this symposium, I will present the results of a novel investigation in which a group of previously sedentary young men and women trained for a year to compete in a marathon, using training techniques typically followed by elite athletes. The results challenge conventional thinking regarding the morphologic response to endurance training. During the early phases of the training programme there was eccentric right ventricular hypertrophy, but concentric left ventricular hypertrophy, characterized by an increase in mass and wall thickness with only a minimal increase in volume. It was not until very prolonged training sessions, as well as high-intensity interval training was incorporated into the training programme that eccentric hypertrophy of the LV developed. Although after 1 year of training, Starling curves approached those observed with elite athletes, LV pressure volume curves did not achieve the same compliance as those observed cross-sectionally in elite athletes. There were remarkable gender differences as well, with females demonstrating substantially less hypertrophy than males, despite identical training regimens. Finally, comprehensive studies in our laboratory involving a wide range of physical activity (from 12 weeks of bed rest to 1 year of training) demonstrate that a remarkable 37 % of the mass of the LV is plastic, and adaptable with changes in exercise training.

SA16

Cardiac hypertrophy mechanisms and complications

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SA17

Effects of endurance training on excitation–contraction coupling in isolated cardiac myocytes from the rat

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Regular exercise enhances cardiac function and modulates myocyte growth in healthy individuals. The talk will describe studies designed to assess contractile function and expression of selected genes associated with intracellular Ca^{2+} regulation after intensity controlled aerobic endurance training in the rat. Female Sprague-Dawley rats were randomly assigned to sedentary control (SED) or treadmill running (TR) 2 h per day, 5 days per week for 2, 4 or 13 weeks. Rats ran for 8 min intervals at 85–90% of $V_{O_2,max}$ separated by 2 min at 50–60%. At the end of the training period, the animals were killed by cervical dislocation and the hearts were removed and ventricular myocytes isolated by perfusion with collagenase-containing solutions. Myocyte length, intracellular Ca^{2+} (fura-2), and intracellular pH (BCECF) were measured in dissociated cells in response to electrical stimulation at a range of stimulation rates. The increase in $V_{O_2,max}$ plateaued after 6–8 weeks, 60% above SED. After 13 weeks, left and right ventricular weights were 39 and 36% higher than in SED. Left ventricular myocytes were 13% longer, whereas width remained unchanged. After 4 weeks of training, myocyte contractility was approximately 20% higher in TR, whereas peak systolic intracellular Ca^{2+} and time for the decay from systole were 20–35% and 12–17% lower, respectively. These results suggest that increased myofilament Ca^{2+} sensitivity is the dominant effect responsible for enhanced myocyte contractility in TR. Western blot analysis indicated 21 and 46% higher myocardial SERCA-2 and Phospholamban, but unaltered Na^+/Ca^{2+} exchanger levels. In conclusion, this study strongly indicates that physical exercise induces adaptive hypertrophy in cardiac myocytes with improved contractile function in marked contrast to the pathological hypertrophy and associated decreased contractile function associated with congestive heart failure.

SA18

Exercise and vascular remodelling

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Endurance exercise training results in structural expansion of the vascular beds of both myocardium and skeletal muscle. In the heart, this involves increases in diameter of the large coronary arteries, and growth of new vessels at the level of resistances arteries and arterioles. Capillary growth by proliferation and sprouting has been demonstrated in trained pig hearts (White *et al.* 1998) but overall, evidence for exercise-induced growth of myocardial capillaries is inconsistent, occurring when it does in young rather than adult animals. This may be due to an already high capillary density, to rapid transformation of capillaries into arterioles during the growth process or to the fact that concurrent cardiac hypertrophy may mask capillary neoformation. Studies in rats, dogs and pigs have used such varying training modes (running, swimming) and periods (4–18 weeks) that it is difficult to deduce a minimum exercise intensity and duration requirement for coronary vascular adaptation. Whilst development of collateral circulation in the healthy heart as a result of exercise training is not proven, exercise-induced structural adaptations to coronary vasculature as a whole allow for improved flow and more homogeneous capillary perfusion.

In contrast to the heart, studies have repeatedly shown capillary growth in trained skeletal muscles of animals and humans, although even now it is not clear whether this occurs by proliferation and sprouting or other means such as vessel elongation or splitting. Growth of resistance arteries and arterioles within trained muscle, on the other hand, is less well established, as are the reasons for differential growth of small *versus* larger vessels. Both cross-sectional and longitudinal studies in humans have confirmed that endurance exercise training induces remodelling of large conduit arteries, such as the aorta and those supplying trained limbs, to have larger diameters and greater distensibility (Huonker *et al.* 1996). This appears to allow for augmented blood flow to exercising muscles whilst maintaining arterial wall shear stress. These structural modifications are likely to involve vascular smooth muscle and elastic tissues, but this remains to be established.

Signals generated during each bout of exercise that could initiate vascular growth and remodelling may be metabolic, relating to production of e.g. adenosine, or tissue ischaemia/hypoxia, haemodynamic effects of increased blood flow and/or pressure, mechanical stresses in cardiac and skeletal muscle arising from repetitive contraction and stretch, and production of growth factors either directly or indirectly.

In the healthy heart, increased blood flow and contraction force have been implicated, with resting bradycardia, as a consequence of training, a prime factor facilitating both of these and leading to capillary growth via vascular endothelial growth factor, VEGF (Brown & Hudlicka, 1999; Zheng *et al.* 1999). Increased blood flow and myocyte stretch are also important in capillary angiogenesis in skeletal muscle in conjunction with VEGF. The enlargement of coronary and peripheral conduit arteries during training is most likely related to flow-mediated increases in shear stress and production of endothelial products such as nitric oxide and prostglandins. Detailed investigations of time-dependent changes in vascular reactivity during training (e.g. Bowles *et al.* 2000) have established that endothelial-dependent dilatation is enhanced early on in advance of structural modifications but that in the fully adapted trained state, metabolic, myogenic and endothelial controls of vascular tone are comparatively little changed. The challenge remains in the application of knowledge of these mechanisms of vascular remodelling by exercise as a preventative strategy in the promotion of health, or to target with site-specific precision pathologies that affect the circulation.

Bowles DK *et al.* (2000). *Exer Sport Sci Rev* **28**, 57–62.
Brown MD & Hudlicka O (1999). *Adv Organ Biol* **7**, 155–196.
Huonker M *et al.* (1996). *Int J Sports Med* **17**, S164–172.
White FC *et al.* (1998). *J Appl Physiol* **85**, 1160–1168.
Zheng W *et al.* (1999). *Circ Res* **85**, 1920198.

SA19

Equine athletes, equine athlete's heart and racing success

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SA20

Human athlete's heart – effects of type of sport, age and ethnicity

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SA21

Sudden cardiac death in young athletes and its impact – the case for screening

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The death of an athlete is a rare event but grabs headlines. The death of a non-athlete is more common but much less likely to get publicity. What is the impact/cost after such an event? These deaths are unaccountable, especially for an exceptionally fit individual. The full implications of the impact of the sudden cardiac death of a young athlete (SCDYA) have not been recognised. The public perception of the death of an athlete is that of suspicion and maybe assumed to be a result of performance enhancing drugs, or excessive training damaging the heart. Further, the sudden death of a young child in sport is seen as an isolated incident and dismissed as a type of cot death. The public cannot relate to SCDYA or apply the potential to their family. Media headlines reporting SCDYA are often dramatic and misleading.

There is a catastrophic impact on the family after such a death. This can make family members disfunctional as all grieve differently. They become vulnerable to nervous breakdown, alcoholism, inability to return to work (especially if parents have witnessed the tragedy), terror of the genetic implications for siblings, guilt of a parent if found to be the genetic carrier. Families become angry that their child has been unwittingly put at risk and this anger translates into targeting associations for not screening.

Within the community there is a need to respond. They know and value the athlete as an individual whose sporting prowess and fitness will have been well known. The peer group is devastated and unnerved. Such deaths haunt those that witness them or know the victims. There is often a need for screening to reassure and sometimes groups affected become involved in campaigning for change. Such tragedies send shudders through the sport and can lead to strident demands for screening from other athletes/parents. The more SCDYAs are publicised the more crucial it is that there should be an approved pro-active response through screening.

Screening is effective in identifying the majority of causes associated with SCYDA, e.g. hypertrophic cardiomyopathy accounts for 50 % of SCYDA; screening is able to identify 97 %. Early diagnosis saves lives. Screening is a positive response to public demand. Most SCDYA are preceded by symptoms that have been dismissed. Screening raises awareness of symptoms amongst coaches, trainers, physiotherapists and parents, and establishes a 'fast-track' pathway for athletes giving cause for concern. Screening feeds valuable research and will help identify the prevalence of diseases. Screening of athletes averts the dangers of potential litigation and should automatically be included in the sophisticated package of medical testing for risk assessment now in place in many sports. Screening offers reassurance for young people who are being urged to stay fit through sport with a confirmation of cardiac health prior to strenuous activity. Safeguarding the health of the athlete should be paramount and therefore there is a moral responsibility to encourage athletes to be screened.

SA22

Causes of SCDYA and their frequency in the general population. Can they be readily distinguished from athlete's heart?

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SA23

How effective is universal screening for causes of SCDYA in Italy?

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SA24

Screening for causes of SCDYA in the USA

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SA25

English Football Association – cardiac screening of young apprentices

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