Coronary flow reserve is supranormal in endurance athletes: an adenosine transthoracic echocardiographic study

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Abstract

Objective—To compare coronary flow reserve in endurance athletes and healthy sedentary controls, using adenosine transthoracic echocardiography.

Methods—29 male endurance athletes (mean (SD) age 27.3 (6.6) years, body mass index (BMI) 22.1 (1.9) kg/m^2) and 23 male controls (age 27.2 (6.1) years, BMI 23.9 (2.6) kg/m^2) with no coronary risk factors underwent transthoracic echocardiographic assessment of distal left anterior descending coronary artery (LAD) diameter and flow, both at rest and during intravenous adenosine infusion (140 µg/kg/min).

Results—Distal LAD diameter and flow were adequately assessed in 19 controls (83%) and 26 athletes (90%). Distal LAD diameter in athletes (2.04 (0.25) mm) was not significantly greater than in sedentary controls (1.97 (0.27) mm). Per cent increase in LAD diameter following 400 µg sublingual nitrate was greater in the athletes than in the controls, at 14.1 (7.2)% *v* 8.8 (5.7)% ($p < 0.01$). Left ventricular mass index in athletes exceeded that of controls, at 130 (19) *v* 98 (14) g/m^2 ($p < 0.01$). Resting flow among the athletes (10.6 (3.1) ml/min; 4.4 (1.2) ml/min/100 g left ventricular mass) was less than in the controls (14.3 (3.6) ml/min; 8.2 (2.2) ml/min/100 g left ventricular mass) (both $p < 0.01$). Hyperaemic flow among the athletes (61.9 (17.8) ml/min) exceeded that of the controls (51.1 (14.6) ml/min; $p = 0.02$), but not when corrected for left ventricular mass (25.9 (5.6) *v* 28.5 (7.4) ml/min/100 g left ventricular mass; NS). Coronary flow reserve was therefore substantially greater in the athletes than in the controls, at 5.9 (1.0) *v* 3.7 (0.7) ($p < 0.01$).

Conclusions—Coronary flow reserve in endurance athletes is supranormal and endothelium independent vasodilatation is enhanced. Myocardial hypertrophy per se does not necessarily impair coronary flow reserve. Adenosine transthoracic echocardiography is a promising technique for the investigation of coronary flow reserve.

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Keywords: coronary flow reserve; athlete; adenosine transthoracic echocardiography

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The concept of coronary flow reserve—the ratio of maximum to resting coronary arterial blood flow—was introduced to clinical practice by Gould and Lipscomb.[1] It has since been used widely, both to assess epicardial coronary stenoses and to examine the integrity of the microvascular circulation. However, measurement of this ratio is limited by the techniques available. The gold standard—intracoronary Doppler[2]—is invasive and therefore unsuited to the acquisition of serial measurements over time, or to the investigation of normal or healthy individuals.

Non-invasive imaging techniques such as magnetic resonance imaging and positron emission tomography are increasingly capable of measuring coronary flow reserve,[3 4] but they are complex, time consuming, and expensive. Transoesophageal echocardiography also allows calculation of coronary flow reserve,[5] though being a semi-invasive technique it is not suited to the investigation of normal subjects. Transthoracic echocardiography has been used to determine distal left anterior descending coronary artery diameter,[6] flow,[7] and more recently coronary flow reserve.[8–11] The non-invasive simplicity of this technique allows measurements to be made on a serial basis, and non-pathological conditions to be investigated.

One such condition in which coronary flow reserve is of interest is athletic training. Pathological left ventricular hypertrophy is known to impair coronary flow reserve[12–17] but the effects of physiological left ventricular hypertrophy on flow reserve are not known. Some animal studies have suggested that exercise training increases coronary sensitivity to vasoactive pharmacological agents,[18–20] and augments maximum coronary flow in response to adenosine.[18 21 22] We therefore undertook to compare coronary flow reserve in endurance athletes with that in sedentary controls using adenosine transthoracic echocardiography.

Methods

SUBJECT SELECTION

Endurance athletes were recruited on a national basis. All participants were male, had competed at national or international level, and were actively training at the time of study. All underwent assessment of maximum oxygen consumption ($\dot{V}O_2$max), following a standard protocol using Douglas bags,[23] to confirm their high performance athletic status. Sedentary controls were recruited locally and were excluded if they had participated in any exercise training programme during the preceding year. Some control subjects had also

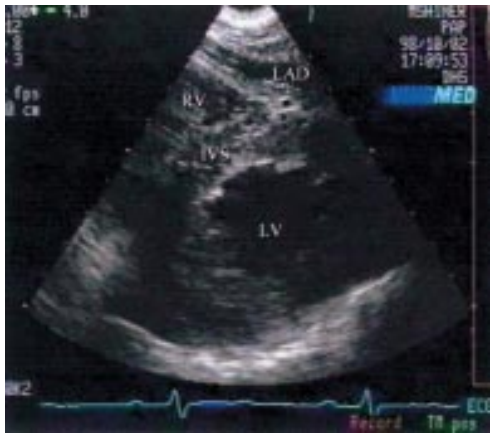

Figure 1 A low left parasternal short axis section through the left ventricle. The distal left anterior descending coronary artery is identified as a circular radiolucency with increased circumferential opacification lying in the anterior interventricular sulcus, barely 3 cm from the chest wall. IVS, interventricular septum; LAD, left anterior descending coronary artery; LV, left ventricle; RV, right ventricle.

been involved in the original feasibility study of coronary flow reserve.[8] Participants were normotensive non-smokers with no history of hypercholesterolaemia and no family history of premature coronary disease. The study was approved by the local regional ethics committee and all participants gave written informed consent.

ECHOCARDIOGRAPHIC EQUIPMENT

Studies were undertaken using a Vingmed CFM 750 ultrasound unit (GE Vingmed Ultrasound, Trondheim, Norway). A 3.5 MHz annular phased array transducer was used for cross sectional and M mode analysis of interventricular septal thickness, end diastolic left ventricular dimension, and posterior wall thickness. Coronary imaging was performed with a broad bandwidth 5 MHz transducer upgraded to 6.3 MHz for maximum near field cross sectional resolution. Doppler frequency was 4 MHz.

TECHNIQUE

Subjects were asked to avoid caffeine containing products for the 12 hours before echocardiography.[24] A venous cannula was sited and subjects were examined in the left lateral decubitus position using a low left parasternal window, as previously described.[6 7] The left ventricle was imaged in short axis and the distal left anterior descending coronary artery (LAD) was identified as a circular radiolucency lying in epicardial fat in the anterior interventricular sulcus (fig 1). Magnified images were committed to videotape for off line analysis of LAD diameter.

Sublingual glyceryl trinitrate 400 µg was given to dilate and fix the epicardial coronary diameter, preventing further flow induced vasodilatation.[25 26] Parasternal short axis cross sectional and M mode images of the left ventricle were then recorded to allow off line caliper analysis of end diastolic interventricular septal thickness, posterior wall thickness, and internal ventricular diameter, according to the recommendations of the American Society of

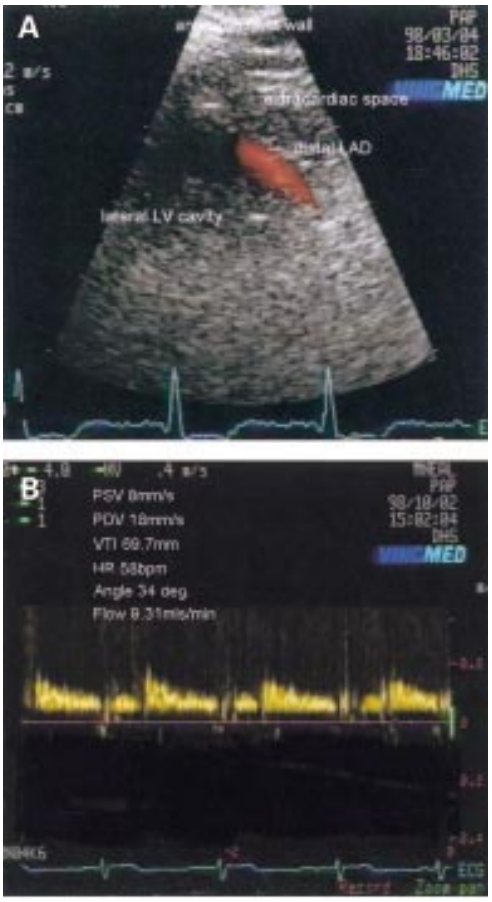

Figure 2 (A) Low left parasternal long axis window. Resting distal left anterior descending coronary artery (LAD) flow is visualised as a thin red diastolic flame once low velocity reject and frame rate are minimised. (B) Spectral Doppler display of resting flow in the distal LAD in an endurance athlete. HR, heart rate; PDV, peak diastolic velocity; PSV, peak systolic velocity; VTI, velocity–time integral.

Echocardiography,[27] and to allow calculation of left ventricular mass according to the Penn convention.[28] Five minutes after administration of glyceryl trinitrate, end diastolic distal LAD diameter was again recorded as above.

The ultrasound probe was then rotated anticlockwise 90° and the probe base angled laterally and downwards to allow Doppler analysis of the anterior interventricular sulcus at the smallest possible incident angle. Magnified colour flow mapping of this region was undertaken. The LAD was identified as a thin red diastolic jet (fig 2A). Doppler sampler width was maximised within the colour signal, and flow was analysed with spectral Doppler (fig 2B). The incident angle at which the Doppler signal intersected LAD blood flow was noted with on line angle correction.

Intravenous adenosine was infused (140 µg/kg/min, right antecubital fossa) for a maximum of six minutes. Colour flow mapping of the distal LAD revealed disturbed flow (fig 3A). Pulsed wave Doppler analysis of hyperaemic LAD flow was recorded as above (fig 3B). During the final minute of infusion, LAD diameter was again recorded in short axis to exclude further flow induced vasodilatation.[29]

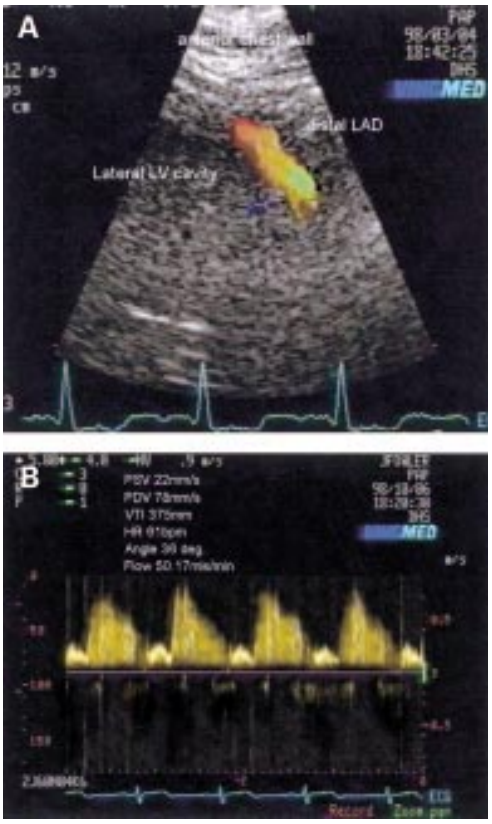

Figure 3 (A) Magnified colour flow mapping of hyperaemic distal left anterior descending coronary artery (LAD) flow. (B) Spectral Doppler trace of hyperaemic distal LAD blood flow (note scale alteration). HR, heart rate; PDV, peak diastolic velocity; PSV, peak systolic velocity; VTI, velocity–time integral.

DATA COLLECTION

The Vingmed CFM 750 incorporates an internal analysis package for use with video playback. Distal LAD diameter was measured at end diastole with internal calipers applied to endothelial borders; therefore intraluminal diameter was gauged as previously validated.[30] Peak velocities were read with spectrum calipers. Velocity–time integral was computed from manually acquired envelope tracings of the Doppler signal over complete cardiac cycles. A mean of three measures was calculated for LAD diameter, flow velocity, and velocity–time integral. Heart rate was calculated from RR intervals of simultaneously recorded ECGs.

MEASUREMENTS AND CALCULATIONS

Velocity distribution within a given cross section of a cylinder is parabolic.[31] Mean velocity is therefore half maximum velocity.[31] In the original validation of coronary Doppler flow wires peak velocities were therefore corrected by a factor of 0.5 for volumetric flow calculations.[32] This correction factor has been adopted by subsequent investigators in the assessment of coronary flow[4 33] and was used throughout this study.

- Body surface area (BSA) was calculated from height and body mass measurements according to the formula of Du Bois and Du Bois[34]: height $(cm)^{0.725} \times$ mass $(kg)^{0.425} \times 0.007184$.
- Body mass index (BMI) was calculated as mass (kg)/height $(m)^2$.
- Diastolic interventricular septal thickness, posterior wall thickness, and internal ventricular diameter were measured according to the recommendations of the American Society of Echocardiography.[27]
- Left ventricular mass (LVM) was calculated according to the Penn convention[28]: left ventricular mass = $1.04((LVID + PWT + IVST)^3 - LVID^3) - 13.6$ g, where LVID = left ventricular internal diameter, PWT = posterior wall thickness, and IVST = interventricular septal thickness.
- Left ventricular mass index (LVMI) was calculated as LVM/BSA.
- Left ventricular hypertrophy was defined according to Devereux's criteria[35] as LVMI > 134 g/m^2 in men and > 110 g/m^2 in women.
- Flow calculations were made according to the method of Doucette and colleagues.[32] Coronary flow was calculated as: $F = \pi(d/2)^2 \times HR \times VTI \times 1/\cos\theta \times 0.5$, where F = flow (ml/min); d = distal LAD diameter (cm); HR = heart rate (beats/min); VTI = velocity–time integral (cm); and $\cos\theta$ = cosine incident angle of Doppler beam.
- Coronary flow reserve was calculated as: $CFR = F^h/F^b$, where CFR = coronary flow reserve; F^h = hyperaemic coronary flow; and F^b = baseline coronary flow.

VARIABILITY

Reproducibility of the technique, interobserver variability, and intraobserver variability were assessed. Intraobserver variability was assessed from 15 studies, with the observer blinded to previous echocardiographic data. For the assessment of interobserver variability, 15 ultrasound studies were examined independently by the unit's senior echocardiographic technician, blinded to prior echocardiographic data. Reproducibility was assessed in five participants who underwent repeat adenosine echocardiography on a separate date. Data for reproducibility, intraobserver variability, and interobserver variability are shown in table 1. They were calculated as the standard deviation of the difference between the two measure-

Table 1 Reproducibility, interobserver variability, and intraobserver variability

Variable	*Interobserver variability (%)*	*Intraobserver variability (%)*	*Reproducibility (%)*
LAD diameter (mm)	3.7	3.2	4.1
IVST (mm)	3.3	2.2	3.6
LVID (mm)	3.5	3.1	4.3
PWT (mm)	4.6	3.5	4.8
LVM (g)	5.1	4.5	6.1
Resting peak diastolic velocity (m/s)	4.7	2.8	5.8
Resting velocity–time integral (mm)	3.7	3.0	3.8
Resting LAD flow (ml/min)	5.5	4.1	6.8
Resting LAD flow (ml/min/100 g LV)	5.8	4.8	7.5
Hyperaemic peak diastolic velocity (m/s)	5.0	4.1	12.1
Hyperaemic velocity time integral (mm)	3.9	3.6	14.1
Hyperaemic LAD flow (ml/min)	6.5	4.5	8.2
Hyperaemic LAD flow (ml/min/100 g LV)	6.7	5.1	7.8
Coronary flow reserve	5.8	3.8	9.1

IVST, interventricular septal thickness; LAD, left anterior descending coronary artery; LV, left ventricle; LVID, left ventricular internal diameter; LVM, left ventricular mass; PWT, posterior wall thickness.

Table 2 Comparative characteristics of endurance athletes and sedentary controls

Variable	*Athletes*	*Controls*	*p Value*
Age (years)	27.3 (6.6)	27.2 (6.1)	NS
BSA (m^2)	1.87 (0.11)	1.87 (0.11)	NS
BMI (kg/m^2)	22.1 (1.9)	23.9 (2.6)	0.01
IVST (mm)	10.8 (0.9)	9.3 (0.9)	< 0.01
LVID (mm)	53.5 (3.4)	49.0 (2.7)	< 0.01
PWT (mm)	9.9 (1.0)	9.2 (0.9)	0.02
LVM (g)	252 (35)	178 (27)	< 0.01
LVMI (g/m^2)	135 (19)	95 (14)	< 0.01
Angle correction	35 (8)	39 (9)	NS
Distal LAD diameter (mm)	2.04 (0.25)	1.98 (0.27)	NS
Distal LAD diameter post-GTN	2.33 (0.33)	2.15 (0.28)	NS (0.06)
Diameter increase post-GTN (%)	14.1 (7.2)	8.8 (5.7)	0.01

Values are mean (SD).
BMI, body mass index; BSA, body surface area; GTN, glyceryl trinitrate; IVST, interventricular septel thickness; LAD, left anterior descending coronary artery; LVID, left ventricular internal diameter; LVM, left ventricular mass; LVMI, left ventricular mass index; PWT, posterior wall thickness.

ments divided by the mean measurement and expressed as per cent of the mean.

STATISTICS

Data distribution was assessed. Within group comparisons were made with the paired Student's *t* test. Between group comparisons were made with the unpaired Student's *t* test. Measurements are given as mean (SD) unless otherwise indicated. Significance was accepted at $p < 0.05$.

Results

Twenty nine male endurance athletes and 23 sedentary male controls were recruited to the study. Subject characteristics are shown in table 2. Of the athletes, 24 were engaged in middle to long distance running (between 800 m and marathon distance) and five were rowers. Duration of intensive training was greater than three years in all athletes, and all were engaged in active training at the time of the study. Mean $\dot{V}O_2$max among the athletes was 71.7 (4.6) ml/kg/min.

ECHOCARDIOGRAPHY

Cross sectional and M mode left ventricular images were adequate in all subjects to allow measurement of interventricular septal thickness, left ventricular internal diameter, and posterior wall thickness, and to allow calculation of left ventricular mass. Twelve athletes (46%) and none of the sedentary controls had left ventricular hypertrophy by echocardiographic criteria (left ventricular mass index > 134 g/m^2).

The distal LAD was adequately imaged in cross section in 20 sedentary controls (87%) and 27 athletes (93%). LAD flow was adequately imaged by spectral Doppler in 19 controls (83%) and 27 athletes (93%). One athlete showed no response to adenosine (see below) and was excluded from further analysis. Paired data were therefore available for 26 athletes and 19 controls.

Subject characteristics, left ventricular indices, and distal LAD diameter measurements are shown in table 2. Ventricular mass and volume were substantially greater among the athletes. Distal LAD diameter was no greater than in the controls. Per cent diameter increase in response to 400 μg sublingual glyceryl trinitrate was, however, significantly greater in the athletes than in the controls.

RESPONSE TO ADENOSINE

Adenosine infusion was tolerated well. No participant requested early termination of the infusion. One athlete developed asymptomatic transient second degree heart block. In one athlete there was no response to adenosine infusion, which has been noted before[36 37] though the mechanism remains unknown. Data relating to this athlete were therefore excluded from subsequent analysis.

Resting and hyperaemic data in athletes and controls are shown in table 3. At rest, heart rate, peak systolic and diastolic velocities, velocity–time integral, and distal LAD flow were all less in the athletes than in the controls. During hyperaemia, peak diastolic velocity, velocity–time integral, and distal LAD flow were all greater among the athletes, while heart rate remained lower. Coronary flow reserve was therefore supranormal in athletes in comparison with controls, at 5.9 (1.0) *v* 3.7 (0.7). Coronary flow reserve in athletes with (n = 12) and without (n = 14) left ventricular hypertrophy did not differ significantly, at 6.0 (0.9) *v* 5.9 (1.2), respectively.

Discussion

We found that coronary flow reserve in endurance athletes is supranormal: resting coronary flow is lower than in sedentary controls while hyperaemic coronary flow is greater. Scaled for left ventricular mass, resting coronary flow remained lower in endurance athletes, while hyperaemic coronary flow was similar to that of controls. The physiological significance of these findings is not clear, but it is likely to reflect the greater efficiency of the exercise trained heart.[21 38–40]

Table 3 Comparison of distal left anterior coronary artery diameter, flow velocities, and flow reserve between endurance athletes and sedentary controls

	Resting			*Hyperaemic*		
Variable	*Athletes*	*Controls*	*p Value*	*Athletes*	*Controls*	*p Value*
PSV (m/s)	0.06 (0.08)	0.11 (0.04)	0.02	0.27 (0.25)	0.21 (0.19)	NS
PDV (m/s)	0.18 (0.06)	0.27 (0.08)	< 0.01	0.90 (0.20)	0.77 (0.19)	0.02
VTI (mm)	90 (21)	117 (26)	< 0.01	432 (83)	364 (62)	< 0.01
Heart rate (beats/min)	56.5 (7.9)	68.7 (8.8)	< 0.01	68.4 (13.7)	77.5 (10.4)	0.02
Flow (ml/min)	10.6 (3.1)	14.3 (3.6)	< 0.01	61.9 (17.8)	51.1 (14.6)	0.02
Flow (ml/min/100 g)	4.4 (1.2)	8.2 (2.2)	< 0.01	25.9 (5.6)	28.5 (7.4)	NS
Coronary flow reserve	–	–	–	5.9 (1.0)	3.7 (0.7)	< 0.01

Peak systolic velocity (PSV), peak diastolic velocity (PDV), and velocity–time integral (VTI) are angle corrected values.

HUMAN STUDIES

There are only four previous studies of coronary flow in athletes.[41–44] Heiss and colleagues compared 11 athletes with 11 untrained students[41] using argon clearance. They found coronary flow to be lower in trained athletes, both at rest and during exercise. The exercise protocol, however, was submaximal, and the argon clearance technique has subsequently been found to underestimate hyperaemic flow.[45] Haskell and colleagues performed coronary angiography on 11 ultradistance runners and 11 controls with atypical chest pain.[42] In contrast to other investigators,[46–49] they found proximal coronary dimensions to be no greater in athletes than in controls. However, coronary dilating response to glyceryl trinitrate was significantly greater in the athletes, implying enhanced endothelium independent vasodilator capacity, as has been shown in exercise trained pigs.[19] Our study corroborates this observation. Radvan and colleagues undertook a study of coronary flow reserve using positron emission tomography in 10 rowers and 10 patients with hypertrophic cardiomyopathy.[43] They found resting coronary flow (per gram left ventricular muscle mass) did not differ significantly between athletes and patients, but that vasodilator reserve in response to intravenous dipyridamole was significantly attenuated in the patients with hypertrophic cardiomyopathy. Recently, Toraa and associates assessed coronary flow reserve in athletes, using positron emission tomography.[44] They found that *total* resting coronary flow was similar in athletes and controls, and calculated a coronary flow reserve of 6.1 in athletes and 3.8 in controls.

ANIMAL STUDIES

Studies of coronary flow in exercise trained animals offer conflicting results. Resting coronary flow per unit left ventricular mass has been described as lower,[38] the same,[22 50] or greater[51] than in the untrained state. Maximum coronary flow has been found to be the same[50 52] or greater than in the untrained state.[18 22] As a result, coronary flow reserve has been either normal[50 52] or supranormal.[18 22 51] The studies which showed no increase in coronary flow reserve, however, employed training regimes of only 4–12 weeks, while those that involved longer exercise training programmes (16–22 weeks) clearly showed increased pharmacological sensitivity to adenosine in the trained state.[19 22] The effects in animals of years of intensive training—such as endurance athletes undergo—are unknown.

ECHOCARDIOGRAPHY

Echocardiographic indices of left ventricular wall thickness and left ventricular mass were similar to published data from normal[53] and athletic individuals.[54] Interventricular septal thickness was greater among the athletes but did not exceed 13 mm in any individual. Internal ventricular diameter was a mean of 9.2% greater among the athletes—similar to the 10% reported by Maron.[55] Left ventricular mass and left ventricular mass index were therefore substantially greater among the athletes, but only 12 of 26 (46%) met Devereux's criteria for left ventricular hypertrophy.[35] Data on the prevalence of left ventricular hypertrophy in athletes vary, but the most comprehensive study found physiological hypertrophy only in a minority.[56]

Distal LAD diameter and blood flow were adequately visualised in 93% of the athletes—a higher proportion than previously reported.[6 30 57 58] This success rate reflects the youth and lean upper body mass of the athletes, which made them ideal echocardiographic subjects.

CORONARY FLOW RESERVE

Resting distal LAD flow was lower in the athletes, while hyperaemic flow was greater. Coronary flow reserve was therefore significantly greater among the athletes. One of the main factors mediating this increased coronary flow reserve is the resting bradycardia. For a given individual, if "resting" heart rate is increased above baseline by pacing, resting coronary flow increases while hyperaemic flow is unchanged. Coronary flow reserve therefore diminishes linearly with increasing resting heart rate.[59–62] Extrapolation of this observation below normal resting heart rates would suggest that resting bradycardia might augment coronary flow reserve, and certainly sedentary pigs subject to long term pacing induced bradycardia do show reduced resting coronary blood flow in comparison with sham operated controls.[38] Resting bradycardia is therefore likely to account for some of the observed increase in coronary flow reserve.

Hyperaemic flow in athletes is increased, but this increase is negated by scaling for left ventricular mass. This might suggest that there is not a genuine increase in peak myocardial vascular capacity in trained individuals, rather that hyperaemic flow for a given area of myocardium remains maximal and is unaffected by exercise training. Histopathological analysis of myocardial structure in exercise trained animals, however, has—with a couple of exceptions[39 50]—provided evidence of increased myocardial capillary density.[63–67] Despite this, not all animal studies have shown an increase in maximum coronary flow per unit mass in the trained state, and therefore any relation between myocardial capillary density and maximum coronary flow remains conjectural.

INFLUENCE OF THE ENDOTHELIUM

Enhanced endothelium dependent coronary vasodilatation,[20 68–70] and enhanced endothelium independent coronary vasodilatation[18 19 21 22 69 71] have both been found in exercise trained animals. More recent human studies have suggested increased endothelium dependent vasodilatation in response to training.[72 73] Our work supports the concept of augmented endothelium independent coronary vasodilatation in the trained state.

INFLUENCE OF HYPERTROPHY

Coronary flow reserve is impaired in pathological hypertrophy[12 13 15–17] owing to coronary

medial thickening,[74] increased perivascular collagen,[75] interstitial fibrosis,[76] and fibre hypertrophy.[77] We have shown, by contrast, that varying degrees of physiological hypertrophy do not necessarily impair coronary flow reserve in man, and this is corroborated by previous research in greyhounds.[78] This could prove useful in differentiating athlete's heart—in which coronary flow reserve is in the normal to supranormal range—from hypertrophic cardiomyopathy, in which coronary flow reserve is markedly attenuated.[79]

LIMITATIONS

In recording blood flow, angles of incidence above 30° are undesirable as cosθ begins to assume greater overall importance (cos 30 = 0.87). However, in all cases flow was individually angle corrected and therefore the potential significance of this variation was minimised.

Cardiac motion dictated that systolic and diastolic envelopes were not always of equal clarity. Therefore systolic and diastolic velocity–time integral could not always be recorded from identical spectral envelopes.

For a given coronary perfusion pressure (within limits of autoregulation) alterations in aortic pressure produce parallel increases in basal and maximum coronary flow, such that coronary flow reserve remains the same.[59 60] Therefore continuous monitoring of systemic arterial pressure was not undertaken.

Accurate measurement of coronary flow reserve depends on obtaining true resting coronary flow. Echocardiography is relatively unstressful, and certainly less so than coronary angiography, but even in this study true resting flow may not have been achieved in all participants, thus artificially reducing coronary flow reserve.

We could not be absolutely sure that our population samples did not include subjects with minor coronary disease or abnormalities, but short of undertaking intravascular ultrasound, a guarantee of this nature was not possible, and adequate precautions were taken to avoid inclusion of those with possible cardiovascular disease. As this was a non-invasive study, we were at least able to avoid the unsatisfactory option of recruiting patients with chest pain but angiographically normal coronary arteries—a practice that has been fairly criticised.[80]

CONCLUSIONS

Coronary flow reserve in endurance athletes is supranormal, largely as a result of diminished resting coronary flow rather than augmented hyperaemic coronary flow. The increase in distal LAD diameter with sublingual glyceryl trinitrate was greater in athletes than controls, suggesting that endothelium independent vasodilatation is enhanced by exercise training. Pathological hypertrophy is known to limit coronary flow reserve, but physiological hypertrophy does not have the same effect. Hypertrophy of the myocardium on its own therefore does not necessarily impair coronary flow reserve. Adenosine transthoracic echocardiography continues to be a promising technique for investigating coronary flow reserve.

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