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Margareetta Tummavuori

Long-Term Effects of Physical Training on Cardiac Function and Structure in Adolescent Cross-Country Skiers

A 6.5-Year Longitudinal Echocardiographic Study

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ABSTRACT

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The purpose of this 6.5-year follow-up study was to investigate the impact of training on the structure and function of the heart of young endurance athletes with the help of echocardiographic variables and maximal oxygen uptake ($\dot{V}O_{2max}$), which indicates the capacity of maximal cardiovascular function. This study also sought to determine at which age the structural and functional changes in the heart took place, the impact on cardiovascular function of short-term (6 - 7 months) training during three different training seasons, and the effect of body size and growth. The significance of the size of the heart at the baseline situation was examined by dividing the 16-year-old male cross-country skiers (n = 20) into two groups one comprising those with small hearts and the other comprising those with large hearts according to the end-diastolic diameter (EDD) of the left ventricle. EDD, septal wall thickness in diastole (SWTd) and posterior wall thickness in diastole (PWTd) of the left ventricle of the heart increased (7.8, 12.5 and 9.8 %, respectively) significantly during the 6.5 years. EDD increased significantly during the first three years (age 16 - 19), while the walls remained unchanged, whereas during the last three years (age 19 - 22) EDD did not increase, but the walls thickened significantly. $\dot{V}O_{2max}$ increased during the first three years and evened out thereafter. The annual echocardiographic changes and those of $\dot{V}O_{2max}$ were small. There were no significant changes during the different training seasons. EDD, normalized with body surface area (BSA), did not change during the study. In the baseline situation the EDD of those with small hearts increased during the study more than the EDD of those with large hearts, but without reaching comparable values even at the end of the study. There were no differences in wall thickness between the groups. EDD increased more in the group where BSA had increased more. The skiers tended to have a larger EDD, and their SWTd and PWTd were thicker than those of control subjects of the same age. In conclusion it can be stated that genetic factors and growth had a great impact on the size of the heart of the skiers and control subjects, and that young cross-country skiers, thanks to endurance training, first experience dilatation of the left ventricle of the heart and then hypertrophy of the walls.

Key words: athlete's heart, echocardiography, end-diastolic diameter, wall thickness, maximal oxygen uptake, long-term training, young cross-country skiers

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Margareetta Tummavuori

This thesis is based on the following international congress presentations (9). Abstracts are given in Appendices 20 - 28. In addition some unpublished results are presented.

LIST OF CONGRESS PRESENTATIONS

Tummavuori, M. & Rusko, H. (1993) Development of the athlete's heart during three year's training in young cross-country skiers. American College of Sports Medicine (ACSM) Annual Meeting. 2-5.6.1993, Seattle, Washington, USA. Med. Sci Sports Exerc. 25: S132, 1993.

Tummavuori, M. & Rusko, H. (1993) Echocardiographic changes during two skiing seasons in young cross-country skiers. FIMS 7th European Sports Medicine Congress, 7-10.10.1993, Nicosia, Kypros. PD42, 217.

Tummavuori, E.A.M. & Rusko, H.K. (1994) The influence of the initial heart size on the development of the athlete's heart. American College of Sports Medicine (ACSM) Annual Meeting 1-4.6.1994, Indianapolis, Indiana, USA. Med. Sci Sports Exerc. 26: S182, 1994.

Tummavuori, M. & Rusko, H. (1994) Development of the athlete's heart during five years' training in young athletes. International congress on applied research in sports, 9-11.8.1994, Helsinki, Finland. P. 40, Proceedings p.271-274.

Tummavuori, M. & Rusko, H. (1995) Maximum oxygen uptake and the development of the athlete’s heart. Third IOC World Congress on Sport Sciences, September 16-22, 1995 Atlanta, Georgia, USA. P. 356.

Tummavuori, M. & Rusko, H. (1996) Training and detraining effects on the athlete’s heart during five years’ echocardiographic study. American College of Sports Medicine (ACSM) Annual Meeting 29.5.-1.6.1996 Cincinnati, Ohio, USA. Med. Sci Sports Exerc. 28: S69, 1996.

Tummavuori, M. and Rusko, H. (1997) Development of the athlete’s heart during six years’ echocardiographic study. Fourth IOC World Congress on Sport Sciences, October 22-25, Monte-Carlo, Monaco.

Tummavuori, M. and Rusko, H. (2002) Influence of endurance training for six years on the heart size and maximal oxygen uptake of young endurance athletes. Science for Success–Current Trends in Applied Research and Competitive Sport in Europe, eds. Siukonen, S., Sinisalo, M., Rusko, H. & Blomqvist, M., KIHU – Research Institute for Olympic Sports, Jyväskylä, Finland, October 2-4. 2002, p.108.

Tummavuori, M. and Rusko, H. (2003) Annual echocardiographic changes in young endurance athletes during six years. 7th IOC Olympic World Congress on Sport Sciences, 7-11 October, Athens, Greece. Book of Abstracts, p. 116B.

ABBREVIATIONS

a-v O_2 difference	arteriovenous oxygen difference
BLa_{max}	maximal blood lactate concentration, mmol · l^{-1}
BSA	body surface area (m^2)
BV	blood volume, l
DBP	diastolic blood pressure, mmHg
dP/dt	rate of change of the ventricular pressure with respect to time
ECG	electrocardiogram
EDD	end-diastolic diameter of left ventricle, cm
EDV	end-diastolic volume, ml
EF	ejection fraction
ESD	end-systolic diameter of left ventricle, cm
ESV	end-systolic volume, ml
FS	fractional shortening, %
HI	hypertrophy-index
HR	heart rate, beats · min^{-1}
HR_{max}	maximal heart rate, beats · min^{-1}
IC	index of contractility
LVM	left ventricular mass, g
MHz	megahertz
MRI	magnetic resonance imaging
MWT	mean wall thickness, cm
O_2	oxygen
PV	plasma volume
PWTd	posterior wall thickness of left ventricle, cm
$\dot{Q}$	cardiac output, l · min^{-1}
$\dot{Q}_{max}$	maximal cardiac output, l · min^{-1}
RCM	red cell mass, l
SaO_2	oxygen saturation, %
SBP	systolic blood pressure, mmHg
SD	stroke dimension, cm
SV	stroke volume, ml
SV_{max}	maximal stroke volume, ml
SWTd	septal wall thickness of left ventricle, cm
$\dot{V}O_2$	oxygen uptake, (l · min^{-1}, ml · kg^{-1} · min^{-1})
$\dot{V}O_{2max}$	maximal oxygen uptake, (l · min^{-1}, ml · kg^{-1} · min^{-1}, ml · $kg^{-2/3}$ · min^{-1})
$\dot{V}O_{2max\ demand}$	peak workload during $\dot{V}O_{2max}$ test
WS	wall stress index

CONTENTS

1 INTRODUCTION

The athlete's heart has been one of the oldest, most interesting and most stimulating objects of study in sports medicine. The first study of dilated human hearts was found by S.E. Henschen, director of the medical clinic of Upsala University in 1899, when he examined cross-country skiers using the percussion method, and compared his discoveries with the hearts of control subjects. Skiing, especially when started already in the years of adolescence easily dilates the heart and the thickness of the dilated heart increases when it has to work harder (Henschen 1899). Henschen (1899) also discovered that both the left and the right side of the heart increase, and according to him "the biggest hearts will win the race". Scientists have debated about Henschen's discovery of the healthy response created by training, claiming that the dilatation of the hearts of athletes is pathological, caused by overexertion, or by possible earlier heart disease (Mills et al. 1997). "Athlete's heart" may be a somewhat misleading term, as not all athletes, even if they have trained and competed at a very high level, develop athlete's heart (Urhausen & Kindermann 1999).

Regular long-term training leads to an increase in heart mass, which is caused by the dilatation of the end-diastolic diameter of the left ventricle (EDD) or hypertrophy of the septal wall (SWTd) or posterior wall (PWTd) of the left ventricle, or both (Maron et al. 1995, Oakley 2001). The physiological enlargement of the heart depends on the sport, intensity of training, volume of training and training years, body size, sex, age and genetic factors (Pelliccia 1996). If the exercise is of the volume overload type the effect is eccentric, and if the exercise is one of pressure overload the effect on the heart muscle is concentric (Richey & Brown 1998). Endurance sports such as cycling, cross-country skiing, canoeing and rowing had the greatest impact on the increase in EDD (Pelliccia et al. 1999). Cycling, rowing, canoeing and swimming had most effect on both EDD and wall thickness, as the training in these sports was of both the volume and pressure overload types (Pelliccia et al. 1991, Shapiro 1992). Power sports such as weight lifting had a strong impact on the wall thickness of the left ventricle, thanks to pressure overload training, but only a mild effect on EDD. Ball games and team sports such as football, ice hockey and

tennis had an intermediate level of impact on EDD and wall thickness (Pelliccia 1996).

Among endurance athletes cross-country skiers have the highest maximal oxygen uptake ($\dot{V}O_{2max}$). The $\dot{V}O_{2max}$ values of 80 - 85 ml · kg^{-1} · min^{-1} reached by Finnish male cross-country skiers in the 1960s and 1970s increased on the average to 90 ml · kg^{-1} · min^{-1} in the 1990s, the highest individual value being 93 ml · kg^{-1} · min^{-1}. The differences in $\dot{V}O_{2max}$ were significant between world-class skiers, medium-class skiers and less successful skiers, equally high values being shown by free and classical technique specialists (Rusko 2003). According to Bergh (1987) an internationally successful cross-country skier must have a $\dot{V}O_{2max}$ at least 350 ml · kg$^{-2/3}$ · min^{-1}. Young Norwegian world-class top skiers had a $\dot{V}O_{2max}$ of about 350 ml · kg$^{-2/3}$ · min^{-1} (Ingjer 1992), and a Finnish world-class top male skier had a $\dot{V}O_{2max}$ of 368 ml · kg$^{-2/3}$ · min^{-1} (Rusko 2003); thus a good $\dot{V}O_{2max}$ led to a good success at competitions.

There exist no previous echocardiographic longitudinal studies where young top-class endurance athletes have been monitored intensively over a period of several years. The purpose of this study was to examine 16-year-old cross-country skiers over a continuous period of 6.5 years, and to collect data on the structural and functional changes that take place in the heart with training. The echocardiographic variables were examined at rest, and maximal cardiovascular capacity was measured during a ski-walking test on the treadmill. The results were used to determine the age at which the structural and functional changes of the heart took place, whether the increase in heart size was linear or whether there were phases of levelling off during this increase. Also, the significance of the size of the heart at baseline was examined by dividing the subjects into groups according to whether they had small or large hearts as determined by the baseline measurements of EDD. The effects of short-term training (6 - 7 months) were examined during three different training seasons. To evaluate the effect of biological growth and training, the results obtained for the athletes were compared with those of control subjects of the same age.

2 REVIEW OF THE LITERATURE

2.1 General Overview on the Long-Term Effects of Physical Exercise Training on Cardiac Structure and Function

2.1.1 The Mechanical Function of the Myocardium

The contraction of a heart muscle cell is regulated by four clearly distinguishable, though interdependent factors: preload, afterload, contractility and heart rate (HR). To be able to estimate the contractile properties of a muscle it is important to know the degree of tension of muscle fibers (preload) when the muscle begins to contract, or the power that stretches the muscle before contraction, the final diastolic length of the fibers compared to the resting length tension of the muscle (Janicki et al. 1996, Richey & Brown 1998). Afterload is a resistance load affecting the contractions of the ventricles, which prevents the heart muscle fibers from shortening during ejection, as an increase in resistance has a negative influence on cardiac performance through increasing heart workload (Janicki et al. 1996). Systolic stress (afterload) is a major determinant of ejection performance (Gunther & Grossman 1979). Endurance training increases venous return and thereby increasing preload and simultaneously, exercise-induced vasodilatation results in a decreased afterload. Resistive training increases blood pressure, which increases afterload and decreases venous return by the Valsalva-maneouvre, thereby decreasing preload (Effron 1989). As for myocardial contraction, blood volume in end-diastolic volume (EDV) is regarded as preload. Preload can also be expressed as end-diastolic pressure at the point when the ventricles have filled. During exercise cardiac output ($\dot{Q}$), blood volume (BV), body position, the pumping action of respiration and skeletal muscles and venous tone affect the preload. The afterload is the pressure in the arteries starting from the ventricles. An increased afterload has a negative influence on cardiac performance, because it increases the workload of the heart (Brooks & Fahey 1985, Janicki et al. 1996). The greater maximal stroke volume (SV_{max}) of young endurance athletes reflects greater ventricular diastolic filling (preload) (Rowland et al. 2002).

The phenomenon which takes place in the cavities of the heart during the cardiac cycle is called the Starling or Frank-Starling law or mechanism. The strength of the cardiac contraction depends on the initial length of the myocardial fibers before the systolic phase. The relationship between contractile force and the resting length of the heart muscle fibers, was first described by the physiologists Otto Frank (1895) and E.H. Starling (1920). According to the Frank-Starling law there is a direct connection between the diastolic volume of the heart and the force of contraction (Janicki et al. 1996).

Conflicting results have been obtained on the effects of the Frank-Starling mechanism regarding the increase in stroke volume (SV), how much is gained through the Frank-Starling mechanism during dynamic exercise and what influence physical stress has on sympathetic overactivity. The mechanism has been found to function with untrained people during light exercise (Plotnick et al. 1986, Poliner et al. 1980, Steingart et al. 1984), and during heavy but not submaximal exercise, where the increase of HR increased $\dot{Q}$ (Weiss et al. 1979). It has been assumed that the increase in SV at maximal or almost maximal exercise rather than the Frank-Starling mechanism has predominantly accounted for the enhanced sympathetic stimulation (Plotnick et al. 1986). During exercise in both supine and upright positions SV was maintained both by the Frank-Starling mechanism and increased contractility (Poliner et al. 1980), whereas according to Fedele et al. (1985) Frank-Starling mechanism functioned at a lower exercise level and $\dot{Q}$ increased. As the exercise became heavier contractility increased, mostly due to the increase in adrenergic activity.

The SV of endurance runners increased progressively during exercise, and was explained by increased contractility, adrenergic stimulation and the Frank-Starling mechanism. $\dot{Q}$ achieved by the control subjects at the end of exercise was maintained with the help of HR and adrenergic stimulation without the operation of the Frank-Starling law. During recovery the EDV of both groups significantly increased as a result of increased venous return and larger filling of the ventricles by the activation of the Frank-Starling mechanism, especially in the athletes, and which increased the ejection fraction (EF) and SV to some extent in both groups (DiBello et al. 1996). The Frank-Starling mechanism functioned with competitive athletes, as both SV and EDV increased (Crawford et al. 1985, Rerych et al. 1980).

The higher HR found in the upright position compared to the supine position compensated for the smaller SV, owing to the larger venous return in the supine position, and the Frank-Starling mechanism functioned better than in upright position (Sato et al. 1999). In the study by Leyk et al. (1994) $\dot{Q}$ was larger in the supine position at rest and during light exercise, but during heavier exercise in the upright position $\dot{Q}$ approached and even exceeded the values obtained for the supine position. Thus diastolic filling is larger in sports where the body is in a horizontal position, such as swimming, and the blood flow to the heart is optimal.

Levine et al. (1991) showed when studying the relations between pulmonary capillary wedge pressure (PCWP) and SV, that endurance athletes had greater ventricular diastolic compliance and distensibility than nonathletes. The study also showed that as PCWP decreased e.g. from 10 to 5 mmHg, as may happen when moving from a supine to upright posture, the SV of athletes decreased significantly more than the SV of nonathletes. This may explain the orthostatic hypotension found in well-trained endurance athletes.

The increase of the size of the heart caused by training may lead to a situation where the pericardium has stretched to a point where its elastic properties restrict the further dilatation of the heart and the use of Frank-Starling mechanism. After a pericardiectomy $\dot{V}O_{2max}$, SV and $\dot{Q}$ increased significantly, maximal heart rate (HR_{max}) remaining unchanged, in untrained dogs (Stray-Gundersen et al. 1986) and pericardiectomy in pigs led to a 30-percent growth in maximal cardiac output ($\dot{Q}_{max}$) and $\dot{V}O_{2max}$ (Hammod et al. 1992). It is difficult to estimate the significance of the pericardium in people, but it has been assumed that it has been the essential reason for atrial and ventricular intracavity pressure in patients who have undergone an aortic valve replacement or coronary artery bypass (Tyberg et al. 1986) and during exercise in patients who suffered from different cardiac insufficiencies (Janicki 1990).

In addition to preload and afterload one of the main mechanisms of the heart muscle is contractility (inotropic action). The positive inotropic action of the sympathetic system enables an increase in contractility, and the ventricles are able to pump a larger SV or to pump against a higher pressure without an increase in EDV. Contractility is independent on the changes in the length of the myocardial fibers and independent of preload and afterload (Janicki et al. 1996). Defining the contractility of the heart is difficult, but often the change in contractility is precisely the opposite of that in HR. Contractility can be defined as the rate of change of ventricular pressure with respect to time (dP/dt, mmHg/sec) or aortic flow (e.g. max dQ/dt), and can be measured with cardiac catheters (Janicki et al. 1996). An improvement in contractility shortens the systolic filling period (Rowland et al. 2000). Endurance athletes have a higher EF both at rest and during exercise than sedentary or moderate active controls indicating an enhanced myocardial contractility (Jensen-Urstad et al. 1998).

The fourth factor affecting the action of the heart is HR or the frequency of the contraction of the heart muscle. A lower HR is the most positive effect created by aerobic training (Charlton & Crawford 1997, Fleck 1988) and it is connected with high $\dot{V}O_{2max}$ (Kenney 1985). Rest heart rate is considered to fall because of the increase in parasympathetic tonus (De Meersman 1992). Static muscle exercise, on the contrary, does not cause changes in the parasympathetic regulation of the heart (Huonker et al. 1996).

The Figure 1 shows the factors that affect $\dot{Q}$ during exercise.

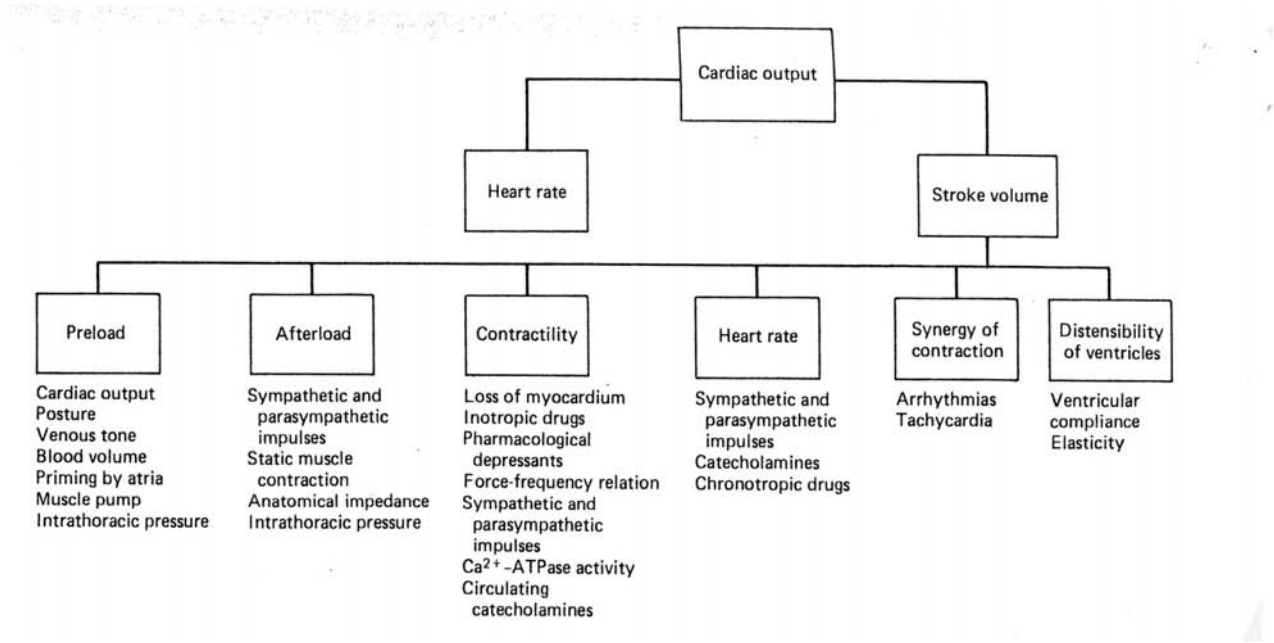

FIGURE 1 Factors affecting cardiac output during exercise (Brooks & Fahey 1985)

2.1.2 The Regulation of the Action of the Heart

The impact of the sympathetic and parasympathetic components of the autonomic nervous system is important for the regulation of the action of the heart. They regulate the rate of HR (chronotropic action), contractile force (inotropic action) and the velocity of the atria-ventricular conduction (dromotropic effect) (Green 1990). Two different types of receptors, α and β can be found in the sympathetic adrenergic effect, and their stimulation can have different impacts. For example, the blood vessels of the muscle and skin α-adrenergic receptors cause vasoconstriction, whereas β-receptors cause vasodilatation (Brooks & Fahey 1985).

During light physical exercise sympathetic activity becomes predominant, and tachycardia is caused by the decrease in parasympathetic tone. As the exercise increases, HR increases because of the further vagal withdrawal and the increase in concomitant sympathetic activation. During maximal exercise parasympathetic activity further diminishes while sympathetic activity increases greatly (Maciel et al. 1986, O'Leary 1996, Rowell & O'Leary 1990, Seals et al. 1988, Yamamoto et al. 1991). Cardiac parasympathetic modulation increased and sympathetic modulation decreased in sedentary men following endurance training varying from five weeks to eight months (Ekblom et al. 1973, Shi et al. 1995). People who are in good physical condition have had more enhanced cardiac vagal function during exercise than those in poor condition (Tulppo et al. 1998). According to Kenney (1985) bradycardia caused by training is due more to increased parasympathetic control than any marked decrease in sympathetic stimulation. Similarly Bryan et al. (1992) and Huston et al. (1985)

found that the bradycardia of the endurance athletes has been caused by increased vagal tonus. According to De Meersman (1992) the improved aerobic capacity caused by an eight-week intensive running training markedly increased parasympathetic activity. During dynamic arm exercise submaximal HR was higher than during leg exercise at the same $\dot{V}O_2$ levels although HR_{max} was lower (Tulppo et al. 1999). Pharmacological blockers have been used to estimate the amount of the size of the sympathetic and parasympathetic nervous systems (Barney et al. 1988, Berntson et al. 1994, Smith et al. 1989). HR increased at rest, when the influence of the parasympathetic nervous system was blocked intravenously with atropine, and HR increased along with doses of atropine (Tulppo et al. 1996). Parasympathetic influence was significantly greater in endurance runners at rest than in controls, and sympathetic influence was slightly inferior in endurance runners (Smith et al. 1989).

The control mechanism outside the heart consists of baroreceptors. The baroreceptors are stimulated by blood pressure created by the stretching of the arterial wall. The impulses starting from the baroreceptors go to the cardioinhibitory and vasomotor centres in the medulla oblongata, where they inhibit the sympathetic system and excite the parasympathetic system. When blood pressure decreases, the rate of excitement of the baroreceptors decreases thereby increasing sympathetic activity and causing the opposite reactions: arterial pressure increases, the velocity of cardiac contraction increases, $\dot{Q}$ increases and blood pressure increases (Brooks & Fahey 1985, Ganong 1999).

Hormones also affect the action of the heart. Catecholamines, thyroid hormones and glucagons have positive inotropic (increase the force of contraction) and chronothropic (increase HR) effects on the heart. Glugocorticoids, mineralocorticoids, adrenocorticotropic hormone, thyroid stimulating hormone and growth hormone have an indirect effect on the heart by changing blood pressure or blood volume (Brooks & Fahey 1985, Ganong 1999, Richey & Brown 1998).

2.1.3 The Pump Function of the Heart

Blood is circulating by two pumping systems: the central cardiac pump and the peripheric pump, which is responsible for systemic venous return (Rowland 2001). The amount of blood pumped by the heart is regulated by the intrinsic regulation of the heart, which reacts to changes in the amount of blood flowing into the heart (Frank-Starling mechanism) and by regulation of the autonomic nervous system. As a result of the increase in venous return caused by the pumping action of exercising muscles SV increases (Booher & Smith 2003). During maximal dynamic exercise a fourfold increase in $\dot{Q}$, threefold increase in HR and twofold increase in SV compared to resting values were found (Charlton & Crawford 1997). The pump function of the skeletal muscles and the suction effect of the left ventricle are principal factors in the systemic venous return, and during exercise the peripheric pump seems to "drive" the circulation (Rowland 2001).

The size of the heart and SV of cross-country skiers are larger than those of untrained people, and they effect the increase in $\dot{Q}_{max}$ and $\dot{V}O_{2max}$ (Rusko 2003). The SV of athletes has been found to increase up to the maximal level of $\dot{V}O_2$ (Crawford et al. 1985, Fleg et al. 1994, Gledhill et al. 1994, Seals et al. 1994). During peak exercise the venous return rises to fourfold its rest value, whereas SV increases by 30 - 40 % and the size of the left ventricle changes slightly. After the initial phases of progressive exercise an increase in progressive venous return occurs together with the stable SV. The increase in HR during exercise must match with the increase in venous return to enable stable diastolic filling and SV (Rowland 2001).

Red cell mass (RCM) and plasma volume (PV) increase with endurance training; hence the venous return from the muscles to the heart grows during exercise. The increased SV stretches the heart muscle, thereby also increasing $\dot{Q}$ (Rusko 2003). During exercise in the upright position the effect of gravity sequesters blood in the lower limbs, thereby creating a difference of pressure of 90 mmHg between the legs and the heart. The peripheric pump mechanism has to be efficient to be able to overcome this pressure differential and thus achieve the sufficient diastolic filling of the heart needed during exercise. The cardiac systolic function maintains but does not dictate circulatory flow (Rowland 2001). A break in a few weeks decreased blood volume during exercise in the upright position, which led to a decrease in SV and $\dot{V}O_{2max}$ and an increase in HR (Coyle et al. 1986). $\dot{Q}$ and $\dot{V}O_2$ correlated positively. $\dot{Q}$ has increased almost linearly with $\dot{V}O_2$ in untrained people and endurance athletes, and neither age nor gender had a significant influence on linearity during submaximal exercise in endurance athletes who had trained for many years (Leyk et al. 1994, Proctor et al. 1998).

2.1.4 Maximal Oxygen Uptake ($\dot{V}O_{2max}$)

According to the Fick equation $\dot{V}O_{2max}$ = maximal cardiac output ($\dot{Q}_{max}$) x maximal arteriovenous oxygen difference (a-v O_2 difference). The maximal a-v O_2 difference changes very little with training; thus the exercise-induced difference between endurance athletes and sedentary individuals is very small (Brooks & Fahey 1985). Because $\dot{Q}$ of top endurance athletes can be up to 40 l · min^{-1}, or almost twofold that of inactive people (Bassett, Jr. & Howley 2000), $\dot{Q}$ is the limiting factor or the most important factor defining $\dot{V}O_{2max}$ (Rowell 1986). $\dot{Q}$ rises nearly linearly with $\dot{V}O_2$. There is a slope of approximately 6 l · min^{-1} of $\dot{Q}$ per 1 l · min^{-1} in $\dot{V}O_2$ in active subjects and endurance athletes (Faulkner et al. 1977, Leyk et al. 1994, Proctor et al. 1998, Rowell 1986). Cardiac functional capacity or the ability to generate $\dot{Q}$ is the predominant factor accounting for the greater $\dot{V}O_{2max}$ in endurance athletes compared with nonathletes (Rowell 1986). The higher $\dot{Q}_{max}$ found in athletes reflects a greater SV_{max} (Gledhill et al. 1994, Hanson & Tabakin 1965, Rowland et al. 2000).

During exercise HR is the most important factor increasing $\dot{Q}$. At rest $\dot{V}O_2$ is fairly high in the tissues of the heart muscle and low in the skeletal muscles. During exercise the $\dot{V}O_2$ of the heart muscle rises to threefold or fourfold its resting level and the $\dot{V}O_2$ of the skeletal muscles involved can increase 20 - 50-fold (Grote 1989). The oxygen transport capacity of the central cardiovascular system is considered the main determinant of $\dot{V}O_{2max}$. Usually 80 - 85 % of the available oxygen is used in $\dot{V}O_{2max}$. SV is the most important variable determining individual differences in $\dot{V}O_{2max}$ (Rowell 1986). Because the HR_{max} of athletes and non-athletes is almost the same, the larger heart and larger SV of athletes increase $\dot{Q}_{max}$ and further $\dot{V}O_{2max}$. The greatest differences between non-athletes and top athletes in $\dot{V}O_{2max}$ can be seen in SV_{max} and $\dot{Q}_{max}$ (Rowell 1986, Rusko 2003): 75 ml vs. 200 ml; 15 l vs. 37 l, respectively, the $\dot{V}O_{2max}$ values being 30 and 87 ml · kg^{-1} · min^{-1} (Rusko 2003). Even though a high $\dot{V}O_{2max}$ is necessary for elite level competitors, it does not necessarily guarantee success; for example, to be successful cross-country skiers must be able to sustain a good speed or high proportion of $\dot{V}O_{2max}$ throughout duration of the race (Hoffman & Clifford 1992). On the basis of measurements of competition (10 and 20 km) HR top skiers rarely ski using less than 85 % of $\dot{V}O_{2max}$ (Bergh 1982), or they ski near the anerobic threshold, during competitions. Droghetti et al. (1985) also found a strong correlation between the anaerobic threshold and average competition speed in skiers in the Italian national team.

Combined upper and lower body training requires increased $\dot{V}O_2$ and blood flow into the muscles, and so higher $\dot{Q}_{max}$ is needed (Rusko 2003). During exercise done with the arms $\dot{V}O_2$ was 20 - 30 % lower than during exercise done with the legs (Miles et al. 1989, Sawka 1986), and a 2 - 3 % higher $\dot{V}O_{2max}$ was reached in a ski-walking test than running uphill (Hermansen 1973, Strømme et al. 1977). $\dot{V}O_{2max}$ was equally high with the same protocol during tests run on the treadmill and on the track, although a greater maximal speed was reached in the field, and the exercise lasted longer, due to the better running economy on the track (Meyer et al. 2003). The upper body $\dot{V}O_{2max}$ of cross-country skiers is nowadays about 90 % of that of the lower body $\dot{V}O_{2max}$ in both male and female skiers (Rusko 2003). Among endurance athletes cross-country skiers have always had the highest $\dot{V}O_{2max}$ among endurance athletes, and whether l · min^{-1} or ml · kg^{-1} · min^{-1} is more important depends on the profile of the ski-track, snow conditions and the gliding characteristics of the skis. During steep uphill skiing and poor gliding conditions ml · kg^{-1} · min^{-1} seems to be more important, whereas l · min^{-1} and large body mass are more important on gently inclined uphill sections, on flat terrain and downhill irrespective of gliding conditions (Rusko 2003). The best index to discriminate between the skiers of different sizes is $\dot{V}O_{2max}$ expressed as ml · $kg^{-2/3}$ · min^{-1}, and it was also a good predictor of success (Bergh 1987). According to Bergh (1987) to be able to win gold medals in the Olympic Games or World Championships you need a

$\dot{V}O_{2max}$ of at least 350 ml · $kg^{-2/3}$ · min^{-1}. In Ingjer's (1992) study two skiers in a sample of 20 to 23-year-old world top skiers had a $\dot{V}O_{2max}$ of this value.

The $\dot{V}O_{2max}$ results of cross-country skiers also depend on whether the measurements are performed in the field or on the treadmill, what skiing technique is used, what class of skier is being examined and which skiing technique the skier specializes in. When skiing uphill on snow $\dot{V}O_2$ was the same, as in ski-walking test on the treadmill, but during uphill skating $\dot{V}O_{2max}$ seemed to be lower than during uphill diagonal skiing. Skiers specialized in classical techniques attained the same $\dot{V}O_{2max}$ during uphill diagonal skiing as in a ski-walking test on the treadmill, but during uphill skating $\dot{V}O_{2max}$ was 0.3 l · min^{-1} lower. Skiers specialized in free style skiing attained the highest $\dot{V}O_{2max}$ during a ski-walking test on the treadmill, a 0.1 l · min^{-1} lower $\dot{V}O_{2max}$ during diagonal uphill skiing, and 0.2 l · min^{-1} lower $\dot{V}O_{2max}$ during uphill skating. All-round skiers had a similar $\dot{V}O_{2max}$ in each exercise. A lower $\dot{V}O_{2max}$ during skating and leg muscle O_2 saturation measurements indicated that blood flow into the leg muscles decreased, because of static muscle contractions during skating. These findings have shown how important a high $\dot{V}O_{2max}$ has been for a good performance both in classical and freestyle ski races, and that it would be useful for skiers who are specialized in skating to train both techniques when training on snow, in order to be able to achieve a good $\dot{V}O_{2max}$ in ski-skating competitions (Rusko 2003).

The transport of oxygen from the air to the mitochondria consists of many phases, each of which can be a potentially impede the transport of O_2. The factors restricting $\dot{V}O_{2max}$ can be divided in four main areas: respiration, central circulation, peripheral circulation and muscle metabolism. The pulmonary system can restrict $\dot{V}O_{2max}$ in certain circumstances. Top athletes are more likely to expose themselves to the desaturation of arteries during a maximal performance compared to sedentary people (Dempsey et al. 1984). The greater $\dot{Q}$ of athletes may lead to a decrease in the transference time of red blood cells in the pulmonary capillary or there may not be enough time to saturate the blood with oxygen before it goes into the pulmonary capillary (Bassett, Jr & Howley 2000). Highly trained athletes have been able to overcome this pulmonary restriction by breathing a 26 % oxygen (O_2) gas mixture, which increased their $\dot{V}O_{2max}$ from 70.1 ml · kg^{-1} · min^{-1} to 74.7 ml · kg^{-1} · min^{-1}, and their arterial O_2 saturation (SaO_2) from 90.6 % to 95.5 %. None of these changes were observed in normal subjects (Powers et al. 1989). Pulmonary limitations have been obvious in people training at the height of 3000 - 5000 meters (Daniels & Oldridge 1970, Faulkner et al. 1968). Another method of changing O_2 transport is to increase the hemoglobin concentration, BV and RCM. The reinfusion of blood has been found to improve $\dot{V}O_{2max}$ with 4 - 9 % (Gledhill 1982, 1985). The mitochondria are the last step in the O_2 transport to muscle fibers, and in theory doubling of the number of mitochondria would also double the VO_2 of muscles. It has, however, been shown that during dynamic exercise of the whole body at the sea level $\dot{V}O_{2max}$ is mostly restricted by the

capacity of the cardiorespiratory system (heart, lungs, blood) to transport O_2 to the muscles, not by the ability of the muscle mitochondria to consume O_2 (Bassett, Jr. & Howley 2000).

2.2 Previous Ultrasound Studies on the Structure and Function of the Heart in the Athletes

2.2.1 Ultrasound

In research ultrasound is used to measure the density of a medium by acoustic reflection. The amount of the ultrasound reflected depends on the degree of the difference between different acoustic impedances: the greater the acoustic mismatch, the greater the amount of sound reflected. Acoustic impedance is the speed with which the sound travels through the medium and varies according to the density of the medium. An acoustic wave is a series of compressions and rarefactions. An acoustic cycle is a combination of compressions and rarefactions, and wavelength is the distance from the beginning of one compression to that of the next compression. Velocity is the speed with which the acoustic waves pass through the medium, and frequency is the number of cycles per time unit. Velocity equals frequency x wavelength, i.e. the higher the frequency, the shorter the wavelength. Velocity depends on the density of the medium and its elastic properties. Sound travels faster through a dense medium than a less dense medium. In a homogenous medium the sound wave travels in a straight line, but as it reaches media possessing different acoustic impedances some of the beams are reflected and some refracted. Acoustic impedance equals the density of the medium x the velocity with which the sound travels through that medium. Echocardiography in adults generally utilizes an ultrasound wave frequency of approximately 2 - 3 megahertz (MHz), enabling clearly distinct echoes that are about 1 mm apart in the medium to be recorded. The less homogenous the medium is, the more difficult it is for the ultrasound wave to penetrate it, owing to reflection and refraction, which are important factors in diminishing the intensity of a beam as it travels through a nonhomogenous medium (Feigenbaum 1986).

The resolution in ultrasound means the ability to distinguish or identify two objects close to each other. If two targets are at one millimetre apart and they can be separately identified and distinguished, we talk about one millimetre resolution. The frequency or the wavelength of the ultrasonic beam is one of the determinants of the capacity of axial resolution. To be able to identify small objects a short wavelength is needed, and the ultrasonic beam should be extremely narrow. Ultrasonic beam can also vary in intensity. The centre of the beam is more clearly distinguishable than the edges. An object with high acoustic impedance produces echoes even from the edges of the ultrasonic beam, but object with low acoustic impedance produce echoes of

reduced amplitude only in the centre of the beam. Too wide a beam can also distort interpretation of the returning echoes. Even where objects are father away from transducer they are recorded simultaneously and successively (Feigenbaum 1986).

2.2.2 Volume and Pressure Overload

Cardiac hypertrophy is associated with large SV, large $\dot{Q}_{max}$ and low HR, and the relative length of the diastolic phase of the cardiac cycle is much longer for athletes than for sedentary subjects both at rest and during submaximal work. This allows greater myocardial perfusion in athletes than in the sedentary subjects (Shephard 1996). Long-term training is mostly connected with the physiological increase in left ventricular mass (LVM), due to the increase in EDD or in the walls thickness or both (Maron et al. 1995). The hypertrophy of the left ventricle is the increase in heart muscle mass, which is primarily caused by the growth of muscle cells (Gosse & Dallocchio 1993, Weber 1988). The hypertrophy of the heart muscle is eccentric in the case of volume overload and concentric in the case of pressure overload exercise (Richey & Brown 1998). As a consequence of chronic volume overload (e.g. endurance running), primarily the EDD (dilatation) increases as relatively also do the thickness of SWT and PWT (Shapiro 1992). Endurance exercise requires an increase in $\dot{Q}$ and hence SV (Mills et al. 1997). During endurance training the heart has to maintain a greatly increased $\dot{Q}$ for a long period, and it has to work against a slightly increased afterload, which is caused by either a minor or a considerable training-induced increase in mean arterial pressure (Bryan et al. 1992). Long-term endurance training increases venous return and BV, which explains the increase in preload. The eccentric growth of the left ventricle is stimulated, which typically increases new sarcomers, leading to myofibril elongation and chamber enlargement (Alpert et al. 1989, Effron 1989, Fleck 1988, Morganroth et al. 1975, Richey & Brown 1998).

According to Laplace's formula (pressure x radius) / (2 x wall thickness) an increase in pressure can be offset by an increase in wall thickness. Consequently, systolic pressure is directly proportional to ventricular wall thickness and indirectly proportional to the size of the ventricular chamber (Lorell & Carabello 2000, Mills et al. 1997). The thickening of SWT and PWT (hypertrophy) without the dilatation of the left ventricle is the result of pressure overload (e.g. weightlifting, shotput) (Morganroth et al. 1975, Pelliccia 1996). In isometric training, peripheric resistance and afterload increase, causing an increase in blood pressure to rise during exercise. Essentially, hypertrophy is an increase in the number of force-generating units (sarcomeres) in the myocyte (Lorell & Carabello 2000). Concentric hypertrophy of the left ventricle is stimulated as the number of parallel myofibrils increases and the walls thicken (Alpert et al. 1989, Fleck 1988, MacDougall et al. 1985, Morganroth et al. 1975, Richey & Brown 1998). The increase in myocardial thickness occurs as a response to the systolic blood pressure produced by resistance during skeletal

muscle contraction (Mills et al. 1997). No significant increase occurs in $\dot{Q}$ during isometric exercise (Bryan et al. 1992). The training of cyclists and rowers is volume and pressure overload, so that both dilatation and hypertrophy occur in the heart (Pelliccia et al. 1991, Shapiro 1992). In cycling keeping the hands on the handlebar is isometric training and pedalling with feet is endurance training (Fagard et al. 1984, Shapiro 1992). Owing to exercise of the upper body cyclists have higher blood pressure than runners during training (Shapiro 1992), and during rowing the peak systolic blood pressure fluctuates around 200 mmHg (Clifford et al. 1994). Since the adoption of the ski-skating technique in cross-country skiing, the strength and muscular endurance of all the muscle groups in the body (arms, shoulders, back, abdominal muscles) have become an important part of cross-country skiing training (Rusko 2003). Thus cross-country skiing cannot be regarded merely as an endurance sport, but as a sport requiring both volume and pressure overload, which can result in both the dilatation and hypertrophy of the heart.

Spirito et al. (1994) calculated the effect of 27 different sports (national and international level competitive athletes, n = 947) an EDD and increase in heart walls thickness. In sports where EDD was large the walls had also thickened. Endurance cycling, cross-country skiing, swimming and pentathlon had the greatest impact on the increase in EDD, and rowing, endurance cycling, swimming and canoeing on the thickening of the walls. Cross-country skiing came 12th as a sport affecting the increase in the walls. Isometric sports, such as weight lifting and wrestling, were ranked high for left ventricular wall thickness relative to cavity dimension, but as absolute values remained within normal limits. The results also showed that gender affected the development of the athlete's heart regardless of sport, age or BSA so that the EDD of female athletes was on the average 2.0 mm less and the walls 0.9 mm thinner than those of male athletes. An increase in BSA of 0.1 m^2 led to a 1.2-mm increase of EDD and a 0.2-mm increase in the walls, and 1-year increase in age was associated with an increase of 0.2 mm in the dimension of the cavity of left ventricle and 0.1 mm in wall thickness.

Urhausen & Kindermann (1999) used a hypertrophy index (HI) in examining athletes drawn from different sports. Bodybuilders who had used anabolic steroids exhibited distinctly higher HI compared athletes from other sports. It was noteworthy that the athletes from strength sports (weightlifters, bodybuilders) who had not used anabolic steroids had significantly lower concentric hypertrophy of the left ventricle than aerobically trained athletes (triathletes, soccer players, rowers) and untrained subjects regardless of BSA. The athletes from combined strength and endurance sports (rowers) and endurance athletes showed a slightly higher HI compared to those from other sports.

2.2.3 Cross-Sectional Studies in Young Athletes

The results of the echocardiographic studies carried out among young athletes (16 - 22 years) (Table 1) have been partly conflicting, when controlled for specific sport training, i.e. where the training is volume or pressure overload or both. The EDD of endurance runners in university and college teams was 5.1 - 5.6 cm and SWTd 0.84 - 1.1 cm and PWTd 0.88 - 1.1 cm. The largest EDD was found among those who trained an average of 113 km · week^{-1}, and the smallest EDD belonged, oddly enough, to the group that trained most (145 km · week^{-1}). The runners who trained least (80 km · week^{-1}) had the thinnest walls (Cohen & Segal 1985, Colan et al. 1987, Conrad et al. 1982, Morganroth et al. 1975, Osborne et al. 1992, Yeater et al. 1996). The EDD of highly ranked international or national orienteers was 5.4 cm, and the thickness of both walls was 1.11 cm (Henriksen et al. 1997). The EDD (5.4 - 5.8 cm) and walls (1.0 - 1.3 cm) of cyclists of the same age were discovered to be somewhat larger than those of runners, and the more successful the cyclists were, the larger were EDD, SWTd and PWTd (Agati et al. 1985, Pellicia et al. 1991, Spataro et al. 1985, Spirito et al. 1994). Seven professional road cyclists out of 30 had an EDD over 6 cm, and eight cyclists had SWTd thicker than 1.5 cm (Spataro et al. 1985). Studies on EDD and wall thickness in rowers and canoers showed similar result to the above-mentioned studies with endurance runners (Agati et al. 1985, Hoyt et al. 1984, Pelliccia et al. 1999, Spataro et al. 1985, Urhausen et al. 1996, Wieling et al. 1981). In Pelliccia et al. (1991) study the largest wall thickness, 1.6 cm, was found in a canoeist, whose EDD was 5.7 cm, and in the 16 elite athletes studied (12 rowers, 3 canoeists, 1 cyclist), whose walls were ≥ 1.3 cm thick, EDD was 5.5 - 6.3 cm. In Hoyt et al. (1984) rowers who had been successful in competitions had significantly larger EDD than rowers with less success. There were no differences in walls, which would indicate that success in competitions was affected more by dilatation of the left ventricle than enlargement of LVM. Pelliccia et al. (1991) shows that after multivariate adjustment wall thickness was associated with BSA, age, male sex and sport (rowing, canoeing, cycling).

Studies done with strength athletes (weightlifting, powerlifting, wrestling, bodybuilding, shotput, throwing events) have shown that these sports require pressure overload, and that the walls, a few cases excepted, have been thicker (1.1 - 1.4 cm) and EDD smaller (4.8 - 5.4 cm) than in endurance athletes (Agati et al. 1985, Cohen & Segal 1985, Haykowsky et al. 2000, Morganroth et al. 1975, Spataro et al. 1985, Van Den Broeke & Fagard 1988). The training of volume had no effect on the size of EDD and the wall thickness in weightlifters who trained under and over 10 hours · week^{-1}, but the walls of lifters who trained over 10 hours · week^{-1} and used anabolic steroids had thicker, although not significant, than the walls of the other lifters (Yeater et al. 1996).

Very few echocardiographic cross-sectional studies have been conducted exclusively among young cross-country male skiers. In Pekkarinens (1986) study of 17-year-old boy skiers in Finnish clubs EDD was 5.2 cm, SWTd 0.98 cm and PWTd 1.18 cm. In Pelliccia et al. (1991, 1999) the EDD of 24 to 25-year-old

elite cross-county skiers was 5.5 cm and the walls 0.96–0.99 cm; however about one third of the subjects in this study were women. According to the study cross-country skiing comes second after cycling as a sport which has had the most effect on EDD, as previously indicated by Spirito et al. (1994). Henriksen et al. (1997) studied elite cross-country skiers and middle-distance runners whose EDD was as large as that of the skiers in Pelliccia et al. (1991, 1999) but the thickness of both walls was as high as 1.2 cm. The EDD of the controls in the various studies has varied from 4.6 cm to 5.3 cm and SWTd from 0.78 cm to 1.06 cm and PWTd from 0.74 cm to 1.06 cm.

2.2.4 Longitudinal Studies in Young Athletes

Few echocardiographic follow-up studies have been done among young people, and in the cases, too, the results are also partly conflicting (Table 2). The impact of training has been followed from one week to three years. After only one week's training the EDD of competitive swimmers increased; however it decreased after nine weeks' training, despite an increase in $\dot{V}O_{2max}$ (Ehsani et al. 1978). Even when the $\dot{V}O_{2max}$ of endurance runners and sprinters increased significantly after two months of training, no changes occurred in the walls of the left ventricle or EDD. Nor were any changes observed in a strength training group which trained for twenty weeks, even if post training LVM was slightly but significantly larger in the endurance group and strength group (Ricci et al. 1982). A 4 - 5-month intensive training period did not affect the EDD or wall thickness of 20-year-old cross-country skiers, but stroke dimension (SD) was significantly larger as was the combined wall thickness (Bienmüller et al. 1982). In both the study by Bonaduce et al. (1998) of the cyclists in the Italian amateur team and in that by Cavallaro et al. (1993) among top class rowers a one-month deconditioning period and it was followed by a five-month intensive training period. In both studies EDD increased significantly over the course of the five-month training period, and was 5.8 cm during the competition season. The walls of the rowers increased more than those of the cyclists, which is due to the fact that training in the rowers was a combination of isotonic and isometric training, with more of the latter than the cyclists had.

A seven-month training season affected the dimensions of the hearts of beginners and experienced rowers differently (Wieling et al. 1981). The EDD, SWTd and PWTd of junior rowers, who had not had daily training before, increased, but in the more experienced senior rowers only EDD increased. A significant difference was found between the junior and senior rowers in EDD and SWTd at both the beginning and end of the study, and in PWTd at the beginning of the study. Pavlik et al. (1988) followed endurance athletes in national teams (cyclists and long-distance runners) over six years between ages 19 – 28, but follow-up subjects were not always the same, and the measurements not performed on all subjects in successive years, some being measured only twice. EDD tended to increase between age 19 - 23, and SWTd increased significantly during the study, but no changes were found in PWTd.

The three-year follow-up study by Tummavuori (1997) indicated that the EDD of 16-year-old cross-country skiers and controls increased while the walls remained almost unchanged. In the controls the walls increased.

The EDD of non-trained 20 to 22-year-old subjects of same baseline EDD increased equally significantly after 11 - 14 weeks' training in both the studies by Adams et al. (1981) and Stein et al. (1980). Perrault et al. (1982) concluded that the effect of training on the adaptation of the left ventricle decreases with age, as a 20-week endurance training program caused an increase in EDD in 19-year-old untrained people, but not in 40-year-olds. Training method also affected the increase in the dimensions of the heart, as a three months' continuous running training increased mean wall thickness (MWT) and LVM, whereas interval type training did not in 15 - 17-year-old untrained boys. $\dot{V}O_{2max}$ increased in both groups, but neither training method had any effect on EDD (Taylor et al. 1979).

2.2.5 Studies in Adults

The morphology of the heart and the changes that are taken place in it are affected by body size, age, gender, genotype, sport and training. Endurance sports, depending on the intensity and volume of training and number of years of training, lead to the physiological dilatation of the heart and increase in wall thickening. Strength sports increase the size of the walls of the left ventricle, induce only a slight increase in the size of the left ventricle itself. Strength athletes usually have very large body size, but the size of the heart when normalized by body weight is normal (Longhurst et al. 1980, Snoeckx et al. 1982, Urhausen & Kindermann 1992) or, after normalizing by lean body weight, the diastolic and systolic dimensions are smaller than those of endurance athletes or control subjects (Fisher et al. 1989).

Fagard (1996, 1997) Maron (1986), Perrault & Turcotte (1994) and Pluim et al. (1999) carried out meta-analyses of echocardiographic studies comparising almost 5500 athletes and about 2700 controls. In endurance athletes EDD was 5.32 - 5.39 cm, SWTd 1.04 - 1.08 cm and PWTd 1.02 - 1.07 cm. The respective values for strength athletes were 5.21 - 5.32 cm, 1.03 - 1.18 cm and 0.95 - 1.12 cm, and those for combined endurance-strength athletes 5.51 - 5.62 cm, 1.13 - 1.17 and 1.10 - 1.16 cm. The EDD of the control subjects was 4.80 - 5.19 cm, SWTd 0.88 - 0.93 cm and PWTd 0.87 - 0.90 cm.

A significant difference was found between athletes (endurance, strength and endurance-strength sports) and control subjects in EDD. The difference in SWTd was significant between endurance athletes and control subjects, and endurance athletes and strength athletes. There was a significant difference in PWTd between control subjects and endurance athletes, but not between endurance athletes and endurance-strength athletes or between endurance athletes and strength athletes (Pluim et al. 1999). In the meta-analyses of both Maron (1986) and Fagard (1996, 1997) a 10 % difference in EDD was found between endurance athletes and controls. The difference between strength

athletes and control subjects in EDD was 2.5 %, and the difference between cyclists and control subjects was 9 % (Fagard 1996, 1997). The MWT of endurance runners was 18 % thicker than that of the controls, the MWT of strength athletes was 15 % thicker than that of the controls, and the MWT of the cyclists was 29 % thicker than that of the controls (Fagard 1996, 1997). Highly trained endurance athletes seem to have the largest absolute dimensions of the left ventricle. A world champion professional cyclist had an EDD as large as 7 cm (Rost 1982), and in Pellicia et al. (1999) an athlete's heart dilatated to the size of 7 cm was also recorded. A small minority of elite athletes (2 %), primarily rowers and canoeists, had a wall thickness of the ventricle that surpassed the normal limits (from 1.3 cm to 1.6 cm) (Pelliccia 1996, Pelliccia & Maron 1996). A wall thickness of up to 1.8 (Van Camp et al. 1995) and 1.9 cm (Reguero et al. 1995) has even been measured.

The physiological thickening of the left ventricle wall of top athletes (12 %) is connected with a dilatated (≥ 5.5 cm) left ventricle. The physiological dilatation of the left ventricle is normally connected with large body size and the training of intensively aerobic sports such as cross-country skiing, cycling and rowing (Pelliccia 1996, Pelliccia & Maron 1996). Women of the same ethnic background, same age and high level of training had a 10 % smaller EDD and 23 % thinner wall thickness than men (Pelliccia 1996). In Huonker et al. (1996) the EDD of endurance athletes was significantly larger than that of weightlifters and controls, the SWTd and PWTd of cyclists, triathlonists, weightlifters and bodybuilders was larger than in control subjects, and relative heart volume was larger in endurance athletes than in strength athletes and controls. Weightlifters had the smallest relative heart volume and significantly smaller values compared to the control group. The athletes had larger LVM than the control subjects, the average difference being 46 % (Maron 1986). The LVM of controls (174 g) was significantly smaller than that of endurance athletes (249 g), of endurance-strength athletes (288 g) or of strength athletes (267 g) (Pluim et al. 1999). There were no significant differences in LVM between the three groups of athletes.

2.2.6 Genotype

The different response to endurance training is affected by age, sex, earlier experiences, initial level and genetics. The differences between individuals in the response to training are considerable (Bouchard & Lortie 1984). A 20-week training program increased the maximal aerobic power of inactive people on the average 33 %, the variance ranging from 5 % to 88 %, showing that there are individuals who react little and those who react strongly (low and high responders) to endurance training (Lortie et al. 1984). Also the baseline level affects the results of endurance training, and training response can be rapid or slow. Individuals who had a lower baseline level of endurance performance level were able through training to attain a higher level of performance, irrespective of rapid or slow endurance training response than individuals who

had a high baseline level of performance (Bouchard & Lortie 1984). It is probable that there is a genetic effect on the dimensions of the heart such that either the training response to the hypertrophy of the heart is genetic or there is a genetic disposition to tolerate intensive training and reach high levels of competitive performance (Pelliccia 1996).

To become a top athlete an individual must have good genes, for it is virtually impossible for an untrained sedentary person who has a $\dot{V}O_{2max}$ of 40 ml · kg^{-1} · min^{-1} to achieve a $\dot{V}O_{2max}$ of 70 ml · kg^{-1} · min^{-1} through training (Levine 1993). Fifteen-week and twenty-week endurance training studies conducted with monozygotic twins proved that the respective 14 and 12 % increase in $\dot{V}O_{2max}$ was greatly dependent on their genes, the intra-class correlation being 0.65 - 0.83 (Hamel et al. 1986, Prud'Homme et al. 1984). The similarity of the twin siblings and thus the effect of genes on the response to the enzyme activities of muscles emerged during the last eight weeks of the 15-week training period (Hamel et al. 1986). Danis et al. (2003) examined the effect of six months' training on 11 – 14-year-old monozygotic twins. The $\dot{V}O_{2max}$ (ml · kg^{-1} · min^{-1}) of the training twins increased by 10.6 %, and absolute $\dot{V}O_{2max}$ improved in both training and untrained twins, but there was no significant difference between the groups. Genes have a considerable impact, as variance analysis estimated the percentages of training, genes and their interaction to be 20, 70 and 10 %. The variance of LVM was explained by genetic factors in 60 % of the boys and 71 % of the girls (Verhaaren et al. 1991). A genetic effect was also found in the study by Roberts et al. (2002), where one of the 24-year-old identical twins had done cross-country skiing training for about 750 hours · year^{-1} during the previous three years, while the other twin was active in another way, which did not include skiing training, for 175 hours · year^{-1}. In absolute $\dot{V}O_{2max}$ there was only a 2 % and in relative $\dot{V}O_{2max}$ 5 % difference between the trained and untrained twins, even where the difference of time over a 42.2-kilometer competition was 15 %. Thus $\dot{V}O_{2max}$ alone is a poor predictor of performance in homogenous groups of athletes.

However in Adams et al. (1985) after a 14-week training period the monozygotic twins showed as much variety in the size of the heart as dizygotic twins and non-twin siblings. The researchers thought that the environment, home, had a larger effect on the size of the heart than genetics. To reduce the effects of environmental factors Bielen et al. (1990) examined 6 - 8-year-old monozygotic and dizygotic twins, but found no genetic effect in EDD or wall thickness. No significant genetic effect was found in either EDD or fractional shortening (FS) in the study by Fagard et al. (1987). However according to Bielen et al. (1991) the increase in EDD and FS under submaximal exercise was considered, unlike at rest, to be caused by a significant genetic component, estimated the genetics to explain 24 % of EDD and 47 % of FS (Bielen et al. 1991).

TABLE 1 Cross-sectional echocardiographic studies of 16 - 22 years old athletes (17 - 25 years old cross-country skiers). EDD = end-diastolic diameter (cm), SWTd = septal wall thickness in diastole (cm), PWTd = posterior wall thickness in diastole (cm), MWT = mean wall thickness. $_a$ = wall not specified

Reference	Year	Subjects	Male/Female	N	Age	EDD	SWTd	PWTd	MWT
Sharma et al.	1999	elite athletes (football, tennis, swimming,	M/F	231/76	16	5.01			0.91_a
		rowing, triathlon)	M	231	16	5.16			0.93_a
Pekkarinen	1986	cross-country skiers	M	12	17	5.17	0.98	1.18	
		controls	M	11	17	4.95	0.86	1.06	
Somauroo et al.	2001	soccer players	M	171	17	5.14	1.03	1.00	
Pelliccia et al.	1991	tennis	M/F	32/15	17	5.00	0.91		
		roller-skating	M/F	32/26	19	4.90	0.90		
		swimming	M/F	26/28	19	5.30	0.93		
		pentathlon	M/F	36/14	19	5.24	0.92		
		volleyball	M/F	36/15	20	5.37	0.94		
		cyclists	M/F	49/15	20	5.48	1.04		
		canoeing	M/F	52/8	20	5.45	1.05		
		rowing	M/F	92/3	21	5.60	1.13		
		alpine skiing	M/F	24/8	21	5.20	0.89		
		tae kwon do	M/F	14/3	21	5.06	0.87		
		fencing	M/F	31/11	22	5.17	0.92		
		team handball	M/F	9/17	22	5.18	0.85		
		roller hockey	M	23/0	22	5.34	0.97		
		boxing	M	14/0	22	5.25	0.98		
		cross-country skiers	M/F	24/7	24	5.45	0.96		
		25 sports	M	738	22	5.42	1.01	0.94	
Pelliccia et al.	1999	gymnastics	M/F	31/56	17	4.64			0.76
		tennis	M/F	36/28	19	5.23			0.90
		swimming	M/F	9/20	20	5.16			0.90
		table tennis	M/F	6/5	20	5.07			0.82
		skating	M/F	22/14	21	5.12			0.90
		boxing	M	15/0	21	5.47			1.01
		rowing	M/F	78/2	22	5.78			1.09
		volleyball	M/F	15/6	22	5.45			0.93
		cross-country skiers	M/F	31/10	25	5.48			0.99

Reference	Year	Subjects	Male/Female	N	Age	EDD	SWTd	PWTd	MWT
Csanady et al.	1986	basketball players	M	14	17	5.36	1.12	0.99	
		basketball players	M	15	25	5.68	1.15	0.98	
Cohen & Segal	1985	distance runners	M	10	19	5.58	1.05	1.14	
		wrestlers	M	10	19	4.88	1.35	1.29	
Spirito et al.	1988	waterpolo and soccer players	M	21	19	5.40	0.99	0.98	
		controls	M	21	19	5.20	0.81	0.74	
Morganroth et al.	1975	long-distance runners	M	15	19	5.41	1.09	1.13	
		swimmers	M	15	20	5.66	1.07	1.06	
		wrestlers	M	12	20	4.78	1.30	1.37	
		controls	M	16	19	4.64	1.03	1.03	
Hoyt et al.	1984	rowers, less successful	M	8	19	5.46	0.93	0.90	
		rowers, successful	M	8	21	5.60	0.95	0.93	
Zemva & Rogel	2001	dancers	M	15	20	5.07	0.89	0.99	
Rowland & von Duvillard	1990	weight lifting, running, racquetball	M	12	20	5.23			
Van Den Broeke & Fagard	1988	throwers	M	10	20	5.28	1.07	1.05	
		controls	M	10	19	5.01	1.06	1.05	
Urhausen et al.	1996	soccer players	M	22	20	5.50	1.03	1.00	
		400-m runners	M	22	21	5.27	1.02	1.02	
Agati et al.	1985	cyclists	M	12	20	5.60	1.30	1.06	
		canoeists	M	12	20	5.70	1.30	1.00	
		anaerobics (wrestlers, weight-lifters, shot putters)	M	18	20	5.20	1.30	0.98	
		controls	M	8	22	5.00	0.87	0.91	
Osborne et al.	1992	long distance runners	M	15	20	5.53	0.84	0.92	
		controls	M	26	22	5.20	0.78	0.87	
Spataro et al.	1985	road cycling	M	30	20	5.77	1.27	1.14	
		track cycling	M	14	20	5.77	1.29	1.23	
		canoing	M	13	20	5.59	1.36	1.22	
		rowing	M	30	21	5.71	1.32	1.23	
		body-building	M	14	22	5.35	1.24	1.13	
Spirito et al.	1994	endurance cycling	M/F	37/13	20	5.50			1.05
		sprint cycling	M/F	13/2	20	5.43			1.01
		27 sports (same subjects as in Pelliccia et al. 1991)	M/F	738/209	22	5.30			1.00
Conrad et al.	1982	aerobically well-trained	M	10	20	5.34	1.03	0.96	
		controls	M	10	24	5.23	0.93	0.88	

Reference	Year	Subjects	Male/Female	N	Age	EDD	SWTd	PWTd	MWT
Urhausen et al.	1996	rowers	M	28	21	5.60	1.12	1.11	
Yeater et al.	1996	runners	M	8	21	5.11	0.98	1.11	
		regular lifters	M	11	21	5.17	0.93	0.99	
		heavy lifters	M	16	21	5.39	0.95	1.16	
		steroid users	M	8	21	5.40	1.24	1.31	
Colan et al.	1987	long-distance runners	M	11	21	5.20		0.99	
		swimmers	M	11	21	5.40		1.00	
		controls	M	33	22	4.80		0.88	
Haykowsky et al.	2000	powerlifters	M	8	21	5.32	0.94	0.92	
		controls	M	8	22	5.21	0.94	0.94	
Henriksen et al.	1997	orienteers	M	96	22	5.39	1.11	1.11	
		cross-country skiers, middle distance runners	M/M	24/23	22	5.48	1.19	1.19	
Heath et al.	1981	middle and/or distance runners, cyclist	M	15/1	22	5.00	1.11	0.95	
Notaristefano et al.	1988	endurance athletes	M	19	22	5.64	1.16	1.03	
		controls	M	10	26	5.33	0.96	0.86	

TABLE 2 Echocardiographic follow-up studies of 16 - 23 years old male athletes. EDD = end-diastolic diameter (cm), SWTd = septal wall thickness in diastole (cm), PWTd = posterior wall thickness in diastole (cm).

Reference	Year	Subjects	N	Age	Follow-up time	EDD	SWTd	PWTd
Tummavuori	1997	cross-country skiers	16	16	0. year	5.56	0.84	0.84
					after 1 year	5.68	0.88	0.85
					after 2 years	5.87	0.89	0.9
					after 3 years	5.87	0.86	0.88
		controls	12	16	0. year	5.23	0.75	0.78
					after 1 year	5.39	0.79	0.8
					after 2 years	5.55	0.84	0.81
					after 3 years	5.58	0.88	0.86
					interaction	n.s.	$p < 0.01$	$p < 0.01$
Ricci et al.	1982	sprint	8	16	pre training	4.78	1.03	1.03
					post training, 8 weeks	4.88	1.05	1.02
		endurance	8	17	pre training	4.79	1.00	1.05
					post training, 8 weeks	4.90	1.09	1.04
		strength	12	19	pre training	4.76	1.07	1.07
					post training, 20 weeks	4.84	1.14	1.09
Ehsani et al.	1978	swimmers (1 woman)	8	18	pretraining	4.87		0.94
					1 week training	5.30 ***		0.95
					3 weeks training	5.20 ***		0.99
					5 weeks training	5.10 ***		1.01 *
					9 weeks training	5.20 ***		1.01 ***
					sedentary control	4.78		0.94
					* $p < 0.02$, ** $p < 0.01$, *** $p < 0.005$ control vs training period			
		runners	6	20	pre detraining	5.10		1.07
					2nd day detraining	5.00		1.00
					4th day detraining	4.94 **		0.96 **
					1 week detraining	4.70 **		0.90 **
					2 weeks detraining	4.68 ***		0.82 ***
					3 weeks detraining	4.63 ***		0.80 ***
					** $p < 0.01$, *** $p < 0.005$ pretraining vs. detraining period			

Reference	Year	Subjects	N	Age	Follow-up time	EDD	SWTd	PWTd
Bienmüller et al.	1982	cross-country skiers	12	20	3-4 months after last ski season	5.20	1.13	0.98
					4-5 months intensive training	5.20	1.22	1.06
						n.s.		
Wieling et al.	1981	oarsmen, freshmen	9	20	0 month	5.15	0.89	0.83
					0.5 month training	5.26	0.89	0.84
					1 month training	5.19	0.88	0.86
					4 months training	5.33 c	0.94 c	0.86
					7 months training	5.33 a, c	1.00 a	0.92 a
		oarsmen, senior	14	23	0 month	5.61 a, b	1.11 a, b	1.01 a, b
					0.5 month training	5.69 b, c	1.11 b	1.02 b
					1 month training	5.72 b, c	1.10 b	0.99 b
					4 months training	5.73 b, c	1.14 b	1.02 b
					7 months training	5.79 a, b	1.15 a, b	0.99 a
		controls	17	22		4.98	0.84	0.80
					a) between oarsmen and controls, b) between freshmen and seniors, c) between first and subsequent examination,	statistical significance if $p < 0.05$		
Bonaduce et al.	1998	cyclists	15	21	1 month deconditioning	5.40	1.10	1.00
					5 month vigorous training	5.80	1.30	1.20
						$p < 0.001$	$p < 0.05$	$p < 0.05$
		nonathletes	15	21		4.70	0.90	0.90
					nonathletes vs. detrained athletes	$p < 0.001$	$p < 0.001$	$p < 0.001$
Cavallaro et al.	1993	rowers	15	22	1 month deconditioning	5.60	1.00	1.00
					5 month vigorous training	5.80	1.20	1.10
						$p < 0.001$	$p < 0.001$	$p < 0.005$

3 PURPOSE OF THE STUDY

The purpose of this 6.5-year follow-up study was to determine the impact of training on the cardiac structure and function of the heart of young endurance athletes. The study also examined the changes in maximal cardiovascular function as indicated by $\dot{V}O_{2max}$ during a 6.5-year training period.

The hypotheses addressed by this study were as follows:

1. What changes does long-term progressive skiing endurance training cause in the size and function of the heart, and what is the significance of the size of the heart at the baseline situation?
   - H_1 EDD, SWTd and PWTd would increase.
   - H_2 The subjects with small and large hearts at the baseline situation; EDD, SWTd and PWTd would increase equally much.
   - H_3 $\dot{V}O_{2max}$ would increase.
   - H_4 A relationship of dependence would be found between EDD and $\dot{V}O_{2max}$ both at the baseline and at the follow-up after 6.5 years.
2. At what age do the structural and functional changes of the heart take place?
   - H_5 The most marked changes would occur between the ages of 16 and 20, after which they would level off.
   - H_6 The echocardiographic changes and those of $\dot{V}O_{2max}$ during different training periods would be small, but the size of the heart and $\dot{V}O_{2max}$ would be at their highest at the beginning of the competition season.
3. How do the size and growth of the subjects affect the structure and function of the heart?
   - H_7 Biological growth increases the size of the heart, but the changes in the size of the heart would be larger in the cross-country skiers training regularly than control subjects.
   - H_8 The subjects who develop biologically early would show smaller changes in the size of the heart than late-developing subjects.

4 MATERIALS AND METHODS

4.1 Experimental Group

The subjects of the study were initially 15 - 17-year-old male cross-country skiers, who were at the top of their age group in Finland. Ninety-eight cross-country skiers were sent an inquiry concerning their willingness to participate. The forty-seven skiers who had given an affirmative answer to the inquiry were invited to participate in the baseline measurements. The baseline measurements consisted of anthropometric variables (height, weight and body fat percentage) and $\dot{V}O_{2max}$ as measured in an exercise test done on a treadmill. Thirty-eight young male skiers were finally selected on the basis of the baseline measurements, training backgrounds and success in competitions. The most important criterion for selection was training. The subjects were required to do a reasonable amount of training even before the beginning of the study, in order to avoid big changes in those who had trained little. The second criterion was success in competitions, and the last criterion $\dot{V}O_{2max}$. The subjects and their parents were informed of the possible risks related to the measurements before they gave their written consent for their sons to participate. The subjects also signed a written consent before every visit to the study centre.

4.2 Control Group

Eighteen school-age volunteers were selected for the control group. They were not competitive athletes in any sport, but they had a positive attitude to exercise and they went in for several sports themselves. It would have been very difficult to have maintained a totally inactive control group throughout the research period. Ten of the eighteen schoolboys participated in the final measurements carried out four years later. The control subjects were also

informed of the possible risks related to the measurements, and they gave their written consent before each measurement occasion.

4.3 Experimental Design

The results of the study were analyzed in eight different sections:

A. Changes during 6.5 years
    1. Two measurements: baseline – follow-up after 6.5 years (***6.5y2m***)
    2. Small (S) and large (L) hearts according to baseline EDD: baseline – follow-up after 6.5 years (***EDD-SL***)
B. Timing of changes
    1. 6.5 years, three measurements: baseline – follow-ups after 3.5 years and 6.5 years (***6.5y3m***)
    2. 6.5 years, seven measurements: baseline – follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years (***Annual)***
    3. Three different training seasons over 6 - 7 months (***Seasonal***)
C. Impact of size and growth of the subjects
    1. 4.5 years, two measurements, skiers (S) and control subjects (C): baseline – follow-up after 4.5 years (***4.5y2mSC***)
    2. Small and large change in BSA (***BSAChange)***
    3. Small (S) and large (L) hearts according to baseline EDD/BSA: baseline – follow-up after 6.5 years (***EDD/BSA-SL***)

A. Changes over 6.5 Years

1. *6.5y2m*

The research period was 6.5 years. The baseline measurements took place in May 1987 and the follow-up after 6.5 years in November 1993. The subjects were 20 cross-country skiers.

2. *EDD-SL*

The research period was 6.5 years. The 20 subjects of study 6.5y2m were divided into two groups according to their baseline EDD. The average EDD for skiers with small hearts (S = small heart, n = 10) was 5.07 ± 0.17 cm (4.72 - 5.28 cm) and for those with large hearts (L = large heart, n = 10) 5.57 ± 0.25 cm (5.29 - 6.11 cm). The measurement dates were the same as in study 6.5y2m: the baseline measurements were done in May 1987 and the follow-up after 6.5 years in November 1993.

B. Timing of Changes

1. *6.5y3m*

The length of the research period was 6.5 years, during which time the subjects were measured at three different time points. The baseline measurements took place in May, the follow-ups after 3.5 years and 6.5 years in November. The subjects were 14 cross-country skiers.

2. *Annual*

The length of the research period was 6.5 years. The subjects were measured annually. There were seven measurement times: baseline in May, follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years in November. The subjects were 8 cross-country skiers.

3. *Seasonal*

The subjects were examined on the fifth research year during three different training periods: Basic Training Season 1 (BTS1) in May – June, Basic Training Season 2 (BTS2) in August – September, and Pre-Competition Season (PCS) in November – December. The study period was 6 - 7 months, depending on the occasion of measurement. The subjects were 15 cross-country skiers.

C. Impact of Size and Growth of the Subjects

1. *4.5y2mSC*

The length of the research period was 4.5 years. The experimental group consisted of 15 cross-country skiers (S) and the control subjects (C) of 10 schoolboys. There were two measurement times: the baseline measurements were carried out with the skiers in March and the control subjects in May; the follow-up approximately 4.5 years later, the skiers in November and the control subjects in August - September.
Table 3 shows the measurement schedule.

2. *BSA Change*

The length of the research period was 6.5 years. The 20 subjects of study 6.5y2m were divided into two groups according to the change in BSA over 6.5 years: small change in BSA (n = 10), 0.05 ± 0.03 m_2 (0.02 - 0.08 m^2), large change in BSA (n = 10) 0.15 ± 0.03 m_2 (0.12 - 0.22 m^2) during 6.5 years. The measurement times were the same as in study 6.5y2m: baseline measurements were performed in May and the follow-up after 6.5 years in November.

3. *EDD/BSA-SL*

The length of the research period was 6.5 years. The 20 subjects of study 6.5y2m were divided into two groups according to baseline EDD/BSA. The average of the skiers with small hearts (S = small heart, n = 10) was 2.76 ± 0.11 (2.49 - 2.87) and the average of the skiers with large hearts (L = large heart, n = 10) 3.3 ± 0.12 (2.91 - 3.26). The measurement times were the same as in study 6.5y2m: the baseline measurements were done in May and the follow-up after 6.5 years in November.

TABLE 3 Measurement schedule: 6.5y2m (6.5 years, two measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), Seasonal (3 different training seasons), 4.5y2mSC (4.5 years, 2 measurements), S = Skiers, C = Control subjects.

	6.5y2m (S, n=20)	6.5y3m (S, n=14)	Annual (S, n=8)	Seasonal (S, n=15)	4.5y2mSC (S, n = 15, C, n = 10)
Baseline, May	x	x	x		
1.5 years, Nov			x		
2.5 years, Nov			x		March (S),May (C),
3.5 years, Nov		x	x		
4.5 years, Nov			x		
5.5 years, Nov			x		
6.5 years, Nov	x	x	x		Nov (S), Aug-Sept (C)
5th year, May-June				x	
5th year, Aug-Sept				x	
5th year, Nov-Dec				x	

4.3.1 Representativeness of the groups

The study group consisted of 38 cross-country skiers, 23 of whom participated in all phase of the study. Data on 20 skiers was used in the analysis of the results as one of the three skiers involved in the follow-up after 6.5 years had stopped training during the first year, one had considerably reduced his level of training, and one had progressively reduced his training during the study and stopped training altogether during the last two years of the study. The 20 subjects for whom complete data were obtained were formed into three groups, according to whether they had been involved only in the baseline and follow-up after 6.5 years (6.5y2m, n = 20), in baseline and follow-ups after 3.5 years and 6.5 years (6.5y3m, n = 14) or in the annual measurements (Annual, n = 8) (Table 4).

TABLE 4 Skiers grouped according to participation in the measurements: 6.5y2m (6.5 years, two measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), Seasonal (3 different training seasons), 4.5y2mSC (4.5 years, 2 measurements), S = skiers.

Subject	6.5y2m	6.5y3m	Annual	Seasonal	4.5y2mS
1	x	x			x
2	x	x	x	x	x
3	x	x		x	x
4	x			x	x
5	x			x	x
6	x	x	x	x	x
7	x				
8	x				
9	x	x	x	x	x
10	x	x	x		x
11	x	x		x	
12	x				
13	x	x	x	x	x
14	x			x	x
15	x	x		x	x
16	x	x		x	
17	x	x		x	x
18	x	x	x	x	x
19	x	x	x	x	x
20	x	x	x	x	x
	(n = 20)	(n = 14)	(n = 8)	(n = 15)	(n = 15)

All three subject groups (6.5y2m, 6.5y3m, Annual) represented well the whole group of subjects (n = 39) at the baseline. There were no significant differences in height, weight, BSA, $\dot{V}O_{2max}$ ($l \cdot min^{-1}$, $ml \cdot kg^{-1} \cdot min^{-1}$, $ml \cdot kg^{-2/3} \cdot min^{-1}$), EDD, SWTd and PWTd between the whole study group (n = 39) and the three subject groups (Table 5). Similarly at the follow-up after 6.5 years no significant differences were observed between the whole group (n = 23) and the three subject groups in the above-mentioned variables (Table 6).

TABLE 5 Representativeness of groups of 6.5y2m (6.5 years two measurements), 6.5y3m (6.5 years 3 measurements) and Annual (6.5 years 7 measurements) at the baseline in height, weight, body surface area (BSA), maximal oxygen uptake ($\dot{V}O_{2max}$), EDD (end-diastolic diameter), SWTd (septal wall thickness in diastole) and PWTd (posterior wall thickness in diastole). Mean ± SD.

Baseline	Whole group (n = 38)	6.5y2m (n = 20)	6.5y3m (n = 14)	Annual (n = 8)	Drop-outs (n = 3)
Height (cm)	177.5 ± 5.5	178.9 ± 5.2	178.8 ± 6.1	181.0 ± 6.2	176.6 ± 1.3
Weight (kg)	64.9 ± 6.5	66.3 ± 5.7	65.8 ± 6.6	67.8 ± 7.9	63.3 ± 5.2
BSA (m^2)	1.81 ± 0.11	1.83 ± 0.10	1.83 ± 0.12	1.87 ± 0.13	1.78 ± 0.06
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	4.5 ± 0.4	4.5 ± 0.4	4.4 ± 0.4	4.5 ± 0.5	4.2 ± 0.6
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	68.7 ± 3.1	68.2 ± 3.0	67.4 ± 3.1	67.0 ± 2.7	66.7 ± 4.9
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	276.0±12.6	275.6±12.4	271.4±12.0	272.6±13.3	264.1±23.2
EDD (cm)	5.28 ± 0.40	5.32 ± 0.33	5.38 ± 0.33	5.45 ± 0.39	4.70 ± 0.43
SWTd (cm)	0.83 ± 0.05	0.84 ± 0.05	0.85 ± 0.05	0.86 ± 0.06	0.81 ± 0.02
PWTd (cm)	0.83 ± 0.04	0.83 ± 0.05	0.83 ± 0.05	0.85 ± 0.04	0.84 ± 0.04

TABLE 6 Representativeness of groups of 6.5y2m (6.5 years two measurements), 6.5y3m (6.5 years 3 measurements) and Annual (6.5 years 7 measurements) at the follow-up after 6.5 years in height, weight, body surface area (BSA), maximal oxygen uptake ($\dot{V}O_{2max}$), EDD (end-diastolic diameter), SWTd (septal wall thickness in diastole) and PWTd (posterior wall thickness in diastole). Drop-outs vs. 6.5y2m, * $p < 0.05$. Mean ± SD.

Follow-up after 6.5 years	Whole group (n = 23)	6.5y2m (n = 20)	6.5y3m (n = 14)	Annual (n = 8)	Drop-outs (n = 3)
Height (cm)	181.0 ± 4.9	181.3 ± 5.2	181.3 ± 6.1	182.6 ± 6.2	179.6 ± 1.5
Weight (kg)	73.9 ± 6.9	73.6 ± 6.4	73.1 ± 7.4	74.2 ± 8.1	76.3 ± 11.1
BSA (m^2)	1.94 ± 0.11	1.94 ± 0.11	1.93 ± 0.12	1.95 ± 0.13	1.95 ± 0.11
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	5.1 ± 0.7	5.1 ± 0.6	5.1 ± 0.6	5.1 ± 0.8	4.5 ± 1.2
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	68.3 ± 6.5	69.2 ± 6.2	68.9 ± 5.6	67.9 ± 5.2	58.7 ± 6.3 **
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	286.8±28.4	290.1±26.8	288.1±24.6	285.1±27.9	248.7±41.0 *
EDD (cm)	5.70 ± 0.48	5.77 ± 0.39	5.74 ± 0.42	5.83 ± 0.49	5.26 ± 0.84
SWTd (cm)	0.96 ± 0.11	0.96 ± 0.12	0.97 ± 0.11	0.98 ± 0.10	0.92 ± 0.09
PWTd (cm)	0.92 ± 0.08	0.92 ± 0.08	0.93 ± 0.09	0.93 ± 0.07	0.89 ± 0.08

4.4 Antropometric Measurements

The skiers and control subjects were measured for height (cm), weight (kg) and percentage body fat; percentage body fat was measured with a John Bull skinfold meter, measuring the following four skinfolds: triceps, biceps, subscapularis, iliaca (Durnin & Rahaman 1967).

Table 7 shows the age, height, weight and percentage body fat of the subjects participating in the different phases of the study.

TABLE 7 Physical characteristics of the subjects at the baseline and at the follow-ups: 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), Seasonal (three different training seasons) and 4.5y2mSC (4.5 years, 2 measurements). S = Skiers, C = Control subjects.

		6.5y2m (S, n=20)	6.5y3m (S, n=14)	Annual (S, n = 8)	Seasonal (S, n=15)	4.5y2mSC (S, n=15)	4.5y2mSC (C, n=10)
Age (years)	Baseline	15.9±0.8	15.7±0.8	16.0±0.9	20.6±0.6	17.6±0.7	17.0±0.2
	Follow-up	22.4±0.8	22.2±0.8	22.5±0.9	21.2±0.6	22.3±0.7	21.6±0.3
Height (cm)	Baseline	178.9±5.2	178.8±6.1	181.0±6.2	180.4±5.2	181.7±5.8	180.1±5.3
	Follow-up	181.3±5.2	181.3±6.1	182.6±6.2	180.4±5.7	181.9±5.8	182.3±5.5
Weight (kg)	Baseline	66.3±5.7	65.8±6.6	67.8±7.9	73.7±6,4	71.5±7.6	64.0±6.2
	Follow-up	73.6±6.4	73.1±7.4	74.2±8.1	72.9±6.4	74.1±7.3	72.0±8.4
Fat %	Baseline	9.3±1.9	9.6±2.2	10.0±2.5	9.7±1.9	9.6±1.7	11.8±2.5
	Follow-up	8.2±1.7	8.2±1.7	8.5±1.8	7.5±1.4	8.3±1.7	12.2±3.3

4.5 Heart Rate and Arterial Blood Pressure at Rest

During the measurements of resting heart rate and resting blood pressure the subjects rested in the supine position 15 - 20 minutes after which the HR was

taken with a heart rate monitor (Polar Sport Tester, Polar Electro Oy, Kempele, Finland) and diastolic (DBP) and systolic (SBP) pressure with a mercury blood pressure meter (BOSO STAT, Bosch & Sohn, Germany).

4.6 Cardiovascular Parameters

$\dot{V}O_{2max}$ of the skiers and control subjects was measured during an exercise test on a treadmill. The treadmill was stopped at three-minute intervals for approximately 15 seconds, and a capillary blood sample was taken from the fingertip in order to determine lactate concentration. The speed and angle of the treadmill were increased simultaneously to allow a 4 - 6 ml · kg^{-1} · min^{-1} increase in theoretical oxygen uptake ($\dot{V}O_{2\ demand}$) to take place compared to that with the previous load (Balke & Ware 1959, Rusko 1987). The exercise patterns of the skiers used in the treadmill tests are given in Appendix 1. Those of the control subjects are given in Appendix 2. $\dot{V}O_{2\ demand}$ of the skiers was 26 ml · kg^{-1} · min^{-1} during the first exercise period, and the load was increased by 6 ml · kg^{-1} · min^{-1} at three-minute intervals. The initial load for the control subjects corresponded to $\dot{V}O_{2\ demand}$ of 15 - 20 ml · kg^{-1} · min^{-1} depending on their physical condition, and the load was increased at three-minute intervals by 4 - 6 ml · kg^{-1} · min^{-1}. The highest maximal oxygen consumption maintained during one minute was defined as $\dot{V}O_{2max}$ (l · min^{-1}, ml · kg^{-1} · min^{-1}, ml · $kg^{-2/3}$ · min^{-1}).

The exercise tests were carried out by ski-walking on the treadmill in both skiers and control subjects. The skiers ran on the treadmill only on the first measurement occasion as thereafter they participated in a 13-week training intervention study, with the emphasis on running training. Therefore running on the treadmill was also used to test $\dot{V}O_{2max}$.

Lactate concentration was analysed from blood samples using the enzymatic method (Roche Lactate Analyzer 640, Roche Bioelectronics, Basel, Switzerland; YSI Model 1500, Yellow Springs Inc., USA; EBIO, 6666 Eppendorf-Netheler-Hinz Gmbh, Germany). The heart rate of the skiers and control subjects was monitored with the help of electrocardiogram (ECG) equipment (recorder Simonsen & Weel SCR-521; Simonsen & Weel, Denmark, and monitor S & W Medico Teknik A/S DS-521;s & W Medico Teknik A/S, Albertslund, Denmark, and Hellige Servomed SMS 182, Hellige GMBH, Freiburg im Breisgau, Germany) throughout the treadmill test. Oxygen uptake ($\dot{V}O_2$) was measured throughout the test to exhaustion with a breathing gas analyzer (Oxycon Mijnhardt OX-4, Breda, Netherlands and Sensor Medics 2900Z, Yorba Linda, CA, USA) which was calibrated with calibration gases before and after every test.

4.7 Training of Skiers

Training time in hours, the time spent on each bout of training and training distances in kilometres were calculated from the training diaries of the skiers separately from each training season (Basic Training Season 1 and 2, Pre-Competition Season, Competition Season, Transition Season). The lengths and points of time of the seasons are shown in Appendix 3. Training information was available only for some of the skiers, as not all of them kept a training diary.

Table 8 shows the skiers' training (6.5y2m) during the 28 weeks after the baseline and before the follow-up after 6.5 years. The 28 weeks comprise two different training periods: Basic Training Season 1 (BTS1), duration 16 weeks, and Basic Training Season 2 (BTS2), duration 12 weeks. Training the whole year (Year, 52 weeks) refers to following the baseline measurements and the follow-up after 6.5 years, and comprises the 28 weeks before the follow-up after 6.5 years plus 24 weeks after the follow-up after 6.5 years.

Table 9 shows the training of the skiers (6y3m) during the 28 weeks after the baseline and before the follow-up after 3.5 years and before the follow-up after 6.5 years. The 28 weeks consists of two different training periods: Basic Training Season 1 (BTS1), duration 16 weeks, and Basic Training Season 2 (BTS2), duration 12 weeks. Training the whole year (Year, 52 weeks) refers to the year after the baseline, the follow-up after 3.5 years and the follow-up after 6.5 years; in all cases 28 weeks before and 24 weeks after the measurements.

Table 10 shows the training of the skiers (4.5y2mSC) during the 28 weeks before the baseline and after the follow-up after 4.5 years. The 28 weeks consists of two different training periods: Basic Training Season 1 (BTS1), duration 16 weeks, and Basic Training Season 2 (BTS2), duration 12 weeks. Training the whole year (Year, 52 weeks) refers to the 28 weeks before and 24 weeks after the measurements.

TABLE 8 Skiers' training (6.5y2m) during the 28 weeks after the baseline and before the follow-up after 6.5 years. 28 weeks consists of Basic training season 1 (BTS1, 16 weeks) and Basic training season 2 (BTS2, 12 weeks). Training during the whole year after the baseline and during the last year (28 weeks before the follow-up after 6.5 years and 24 weeks after the follow-up after 6.5 years). P - value pre vs. post, Friedman's test. Mean ± SD.

		N	Baseline	6.5 years	p-value
BTS1	km · week^{-1}	7	78.4 ± 31.9	123.2 ± 64.5	n.s.
(16 weeks)	h · week^{-1}	15	9.4 ± 4.6	10.5 ± 4.9	n.s.
	times · week^{-1}	12	6.3 ± 3.8	7.0 ± 3.7	n.s.
BTS2	km · week^{-1}	6	85.4 ± 28.8	134.4 ± 61.4	n.s.
(12 weeks)	h · week^{-1}	15	9.6 ± 3.5	10.8 ± 4.8	n.s.
	times · week^{-1}	12	7.6 ± 3.7	7.3 ± 3.2	n.s.
Year	km · year^{-1}	8	4397 ± 1254	6695 ± 1818	$p < 0.01$
(52 weeks)	h · year^{-1}	14	486 ± 189	503 ± 221	n.s.
	times · year^{-1}	11	366 ± 216	405 ± 184	n.s.

TABLE 9 Skiers' training (6.5y3m) during the 28 weeks after the baseline and before the follow-up after 3.5 years and 6.5 years. 28 weeks consists of Basic training season 1 (BTS1, 16 weeks) and Basic training season 2 (BTS2, 12 weeks). Training during the whole year after the baseline and during the follow-up after 3.5 years and the follow-up after 6.5 years (28 weeks before the baseline and follow-ups and 24 weeks after the baseline and follow-ups). P-value pre vs. post, Friedman's test. Mean ± SD.

		N	Baseline	3.5 years	6.5 years	ANOVA
BTS1	km · week^{-1}	5	64.1 ± 20.0	104.5 ± 25.1	105.7 ± 35.0	n.s.
(16 weeks)	h · week^{-1}	11	8.8 ± 4.6	11.5 ± 3.0	10.3 ± 4.5	n.s.
	times · week^{-1}	9	6.1 ± 4.0	8.0 ± 3.5	7.5 ± 3.9	$p < 0.01$
BTS2	km · week^{-1}	5	77.2 ± 23.1	114.7 ± 42.6	113.6 ± 38.1	n.s.
(12 weeks)	h · week^{-1}	7	9.5 ± 3.9	13.0 ± 2.6	10.7 ± 4.6	$p < 0.05$
	times · week^{-1}	6	7.6 ± 4.3	8.3 ± 2.9	7.6 ± 3.4	n.s.
Year	km · year^{-1}	4	3856 ± 1248	5123 ± 768	6124 ± 1260	n.s.
(52 weeks)	h · year^{-1}	11	454 ± 199	569 ± 85	475 ± 239	n.s.
	times · year^{-1}	9	353 ± 238	389 ± 132	404 ± 205	n.s.

TABLE 10 Skiers' training (4.5y2mSC) during the 28 weeks before the baseline and before the follow-up after 4.5 years. 28 weeks consists of Basic training season 1 (BTS1, 16 weeks) and Basic training season 2 (BTS2, 12 weeks). Training during the whole year (28 weeks before the baseline and follow-up and 24 weeks after the baseline and follow-up). P-value pre vs. post, Friedman's test. Mean ± SD.

		N	Baseline	4.5 years	p-value
BTS1	km · week^{-1}	6	92.1 ± 54.2	132.9 ± 65.1	$p < 0.05$
(16 weeks)	h · week^{-1}	12	10.5 ± 3.1	11.9 ± 4.3	n.s.
	times · week^{-1}	9	8.2 ± 4.9	8.6 ± 3.6	n.s.
BTS2	km · week^{-1}	6	114.1 ± 53.0	151.0 ± 46.9	n.s.
(12 weeks)	h · week^{-1}	11	14.2 ± 5.9	12.7 ± 3.3	n.s.
	times · week^{-1}	8	9.3 ± 3.3	8.7 ± 2.9	n.s.
Year	km · year^{-1}	6	5148 ± 2368	7240 ± 1910	$p < 0.05$
(52 weeks)	h · year^{-1}	10	563 ± 185	537 ± 235	n.s.
	times · year^{-1}	8	450 ± 259	445 ± 209	n.s.

4.8 Physical Activity of Control Subjects

The physical activity of the control subjects was studied with a questionnaire, in which they were asked about the sports practised during the year, the duration of active periods in a year (months, weeks), the frequency of activity (times · month^{-1}, times · vk^{-1}) and the bout spent on each time of activity (minutes). The physical activity of the year preceding the beginning of the study was 195.5 ± 143.4 hours · year^{-1}, and at the end of the study 73.7 ± 79.4 hours · year^{-1} ($p < 0.05$). The control subjects went in for 2.6 ± 1.7 different sports at the beginning of the study, and 2.2 ± 2.2 at its end. The time spent on different sports during the year preceding the measurements was 3.7 ± 2.8 hours · week^{-1}, and 1.5 ± 1.5 hours · year^{-1} ($p < 0.05$). The sports engaged in by the control subjects were walking, ball games (rink bandy, floorball, tennis, football, badminton, and volleyball), gym, cycling and skiing. The reduction in the time spend on sports at the end of the study was due to engagement on military

service during which period the amount of non-sport physical stress was, however, considerably greater than experienced during their school years.

4.9 Echocardiographic Measurements

The echocardiographic measurements were taken with an ultrasound scanner (ALOKA ECHO CAMERA LS, model SSD-280 LS, Aloka Co. Ltd, Tokyo, Japan). The transducer was a mechanical 3.0 MHz sector transducer. The device gave a B- and M-mode real time or still display. The frame rate of the B-mode was 30 FPS (frame · sec^{-1}). The M-mode display had three alternatives in scanning rate: the time needed to scan one image F (fast) 1.6 seconds, M (middle) 3.8 seconds, and S (slow) 7.6 seconds. A printer (ALOKA ULTRASONO RECORDER, SSZ-95, Aloka Co., Ltd, Tokyo, Japan) was used to print the B- and M-mode images on special paper (3M, 7772 Dry Silver, St Paul, MN, USA).

Echocardiograms at rest were taken when the subjects were lying on their left side, the left hand being bent under the head. The examining point in the heart was 1 - 3 cm from the left edge of the sternum between the third or fourth rib. By using the B-mode image as guide, the M-mode recordings were registered on the apical side of the points of the mitral valve leaflets before the level of the papillary muscle as much in the middle of the ventricle as possible and as vertically as possible towards both the septal and posterior wall. The echoes were measured using the leading edge method, where the measurement is carried out from the front edge of the echo (the edge of the echo closest to the transducer) to the front edge of the following echo (Feigenbaum 1986). The electrocardiogram (ECG) of the subjects was monitored throughout the test with the ECG equipment of the echo camera.

4.10 Analysis of the Echocardiograms

The echocardiograms were analysed according to the recommendations of the American Society of Echocardiography (A.S.E.) (Feigenbaum 1986, Figure 2). The echocardiographic variables were determined from rest echocardiograms with the help of a computer program developed at the Department of Biology of Physical Activity of Jyväskylä University with digitalizing table (Summagraphics, SummaSketch, Model MM1201, Summagraphics Corporation, Fairfield, Connecticut, USA) and cursor. The resolution of the digitalizing table was 0.05 mm. The digitalizing was done during 3 - 5 cycles. The speed of the paper (1 mark interval = 1/2 second) was calibrated from the M-mode along the x - axis and the size of the strengthening interval (1 mark interval = 1 cm) was calibrated along the y - axis. The pulse was recorded from

the ECG in the M-mode. If the measuring point was not, when observed from the B-mode, vertically towards the walls of the left ventricle, an edge correction was made in order to obtain digitalized variables of the correct size.

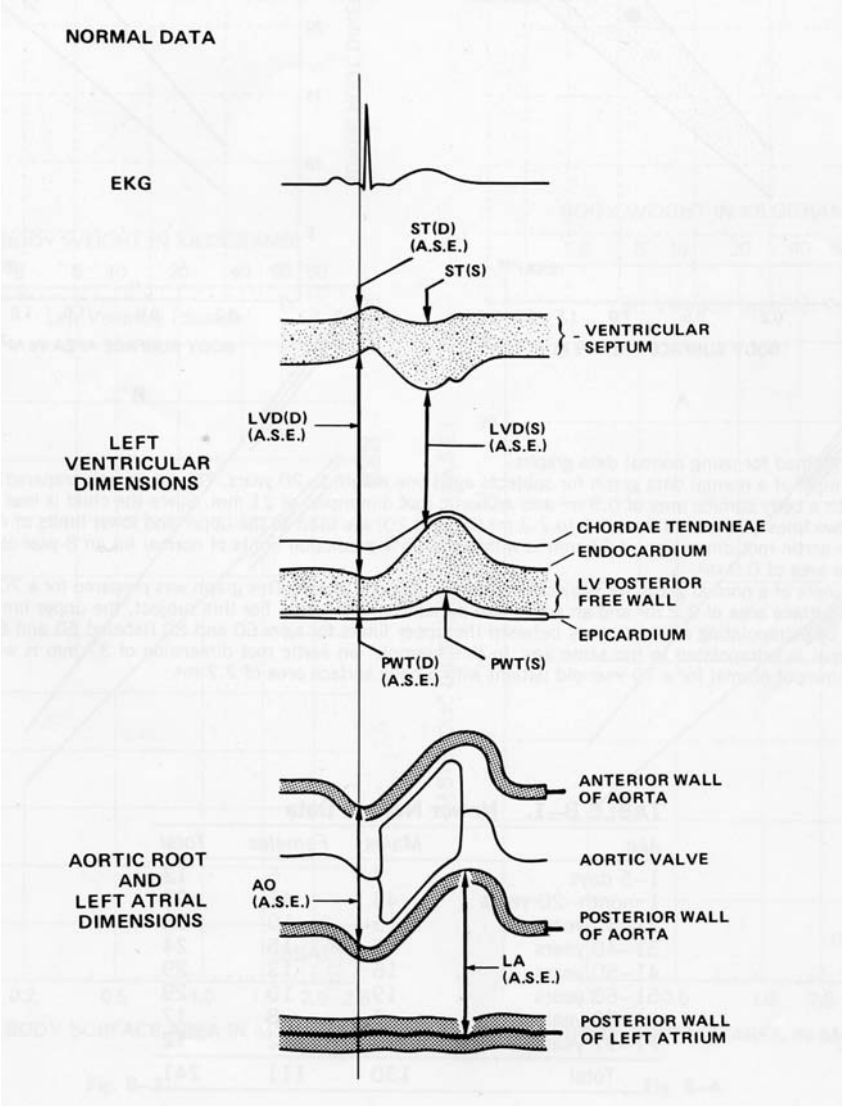

FIGURE 2 Methods of measurement according to the American Society of Echocardiography (Feigenbaum 1986).

4.11 Echocardiographic Variables

The following echocardiographic variables were determined from the M-mode:

1. Left ventricular end-diastolic diameter (EDD)
2. Left ventricular end-systolic diameter (ESD)
3. Septal wall thickness in diastole (SWTd)
4. Posterior wall thickness in diastole (PWTd)

Calculated parameters and calculation equations:

1. Stroke dimension (SD) = EDD – ESD
2. End-diastolic volume (EDV) = $\{7.0 \times (EDD)^3\} / (2.4 + EDD)$ (Teichholz et al. 1976)
3. End-systolic volume (ESV) = $\{7.0 \times (ESD)^3\} / (2.4 + ESD)$ (Teichholz et al. 1976)
4. Stroke volume (SV) = EDV – ESV
5. Left ventricular mass (LVM) = $0.80 \times \{1.04 ((SWTd + EDD + PWTd)^3 – EDD)^3 \} + 0.6$ g (Devereux et al. 1986)
6. Fractional shortening (FS) = $\{(EDD – ESD) / EDD\} \times 100$
7. Hypertrophy index (HI) = (SWTd + PWTd) / EDD
8. Wall stress index (WS) = $\frac{\text{Systolic blood pressure}}{\frac{EDD}{2} \times \frac{1}{PWTd}}$ (Graettinger 1984)
9. Index of contractility (IC) = Systolic blood pressure / ESV (Crawford et al. 1985)

The absolute values of the echocardiographic variables were normalized using the subjects' body surface (BSA, m^2 = (weight, kg)$^{0.425}$ · (height, cm)$^{0.725}$ · 0.007184) (DuBois & DuBois 1916), height and weight.

4.12 Reproducibility

The reproducibility of the echocardiographic variables was determined at the baseline from two series of measurements so that two readers (R1 and R2) digitized the same variable once (intersubject reproducibility) and one reader (R1) digitized the same variable twice (intrasubject reproducibility). The reproducibility of the echocardiographic variables was tested using the Interclass Correlation Coefficient (ICC) and Coefficient of Variation (CV) tests (Tables 11 and 12).

TABLE 11 Reproducibility of echocardiographic variables in two series of measurements (a and b) at the baseline between two readers (R1 and R2) (Inter). EDD = end-diastolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, ESD = end-systolic diameter. ICC = Interclass correlation, CV = Coefficient of variation.

		INTER			
		Mean ± SD (R1)	Mean ± SD (R2)	ICC	CV (%)
EDD (n = 6)	a	5.51 ± 0.41	5.64 ± 0.45	.90	3.3
EDD (n = 10)	b	5.68 ± 0.34	5.83 ± 0.37	.90	2.8
SWTd (n = 6)	a	0.95 ± 0.08	0.89 ± 0.04	.71	5.1
SWTd (n = 11)	b	0.96 ± 0.13	0.90 ± 0.07	.74	6.8
PWTd (n = 6)	a	0.99 ± 0.05	0.88 ± 0.03	.77	2.9
PWTd (n = 11)	b	0.96 ± 0.08	0.89 ± 0.05	.76	5.0
ESD (n = 6)	a	4.11 ± 0.27	3.94 ± 0.21	.72	4.5
ESD (n = 10)	b	3.87 ± 0.25	4.00 ± 0.29	.86	3.8

TABLE 12 Reproducibility of echocardiographic variables in two series of measurements (a and b) at the baseline between the same reader (R1) (Intra). EDD = end-diastolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, ESD = end-systolic diameter. ICC = Interclass correlation, CV = Coefficient of variation.

		INTRA			
		Mean ± SD (R1)	Mean ± SD (R1)	ICC	CV (%)
EDD (n = 6)	a	5.51 ± 0.41	5.57 ± 0.42	.98	1.6
EDD (n = 10)	b	5.68 ± 0.34	5.75 ± 0.35	.97	1.4
SWTd (n = 6)	a	0.95 ± 0.08	0.92 ± 0.06	.93	2.7
SWTd (n = 11)	b	0.96 ± 0.13	0.93 ± 0.10	.93	3.7
PWTd (n = 6)	a	0.99 ± 0.05	0.94 ± 0.04	.88	2.2
PWTd (n = 11)	b	0.96 ± 0.08	0.92 ± 0.06	.87	3.6
ESD (n = 6)	a	4.11 ± 0.27	4.02 ± 0.23	.93	2.3
ESD (n = 10)	b	3.87 ± 0.25	3.93 ± 0.26	.96	1.9

When digitalizing the echocardiograms the reader did not know the earlier values of the subjects' echocardiographic variables. The name of the subject was known because the computer program developed to digitalize echocardiograms required data on the subject's height, weight and blood pressure. This data was needed in normalizing the echocardiographic variables and in calculating the formulas of the variables.

4.13 Statistical Methods

The statistical analyses were done using SPSSWIN 10.1 and 11.1 programs (SPSS inc., Chicago, Ill., USA). The results are shown as means with standard deviations (SD). The significance of the differences between means was tested using the paired, 2-tailed Student's t-test. Analysis of variance (ANOVA) was used to calculate the significances between the within-group measurement times. The matrix was simple (1), when other measurement times were compared to the first measurement time, and repeated, when successive measurement times were compared. Multiple analysis of variance (MANOVA) was used to determine the group effect of the repeated measurements and interaction between them. If there were differences between the test and control groups, analysis of covariance (ANCOVA) was used in order to determine the differences between the groups in the final measurements. Friedman's test was used as the non-parametric test of repeated measurements. The relation between the variables was studied with Pearson's correlation coefficient, linear regression and multivariable linear regression. The reproducibility of the echocardiographic variables was assessed with the Interclass Correlation (ICC) and Coefficient of Variation (CV) tests. The level of statistical significance was set at $p < 0.05$. All values given in the text are mean ± SD, unless otherwise stated.

5 RESULTS

5.1 Changes during 6.5 years

5.1.1 6.5y2m (6.5 years 2 measurements)

5.1.1.1 Height, Weight, BSA, HR, BP and $\dot{V}O_{2max}$

HR rate and both SBP and DBP decreased at rest significantly during the study ($p < 0.001$) (Table 13).

During the six and a half years the $\dot{V}O_{2max}$ of the skiers improved by 0.6 ± 0.5 $l \cdot min^{-1}$ ($p < 0.001$) and their theoretical maximal oxygen uptake ($\dot{V}O_{2max\ demand}$) by 10.5 ± 6.4 $ml \cdot kg^{-1} \cdot min^{-1}$ ($p < 0.001$). Maximal blood lactate concentration (BLa_{max}) increased during the study by 5.09 ± 2.34 $mmol \cdot l^{-1}$ ($p < 0.001$) (Table 14).

TABLE 13 Height, weight, body surface area (BSA), heart rate (HR) and systolic (SBP) and diastolic (DBP) blood pressure at baseline and at follow-up after 6.5 years. N = 20, p-value baseline vs. 6.5 years.

	Baseline	6.5 years	p-value
Height (cm)	178.9 ± 5.2	181.3 ± 5.2	$p < 0.001$
Weight (kg)	66.3 ± 5.7	73.6 ± 6.4	$p < 0.001$
BSA (m^2)	1.83 ± 0.10	1.94 ± 0.11	$p < 0.001$
HR ($beats \cdot min^{-1}$)	64.5 ± 14.3	52.3 ± 6.7	$p < 0.001$
SBP (mmHg)	125.6 ± 10.4	119.4 ± 8.7	$p < 0.05$
DBP (mmHg)	74.1 ± 11.4	60.8 ± 13.5	$p < 0.001$

TABLE 14 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) at baseline and at follow-up after 6.5 years. N = 20, p-value baseline vs. 6.5 years.

	Baseline	6.5 years	p-value
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	4.5 ± 0.4	5.1 ± 0.6	$p < 0.001$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	68.2 ± 3.0	69.2 ± 6.2	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	275.6 ±12.4	290.1 ± 26.8	$p < 0.05$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}_{(demand)}$)	59.9 ± 3.6	70.4 ± 6.6	$p < 0.001$
HR_{max} ($beats \cdot min^{-1}$)	194.1 ± 8.8	190.7 ± 6.6	$p < 0.05$
BLa_{max} ($mmol \cdot l^{-1}$)	6.77 ± 1.55	11.86 ± 1.71	$p < 0.001$

5.1.1.2 Echocardiographic Variables

EDD of the left ventricle increased by 0.45 ± 0.40 cm ($p < 0.001$) the percentage change being 8.7 %. SWTd became 0.12 ± 0.12 cm thicker ($p < 0.001$) and PWTd 0.09 ± 0.08 cm ($p < 0.001$) thicker (Table 15).

LVM increased by 55.2 ± 34.1 g during the study ($p < 0.001$). Among the volume variables EDV increased by 28.2 ± 25.5 ml ($p < 0.001$) and SV by 25.3 ± 17.9 ml ($p < 0.001$) (Table 16).

At baseline HR correlated negatively with EDD ($r = -.48$, $p < 0.05$), EDV ($r = -.48$, $p<0.05$) and SV ($r = -.47$, $p < 0.05$). EDD did not explain the changes in $\dot{V}O_{2max}$.

The correlations between $\dot{V}O_{2max}$ and echocardiographic variables at follow-up after 6.5 years are shown in Table 17. Fifty-six percent of the changes in EDD and 60 % in LVM were explained by VO_{2max} (Figure 3 and 4). The changes in EDV and SV were explained by VO_{2max} ($l \cdot min^{-1}$) 58 and 49 %, respectively.

TABLE 15 Echocardiographic variables in left lateral position at baseline and at follow-up after 6.5 years. EDD = end-diastolic diameter, ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension. N = 20, p-value baseline vs. 6.5 years.

	Baseline	6.5 years	p-value
EDD (cm)	5.32 ± 0.33	5.77 ± 0.39	$p < 0.001$
EDD/BSA	2.90 ± 0.17	2.98 ± 0.14	n.s.
ESD (cm)	3.83 ± 0.38	3.90 ± 0.41	n.s.
SWTd (cm)	0.84 ± 0.05	0.96 ± 0.12	$p < 0.001$
PWTd (cm)	0.83 ± 0.05	0.92 ± 0.08	$p < 0.001$
SD (cm)	1.39 ± 0.09	1.49 ± 0.11	$p < 0.001$

TABLE 16 Systolic (SBP) and diastolic (DBP) blood pressure and echocardiographic variables in left lateral position at baseline and at follow-up after 6.5 years. LVM = left ventricular mass, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress index, IC = index of contractility. N = 20, p-value baseline vs. 6.5 years.

	Baseline	6.5 years	p-value
SBP (mmHg)	121.0 ± 10.7	132.9 ± 10.2	p < 0.001
DBP (mmHg)	72.8 ± 7.7	81.0 ± 8.8	p < 0.001
LVM (g)	158.9 ± 21.7	214.1 ± 36.1	p < 0.001
LVM/weight	2.44 ± 0.21	2.91 ± 0.41	p < 0.001
LVM/BSA	87.8 ± 4.7	110.5 ± 16.0	p < 0.001
EDV (ml)	137.4 ± 20.0	165.6 ± 26.3	p < 0.001
ESV (ml)	64.2 ± 15.3	67.1 ± 16.2	n.s.
SV (ml)	73.2 ± 11.9	98.5 ± 19.1	p < 0.001
FS (%)	28.0 ± 4.5	32.3 ± 5.1	p < 0.001
HI	0.32 ± 0.02	0.33 ± 0.04	n.s.
WS	37.7 ± 5.2	42.7 ± 6.2	p < 0.001
IC	2.00 ± 0.58	2.10 ± 0.59	n.s.

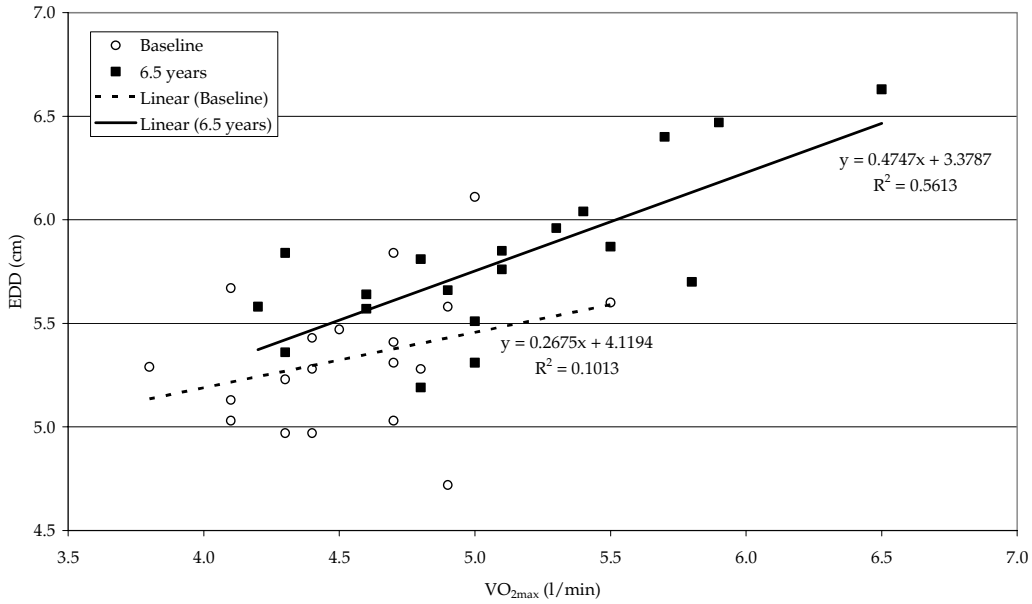

FIGURE 3 The changes in end-diastolic diameter of the left ventricle (EDD) explained by maximal oxygen uptake (VO_{2max}, $l \cdot min^{-1}$) at baseline and at follow-up after 6.5 years.

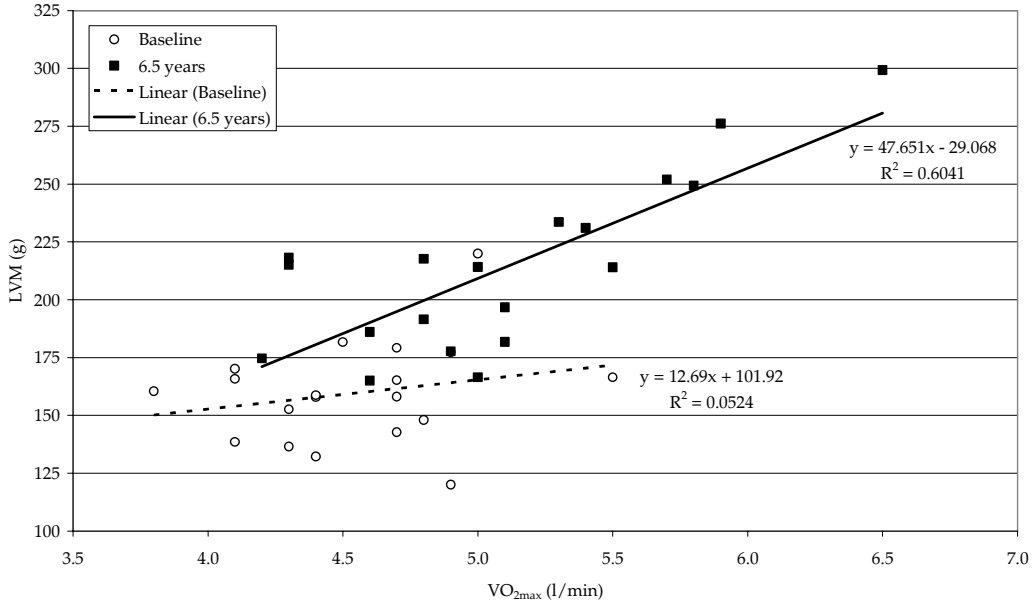

FIGURE 4 The changes in left ventricle mass (LVM) explained by the maximal oxygen uptake ($\dot{V}O_{2max}$, $l \cdot min^{-1}$) at baseline and at follow-up after 6.5 years.

TABLE 17 Correlation coefficients between maximal oxygen uptake ($\dot{V}O_{2max}$) and echocardiographic variables at follow-up after 6.5 years. EDD = end-diastolic diameter, LVM = left ventricular mass, EDV = end-diastolic volume, SV = stroke volume. N = 20, *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

	EDD	LVM	EDV	SV
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	.75 ***	.78 ***	.76 ***	.70 ***
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)		.49 *		
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	.51 *	.65 **	.51 *	.51 *
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}_{(demand)}$)		.56 *		

When examining the individual changes in heart growth of the subjects, the largest value of 1.03 cm was found in subject no. 6 and the next largest value of 0.6 cm in subject no. 16. EDD of subject no. 2 was 6.11 cm already at baseline. In four subjects (nos. 3, 9, 10 and 11) EDD remained almost the same or decreased a little. $\dot{V}O_{2max}$ in proportion to body weight was the highest at the follow-up after 6.5 years in subjects 14 and 16. (Table 18, Figure 5).

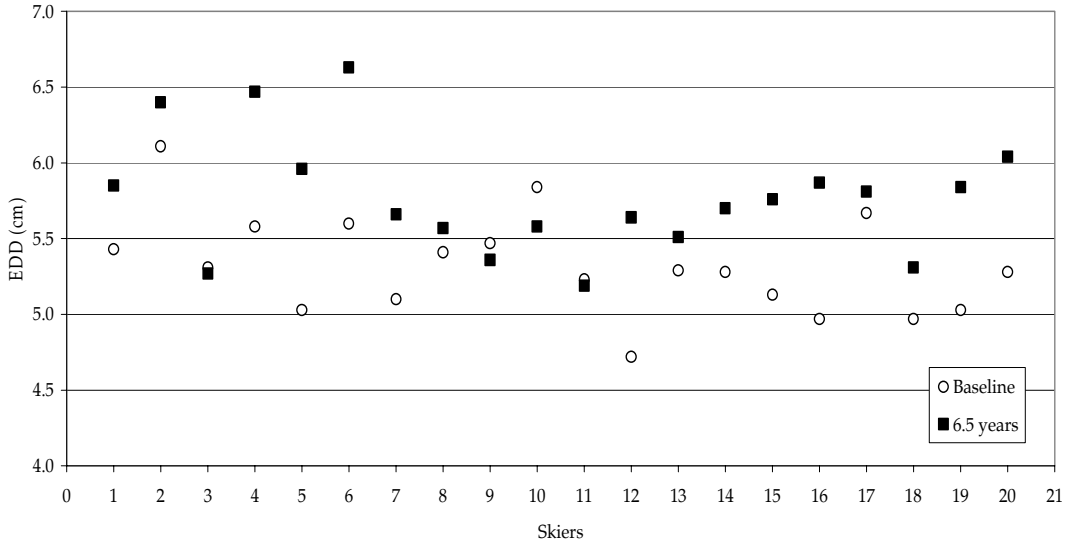

FIGURE 5 Individual values of skiers' end-diastolic diameter (EDD) in left lateral position at baseline and at follow-up after 6.5 years.

TABLE 18 End-diastolic diameter (EDD), maximal oxygen uptake ($\dot{V}O_{2max}$), height and weight of skiers 2, 6, 14 and 16 (three skiers showed the largest increase in EDD) and the skiers 9 and 10 (EDD decreased) at baseline and at follow-up after 6.5 years.

		EDD (cm)	$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	Height (cm)	Weight (kg)
Skier 2	Baseline	6.11	5.0	69	192.8	73.0
	6.5 years	6.40	5.7	74	194.5	76.9
Skier 6	Baseline	5.60	5.5	68	185.7	80.2
	6.5 years	6.63	6.5	72	188.0	90.1
Skier 14	Baseline	5.28	4.8	71	177.6	67.4
	6.5 years	5.70	5.8	82	178.2	70.4
Skier 16	Baseline	4.97	4.4	71	175.8	62.3
	6.5 years	5.87	5.5	77	178.5	71.6
Skier 9	Baseline	5.47	4.5	70	178.7	64.3
	6.5 years	5.36	4.3	62	180.0	69.5
Skier 10	Baseline	5.84	4.7	64	182.1	73.2
	6.5 years	5.58	4.2	59	182.1	71.2

5.1.2 EDD-SL

Skiers were divided in two groups according to the baseline EDD. The average EDD of skiers with small hearts (S, n = 10) was 5.07 ± 0.17 cm (4.72 - 5.28 cm) and the average EDD of skiers with large hearts (L, n = 10) was 5.57 ± 0.25 cm (5.29 - 6.11 cm). The EDD of skiers with small hearts increased by 0.63 cm (12.4%) and the EDD of skiers with large hearts by 0.28 cm (4.9 %) during the 6.5-year research period. The EDD values of skiers with small hearts were not, however, as big as those of skiers with large hearts at the follow-up after 6.5 years. EDD and EDD/BSA showed interaction, but analysis of covariance

revealed no notable difference between the groups at the follow-up after 6.5 years (Table 19).

LVM, EDV, ESV and SV of the small-hearted subjects also increased more than those of the large-hearted, though not significantly. The final SV value was almost identical in both groups. Examination of percentage changes showed that the echocardiographic variables of the small-hearted subjects had grown more than those of the large-hearted subjects, PWTd, HI, WS and IC variables excluded (Table 20).

TABLE 19 Height, weight, body surface area (BSA) and echocardiographic variables of the skiers with small (S, n = 10) and large (L, n = 10) end-diastolic diameter at baseline and at follow-up after 6.5 years. EDD = end-diastolic diameter, ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension.

	Group	Baseline	6.5 years	ANOVA, interaction
Height (cm)	S	176.8 ± 2.9	179.6 ± 2.9	
	L	181.0 ± 6.2	182.9 ± 6.6	n.s.
Weight (kg)	S	64.9 ± 3.6	72.1 ± 4.7	
	L	67.8 ± 7.2	75.0 ± 7.8	n.s.
BSA (m^2)	S	1.80 ± 0.06	1.91 ± 0.08	
	L	1.87 ± 0.12	1.96 ± 0.13	n.s.
EDD (cm)	S	5.07 ± 0.17	5.70 ± 0.27	
	L	5.57 ± 0.25	5.85 ± 0.49	$p < 0.05$
EDD/BSA	S	2.82 ± 0.15	2.99 ± 0.14	
	L	2.99 ± 0.16	2.98 ± 0.15	$p < 0.05$
ESD (cm)	S	3.58 ± 0.26	3.76 ± 0.38	
	L	4.09 ± 0.30	4.05 ± 0.40	n.s.
SWTd (cm)	S	0.84 ± 0.06	0.98 ± 0.11	
	L	0.85 ± 0.04	0.94 ± 0.12	n.s.
PWTd (cm)	S	0.82 ± 0.05	0.89 ± 0.08	
	L	0.84 ± 0.04	0.96 ± 0.08	n.s.
SD (cm)	S	1.42 ± 0.10	1.52 ± 0.09	
	L	1.37 ± 0.08	1.45 ± 0.13	n.s.

TABLE 20 Echocardiographic variables of skiers with small (S, n = 10) and large (L, n = 10) end-diastolic diameter at baseline and at follow-up after 6.5 years. LVM = left ventricular mass, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress, IC = index of contractility.

	Group	Baseline	6.5 years	ANOVA, interaction
LVM (g)	S	144.1 ± 13.3	207.3 ± 27.7	
	L	173.6 ± 18.4	220.9 ± 43.3	n.s.
LVM/weight	S	2.48 ± 0.13	2.88 ± 0.41	
	L	2.39 ± 0.27	2.94 ± 0.43	n.s.
LVM/BSA	S	89.2 ± 3.0	108.8 ± 14.7	
	L	86.4 ± 5.8	112.1 ± 17.9	n.s.
EDV (ml)	S	122.5 ± 9.5	160.3 ± 17.0	
	L	152.2 ± 16.3	171.0 ± 33.2	n.s.
ESV (ml)	S	54.0 ± 9.7	61.2 ± 14.3	
	L	74.3 ± 13.0	73.0 ± 16.6	n.s.
SV (ml)	S	68.5 ± 10.7	99.1 ± 7.1	
	L	77.9 ± 11.6	98.0 ± 26.8	n.s.
FS (%)	S	29.4 ± 4.8	34.1 ± 4.0	
	L	26.6 ± 3.9	30.6 ± 5.7	n.s.
HI	S	0.33 ± 0.02	0.33 ± 0.04	
	L	0.30 ± 0.02	0.33 ± 0.04	n.s.
WS	S	40.0 ± 6.0	42.3 ± 7.0	
	L	35.4 ± 3.1	43.2 ± 5.7	$p < 0.05$
IC	S	2.37 ± 0.53	2.32 ± 0.62	
	L	1.64 ± 0.35	1.89 ± 0.49	n.s.

The only notable change in $\dot{V}O_{2max}$ took place in variable ml · $kg^{-2/3}$ · min^{-1}, the values of which increased more in small-hearted subjects (19.7 ml, $p < 0.05$) than large-hearted subjects (9.1 ml, n.s.) (Table 21).

No statistical differences were observed between the groups in the training during the study.

The individual EDD values of small-hearted and large-hearted subjects with their changes are shown in Figure 6. EDD increased most in large-hearted subject no. 6 (1.03 cm) followed by small-hearted subjects nos. 5 (0.93 cm), 12 (0.92 cm) and 16 (0.90 cm).

TABLE 21 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) of the skiers with small (S, n = 10) and large (L, n = 10) hearts at baseline and at follow-up after 6.5 years.

	Group	Baseline	6.5 years	ANOVA, interaction
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	S	4.4 ± 0.3	5.1 ± 0.5	
	L	4.6 ± 0.5	5.1 ± 0.8	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	S	68.4 ± 3.5	70.9 ± 6.1	
	L	67.9 ± 2.4	67.6 ± 6.2	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	S	274.6 ± 13.3	294.3 ± 24.7	
	L	276.7 ± 12.2	285.8 ± 29.5	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}_{demand}$)	S	60.4 ± 4.0	71.2 ± 6.8	
	L	59.1 ± 3.5	69.7 ± 7.1	n.s.
HR_{max} ($beats \cdot min^{-1}$)	S	195.8 ± 7.9	192.9 ± 6.4	
	L	191.1 ± 9.1	188.0 ± 6.4	n.s.
BLa_{max} ($mmol \cdot l^{-1}$)	S	6.74 ± 1.33	12.10 ± 1.44	
	L	6.78 ± 1.91	11.64 ± 2.07	n.s.

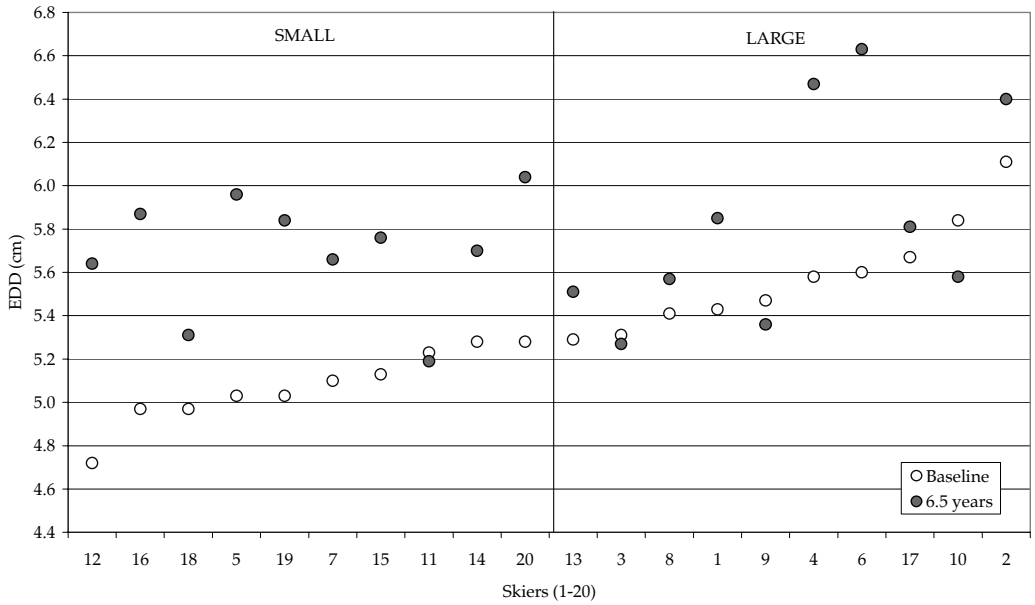

FIGURE 6 Individual values of end-diastolic diameter (EDD) of the skiers with small (n = 10) and large (n = 10) hearts at baseline and at follow-up after 6.5 years.

The effect of growth in height on the EDD values of both small- and large-hearted subjects was studied. At baseline there was a negative correlation in the group with small heart, which became positive at follow-up after 6.5 years; the explanation percentage, however, was low. The explanation percentage for the large-hearted was almost the same both at baseline and at follow-up after 6.5 years (Figures 7 and 8).

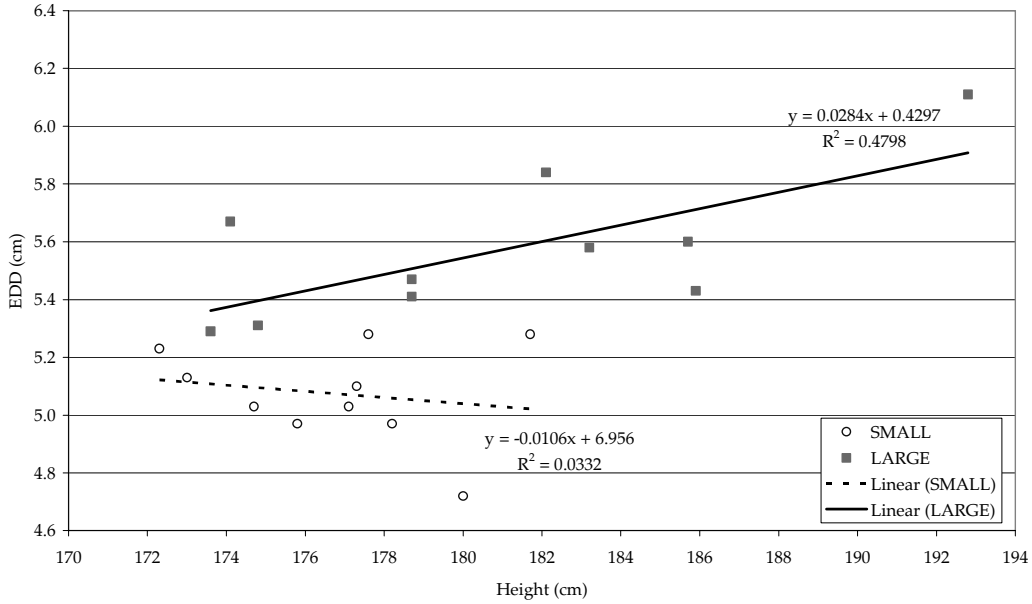

FIGURE 7 Height as the explanation of changes in end-diastolic diameter (EDD) in skiers with small and large hearts at baseline.

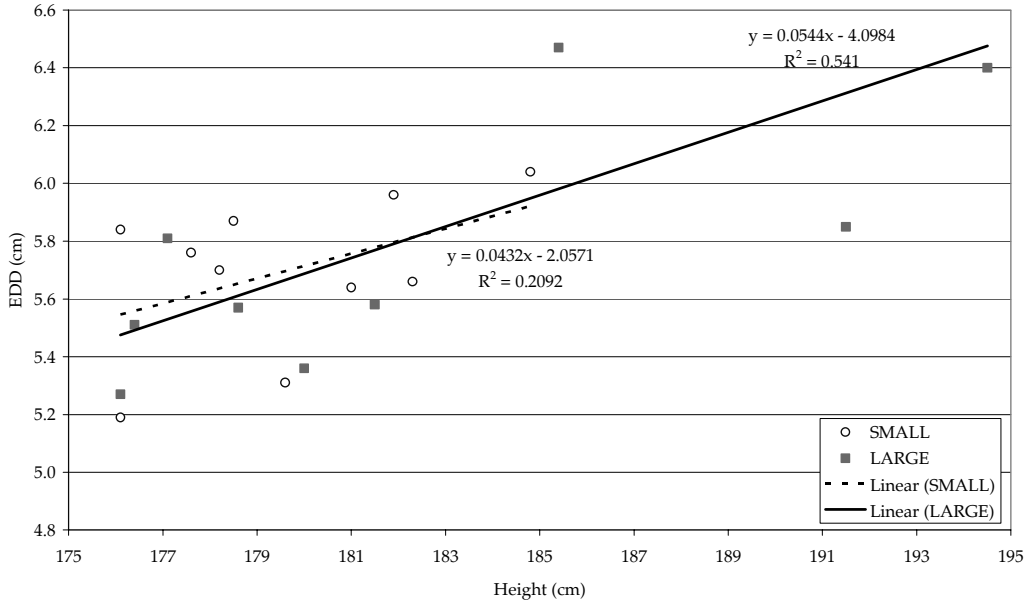

FIGURE 8 Height as the explanation of changes in end-diastolic (EDD) in skiers with small and large hearts at follow-up after 6.5 years.

5.2 Timing of Changes

5.2.1 6.5y3m (6.5 years 3 measurements)

5.2.1.1 Height, Weight, BSA, HR, BP and $\dot{V}O_{2max}$

HR and SBP and DBP decreased at rest during the study ($p < 0.05$) (Table 22). Absolute $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) improved in the skiers during the first 3.5- year period 0.6 $l \cdot min^{-1}$ ($p < 0.001$), but remained almost unchanged during the last 3-year period. $\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$) improved by 16.7 $ml \cdot kg^{-2/3} \cdot min^{-1}$ ($p < 0.05$) and $ml \cdot kg^{-1} \cdot min^{-1}$ (demand) by 11.1 $ml \cdot kg^{-1} \cdot min^{-1}$ ($p < 0.001$) during the study, the biggest changes occurring during the first 3.5-year period ($p < 0.001$ correspondingly) (Table 23).

TABLE 22 Height, weight, body surface area (BSA), heart rate (HR) and systolic (SBP) and diastolic (DBP) blood pressure at baseline and at follow-ups after 3.5 years and 6.5 years. N = 14.

	Baseline	3.5 years	6.5 years	ANOVA
Height (kg)	178.8 ± 6.1	181.1 ± 6.2	181.3 ± 6.1	$p < 0.001$
Weight (kg)	65.8 ± 6.6	73.3 ± 8.5	73.1 ± 7.4	$p < 0.001$
BSA (m^2)	1.83 ± 0.12	1.93 ± 0.13	1.93 ± 0.12	$p < 0.001$
HR ($beats \cdot min^{-1}$)	64.7 ± 14.8	54.8 ± 9.2	52.1 ± 7.1	$p < 0.05$
SBP (mmHg)	125.4 ± 10.6	123.6 ± 8.4	117.4 ± 7.8	$p < 0.05$
DBP (mmHg)	71.8 ± 11.9	62.1 ± 4.7	57.4 ± 14.2	$p < 0.01$

TABLE 23 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) at baseline and at follow-ups after 3.5 years and 6.5 years. N = 14.

	Baseline	3.5 years	6.5 years	ANOVA
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	4.4 ± 0.4	5.0 ± 0.6	5.1 ± 0.6	$p < 0.001$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	67.4 ± 3.1	67.7 ± 2.4	68.9 ± 5.6	n.s.
$\dot{V}O_{2max}$($ml \cdot kg^{-2/3} \cdot min^{-1}$)	271.4 ± 12.0	283.1 ± 15.1	288.1 ± 24.6	$p < 0.05$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$(demand))	59.4 ± 4.0	69.5 ± 3.0	70.5 ± 6.2	$p < 0.001$
HR_{max} ($beats \cdot min^{-1}$)	192.8 ± 8.6	194.5 ± 8.5	189.5 ± 6.3	$p < 0.01$
BLa_{max} ($mmol \cdot l^{-1}$)	6.36 ± 1.43	10.55 ± 1.36	11.96 ± 1.98	$p < 0.001$

5.2.1.2 Echocardiographic Variables

EDD of the left ventricle increased by 0.3 cm ($p < 0.001$) during the first 3.5-year period, but the walls increased most during the last 3-year period (SWTd, $p < 0.001$; PWTd, $p < 0.01$). SD also showed a greater increase during the last three years ($p < 0.01$) than first 3.5 years (n.s.) (Table 24).

TABLE 24 Echocardiographic variables in left lateral position at baseline and at follow-ups after 3.5 years and 6.5 years. EDD = end-diastolic diameter, EDD/BSA = EDD/body surface area, ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension. N = 14.

	Baseline	3.5 years	6.5 years	ANOVA
EDD (cm)	5.38 ± 0.33	5.68 ± 0.37	5.74 ± 0.42	$p < 0.01$
EDD/BSA	2.95 ± 0.16	2.94 ± 0.16	2.98 ± 0.15	n.s.
ESD (cm)	3.88 ± 0.43	4.07 ± 0.36	3.92 ± 0.37	$p < 0.01$
SWTd (cm)	0.85 ± 0.05	0.85 ± 0.05	0.97 ± 0.11	$p < 0.01$
PWTd (cm)	0.83 ± 0.05	0.86 ± 0.05	0.93 ± 0.09	$p < 0.01$
SD (cm)	1.39 ± 0.11	1.40 ± 0.08	1.47 ± 0.11	$p < 0.01$

LVM increased during the last 3-year period more ($p < 0.001$) than during the first 3.5-year period ($p < 0.01$), the total growth being 50.6 ± 32.2 g ($p < 0.001$) in 6.5 years. EDV increased more during the first 3.5-year period (18.5 ± 17.0 ml, $p < 0.001$) than during the last 3-year period (4.6 ± 16.7 ml, n.s.), the growth being 23.1 ± 26.4 ml ($p < 0.01$) in 6.5 years. SV increased almost as much during both three-year periods ($p < 0.05$). SV increased by 22.0 ± 19.3 ml ($p < 0.001$) during 6.5 years (Table 25).

HR correlated negatively ($r = -.56$, $p < 0.05$) with SV only at the baseline. LVM, EDV and SV correlated with $\dot{V}O_{2max}$ at every measurement time and EDD at the baseline and at the follow-up after 6.5 years. The correlations were statistically more significant at the end of the study. $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) explained 36 % of the changes in SV at the baseline and 69 % at the end of the follow-up after 6.5 years (Figure 9).

TABLE 25 Systolic (SBP) and diastolic (DBP) blood pressure and echocardiographic variables at baseline and at follow-ups after 3.5 years and 6.5 years. LVM = left ventricular mass, LVM/BSA = LVM/body surface area, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress index, IC = index of contractility. N = 14.

	Baseline	3.5 years	6.5 years	ANOVA
SBP (mmHg)	122.5 ± 10.3	131.4 ± 13.3	133.1 ± 9.0	$p < 0.01$
DBP (mmHg)	74.3 ± 7.8	80.4 ± 9.8	81.1 ± 8.8	$p < 0.01$
LVM (g)	163.3 ± 22.1	183.7 ± 22.4	213.9 ± 33.7	$p < 0.001$
LVM/weight	2.46 ± 0.24	2.52 ± 0.27	2.93 ± 0.36	$p < 0.01$
LVM/BSA	88.2 ± 5.5	95.2 ± 9.0	110.7 ± 14.1	$p < 0.001$
EDV (ml)	140.9 ± 20.6	159.4 ± 23.8	164.0 ± 28.1	$p < 0.01$
ESV (ml)	66.4 ± 17.2	73.5 ± 14.6	67.5 ± 14.2	$p < 0.05$
SV (ml)	74.5 ± 12.4	85.9 ± 15.3	96.5 ± 22.4	$p < 0.01$
FS (%)	27.9 ± 5.2	28.4 ± 4.0	31.7 ± 5.0	$p < 0.01$
HI	0.31 ± 0.02	0.30 ± 0.03	0.33 ± 0.05	$p < 0.05$
WS	37.9 ± 5.7	40.2 ± 6.3	43.4 ± 6.9	$p < 0.05$
IC	1.99 ± 0.64	1.88 ± 0.57	2.07 ± 0.57	$p < 0.05$

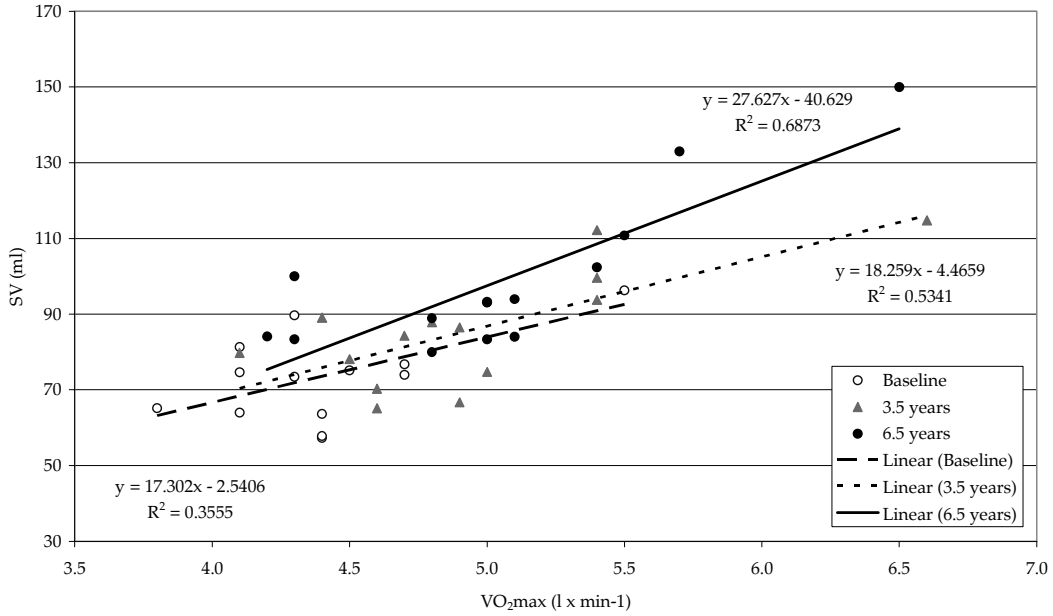

FIGURE 9 Changes in stroke volume (SV) explained by maximal oxygen uptake ($\dot{V}O_{2max}$, $l \cdot min^{-1}$) at baseline and at follow-ups after 3.5 years and 6.5 years.

On the individual level four subjects showed 0.61 cm growth in EDD during the first 3.5 years (subjects 6, 16, 19 and 20). EDD also increased most in the same subjects during 6.5 years: no. 6 (1.03 cm), no. 16 (0.90 cm), no. 19 (0.81 cm) and no. 20 (0.76 cm). Four subjects had a smaller EDD at the follow-up after 6.5 years than at baseline. EDD increased most in the group LargeBSAchange (Figure 10). Those subjects whose EDD had increased during 6.5 years also showed a 1-6 ml $\cdot$ kg^{-1} $\cdot$ min^{-1} improving in $\dot{V}O_{2max}$ whereas those subjects whose EDD had diminished also had a reduction in $\dot{V}O_{2max}$ of 2 - 8 ml $\cdot$ kg^{-1} $\cdot$ min^{-1} (Table 26).

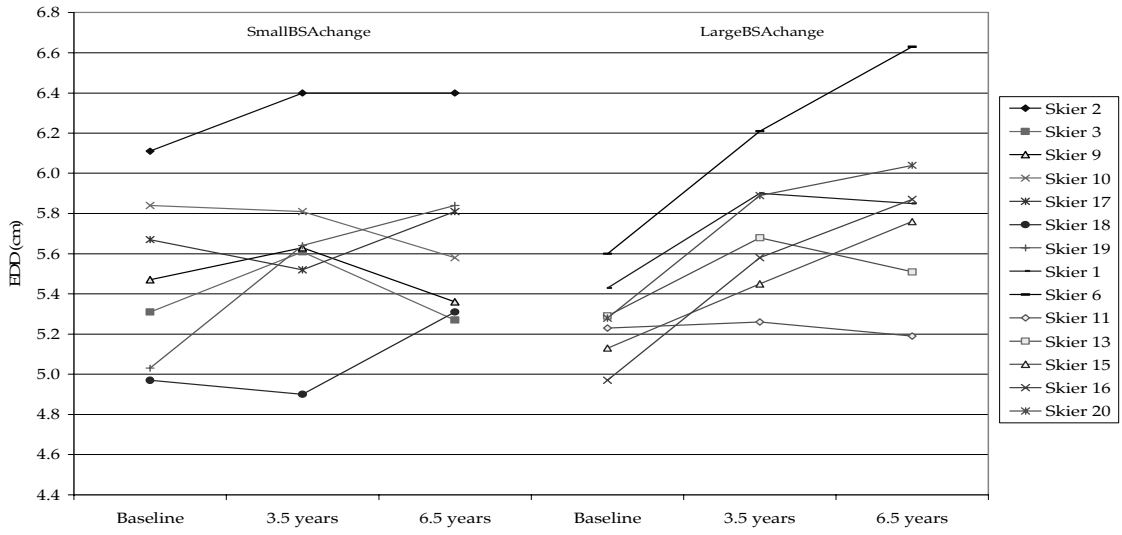

FIGURE 10 The individual end-diastolic diameter (EDD) values of skiers showing small and large change in BSA at baseline and at follow-ups after 3.5 years and 6.5 years.

TABLE 26 The end-diastolic diameter (EDD), maximal oxygen uptake ($\dot{V}O_{2max}$), height and weight of skiers 6, 16, 19 and 20 at baseline and at follow-ups after 3.5 years and 6.5 years. The increase in EDD of these skiers was largest both after three and a half and after six and a half years.

		EDD (cm)	$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	Height (cm)	Weight (kg)
No. 6	Baseline	5.60	5.5	68	185.7	80.2
	3.5 years	6.21	6.6	69	187.3	95.4
	6.5 years	6.63	6.5	72	188.0	90.1
No. 16	Baseline	4.97	4.4	71	175.8	62.3
	3.5 years	5.58	4.9	72	178.4	69.1
	6.5 years	5.87	5.5	77	178.5	71.6
No. 19	Baseline	5.03	4.1	66	174.7	62.8
	3.5 years	5.64	4.4	68	175.8	64.6
	6.5 years	5.84	4.3	67	176.1	64.6
No. 20	Baseline	5.28	4.4	63	181.7	69.6
	3.5 years	5.89	5.4	66	184.6	82.4
	6.5 years	6.04	5.4	67	184.8	81.0

5.2.2 Annual Changes (6.5 years 7 measurements)

5.2.2.1 Height, Weight, BSA, HR, BP and $\dot{V}O_{2max}$

This study measured the skiers annually over six and a half years. Eight skiers were present at every measurement time. Changes occurred in HR, SBP and DBP ($p < 0.05$). HR rate was lowest compared to the baseline measurements (64.9 ± 16.8 beats $\cdot$ min^{-1}) during the follow-up after 5.5 years (50.9 ± 8.2 beats $\cdot$ min^{-1}, $p < 0.01$). SBP decreased by 12.1 ± 12.4 mmHg by the follow-up after 6.5 years compared to the baseline ($p < 0.054$), and DBP by 21.5 ± 13.3 mmHg ($p < 0.01$) (Table 27).

In all $\dot{V}O_{2max}$ variables significant changes were found during the 6.5 years. The highest $\dot{V}O_{2max}$ values ($l \cdot min^{-1}$, $ml \cdot kg^{-1} \cdot min^{-1}$, $ml \cdot kg^{-2/3} \cdot min^{-1}$) were recorded at the follow-up after 4.5 years and the highest performance values ($ml \cdot kg^{-1} \cdot min^{-1}_{demand}$) in the follow-up after 5.5 years. The most significant change between the baseline and the follow-up after 4.5 years was in the absolute $\dot{V}O_{2max}$ ($p < 0.01$) and size changes eliminating $\dot{V}O_{2max}$ ($p < 0.05$) variable. $\dot{V}O_{2max}$, measured as demand, grew statistically most between the baseline measurements and the follow-up after 3.5 years ($p < 0.001$) and between the baseline and the follow-up after 5.5 years ($p < 0.001$). $\dot{V}O_{2max}$ per kilo increased most between the follow-up after 3.5 years and after 4.5 years ($p < 0.05$) (Table 28).

5.2.2.2 Echocardiographic Variables

EDD showed no statistically significant changes at any of the measurement points, even if EDD was 0.38 ± 0.45 cm larger at the follow-up after 6.5 years than at the baseline ($p < 0.05$). The largest changes took place between the baseline and at follow-up after 1.5 years ($p < 0.01$) and baseline and fourth year ($p < 0.01$) measurements. The walls were at their biggest at the follow-up after

5.5 years: SWTd increased by 0.11 ± 0.04 cm between the baseline and the follow-up after 5.5 years ($p < 0.001$) and PWTd by 0.11 ± 0.08 cm ($p < 0.01$). LVM increased by 50.3 ± 40.1 g during 6.5 years, but the most statistically significant difference was between the baseline and the follow-up after 4.5 years ($p < 0.001$) and the baseline and the follow-up after 5.5 years ($p < 0.001$). The statistically most significant growth in SV was between the baseline and the follow-up after 1.5 years ($p < 0.01$) and the baseline and the follow-up after 4.5 yeras ($p < 0.01$). SV was biggest in absolute values at the follow-up after 6.5 years and it increased 25.7 ± 16.5 ml ($p < 0.05$) during the 6.5 years (Table 29, Figure 11).

Figure 12 shows how $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) predicted the changes in EDD annually. The explanation percentage was, across all measurement points, 75.

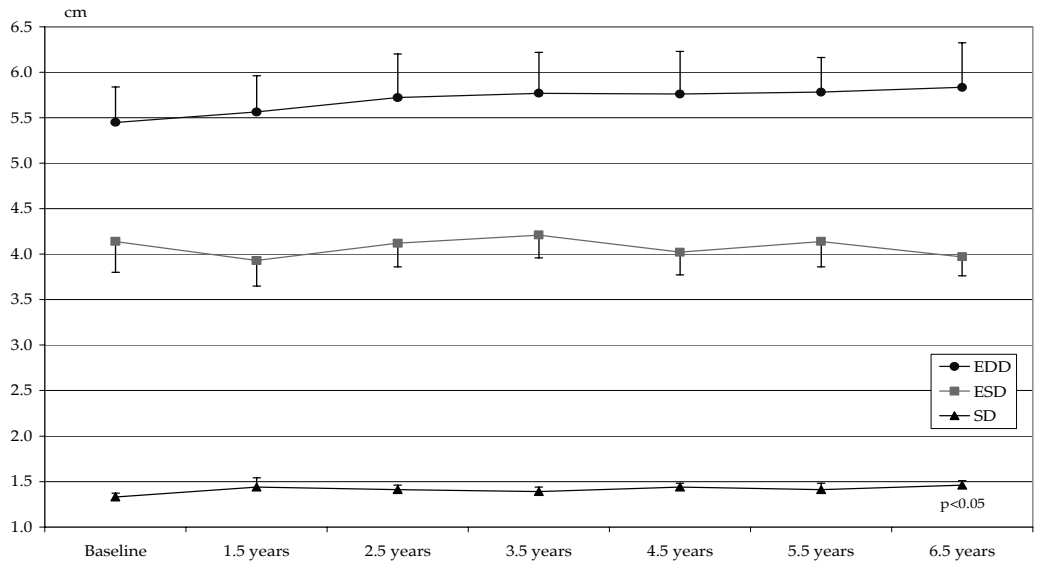

FIGURE 11 End-diastolic diameter (EDD), end-systolic diameter (ESD) and stroke dimension (SD) at baseline and at follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years.

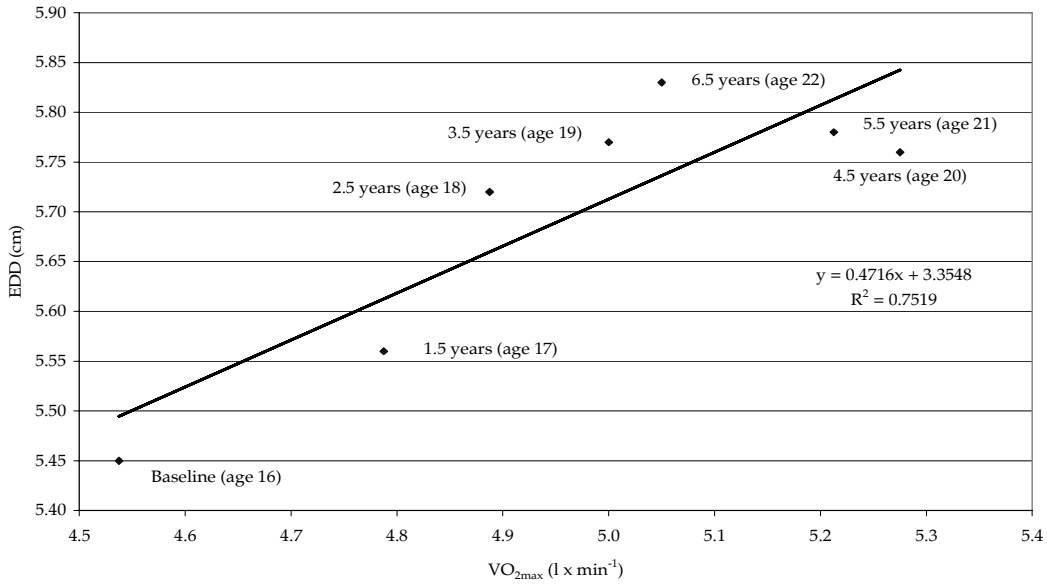

FIGURE 12 End-diastolic diameter (EDD) in relation to maximal oxygen uptake ($\dot{V}O_{2max}$, $l \cdot min^{-1}$) at seven different measurement points (baseline and follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years).

TABLE 27 Height, weight, body surface area (BSA) and heart rate (HR) and systolic (SBP) and diastolic (DBP) blood pressure at baseline and at follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years. N = 8, * Friedman's test.

	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years	npar test*
Height (cm)	181.0 ± 6.2	182.3 ± 5.8	182.3 ± 6.3	182.4 ± 6.3	182.4 ± 6.2	182.6 ± 6.6	182.6 ± 6.2	$p < 0.05$
Weight (kg)	67.8 ± 7.9	70.4 ± 9.1	73.3 ± 10.7	74.4 ± 10.8	73.4 ± 7.5	74.1 ± 7.6	74.2 ± 8.1	$p < 0.001$
BSA (m^2)	1.87 ± 0.13	1.91 ± 0.14	1.94 ± 0.16	1.95 ± 0.16	1.94 ± 0.13	1.95 ± 0.12	1.95 ± 0.13	n.s.
HR (beats · min^{-1})	64.9 ± 16.8	59.6 ± 11.6	56.3 ± 10.7	57.4 ± 9.7	58.4 ± 13.0	50.9 ± 8.2	52.3 ± 8.4	$p < 0.05$
SBP (mmHg)	133.3 ± 9.3	122.5 ± 5.2	130.8 ± 12.0	128.3 ± 8.2	128.0 ± 10.2	122.8 ± 11.1	121.2 ± 10.4	$p < 0.05$
DBP (mmHg)	79.2 ± 9.2	68.3 ± 4.1	67.5 ± 4.2	63.3 ± 5.2	65.2 ± 10.3	61.5 ± 11.2	57.7 ± 15.2	$p < 0.05$

TABLE 28 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) at baseline and at follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years. N = 8, * Friedman's test.

	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years	npar test*
$\dot{V}O_{2max}$ (l · min^{-1})	4.5 ± 0.5	4.8 ± 0.7	4.9 ± 0.8	5.0 ± 0.8	5.3 ± 0.8	5.2 ± 0.7	5.1 ± 0.8	$p < 0.01$
$\dot{V}O_{2max}$ (ml · kg^{-1} ·min^{-1})	67.0 ± 2.7	67.8 ± 3.6	66.5 ± 4.0	67.1 ± 1.9	71.5 ± 5.4	70.1 ± 4.9	67.9 ± 5.2	$p < 0.05$
$\dot{V}O_{2max}$ (ml · $kg^{-2/3}$ ·min^{-1})	272.6 ± 13.3	280.0 ± 21.5	278.6 ± 20.4	281.8 ± 18.3	300.1 ± 26.7	295.0 ± 23.6	285.1 ± 27.9	$p < 0.01$
$\dot{V}O_{2max}$ (ml · kg^{-1} ·min^{-1}_{demand})	59.5 ± 3.0	65.0 ± 3.6	70.5 ± 4.3	69.4 ± 3.3	69.1 ± 6.6	71.8 ± 5.5	69.3 ± 6.7	$p < 0.001$
HR_{max} (beats · min^{-1})	191.0 ± 8.8	193.1 ± 8.4	191.9 ± 8.1	192.9 ± 9.5	189.4 ± 8.5	188.4 ± 5.8	187.8 ± 7.4	$p < 0.01$
BLa_{max} (mmol · l^{-1})	6.48 ± 1.50	8.49 ± 1.58	10.3 ± 2.00	10.58 ± 1.50	9.00 ± 2.40	9.98 ± 1.04	11.26 ± 1.91	$p < 0.05$

TABLE 29 Echocardiographic variables at baseline and at follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years. EDD = end-diastolic diameter, ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension, LVM = left ventricular mass, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress index, IC = index of contractility. N = 8, * Friedman's test.

	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years	npar test *
EDD (cm)	5.45 ± 0.39	5.56 ± 0.40	5.72 ± 0.48	5.77 ± 0.45	5.76 ± 0.47	5.78 ± 0.38	5.83 ± 0.49	n.s.
EDD/BSA	2.92 ± 0.17	2.92 ± 0.18	2.96 ± 0.22	2.96 ± 0.20	2.97 ± 0.15	2.97 ± 0.16	2.99 ± 0.14	n.s.
ESD (cm)	4.14 ± 0.34	3.93 ± 0.28	4.12 ± 0.26	4.21 ± 0.25	4.02 ± 0.25	4.14 ± 0.28	3.97 ± 0.21	n.s.
SWTd (cm)	0.86 ± 0.06	0.85 ± 0.08	0.88 ± 0.06	0.84 ± 0.06	0.93 ± 0.11	0.97 ± 0.07	0.98 ± 0.10	$p < 0.001$
PWTd (cm)	0.85 ± 0.04	0.85 ± 0.04	0.90 ± 0.06	0.86 ± 0.04	0.90 ± 0.08	0.96 ± 0.08	0.93 ± 0.07	$p < 0.05$
SD (cm)	1.33 ± 0.04	1.44 ± 0.10	1.41 ± 0.05	1.39 ± 0.05	1.44 ± 0.04	1.41 ± 0.07	1.46 ± 0.05	$p < 0.05$
LVM (g)	171.1 ± 24.1	175.7 ± 23.4	195.3 ± 24.8	187.4 ± 25.7	205.0 ± 27.6	220.9 ± 31.6	221.4 ± 42.1	$p < 0.001$
LVM/weight	2.40 ± 0.29	2.51 ± 0.33	2.69 ± 0.40	2.54 ± 0.35	2.81 ± 0.41	3.00 ± 0.49	2.97 ± 0.38	$p < 0.001$
LVM/BSA	86.5 ± 6.3	92.1 ± 10.4	100.9 ± 12.1	96.1 ± 11.2	105.7 ± 13.7	113.4 ± 16.7	112.9 ± 16.1	$p < 0.001$
EDV (ml)	145.2 ± 24.3	152.3 ± 26.0	162.8 ± 30.5	165.8 ± 28.7	165.3 ± 32.1	166.2 ± 24.5	170.2 ± 33.2	n.s.
ESV (ml)	76.5 ± 14.7	67.4 ± 11.7	75.5 ± 11.6	79.3 ± 11.3	71.1 ± 11.0	76.5 ± 12.4	68.9 ± 8.6	n.s.
SV (ml)	72.0 ± 12.6	90.6 ± 22.8	91.6 ± 13.6	90.5 ± 13.1	94.7 ± 21.0	91.8 ± 9.3	97.7 ± 19.3	$p < 0.05$
FS (%)	24.8 ± 2.3	30.5 ± 5.1	29.0 ± 2.5	27.9 ± 2.7	30.3 ± 1.7	28.9 ± 3.4	31.4 ± 2.4	$p < 0.05$
HI	0.32 ± 0.03	0.31 ± 0.03	0.31 ± 0.04	0.30 ± 0.03	0.32 ± 0.05	0.34 ± 0.03	0.33 ± 0.04	$p < 0.05$
WS	38.9 ± 5.8	38.6 ± 4.6	43.2 ± 6.2	39.8 ± 5.3	43.2 ± 5.7	45.2 ± 4.6	42.3 ± 3.8	n.s.
IC	1.67 ± 0.46	1.87 ± 0.40	1.80 ± 0.28	1.67 ± 0.33	1.96 ± 0.30	1.80 ± 0.32	1.94 ± 0.23	n.s.

5.2.3 Seasonal Changes

5.2.3.1 HR, BP and $\dot{V}O_{2max}$

In this study the skiers were measured during the fifth year of the study during three different training seasons: Basic training season 1 (BTS1) and 2 (BTS2) and Pre-competition season (PCS). The total research period was 6-7 months and the number of subjects was 15.

HR and SBP and DBP tended to decrease during the period, although not significantly (Table 30). The variables of $\dot{V}O_{2max}$ ($l \cdot min^{-1}$, $ml \cdot kg^{-1} \cdot min^{-1}$, $ml \cdot kg^{-2/3} \cdot min^{-1}$) improved between BTS1 and BTS2 ($p < 0.001$), but no further during the PCS. $\dot{V}O_{2max}$ in proportion to weight improved by 4.5 $ml \cdot kg^{-1} \cdot min^{-1}$, and the variable of $\dot{V}O_{2max}$, in which the effect of body size has been eliminated, by 17.7 $ml \cdot kg^{-2/3} \cdot min^{-1}$. The weight of the subjects remained unchanged during the research period. There were no changes in HR_{max} or maximal blood lactate concentration (BLa_{max})(Table 31).

TABLE 30 Heart rate (HR) and systolic (SBP) and diastolic (DBP) blood pressure during three different training seasons: Basic training season 1 and 2 (BTS1, BTS2) and Pre competition season (PCS). N = 15.

	BTS1	BTS2	PCS	ANOVA
HR (beats · min^{-1})	52.9 ± 0.8	51.0 ± 9.6	50.5 ± 7.1	n.s.
SBP (mmHg)	127.1 ± 10.3	125.0 ± 9.8	121.5 ± 9.0	n.s.
DBP (mmHg)	67.9 ± 14.0	65.7 ± 10.4	63.7 ± 11.9	n.s.

TABLE 31 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) during three different training seasons: Basic training season 1 and 2 (BTS1, BTS2) and Pre competition season (PCS). N = 15.

	BTS1	BTS2	PCS	ANOVA
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	5.1 ± 0.6	5.3 ± 0.5	5.3 ± 0.5	$p < 0.01$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	68.4 ± 4.3	72.9 ± 3.9	72.9 ± 3.5	$p < 0.001$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	286.9 ± 21.8	304.2 ± 15.5	304.6 ± 14.9	$p < 0.001$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}_{(demand)}$)	70.1 ± 5.8	73.4 ± 4.0	73.4 ± 3.6	n.s.
HR_{max} (beats · min^{-1})	190.8 ± 6.3	191.7 ± 7.7	189.9 ± 6.6	n.s.
BLa_{max} ($mmol \cdot l^{-1}$)	11.4 ± 2.9	11.3 ± 1.6	10.1 ± 1.2	n.s.

5.2.3.2 Echocardiographic Variables

There were no statistically significant changes in echocardiographic variables during the different training seasons (Table 32). EDD, EDV and SV correlated with $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) during Basic training seasons 1 and 2, but no longer during Pre-competition season (Table 33).

TABLE 32 Echocardiographic variables during three different training seasons: Basic training seasons 1 and 2 = BTS1 and BTS2, Pre competition season = PCS. EDD = end-diastolic diameter, EDD/BSA (EDD/body surface area), ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension, LVM = left ventricular mass, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress index, IC = index of contractility. N = 15.

	BTS1	BTS2	PCS	ANOVA
EDD (cm)	5.72 ± 0.38	5.77 ± 0.34	5.78 ± 0.39	n.s.
EDD/BSA	2.96 ± 0.14	2.99 ± 0.12	3.01 ± 9.15	n.s.
ESD (cm)	3.99 ± 0.34	3.99 ± 0.44	4.01 ± 0.44	n.s.
SWTd (cm)	0.97 ± 0.07	0.99 ± 0.08	0.99 ± 0.08	n.s.
PWTd (cm)	0.96 ± 0.08	0.95 ± 0.10	0.94 ± 0.10	n.s.
SD (cm)	1.43 ± 0.10	1.45 ± 0.13	1.44 ± 0.10	n.s.
LVM (g)	216.6 ± 29.1	220.8 ± 23.0	220.3 ± 32.8	n.s.
LVM/weight	2.95 ± 0.39	3.01 ± 0.32	3.03 ± 0.42	n.s.
LVM/BSA	112.1 ± 13.9	114.6 ± 11.1	114.6 ± 15.6	n.s.
EDV (ml)	162.3 ± 25.0	165.5 ± 22.8	166.2 ± 25.9	n.s.
ESV (ml)	70.3 ± 14.0	70.6 ± 17.4	71.7 ± 17.8	n.s.
SV (ml)	88.8 ± 18.0	91.6 ± 14.7	92.2 ± 13.3	n.s.
FS (%)	29.7 ± 4.9	30.4 ± 5.9	30.3 ± 4.5	n.s.
HI	0.34 ± 0.03	0.34 ± 0.04	0.34 ± 0.04	n.s.
WS	45.8 ± 6.2	44.7 ± 7.2	44.0 ± 8.8	n.s.
IC	2.00 ± 0.52	2.06 ± 0.72	1.99 ± 0.70	n.s.

TABLE 33 Correlation coefficients between maximal oxygen uptake ($\dot{V}O_{2max}$) and echocardiographic variables during Basic training seasons 1 (BTS1) and 2 (BTS2). EDD = end-diastolic diameter, EDV = end-diastolic volume, SV = stroke volume. N = 15, ** $p < 0.01$, * $p < 0.05$.

	$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)		$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)
	BTS1	BTS2	BTS1
EDD	.52 *	.63 *	
EDV	.55 *	.65 **	
SV	.69 **	.56 *	.59 *

5.3 Impact of Size and Growth of the Subjects

5.3.1 Skiers and Control Subjects

Two years after the baseline with skiers, schoolboy controls matched for age were followed for 4.5 years. There were 15 skiers and 10 control subjects. Both groups were measured for HR, SBP and DBP, anthropometrics, $\dot{V}O_{2max}$ and echocardiographic variables. The baseline measurements of the skiers took place in March, during their competition season. The control subjects were measured in May. The final measurements of the skiers took place in November in the basic training season and of the control subjects in August - September.

5.3.1.1 Height, Weight, BSA, HR, BP and $\dot{V}O_{2max}$

Interaction was found in height ($p < 0.001$), weight ($p < 0.01$) and BSA ($p < 0.01$), and analysis of covariance located differences between the groups (Table 34). In the $\dot{V}O_{2max}$ variables, apart from HR_{max}, differences emerged between skiers and controls ($p < 0.001$). After analysis of covariance no statistical differences were found between the groups at the follow-up after 4.5 years (Table 35).

TABLE 34 Height, weight, BSA, heart rate (HR) and systolic (SBP) and diastolic (DBP) blood pressure in skiers (S, n = 15) and controls (C, n = 10) at baseline and at follow-up after 4.5 years.

		Baseline	4.5 years	ANOVA, interaction	ANCOVA
Height (cm)	S	181.7 ± 5.8	181.9 ± 5.8		
	C	180.1 ± 5.3	182.3 ± 5.5	$p < 0.001$	$p < 0.001$
Weight (kg)	S	71.5 ± 7.6	74.1 ± 7.3		
	C	64.0 ± 6.2	72.2 ± 8.4	$p < 0.01$	$p < 0.05$
BSA (m^2)	S	1.92 ± 0.12	1.95 ± 0.12		
	C	1.82 ± 0.10	1.92 ± 0.12	$p < 0.001$	$p < 0.01$
HR (beats · min^{-1})	S	55.2 ± 9.0	51.2 ± 7.2		
	C	78.3 ± 13.2	65.9 ± 9.5	n.s.	n.s.
SBP (mmHg)	S	128.8 ± 8.3	118.4 ± 7.8		
	C	131.0 ± 14.3	120.4 ± 7.5	n.s.	n.s.
DBP (mmHg)	S	73.3 ± 10.5	57.2 ± 14.2		
	C	76.5 ± 13.3	63.8 ± 5.5	n.s.	n.s.

TABLE 35 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) of skiers (S, n = 15) and controls (C, n = 10) at baseline and at follow-up after 4.5 years.

		Baseline	4.5 years	ANOVA, group effect
$\dot{V}O_{2max}$ (l · min^{-1})	S	5.1 ± 0.6	5.2 ± 0.7	
	C	3.4 ± 0.4	3.8 ± 0.6	$p < 0.001$
$\dot{V}O_{2max}$ (ml · kg^{-1} · min^{-1})	S	70.8 ± 1.8	69.5 ± 6.2	
	C	52.7 ± 3.7	52.8 ± 7.0	$p < 0.001$
$\dot{V}O_{2max}$ (ml · $kg^{-2/3}$ · min^{-1})	S	293.6 ± 14.3	292.1 ± 27.3	
	C	211.7 ± 15.3	218.9 ± 30.2	$p < 0.001$
$\dot{V}O_{2max}$ (ml · kg^{-1} · $min^{-1}_{(demand)}$)	S	69.9 ± 2.8	71.1 ± 6.7	
	C	48.0 ± 3.6	50.5 ± 6.9	$p < 0.001$
HR_{max} (beats · min^{-1})	S	192.4 ± 6.9	188.6 ± 6.2	
	C	194.1 ± 6.8	195.0 ± 6.8	n.s.
BLa_{max} (mmol · l^{-1})	S	10.00 ± 2.41	10.69 ± 1.78	
	C	6.34 ± 1.77	10.29 ± 3.05	$p < 0.001$

5.3.1.2 Echocardiographic Variables

There were no significant differences between the groups in EDD and EDD/BSA. When examining the changes within the groups it was discovered that EDD increased more in controls than skiers at the follow-up after 4.5 years, although the final values of controls did not reach the baseline measurement values of the skiers. There were differences in SWTd and PWTd between the groups ($p < 0.001$), and even with analysis of covariance a significant difference was found between the groups ($p < 0.05$). Both SWTd and PWTd increased more in skiers than controls. Wall growth was biggest in the SWTd of the skiers (0.12 ± 0.09 cm) (Table 36, Figure 13).

LVM, LVM/BSA, ESV, HI and IC differed between skiers and controls ($p < 0.001$), although analysis of covariance found a significant between-groups difference only in LVM at the follow-up after 4.5 years ($p < 0.05$). Examining the changes inside the groups showed that EDV increased by 36.5 ± 29.7 g in skiers and 25.3 ± 16.6 g in controls (Table 37).

TABLE 36 Echocardiographic variables of skiers (S, n = 15) and controls (C, n = 10) at baseline and at follow-up after 4.5 years. EDD = end-diastolic diameter, ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension.

				ANOVA	
		Baseline	4.5 years	Group effect	ANCOVA
EDD (cm)	S	5.69 ± 0.35	5.83 ± 0.43		
	C	5.34 ± 0.39	5.60 ± 0.54	n.s.	n.s.
EDD/BSA	S	2.99 ± 0.16	3.02 ± 0.13		
	C	2.95 ± 0.23	2.91 ± 0.28	n.s.	n.s.
ESD (cm)	S	4.21 ± 0.17	4.03 ± 0.20		
	C	3.78 ± 0.29	3.78 ± 0.37	$p < 0.01$	n.s.
SWTd (cm)	S	0.86 ± 0.06	0.98 ± 0.11		
	C	0.75 ± 0.07	0.81 ± 0.08	$p < 0.001$	$p < 0.05$
PWTd (cm)	S	0.87 ± 0.05	0.94 ± 0.08		
	C	0.76 ± 0.05	0.81 ± 0.10	$p < 0.001$	$p < 0.05$
SD (cm)	S	1.36 ± 0.06	1.44 ± 0.08		
	C	1.42 ± 0.12	1.48 ± 0.08	n.s.	n.s.

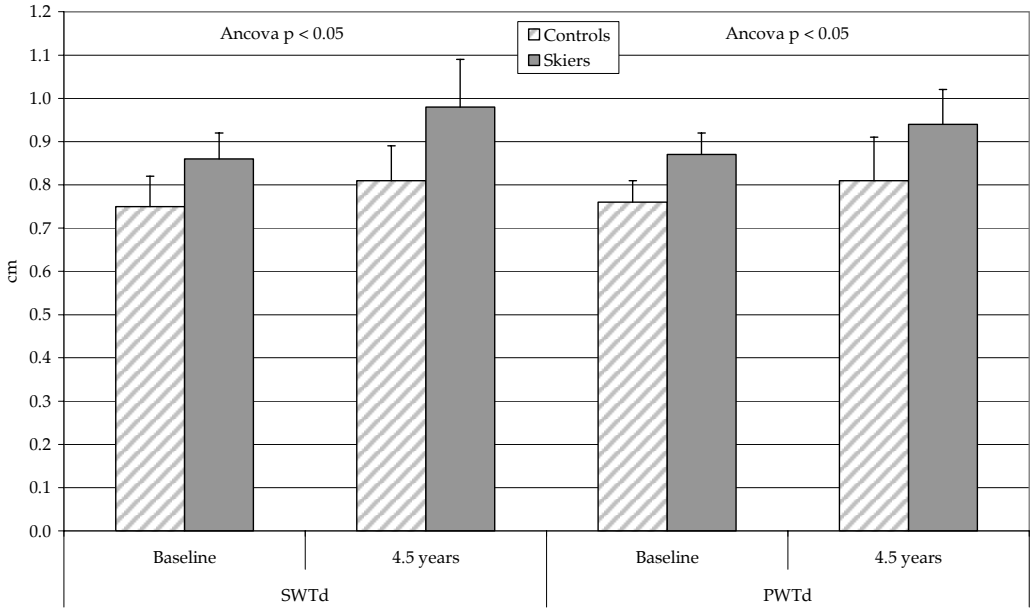

FIGURE 13 Septal wall thickness (SWTd) and posterior wall thickness (PWTd) in diastole of skiers (n = 15) and controls (n = 10) at baseline and at follow-up after 4.5 years.

TABLE 37 Echocardiographic variables of skiers (S, n = 15) and controls (C, n = 10) at baseline and at follow-up after 4.5 years. LVM = left ventricular mass, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress index, IC = index of contractility.

				ANOVA	
		Baseline	4.5 years	Group effect	ANCOVA
LVM (g)	S	186.7 ± 19.4	223.2 ± 38.0		
	C	141.1 ± 27.1	166.4 ± 27.4	$p < 0.001$	$p < 0.05$
LVM/BSA	S	98.0 ± 8.5	115.1 ± 15.2		
	C	77.7 ± 14.4	86.4 ± 12.6	$p < 0.001$	n.s.
EDV (ml)	S	160.3 ± 21.9	169.8 ± 29.3		
	C	138.8 ± 23.9	155.3 ± 34.9	n.s.	n.s.
ESV (ml)	S	79.0 ± 7.8	71.6 ± 8.6		
	C	61.7 ± 11.6	62.0 ± 14.5	$p < 0.01$	n.s.
SV (ml)	S	82.7 ± 14.3	97.3 ± 23.5		
	C	77.1 ± 22.5	93.2 ± 22.9	n.s.	n.s.
FS (%)	S	26.4 ± 3.3	30.5 ± 3.9		
	C	29.1 ± 5.7	32.4 ± 3.6	n.s.	n.s.
HI	S	0.30 ± 0.02	0.33 ± 0.04		
	C	0.28 ± 0.02	0.29 ± 0.05	$p < 0.05$	n.s.
WS	S	35.9 ± 3.9	42.2 ± 5.5		
	C	35.0 ± 5.7	39.1 ± 8.9	n.s.	n.s.
IC	S	1.50 ± 0.22	1.86 ± 0.20		
	C	2.04 ± 0.38	2.26 ± 0.65	$p < 0.01$	n.s.

At the baseline the skiers showed a correlation only between SV and $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) ($r = .71$, $p < 0.01$). At the follow-up after 4.5 years all the $\dot{V}O_{2max}$ variables correlated with the echocardiographic variables (Table 38, Figure 14). In the controls only the correlations presented in Table 39 were found at the end of the study.

TABLE 38 Correlation coefficients between echocardiographic variables and maximal oxygen uptake ($\dot{V}O_{2max}$) of skiers at follow-up after 4.5 years. EDD = end-diastolic diameter, LVM = left ventricular mass, EDV = end-diastolic volume, SV = stroke volume. N = 15, *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

	$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	($ml \cdot kg^{-1} \cdot min^{-1}$)	($ml \cdot kg^{-2/3} \cdot min^{-1}$)	($ml \cdot kg^{-1} \cdot min^{-1}_{(demand)}$)
EDD	.76 **			
EDD/height	.67 **			.57 *
LVM	.78 ***		.63 *	
LVM/weight		.58 *	.57 *	.58 *
EDV	.76 ***			
SV	.76 ***		.58 *	.53 *

TABLE 39 Correlation coefficients between echocardiographic variables and maximal oxygen uptake ($\dot{V}O_{2max}$) of the controls at follow-up after 4.5 years. ESD = end-systolic diameter, ESV = end-systolic volume, IC = index of contractility. N = 10, ** $p < 0.01$, * $p < 0.05$.

	$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)
ESD	.81 **	.71 *
ESV	.82 **	.73 *
IC	-.72 *	-.65 *

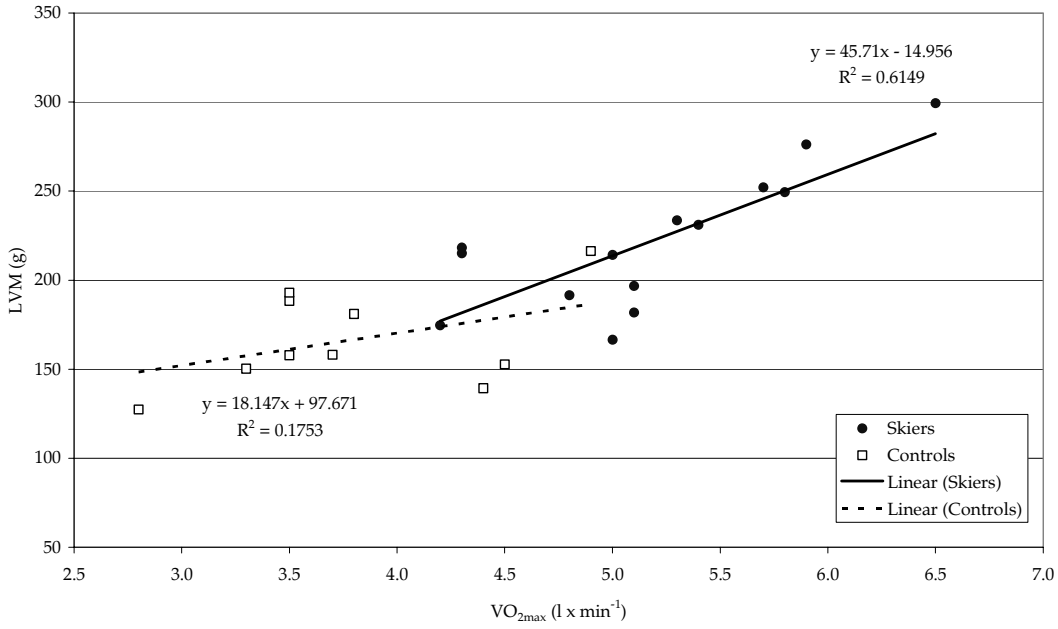

FIGURE 14 Maximal oxygen uptake ($\dot{V}O_{2max}$, $l \cdot min^{-1}$) in relation to left ventricular mass (LVM) at follow-up after 4.5 years in skiers and controls.

Examination of the individual values of the control subjects revealed that control subject no. 8 showed the biggest growth in EDD (0.82 cm). EDD in control subject no. 2 grew by 0.62 cm and in control subject no. 5 by 0.48 cm; the latter also had the biggest EDD value of 6.64 cm. $\dot{V}O_{2max}$ in control subjects 2 and 5 improved by 7 and 10 ml · kg^{-1} ·min^{-1} (Table 40).

TABLE 40 End-diastolic diameter (EDD), maximal oxygen uptake ($\dot{V}O_{2max}$), height and weight of controls 2, 5 and 8 at baseline and at follow-up after 4.5 years. The increase in EDD was largest in these controls during the study.

		EDD (cm)	$\dot{V}O_{2max}$ (l · min^{-1})	$\dot{V}O_{2max}$ (ml · kg^{-1} ·min^{-1})	Height (cm)	Weight (kg)
Control 2	Baseline	5.32	3.7	50	188.5	73.6
	4.5 years	5.94	4.5	57	189.2	79.3
Control 5	Baseline	6.16	3.7	56	180.0	66.3
	4.5 years	6.64	4.9	66	184.0	74.0
Control 8	Baseline	4.98	3.2	56	173.0	56.5
	4.5 years	5.80	3.3	53	177.0	62.1

5.3.2 Change in BSA over 6.5 years

Skiers were divided in two groups according to changes in BSA: small change (n = 10) 0.05 ± 0.03 m^2 (-0.02 - 0.08 m^2) and large change (n = 10) 0.15 ± 0.04 m^2 (0.12 - 0.22m^2).

There was an interaction between height ($p < 0.001$), weight ($p < 0.001$), BSA ($p < 0.001$), EDD ($p < 0.05$), LVM ($p < 0.05$) and EDV ($p < 0.05$). The difference remained after analysis of covariance in height ($p < 0.001$), weight ($p < 0.001$), BSA ($p < 0.001$) and EDV ($p < 0.05$) (Tables 41 and 42). On the individual level no differences between groups were found in EDD and BSA. Figure 15 shows the EDD results of the skiers at the baseline and the follow-up after 6.5 years.

There was an interaction in $\dot{V}O_{2max}$: l · min^{-1}, $p < 0.01$; ml · kg$^{-2/3}$ · min^{-1}, $p < 0.05$, and analysis of covariance showed a between-groups difference at the follow-up after 6.5 years ($p < 0.01$, $p < 0.05$). The change was greatest in the variable of $\dot{V}O_{2max}$ in which body size was eliminated, which increased in the group LargeBSAChange by 26.3 ml · kg$^{-2/3}$ · min^{-1} and in SmallBSAChange only by 2.7 ml · kg$^{-2/3}$ · min^{-1} (Table 43).

TABLE 41 Height, weight, BSA and echocardiographic variables of skiers with small (S, n = 10) and large (L, n = 10) change in BSA at baseline and at follow-up after 6.5 years. BSA = body surface area, EDD = end-diastolic diameter, EDD/BSA (EDD/body surface area), ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension.

	Group	Baseline	6.5 years	ANOVA, interaction
Height (cm)	S	179.2 ± 5.4	180.3 ± 5.4	
	L	178.6 ± 5.2	182.3 ± 5.2	$p < 0.001$
Weight (kg)	S	67.1 ± 4.2	70.5 ± 3.7	
	L	65.6 ± 7.1	76.6 ± 7.3	$p < 0.001$
BSA (m^2)	S	1.85 ± 0.09	1.90 ± 0.08	
	L	1.82 ± 0.12	1.98 ± 0.12	$p < 0.001$
EDD (cm)	S	5.38 ± 0.42	5.65 ± 0.33	
	L	5.26 ± 0.22	5.89 ± 0.42	$p < 0.05$
EDD/BSA	S	2.92 ± 0.20	2.98 ± 0.15	
	L	2.89 ± 0.14	2.98 ± 0.13	n.s.
ESD (cm)	S	3.90 ± 0.41	3.82 ± 0.35	
	L	3.77 ± 0.36	3.99 ± 0.46	n.s.
SWTd (cm)	S	0.84 ± 0.07	0.94 ± 0.14	
	L	0.84 ± 0.03	0.98 ± 0.09	n.s.
PWTd (cm)	S	0.85 ± 0.04	0.93 ± 0.09	
	L	0.81 ± 0.04	0.92 ± 0.09	n.s.
SD (cm)	S	1.39 ± 0.06	1.49 ± 0.12	
	L	1.40 ± 0.12	1.49 ± 0.12	n.s.

TABLE 42 Echocardiographic variables of skiers with small (S, n = 10) and large (L, n = 10) change in BSA at baseline and at follow-up after 6.5 years. LVM = left ventricular mass, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress index, IC = index of contractility.

	Group	Baseline	6.5 years	ANOVA, interaction
LVM	S	164.5 ± 27.3	204.0 ± 32.1	
	L	153.3 ± 13.7	224.2 ± 38.6	$p < 0.05$
LVM/weight	S	2.40 ± 0.15	2.90 ± 0.46	
	L	2.47 ± 0.26	2.93 ± 0.38	n.s.
LVM/BSA	S	87.2 ± 3.9	107.7 ± 16.3	
	L	88.4 ± 5.6	113.2 ± 16.1	n.s.
EDV	S	141.2 ± 25.4	157.4 ± 21.8	
	L	133.5 ± 12.9	173.9 ± 28.8	$p < 0.05$
ESV	S	66.8 ± 16.8	63.5 ± 13.6	
	L	61.6 ± 13.9	70.7 ± 18.5	n.s.
SV	S	74.5 ± 10.8	93.8 ± 18.4	
	L	71.9 ± 13.3	103.2 ± 19.5	n.s.
FS	S	27.7 ± 3.0	32.3 ± 5.3	
	L	28.3 ± 5.6	32.4 ± 5.2	n.s.
HI	S	0.32 ± 0.03	0.33 ± 0.04	
	L	0.32 ± 0.01	0.33 ± 0.04	n.s.
WS	S	39.3 ± 5.6	43.6 ± 5.4	
	L	36.2 ± 4.5	41.8 ± 7.1	n.s.
IC	S	1.98 ± 0.59	2.18 ± 0.51	
	L	2.02 ± 0.59	2.03 ± 0.67	n.s.

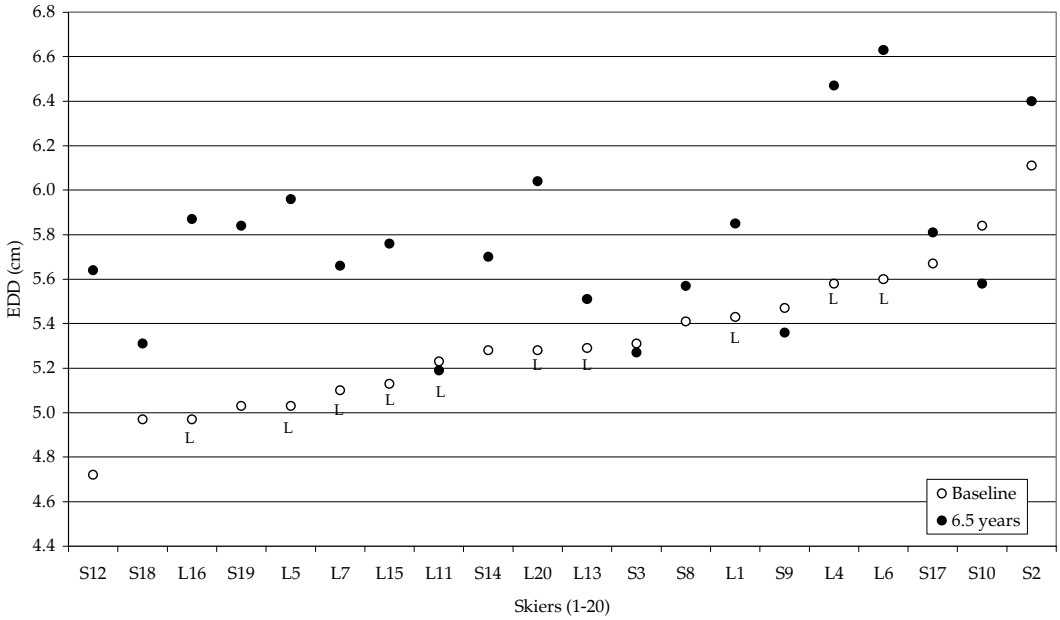

FIGURE 15 Individual values of end-diastolic diameter (EDD) in skiers with small (S, n = 10) and large (L, n = 10) change in BSA at baseline and at follow-up after 6.5 years.

TABLE 43 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) of skiers with small (S, n = 10) and large (L, n = 10) BSAChange at baseline and at follow-up after 6.5 years.

	Group	Baseline	6.5 years	ANOVA
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	S	4.5 ± 0.3	4.7 ± 0.5	
	L	4.6 ± 0.5	5.5 ± 0.5	$p < 0.01$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	S	67.3 ± 2.6	66.4 ± 7.5	
	L	69.0 ± 3.2	72.0 ± 3.0	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	S	273.0 ± 11.4	275.7 ± 29.2	
	L	278.2 ± 13.5	304.5 ± 14.1	$p < 0.05$
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}_{(demand)}$)	S	58.4 ± 3.4	67.4 ± 7.5	
	L	61.2 ± 3.4	73.1 ± 4.5	n.s.
HR_{max} ($beats \cdot min^{-1}$)	S	190.2 ± 10.6	189.7 ± 8.0	
	L	197.5 ± 5.1	191.7 ± 5.5	$p < 0.05$
BLa_{max} ($mmol \cdot l^{-1}$)	S	6.37 ± 1.70	11.52 ± 1.81	
	L	7.14 ± 1.39	12.17 ± 1.64	n.s.

5.3.3 EDD/BSA-SL

Skiers were divided in two groups according to their baseline EDD/BSA measurement values. Mean EDD/BSA of the small-hearted (S, n = 10) skiers was 2.78 ± 0.11 (2.49 - 2.87) and of the large-hearted (L, n = 10) skiers 3.03 ± 0.12 (2.91 - 3.26).

There were no changes in height, weight or BSA. An interaction was found between EDD/BSA ($p < 0.01$) and EDD ($p < 0.001$), but analysis of covariance

revealed only EDD to have statistical significance ($p < 0.05$). The EDD of the small-hearted skiers increased more (0.71 cm, $p < 0.001$) than that of the large-hearted ones (0.19, n.s.) (Table 44).

Interaction ($p < 0.01$) was also seen between EDV and SV, the differences remaining statistically significant even after analysis of covariance: EDV $p < 0.05$, SV $p < 0.01$ (Table 45).

When examining the individual EDD/BSA values of the skiers, it became evident that the values of six skiers from the group LARGE decreased between the baseline and the follow-up after 6.5 years. Only one skier from the group SMALL saw diminished value (Figure 16). When the changes in EDD between the groups were studied, it was found that in the group SMALL EDD increased in every subject and that the changes were larger compared to those in the group LARGE, where, in addition diminished EDD was found in four subjects (Figure 17). No notable changes took place in $\dot{V}O_{2max}$ during the 6.5 years (Table 46).

TABLE 44 Height, weight, BSA and echocardiographic variables of skiers with small (S, n = 10) and large (L, n = 10) EDD/BSA at baseline and at follow-up after 6.5 years. BSA = body surface area, EDD = end-diastolic diameter, ESD = end-systolic diameter, SWTd = septal wall thickness in diastole, PWTd = posterior wall thickness in diastole, SD = stroke dimension.

	Group	Baseline	6.5 years	ANOVA, interaction
Height (cm)	S	179.4 ± 3.9	182.2 ± 4.7	
	L	178.3 ± 6.4	180.3 ± 5.8	n.s.
Weight (kg)	S	67.5 ± 5.4	75.9 ± 7.7	
	L	65.2 ± 6.1	71.3 ± 4.2	n.s.
BSA (m^2)	S	1.85 ± 0.09	1.97 ± 0.12	
	L	1.82 ± 0.11	1.90 ± 0.09	n.s.
EDD/BSA	S	2.78 ± 0.11	2.98 ± 0.15	
	L	3.03 ± 0.12	2.99 ± 0.13	$p < 0.01$
EDD (cm)	S	5.14 ± 0.26	5.85 ± 0.34	
	L	5.50 ± 0.30	5.69 ± 0.44	$p < 0.001$
ESD (cm)	S	3.71 ± 0.32	3.89 ± 0.37	
	L	3.96 ± 0.41	3.92 ± 0.46	n.s.
SWTd (cm)	S	0.84 ± 0.05	0.97 ± 0.10	
	L	0.84 ± 0.05	0.95 ± 0.13	n.s.
PWTd (cm)	S	0.81 ± 0.05	0.89 ± 0.06	
	L	0.84 ± 0.04	0.95 ± 0.10	n.s.
SD (cm)	S	1.39 ± 0.09	1.51 ± 0.10	
	L	1.40 ± 0.09	1.46 ± 0.13	n.s.

TABLE 45 Echocardiographic variables of skiers with small (S, n = 10) and large (L, n = 10) EDD/BSA at baseline and at follow-up after 6.5 years. LVM = left ventricular mass, EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, FS = fractional shortening, HI = hypertrophy index, WS = wall stress index, IC = index of contractility.

	Group	Baseline	6.5 years	ANOVA, interaction
LVM	S	147.5 ± 15.1	216.9 ± 39.1	
	L	170.3 ± 21.9	211.3 ± 34.7	n.s.
LVM/weight	S	2.39 ± 0.18	2.87 ± 0.45	
	L	2.49 ± 0.24	2.96 ± 0.39	n.s.
LVM/BSA	S	86.9 ± 3.9	110.2 ± 17.7	
	L	88.7 ± 5.5	110.8 ± 15.1	n.s.
EDV	S	126.5 ± 14.7	170.6 ± 23.4	
	L	148.2 ± 19.1	160.7 ± 29.2	$p < 0.01$
ESV	S	59.1 ± 12.7	66.4 ± 14.4	
	§	69.3 ± 16.5	67.9 ± 18.7	n.s.
SV	S	67.4 ± 12.6	104.2 ± 17.9	
	L	79.0 ± 8.0	92.8 ± 19.5	$p < 0.01$
FS	S	27.8 ± 4.8	33.5 ± 4.6	
	L	28.2 ± 4.4	31.2 ± 5.6	n.s.
HI	S	0.32 ± 0.02	0.32 ± 0.02	
	L	0.31 ± 0.02	0.34 ± 0.05	n.s.
WS	S	38.7 ± 5.8	41.4 ± 3.4	
	L	36.7 ± 4.5	44.0 ± 8.1	n.s.
IC	S	2.16 ± 0.50	2.13 ± 0.46	
	L	1.85 ± 0.63	2.08 ± 0.71	n.s.

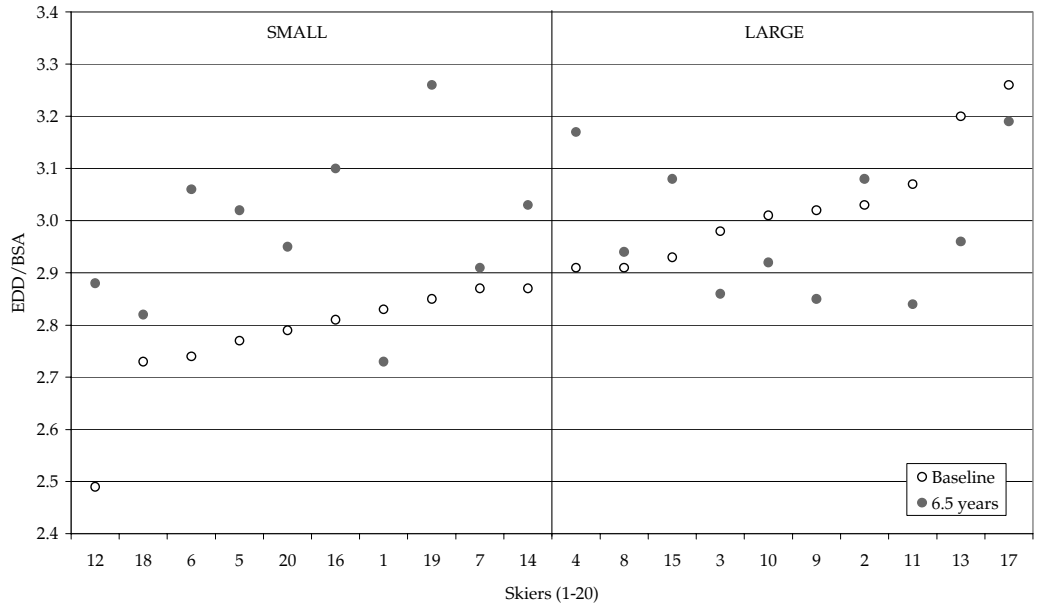

FIGURE 16 Individual values of EDD/BSA of skiers with small (n = 10) and large (n = 10) EDD/BSA at baseline and at follow-up after 6.5 years.

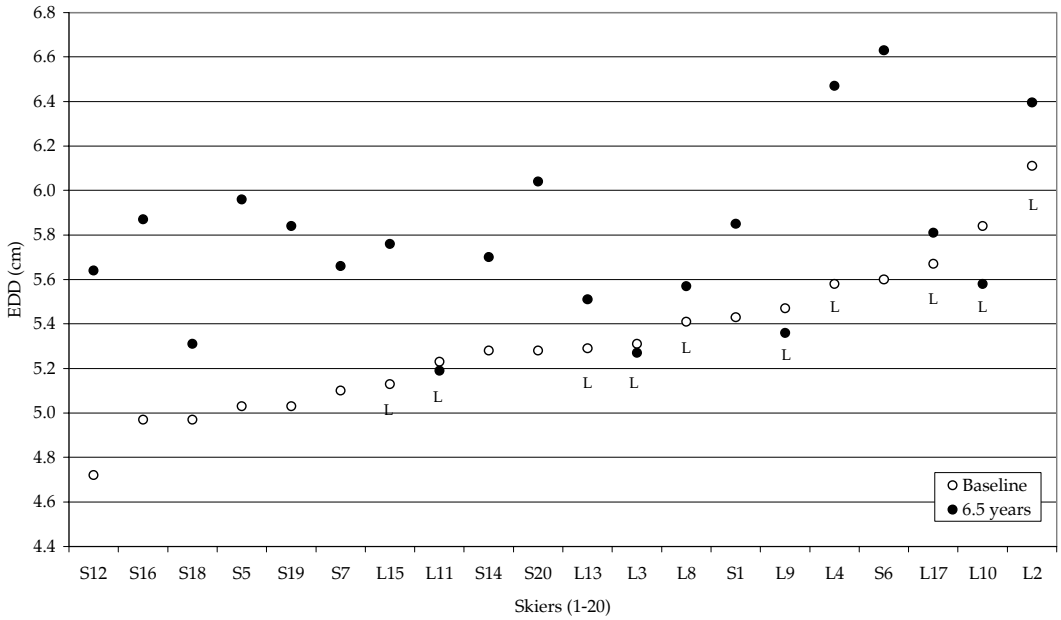

FIGURE 17 Individual values of end-diastolic diameter (EDD) of skiers with small (S, n=10) and large (L, n=10) EDD/BSA at baseline and at follow-up after 6.5 years.

TABLE 46 Maximal oxygen uptake ($\dot{V}O_{2max}$), maximal heart rate (HR_{max}) and maximal blood lactate concentration (BLa_{max}) of skiers with small (S, n = 10) and large (L, n = 10) EDD/BSA at baseline and at follow-up after 6.5 years.

	Group	Baseline	6.5 years	ANOVA
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	S	4.6 ± 0.4	5.3 ± 0.6	
	L	4.5 ± 0.4	4.9 ± 0.6	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	S	67.8 ± 3.2	69.6 ± 7.0	
	L	68.6 ± 2.8	68.9 ± 5.7	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$)	S	276.4 ± 14.6	294.1 ± 28.8	
	L	274.9 ± 10.7	286.0 ± 25.6	n.s.
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}_{demand}$)	S	60.4 ± 4.1	70.6 ± 7.2	
	L	59.3 ± 3.2	70.2 ± 6.3	n.s.
HR_{max} ($beats \cdot min^{-1}$)	S	195.0 ± 8.9	193.0 ± 5.9	
	L	193.0 ± 9.0	188.2 ± 6.9	n.s.
BLa_{max} ($mmol \cdot l^{-1}$)	S	6.15 ± 1.63	11.95 ± 1.18	
	L	7.47 ± 1.19	11.77 ± 2.23	n.s.

5.4 Other Results

The results of echocardiographic (EDD, ESD, SWTd, PWTd, SD, EDV, ESV, SV, LVM, FS, HI, WS and IC) and $\dot{V}O_{2max}$ ($l \cdot min^{-1}$, $ml \cdot kg^{-1} \cdot min^{-1}$, $ml \cdot kg^{-2/3} \cdot min^{-1}$) variables are presented in appendices 4 - 19.

6 DISCUSSION

The aim of this study was to determine the impact of long-term (6.5 years) endurance training on the structural and functional changes in the hearts of young endurance athletes (aged 15.9) at rest. Maximal cardiovascular capacity ($\dot{V}O_{2max}$) was measured with a ski-walking test on a treadmill. The role of the size of the heart on the training-induced changes was studied by dividing the subjects into those with small hearts and those with large hearts on the basis of the baseline measurements of the EDD of the left ventricle of the heart. Also of interest was age at which the structural and functional changes in the heart took place and whether the growth of the size of the heart was linear or whether it was punctuated by phases of evening out. For this reason an intermediate measurement after three and a half years and annual measurements were performed. The impact of short-term training (6 - 7 months) on the changes in echocardiographic and $\dot{V}O_{2max}$ was studied during three different training periods. The impact of the size and growth of the subjects on the structure and function of the heart was studied on the basis of the changes in BSA, i.e. how the size of the heart changed in subjects who developed early and in those who developed late. To determine the influence of biological growth and training the study included a control group of schoolboys of the same age, observed during the last four years of the study.

In this study the EDD, SWTd, PWTd and $\dot{V}O_{2max}$ of the cross-country skiers increased during the 6.5-year follow-up. At the follow-up after 6.5 years a correlation was found between EDD and absolute $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) and $\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$), which predicts the performance of cross-country skiers. In investigating the significance of the baseline values it emerged that the EDD of those with small hearts increased more during the 6.5 years than that of those with large hearts, although those with small hearts did not attain the same EDD of those with large hearts. There were no differences in the thickness of the walls between those with small and large hearts. There were two phases in the timing of the growth of the heart. EDD increased during the first three and a half years (age 16 - 19), when the walls remained unchanged, whereas during the last three years (age 19 - 22) EDD ceased to increase and the walls grew

thicker. $\dot{V}O_{2max}$ increased most during the first three and a half years, thereafter evening out. The annual echocardiographic and $\dot{V}O_{2max}$ changes were small, and there were no significant changes in variables between different training seasons. The impact of body size on EDD became evident when the subjects were divided into two groups on the basis of the changes in their BSA that occurred during 6.5 years. EDD increased more in the group where BSA had increased more. There were no differences in wall thickness between the groups. When the subjects were divided into those with small or large hearts on the basis of baseline EDD/BSA, the echocardiographic results changed a little. The skiers had larger EDD than their age-matched controls both at the baseline (17 years) and at the follow-up after 6.5 years (22 years) of the study. The EDD of the controls increased more during the four and a half years than that of the skiers, as the skiers had a large EDD already at the baseline, owing to several years of training. The walls of the left ventricle of the skiers were thicker than those of the control subjects, and they increased almost equally in both groups.

6.1 Changes during 6.5 years

Cross-country skiing as a sport is the most dynamic sport as far as the circulatory organs are concerned; it is more dynamic than e.g. rowing or cycling. Cross-country skiers attain the highest $\dot{V}O_{2max}$ per kilo among endurance athletes, because in skiing large muscle masses are in use in both the upper and lower body. Cross-country skiing cannot be considered merely an endurance sport demanding volume overload, but due to the many-sidedness of the sport also strength training, pressure overload, which contributes to thickening the walls of the left ventricle of the heart. Along with the increase in ski-skating and double-poling the strength training of the arms and body has received more emphasis in training, in order to exploit best the forces pushing the skier forwards while skiing. During the last 10 - 15 years not only has the significance of the strength of the arms and body increased but so also has that of the $\dot{V}O_{2max}$ of the upper body which at present is approximately 90 % of the $\dot{V}O_{2max}$ of the lower body in both men and women (Rusko 2003). The fastest skiers have had a longer cycle due to better strength in arms, legs and body, so that their kicking and poling phases were better than those in slower skiers (Bilodeau et al. 1996). The strength of the upper body has been one factor predicting competition speed, and there has been a strong correlation between them (r = . 89) (Gaskill et al. 1999). The strength of the upper body also proved significant in the study by Mahood et al. (2001), where the time of a one-kilometre double-poling test predicted best the ranking situation and the time over a single 10-kilometer ski-skating test. The efficiency of the upper body, measured with a double-poling ergometer, was important, but in the double-poling test taken on the treadmill, $\dot{V}O_{2max}$ was a better predictor of skiing performance among 20-year-old top skiers (Staib et al. 2000).

6.1.1 EDD

At the baseline of the present study the EDD of 15.9-year-old skiers was comparable to the values found in 16.6-year-old cross-country skiers (Pekkarinen 1986) and 16 - 22-year-old cross-country skiers (Bienmüller et al. 1982). The skiers had trained for 5 - 6 years before the baseline of the present study, and their training had consisted mostly of volume overload, which had led to the increase in EDD. As the training further increased and became more systematic and specific during the study, EDD increased significantly. During the 6.5 years EDD increased to a level similar to that of rowers of the same age (Wieling et al. 1981) and long-distance runners 2 - 4 years older (MacFarlane et al. 1991, Schmidt-Trucksäss et al. 2000), and larger than that of 22-year-old top cross-country skiers and middle distance runners or orienteers (Henriksen et al. 1996). In the studies by Pelliccia et al. (1991, 1999) the EDD of approximately 25-year-old cross-country skiers has 5.5 cm, and Stolt et al. (2000) obtained the same result with top endurance athletes of the same age and Zakynthinos et al. (2001) with top water polo players. In the study by Pelliccia et al. (1999) 14 % of the 1309 male and female subjects representing 38 different sports had an EDD of ≥ 6 cm. At the follow-up after 6.5 years in the present study, 4 subjects had an EDD of > 6 cm (20 %). According to Urhausen & Kindermann (1992) the EDD of adult endurance athletes of normal size is usually less than 6 cm.

The meta-analytical study by Pluim et al. (1999) found the following variation in EDD: endurance athletes 5.37 cm, endurance - strength athletes 5.62 cm and strength athletes 4.96 cm. The training of most sports affects both the diameter of the left ventricle and the thickening of the walls. Cross-country skiing came second on a ranking list of 27 different sports after endurance cycling in estimates of the influence of the sport on EDD. At the top of the ranking list of sports with an impact on both EDD and the walls were endurance cycling, swimming, rowing and canoeing as sports with an impact on both EDD and the walls (Spirito et al. 1994). The main factors deciding the size of the left ventricle were BSA and certain endurance sports (cycling, cross-country skiing and canoeing), after which came HR, gender and age (Pelliccia et al. 1999). EDD/BSA did not change in the present study during the 6.5 years, as both EDD and BSA grew. The final EDD/BSA values of the skiers in the present study were similar to those recorded for endurance runners, cyclists, canoeists and football players (Spataro et al. 1985).

The hypothesis that EDD would increase was supported. The 0.5-cm increase (7.8 %) in EDD of the young cross-country skiers as the result of the 6.5-year intensive volume overload training can be regarded as small, although the results cannot be compared to those of other studies, as no follow-up studies of the same length exist. The results were influenced by factors other than purely the many years of training done prior to the study. One factor was the military service done during the 6.5-year research period, which caused breaks in training. Some subjects also suffered injuries during the study, which also interrupted and/or caused changes in training. Earlier study results on the

impact of detraining on echocardiographic variables have been conflicting. EDD began to decrease after a detraining period of no more than four days and significantly after three weeks (Ehsani et al. 1978). Nevertheless, a 10-day break in training did not influence the echocardiographic results of endurance athletes (Cullinane et al. 1986). Neither did EDD decrease in 24-year-old top rowers and canoeists, when they considerably decreased their training for an average of 13 weeks after the Olympic Games (Maron et al. 1993). However, the above-mentioned athletes' LVM decreased. A 4 - 6-week non-training period decreased the EDD, walls and LVM of professional football players (Galanti et al. 1987). The EDD of endurance athletes and cross-country skiers decreased by one centimeter six months after detraining (Schumacher & Howald 1984), and the EDD of top endurance athletes decreased after detraining (1 - 13 years) (Pelliccia et al. 2002). The small growth of EDD in the present study could also be explained by the fact that the mean baseline EDD of the 15 to 17-year-old subjects, i.e. 5.32 cm, was already the same as that of grown-up athletes, as the value of 5.4 cm found in the meta-analysis of Maron (1986) and Perrault & Turcotte (1994) has shown.

6.1.2 SWTd and PWTd

The walls of the left ventricle increased considerably during the 6.5-year study, and thus the results were in accordance with the hypothesis. The SWTd and PWTd of the left ventricle were of the same size at the baseline, but SWTd showed a tendency to thicken slightly more (12.5 %) than did PWTd (9.8 %). It can be assumed that, as the wall of both ventricles, SWT increases more than PWT. The increase in SWTd has generally been larger than that of PWTd (Karjalainen et al. 1997, MacFarlane et al. 1991, Nishimura et al. 1980, Shapiro 1987, Spataro et al. 1985, Stolt et al. 2000, Zakynthinos et al. 2001) or the walls have been of the same thickness (Hendriksen et al. 1996, Whyte et al. 1999). There were no differences in the thickness of SWTd and PWTd in top endurance athletes, whereas in athletes from different sports PWTd was thicker than SWTd (George et al. 1999, Osborne et al. 1992, Zemva & Rogel 2001). Blood pressure also increases during endurance training, though less than during strength training. Endurance training is not only volume overload training, but it leads to an increase in both the diameter of the left ventricle and wall thickness (Pluim et al. 1999). The effect of blood pressure on wall thickness was very evident in blood pressure patients, whose wall thickness was as large as that of weight lifters and larger than that of controls (Abinader et al. 1996).

It was assumed that the walls of the skiers in the present study would be thicker, as their training includes power training for legs, arms and upper body. The highest arterial pressure and therefore the greatest stimulus to ventricular hypertrophy are achieved when a high intensity contraction involving a large number of muscles. The Valsalva-type manoeuvre during the double-pole and push phase of the skating style may also increase arterial pressure and further affect the hypertrophication of the walls. The Valsalva-manoeuvre, which is performed at each stroke in rowing, generated large pressure fluctuations

which, added to the rowers' normal pulse pressure, possible caused the hypertrophication of the hearts of these rowers (Clifford et al. 1994). However ski training cannot be directly compared with e.g. rowing training, even though ski training consists of considerably more volume overload than pressure overload training.

The final SWTd of the cross-country skiers in the present study was equal to that of the 17-year-old cross-country skiers in Pekkarinen's (1986) study, although PWTd was smaller (0.92 cm vs. 1.18 cm). The walls in the present study were almost as thick as those of the volume overload training long-distance runners in Fagard's (1996) study. In both walls and EDD the present skiers were similar to the Italian skiers, as at the follow-up after 6.5 years the wall thickness of the 22-year-old skiers was only slightly below the average of the 2-3 year older Italian skiers (Pelliccia et al. 1991, 1999). The wall thickness found in the present study and in the Italian cross-country skiers (Pelliccia et al. 1991, Spirito et al. 1994) seems to be thinner than the wall thickness of endurance athletes in other countries. There can hardly be a basic difference in training. Is it possible that genes or racial characteristics are the cause of these differences? The largest wall thickness (SWTd even 1.9 cm) has been found, according to Pelliccia et al. (1991, 1999), Reguero et al. (1995) and Zakynthinos et al. (2001) in rowers, water polo players, canoeists, cyclists and weightlifters, though 25-year-old top endurance athletes (long-distance runners, orienteers, ski-orienteers) also had thick walls (Stolt et al. 2000). A four-week progressively increasing training regimen did not affect the wall thickness of endurance runners (Lehmann et al. 1990). The increased training contributed to thickening the walls in 20-year-old rowers over seven months, but not in 23-year-old more successful rowers, who increased training (Wieling et al. 1981). The meta-analytical study by Pluim et al. (1999) indicated that endurance athletes had significantly thinner SWTd than strength athletes, although there was no difference between endurance athletes and endurance-strength athletes in either SWTd or PWTd. The PWTd (1.02 cm) of endurance athletes in the meta-analysis carried by Perrault & Turcotte (1994) was larger than that of the cross-country skiers of the present study, whereas the SWTd and PWTd of top athletes representing 28 different sports (Pelliccia et al. 1999) were of the same size as found in the present study. Cross-country skiing was ranked 12 out of 28 different sports, when the impact of different sports on the wall thickness of the left ventricle was measured (Spirito et al. 1994). When endurance training was intensified, the wall thickness of the left ventricle also increased (Adams et al. 1985, Ehsani et al. 1978, Keul et al. 1982). Thirteen percent of top endurance athletes had a wall thickness of over 1.3 cm, but none over 1.5 cm in the study by Henriksen et al. (1997), where the subjects were orienteers, cross-country skiers and middle distance runners.

The results on the connection between EDD and the increase in wall thickness have been conflicting. In the present study there was no correlation between EDD and the walls either at the baseline or at the follow-up after 6.5 years. According to Pelliccia et al. (1999) and Spirito et al (1994), the thickening

of the walls was also connected with an enlarged left ventricle, whereas in the study by Whyte et al. (1999) there was no change in EDD, even where the walls thickened but EDD was increased more often than wall thickness in the study by Douglas et al. (1997). The walls of the cross-country skiers of the present study were thinner than those of endurance runners (MacFarlane et al. 1991) and rowers (Wieling et al. 1981), whose EDD was similar found for skiers in the present study, or those of the endurance runners, whose EDD was larger (Schmidt-Trucksäss et al. 2000) than that of the skiers in the present study.

6.1.3 $\dot{V}O_{2max}$

The impact of the biological growth of the subjects could be seen in $\dot{V}O_{2max}$ values. Theoretically the performance of cross-country skiers does not depend on body mass, as light skiers benefit from steep gradients, whereas heavier skiers benefit from other parts of the terrain (Bergh 1987). In the present study absolute $\dot{V}O_{2max}$ ($l \cdot min^{-1}$) increased significantly during the 6.5 years, but $\dot{V}O_{2max}$ in adjusted for weight ($ml \cdot kg^{-1} \cdot min^{-1}$) did not increase, whereas the results were influenced by changes of body size during the study. However, the $\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$) values, used especially with cross-country skiers as this variable predicts capacity well (Bergh 1987), improved during the follow-up after 6.5 years. The $\dot{V}O_{2max}$ of Norwegian top cross-country skiers increased up to the age of 17, thereafter leveling out at 82 $ml \cdot kg^{-1} \cdot min^{-1}$, while absolute $\dot{V}O_{2max}$ increased up to age 20 (5.9 $l \cdot min^{-1}$) as did $\dot{V}O_{2max}$ expressed as $ml \cdot kg^{-2/3} \cdot min^{-1}$ (338 $ml \cdot kg^{-2/3} \cdot min^{-1}$) (Ingjer 1992). What is notable is the large difference between $\dot{V}O_{2max}$ results in Ingjer's (1992) study compared to the present study and the fact that the Norwegian values increased more than those of the present subjects. It is likely that the extremely high $\dot{V}O_{2max}$ values in Ingjer's (1992) study are due to favorable genes and hard training: the subjects studied were top international skiers. Only some of the skiers of the present study remained at national level throughout the follow-up. In Rusko's (1987) study $\dot{V}O_{2max}$ increased from age 15 to age 20 by 1 - 3 $ml \cdot kg^{-1} \cdot min^{-1}$ over a year, leveling off thereafter, whereas the $\dot{V}O_{2max}$ of international level skiers continued to improve with increased volume of training and volume of intensive training. Heart volume increased most from age 16 to 18, and in international level skiers increased even after age 20. An increase in highly intensive training considerably improved the $\dot{V}O_{2max}$ of cross-country skiers during one year and also improved the performance in competition of skiers who got no help from an increase in low-intensity training (Gaskill et al. 1999). Also in Bergh's study (1987) the $\dot{V}O_{2max}$ of international top skiers was high, 83.8 $ml \cdot kg^{-1} \cdot min^{-1}$, and in skiers with less success reached 79.6 $ml \cdot kg^{-1} \cdot min^{-1}$. The $\dot{V}O_{2max}$ of the 15-year-old cross-country skiers in the studies by Pekkarinen (1986) and Murase et al. (1981) was smaller than that of the skiers in the present study, but the $\dot{V}O_{2max}$ of 18 - 19-year-old skiers was better (Larsson et al. 2002, Murase et al. 1981). Also, 24 to 25-year-old endurance runners have

attained better $\dot{V}O_{2max}$ values (Jensen-Urstad et al. 1998, MacFarlane et al. 1991) than the present skiers. Reaching a high $\dot{V}O_{2max}$ (approximately 350 ml · $kg^{-2/3}$ · min^{-1}) at junior age was connected with good success in competitions and remaining in the world elite at a later age (Ingjer 1992). According to Larsson et al. (2002) a high OBLA (onset of blood lactate accumulation, 4 mmol · l^{-1}) in treadmill test correlated with a good ranking in 18-year-old young top skiers.

The increase in maximal performance (ml · kg^{-1} · min^{-1} demand) in the present study was significant during the 6.5 years. The importance of muscular strength is also emphasized in endurance sport performance (Rusko & Nummela 1996). During uphill skiing the need for energy exceeds the maximal aerobic capacity and skiers have to be able to keep up a relatively fast speed despite high lactate content throughout the competition (Norman et al. 1989). Simultaneous explosive-strength and endurance training (Paavolainen et al. 1999) and endurance and strength training (Millet et al. 2002) increased endurance performance in well-trained endurance athletes and moderately trained cross-country skiers (Hoff et al. 1999) without any changes in $\dot{V}O_{2max}$. The performance was thought to be caused by improved neuromuscular power producing qualities transferred into improved running economy and muscle power (Paavolainen et al. 1999). In the present study the maximal lactate values increased indicating improved lactate tolerance.

Military service during the study and the injuries some skiers suffered also had an impact on $\dot{V}O_{2max}$ results. The higher the $\dot{V}O_{2max}$ the more it decreased because of the lack of training (Coyle et al. 1984) or the break in training had no impact on $\dot{V}O_{2max}$ (Cullinane et al. 1986). After only three days' bed rest $\dot{V}O_{2max}$ decreased in cyclists by 17 % (Smorawinski et al. 2001), and in another study (Convertino 1997) the same result was found after 2 - 3 weeks' bed rest. In most studies a training break of less than four weeks has caused a 4 to 14 % decrease in $\dot{V}O_{2max}$ (Coyle et al. 1984, Houmard et al. 1992, Martin et al. 1986, Moore et al. 1987).

The hypothesis that a correlation would exists between EDD and $\dot{V}O_{2max}$ held only in the case of the follow-up after 6.5 years. The significant correlation between EDD and $\dot{V}O_{2max}$ (l · min^{-1}) at the end of the study proved that the subjects who were larger had the largest EDD, which indicated a connection with absolute $\dot{V}O_{2max}$. Training was considered to have an impact on the correlation between EDD and $\dot{V}O_{2max}$ (ml · $kg^{-2/3}$ · min^{-1}), important for cross-country skiers, at the end of the study. The 17-year-old skiers in Pekkarinen's (1986) study had a better correlation between LVM and $\dot{V}O_{2max}$ (l · min^{-1}, ml · kg^{-1} · min^{-1}) than younger (12 - 14 years) skiers, and in the studies by Bekaert et al. (1981), Dickhuth et al. (1987), Milliken et al. (1988), Osborne et al. (1992) and Pannier et al. (1982) EDD and LVM correlated with $\dot{V}O_{2max}$. $\dot{V}O_{2max}$ has also been found to increase without simultaneous changes in EDD (Perrault et al. 1982, Ricci et al. 1982, Thompson et al. 1981, Wieling et al. 1981).

6.1.4 The Significance of the Baseline Measurements

The significance of the baseline measurement on the growth of the size of the heart was clarified by dividing the skiers into those with small hearts (5.07 cm: 4.72 - 5.28 cm) and those with large hearts (5.57 cm: 5.29 - 6.11 cm) on the basis of their baseline EDD values. This particular division and with a follow-up over several years has not previously been done. The EDD of those with small hearts increased more (0.63 cm) than that of those with large hearts (0.28 cm) during the 6.5 years, but those with small hearts did not quite attain the EDD values of those with large hearts by the end of the study. In the earlier 3-year follow-up study, however, EDD increased equally in the two groups of small hearts and large hearts (Tummavuori 1997). Because there were no significant differences in BSA between the groups, EDD/BSA increased in the group with small hearts, whereas in the group with large hearts it remained unchanged. When examining the data after three and a half years of those for whom results were also available for the intermediate measurements, it was found that the EDD of those with large hearts had attained the 6.5-year follow-up value already during the first three and a half years (16 - 19 years old), whereas the growth in those with small hearts continued throughout the whole study period (16 - 22 years old). It can be assumed that those with large hearts had received better training and shown a better training response before the beginning of the study than those with small hearts, and this had led to a larger EDD already at the baseline. Could the EDD of those with large hearts also have been increased during the last three years by different training? The volume of intensive training should perhaps have been increased, as Rusko (1992) has reported. Alternatively, it is possible that a ceiling had been reached in EDD, just as in the case of endurance athletes, who continued training for years, and their controls in $\dot{V}O_{2max}$ (Ekblom 1969, Martin et al. 1986). Would the EDD of those with small hearts in the present subjects have continued to increase with training? The training response was apparently better in the small heart group during the 6.5-year period as there were no differences in the volume of training between the groups. Those with small hearts were expected to be biologically slower developers than those with large hearts. This assumption was proved wrong, as in both groups the changes in height, weight and BSA were similar.

The hypothesis that in both groups EDD would show equal increase was not supported, as in those with small hearts EDD increased more than in those with large hearts. The larger increase of EDD in the small-hearted group may partly explain the larger increase in $\dot{V}O_{2max}$ in this group. $\dot{V}O_{2max}$ in the small-hearted group improved by 19.7 ml $\cdot$ kg$^{-2/3}$ $\cdot$ min^{-1}, compared to only 9.1 ml $\cdot$ kg$^{-2/3}$ $\cdot$ min^{-1} in the large-hearted group. $\dot{V}O_{2max}$ adjusted for weight also increased in the former group, whereas the value for those with large hearts remained unchanged. The only correlation connected with biological growth was that between growth in height and EDD: in the group of those with large hearts this was positive both at the baseline and at the follow-up after 6.5 years; in the group with small hearts the correlation was negative at the baseline and

positive at the follow-up after 6.5 years. The explanatory percentages were, however, low, and the small number of subjects also has to be taken into account when interpreting the results.

There were no differences in walls between those with small and those with large hearts. The trend seemed to be that in those with small hearts SWTd increased more, whereas in those with large hearts PWTd increased more. The walls did not grow during the first three years, as happened in a previous three-year follow-up study (Tummavuori 1997) of skiers of the same age, but only during the last three years.

6.2 Timing of Changes

6.2.1 Changes at the Follow-Ups after 3.5 and 6.5 years

To determine the timing of the changes in EDD an intermediate measurement, three and a half years after the baseline was performed. Two significant findings emerged. The first was the fact that EDD was largest during the first three and a half years, when it increased by 5.2 % compared to the only 1 % increase over the last three years. During this particular period, from age 16 to 19, the largest increase in height, weight and $\dot{V}O_{2max}$, except for $\dot{V}O_{2max}$ adjusted to body weight also occurred. The annual measurements of a three-year follow-up study of cross-country skiers of the same age showed that EDD increased significantly during the first two years, remaining unchanged during the last year (Tummavuori 1997). In Rusko's (1992) study the relative heart volume of junior cross-country skiers continued to increase up to the age of 20 before levelling off, while the relative heart volume of world class skiers increased even after that, when the volume of training and intensive training increased. In the present study after three and a half years, when the skiers were 19 years old their EDD (5.68 cm) was larger than that of well-trained athletes of the same age (Spirito et al. 1988) and similar to that of approximately 20-year-old endurance athletes (Agati et al. 1985, Cohen & Segal 1985, Morganroth et al. 1975).

The other significant finding was, that no changes occurred in the walls during the first three and a half years, whereas during the last three years, when the skiers were 19 - 22 years old the walls thickened, SWTd by 12.4 % and PWTd by 7.5 %. During the last three years the skiers did their 8 - 11 months' military service, which may have had an impact on the growth of the walls, as the draftees had to carry loads up to 30 kg while marching. Even the lighter equipment included a nearly 10-kg fatigue belt and a 3.5 kg assault rifle.

Thus the hypothesis was supported for EDD, but not the walls. The changes in $\dot{V}O_{2max}$ occurred in accordance with the hypothesis simultaneously with an increase in EDD, except for $\dot{V}O_{2max}$ adjusted for body weight, the values of which did not show any marked increase during the 6.5-year period. On the

basis of the $\dot{V}O_{2max}$ ($l \cdot min^{-1}$, $ml \cdot kg^{-2/3} \cdot min^{-1}$) results it could be assumed that functional cardiovascular changes had taken place.

6.2.2 Annual Changes

In order to determine the timing of the EDD changes more accurate the values of eight skiers were also measured annually. EDD increased during the first three and a half years up to the age of 19, and levelled off thereafter. The statistically greatest change took place between the baseline and the follow-up after 2.5 years and between the baseline and the follow-up after 4.5 years. Although the number of subjects was small, it seems clear that in young cross-country skiers EDD increased up to age 18 - 19, after which it remained unchanged. $\dot{V}O_{2max}$ increased up to age 20, after which the values began to fall. The eight skiers represented the larger groups of the present study reasonably well, as the values of the latter were similar to those recorded at the six-and-a half-year and three-and a half-year follow-up.

The annual measurements also clarified the connections between the $\dot{V}O_{2max}$ and echocardiographic variables, showing that 75 % the annual variation in absolute $\dot{V}O_{2max}$ was explained by the annual changes in EDD. The decrease in $\dot{V}O_{2max}$ and the levelling off the increase in EDD during the last two years of the present study was seen in the reduced correlation between SV and $\dot{V}O_{2max}$ during the corresponding period. Because $\dot{V}O_{2max}$ decreased despite unchanged weight it would also seem that the training of the present skiers was not sufficient either in volume or in intensity to match the results of top skiers. In Rusko's (1992) study the increase in $\dot{V}O_{2max}$ of national-level skiers also evened out after age 21, but the $\dot{V}O_{2max}$ of world-class skiers continued to increase.

The hypothesis that large changes occur in the structure and function of the heart up to the ages of 19 and 20 and level off after that was supported. The three and a half years' endurance training and the growth of subjects led to an increase in EDD; the annual follow-up also indicated that EDD increased during the first three and a half years, ceasing thereafter. The changes in the annual measurements were small, in accordance with the hypothesis. At the beginning of the present study the skiers were the best 15 to 16-year-old cross-country skiers in Finland, and it was expected that they would keep their places among the most successful skiers in the country, and that some of them would become international-level top skiers. This expectation held during the first 3 - 4 years, as in Rusko's (1992) study, but subsequently continued to hold for only some of the skiers, stopping or decreasing in the case of others.

The increase in wall thickness took place during the last three-year period of the study. At the baseline and at the follow-up after 3.5 years the walls were almost the same size, but during the last three years SWTd and PWTd increased. The timing of the increase in the walls during the last three years was supported by the results of Tummavuori's (1997) three-year follow-up study, according to which hardly any changes in the thickness of walls were observed

between at ages 16 to 19. SWTd and PWTd were thickest at the follow-ups after 5.5 and 6.5 years, when the skiers were 21.5 - 22.5 years old. The largest individual increase in SWTd and PWTd during the last three years was 0.24 cm. Probably more strength training had been added to the training programme which as pressure-overload training, affects the thickening of the walls, even if no correlations were found between the thickness of walls and training.

The hypothesis that long-term progressive endurance training would lead to an increase in the SWTd and PWTd of the left ventricle of the heart up to the age of 19 - 20 and even out thereafter was not supported. No changes occurred in the thickness of the walls during the first three years: it was only during the last three years that the thickness of the walls increased. The annual changes were small in accordance with the hypothesis.

6.2.3 Seasonal Changes

Training is planned with the aim of achieving the best possible condition during the competition season as well as peak performance. Cross-country skiers train quantitatively a lot during the basic training seasons. Towards the end of the pre-competition season volume decreases and training intensity increases. The $\dot{V}O_{2max}$ of young cross-country skiers varies from 5 to 15 % and that of adults from 3 to 10 % during a training year, and world-class skiers were able to increase $\dot{V}O_{2max}$ from summer to winter more than less successful skiers (Rusko 2003). During puberty a noteworthy training effect can be induced, if the training loads sufficiently and varies in volume during the year. According to Ingjer's (1992) study, differences in training seasons were noticed in $\dot{V}O_2$ (ml $\cdot$ kg^{-1} $\cdot$ min^{-1}) in boys as young as 14.

There were no statistically significant changes in the echocardiographic variables in the present study between the three different training seasons. The results showed that the adaptation of the heart to physical exercise varying in intensity and volume was reasonably slow, despite the changes in the training programme over the six-month periods. The walls of cross-country skiers of the same age increased during a simultaneous intensive training period of almost the same length, but no change was found in EDD (Bienmüller et al. 1982). A progressive and quantitative increase in training over four weeks increased EDD, EDV and SV, but not the wall thickness in the runners (Lehmann et al. 1990), whereas 13 weeks' interval-type training induced an increase in both EDD and PWTd in university team female swimmers (Lamont 1980). Long-distance runners have had a notable larger EDD during the competition season compared to their rest period values, but no significant differences in walls occurred during different periods, neither in runners nor cyclists (Snoeckx et al. 1983), whereas in the study by Fagard et al. (1983) the walls but not EDD of cyclists increased during the competition season. During seven months of increasing training in junior rowers both EDD and walls increased, whereas in senior rowers only EDD increased (Wieling et al. 1981). $\dot{V}O_{2max}$, EDD, SWTd and PWTd of cyclists who trained hard for five months before the beginning of

the competition season increased significantly, even if they detrained for one month before this particular training period (Bonaduce et al. 1998). On the basis of the above-mentioned results it would seem that the dimensions of the hearts of the present cross-country skiers were already so large, due to the accumulated training of the previous years, that either a six-month training period was unable to induce any changes or the training was not, despite its goals, sufficient in volume and intensity to increase the diameter and wall thickness of the left ventricle.

The $\dot{V}O_{2max}$ of the skiers in the present study changed significantly during the 6 to 7-month follow-up. In the study by Wieling et al. (1981) the $\dot{V}O_{2max}$ of both junior and senior rowers also increased, and in the study by Fagard et al. (1983) that of cyclists increased between the resting and competition season. No changes were found in $\dot{V}O_{2max}$ of long-distance runners between seasons, but competition cyclists showed remarkably higher power in bicycle ergometer tests during preparation, competition and slowing-down seasons than during a resting season (Snoeckx et al. 1982). It was assumed that the $\dot{V}O_{2max}$ of the present study would improve during the training seasons, peaking just before the competition season, but in fact it did not increase above the values reached during basic training season 2. Although not much seemed to happen structurally in the heart during the training seasons in this study, some functional change took place along with the improvement in $\dot{V}O_{2max}$ during the basic training seasons.

The hypothesis that the echocardiographic and $\dot{V}O_{2max}$ changes would be small during different training seasons was supported. However, the hypothesis that size of the heart and $\dot{V}O_{2max}$ would be at their highest at the beginning of the competition season, were not supported. The training done in the basic training seasons and pre-competition season did not affect the structure of the heart in these 21-year-old cross-country skiers, who could already be considered to possess the EDD of top endurance athletes. It is assumed that the training should have been significantly greater in volume and intensity during the seasons in order to induce changes in the heart.

6.3 Impact of Body Size and Growth

6.3.1 Skiers and Control Subjects

In order to evaluate the importance of body size and growth to the growth of the size of the heart age-matched control subjects were examined over a period of four and a half years from age 17 to age 22. An unexpected result was the large EDD (5.6 cm) of the control subjects at the end of the study and the fact that EDD of the skiers was larger by only 0.23 cm. There was no statistical difference between the groups, which was due to the large variance of EDD in the control subjects. According the meta-analytical studies by Fagard (1996) and

Maron (1986) a 10 % significant difference has been found between EDD of endurance runners and control subjects. Also according to the meta-analysis of Perrault & Turcotte (1994) the EDD of control subjects (4.82 cm), has been considerably smaller than the EDD found for the present control subjects. The baseline EDD value of the skiers was almost their maximal value. The change in EDD in the skiers during the study was smaller than the change in the control group. The difference in EDD between the skiers and controls was 6.1 % at the baseline and 3.9 % at the follow-up after 4.5 years.

No statistical significance in the relation between EDD/BSA was found in the present study either, although it was assumed that, due to lack of training and increase in BSA, the EDD/BSA of the control subjects would decrease. A significant difference has been found between long-distance runners, cyclists and water- polo players and control subjects in EDD/BSA (Bekaert et al. 1981, Child et al. 1984, Granger et al. 1985, Osborne at al. 1992, Zakynthinos et al. 2001). The EDD/BSA of rowers, track and road cyclists, long-distance runners, canoers and football players was significantly larger than that of controls, whereas the EDD/BSA of weight-lifters and basketball players was significantly smaller than the values recorded to the control subjects (Spataro et al. 1985). In the study by Csanady et al. (1986) there were no differences between junior or senior basketball players and controls, and neither between junior and senior weight-lifters and controls (Haykowsky et al. 1998). Weight-lifters and other strength athletes usually have normal dimensions, when normalized by BSA, in the left ventricles, owing to their big body size, even if the results have been contradictory. No difference in EDD/BSA was found between weight lifters and the control subjects in the studies by Deligiannis et al. (1988) and Menapace et al. (1982), whereas in the study by Brown & Thompson (1987) the difference was significant.

Only group differences were found in the present study in the echocardiographic volume variables. The only variable showing differences between groups at the follow-up after 4.5 years was LVM. The LVM of the skiers increased by 37 g and that of the control subjects by 25 g. According to Fagard's (1996) meta-analysis LVM in long-distance runners has been 48 % larger than that of control subjects, while the difference at the end of the present study was 25 %. The difference in the present study was expected to be larger, because the role of the body and upper limbs has become stronger in training due to ski-skating and double-poling, as this training also includes pressure overload.

Although the $\dot{V}O_{2max}$ of the skiers of the present study was higher than that of endurance athletes, who were on the average three years older, their LVM was lighter. Although the $\dot{V}O_{2max}$ of the controls of the present study was bigger their LVM was lighter than that of controls five years older in the study by Wernstedt et al. (2002).

The results of the present study were, however, similar to those of other studies, where EDD in endurance athletes has proved to be larger than that in the untrained controls (Child et al. 1984, Colan et al. 1985, Dickhuth et al. 1987,

George et al. 1999, MacFarlane et al. 1991, Osborne et al. 1992, Pavlik et al. 1986, Schmidt-Trucksäss et al. 2000, Sépulveda et al. 1989, Swan & Spitler 1989, Turpeinen et al. 1996, Wernstedt et al. 2002). According to Pekkarinen's (1986) study EDD in almost 17-year-old competing cross-country skiers in Finnish skiing clubs was only 5.17 cm and that of the corresponding control subjects 4.95 cm. However, in endurance runners who were two years older EDD was of the same size as that of the present cross-country skiers, although the endurance runners had a bigger $\dot{V}O_{2max}$ (MacFarlane et al. 1991) than the skiers. The $\dot{V}O_{2max}$ and EDD of the control subjects in the study by MacFarlane et al. (1991) were, however, smaller than the values of the present control.

The differences in wall thickness between the skiers and the controls were caused by the fact that in the skiers the thickening of the walls took place during the last three years, as described earlier in connection with the results of the annual measurements. PWTd increased equally in both groups over four and a half years, but the SWTd of the skiers increased more than that of the controls. The PWTd of the control subjects at the end of the study was similar to that of the control subjects of the meta-analysis (Perrault & Turcotte 1994). According to Fagard's (1996) meta-analysis SWTd in the controls has been 0.93 cm and PWTd 0.89 cm, and the differences between them and long-distance runners have been similar to the differences found in the present study between skiers and controls in left ventricle wall thickness. The final wall thickness found here was almost as large as that of the age-matched controls in the study by Wieling et al. (1981). In the study by Agati et al. (1985) the walls of age-matched controls were thicker than those of the the present controls. In Pekkarinen's (1986) study SWTd in skiers and controls was similar, but in both skiers and controls PWTd was thicker than in the present skiers and controls. The walls of long-distance runners have also been found to be thicker than the walls of controls (Colan et al. 1987, Granger et al. 1985, Ikäheimo et al. 1979, Niemelä et al. 1984, Osborne et al. 1992, Wernstedt et al. 2002).

As far as the skiers are concerned the baseline measurements were performed during the competition season and the final measurements during the pre-competition season. The fact that $\dot{V}O_{2max}$ did not change after the four years' training, taking into account the small changes in the biological growth of the skiers, indicates that already by the age of 18 the skiers had reached their peak $\dot{V}O_{2max}$ level with their training. These results were backed up by the results of a three-year follow-up study with skiers of the same age (Tummavuori 1997). The increase in $\dot{V}O_{2max}$ was due to the growth of the control subjects. Even if their physical activity decreased significantly during the four and a half years they were studied they still cannot be regarded as inactive control subjects, which was proved by their $\dot{V}O_{2max}$ of 53 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}. The $\dot{V}O_{2max}$ increased in half of the control subjects and decreased in half by 6 ml $\cdot$ kg^{-1} $\cdot$ min^{-1} on average during the study.

The largest EDD recorded in this study, 6.64 cm, was that of a control subject whose EDD increased by 0.48 cm during the four years and whose walls were 0.8 cm thick at both the baseline and the follow-up after 4.5 years. The

$\dot{V}O_{2max}$ of this control subject was 66 ml · kg^{-1} · min^{-1} at the end of the study, which shows an increase of 10 ml · kg^{-1} · min^{-1} in spite of an almost 8-kg increase in weight. According to Martino et al. (2002) 19 to 22-year-old men with no training background, who have a high $\dot{V}O_{2max}$ (63 - 67 ml · kg^{-1} · min^{-1}) may naturally have a, possibly genetically inherited large BV, which leads to a large SV and Q, and those with a high $\dot{V}O_{2max}$, SV_{max} and Q_{max} may possibly have these because a larger part of their BV has been hemodynamically active. Hypervolemia was assumed to increase Q_{max} and SV_{max} because of increase in venous return and preload, when diastolic filling speed improved and the Frank-Starling mechanism came into play (Convertino 1991, Krip et al. 1997). Genotype has also had an impact on how much influence training has had on aerobic capacity (Hamel et al. 1986, Prud-Homme et al. 1984).

All control subjects but two in the present study showed reduced voluntary physical activity during military service. The increase in EDD could be explained by the fact that, above all, the volume of aerobic exercise increased substantially during that period. After military service less than half of the control subjects showed an increase in voluntary physical activity, and their sports were mostly aerobic. When examining the results of the control subjects it is important to note that even short-term training has impacts on the cardiological variables of untrained people. Ten-day bicycle ergometer training in inactive people increased $\dot{Q}$ by 12 % in an $\dot{V}O_{2peak}$ exercise test, and this increase was solely the result of the increase in SV, as HR remained changed (Mier et al. 1997). However, although the $\dot{V}O_{2max}$ and $\dot{Q}_{max}$, respectively, of untrained people increased as the result of one-year training respectively, no significant changes occurred in either the diastolic or systolic size or function of the left ventricle of the heart (Sadaniantz et al. 1996).

Findings on the impact of genotype on the heart size have been conflicting. Twins seem to show significant genetic influence in MTW, but not in EDD or FS in submaximal exercise (Bielen et al. 1991). Genetic influence on LVM, but not on EDD or on the walls was found in child twins (Bielen et al. 1990). Genetic factors were found to influence LVM in endurance athletes (Karjalainen et al. 1999) but the amount of training accounts for only 11 % of the variability in LVM (Karjalainen et al. 1997). According to Adams et al. (1985) the environment (home) has a greater influence on the size of the heart than genotype, whereas Bouchard & Lortie (1984) and Landry et al. (1985) believe that genetic and other environmental factors influence the size of the heart. Five-month endurance training increased the diameter of the left ventricle and wall thickness in untrained people, but not in monozygotic twins (Landry et al. 1985).

The correlations between echocardiographic and $\dot{V}O_{2max}$, especially the absolute $\dot{V}O_{2max}$ variable, in the skiers at the end of the present study indicated that training and growth had an impact on the results. There was a correlation between the $\dot{V}O_{2max}$ variable (ml · kg$^{-2/3}$ · min^{-1}), indicating performance in cross-country skiing, and SV, and between $\dot{V}O_{2max}$ and LVM. Osborne et al.

(1992) found correlations between $\dot{V}O_{2max}$ and the dimensions of the left ventricle in both athletes and inactive controls. According to their results, the variance of $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) was explained by the degree of endomorphy, chest circumference and EDD. Because no connection was discovered between echocardiographic and $\dot{V}O_{2max}$ variables during seven months in which the training of rowers increased, resting echocardiographic measurements alone cannot be used as variables measuring $\dot{V}O_{2max}$ and performance (Wieling et al. 1981). Björnstad et al. (1993) discovered a significant correlation between LVM/m^2 and hours spent on training. The control subjects of the present study showed no correlations between $\dot{V}O_{2max}$ and EDD or the walls. According to Eisenmann et al. (2000), the dimensions of the hearts of 9 to 18-year-olds were not connected with habitual physical activity or test results in a bicycle ergometer test, but seemed to be defined primarily through normal growth and maturation.

The hypothesis that biological growth causes the increase in heart size in young people was supported. This was, however, contrary to the hypothesis that EDD increased more during the four and a half years in the control subjects than cross-country skiers. The smaller increase in EDD of the skiers was caused by the fact that they had been training for years before the age of 17, and their baseline EDD values were already close to their final values. No differences were found in EDD/BSA variable between the groups either. The increase in EDD in the control subjects was affected by biological growth and probably genetic factors.

6.3.2 Change in BSA

According to Spirito et al. (1994), a larger BSA was connected with a larger diameter and wall thickness of the left ventricle: if BSA increased by 0.1 m^2, the ventricle dimensions increased by 1.2 mm and the walls by 0.2 mm. Also according to Pelliccia et al. (1999), the greatest factor determining the size of the ventricle has been a large BSA and certain endurance sports (cycling, cross-country skiing and canoeing). The impact of body growth on the structural changes in the heart was also examined in the present study, with the division of the skiers into two groups on the basis of the changes taking place in BSA over six years. The significance of the impact of the increase in body growth on the size of the heart was emphasized in the group showing a large change in BSA during the study, as EDD increased more in that group than in the one where the BSA change was small. The group with a small change in BSA were early developers in biological growth and thus also in the size of the heart. There were no differences in walls during the study. At the follow-up after 6.5 years LVM was also heavier and EDV larger in the large change group than small change group.

$\dot{V}O_{2max}$ tended to improve more in the BSA large change group. $\dot{V}O_{2max}$ ($ml \cdot kg^{-2/3} \cdot min^{-1}$) of the skiers in the BSA large change group was better at the end of the study than the final results of the whole group of skiers (n = 20).

However, the $\dot{V}O_{2max}$ of the group of the BSA large change group was smaller than that of Norwegian top skiers, two years younger (Ingjer 1992). The BSA large change skiers trained more than those with the small change in BSA. The hypothesis regarding the impact of body size and growth on the structure and function of the heart was supported, as the early developers showed smaller changes in heart size and $\dot{V}O_{2max}$ than late developers.

6.3.3 EDD/BSA and EDD/BSA-SL

There were no significant changes at any measurement points in EDD/BSA during the study, which leads to the conclusion that body size and growth influenced the changes in the size of the heart. Had EDD/BSA increased during the study, this would probably have been explained by the effect of training on EDD. An alternative explanation would have been loss of weight.

The significance of body size at the baseline was also explored by dividing the subjects into the groups with small or large hearts on the basis of their baseline EDD/BSA values. Because height and weight increased, so the EDD/BSA values did not change during the study despite the increase in EDD. Although the groups did not differ during the study as far height, weight and BSA were concerned, it was apparent that in the group with small hearts the variables in question tended to grow more than in the group with large hearts. Biological growth had taken place; BSA increased, and EDD increased, which was especially noticeable in with small hearts.

The examining of individual results showed that the four subjects whose EDD increased most during the study all belonged to the group with small hearts. In the group with large hearts the EDD/BSA of six subjects decreased, whereas in the group with small hearts this happened only to one skier. It can be said that no large changes in the size of the heart took place in the group with large hearts. There were no between-group differences in the walls of the left ventricle at the initial or final measurements. The thickening of the walls also took place according to this grouping of the skiers during the last three years of the study. At the end of the study the group with small hearts had larger LVM, EDV and SV than the group with large hearts.

In the group with small hearts $\dot{V}O_{2max}$ tended to improve more than in the group with large hearts. Performance improved equally in both groups, which shows improvement in the muscle force production qualities of the nerve muscle system and further improvement in economy of performance and muscle power (Paavolainen et al. 1999). The response to training was similar in both groups. The hypothesis that there would be a similar increase in EDD was not supported, as in the group with small hearts EDD increased more than in the group with large hearts, when the division was made on the basis of baseline EDD/BSA. Further biological development occurred in the group with small hearts: BSA increased and EDD increased.

The main results of the present study are presented in Figures 18 – 20.

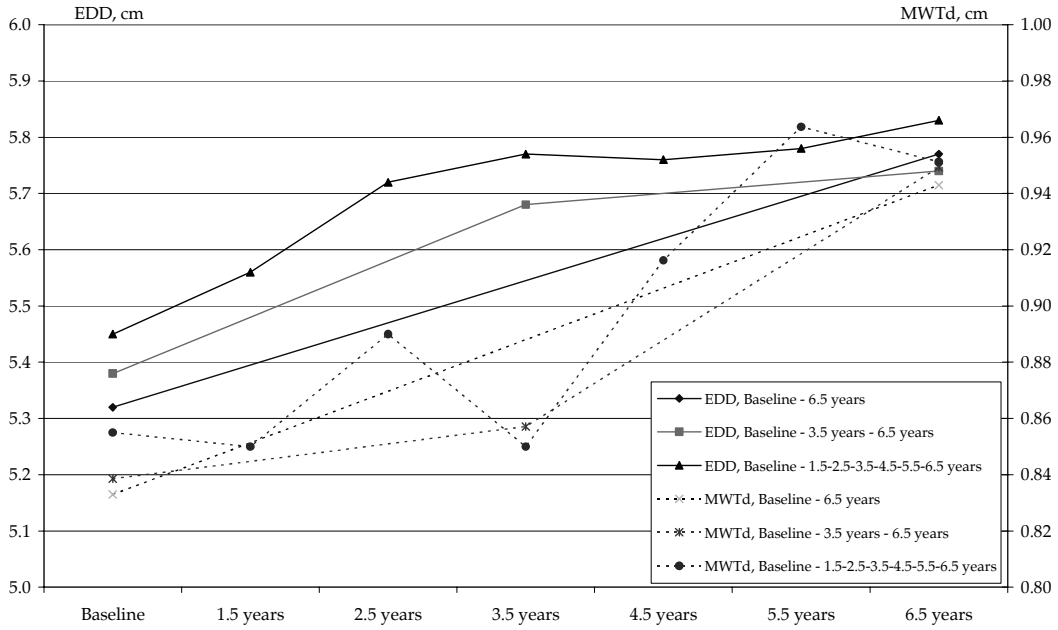

FIGURE 18 End-diastolic diameter (EDD) and mean wall thickness (MWT) of skiers at baseline and at follow-ups after 1.5, 2.5, 3.5, 4.5, 5.5 and 6.5 years.

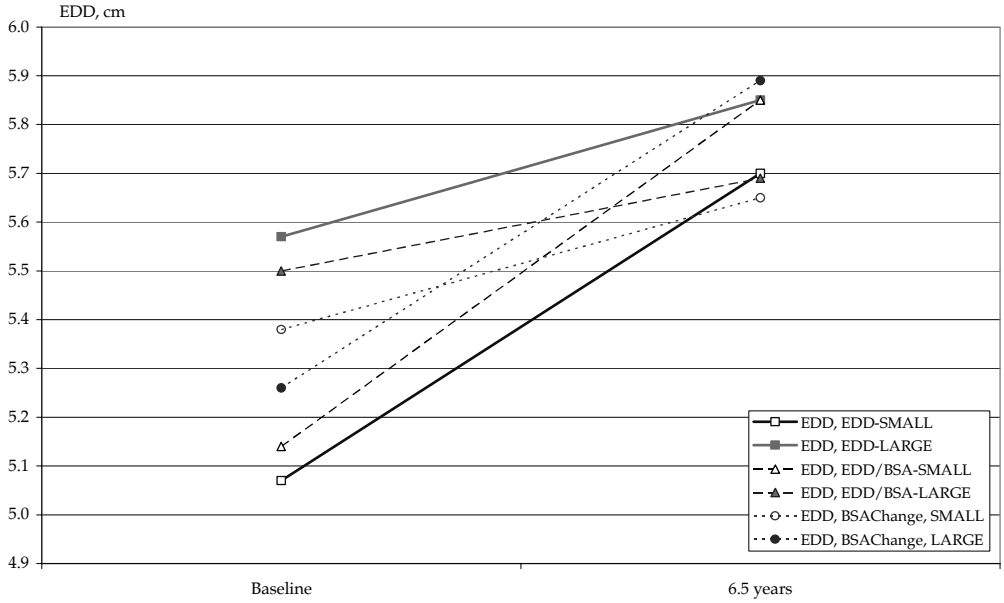

FIGURE 19 End-diastolic diameter (EDD) of skiers at baseline and at follow-up after 6.5 years (EDD-SL, EDD/BSA-SL, change in BSA).

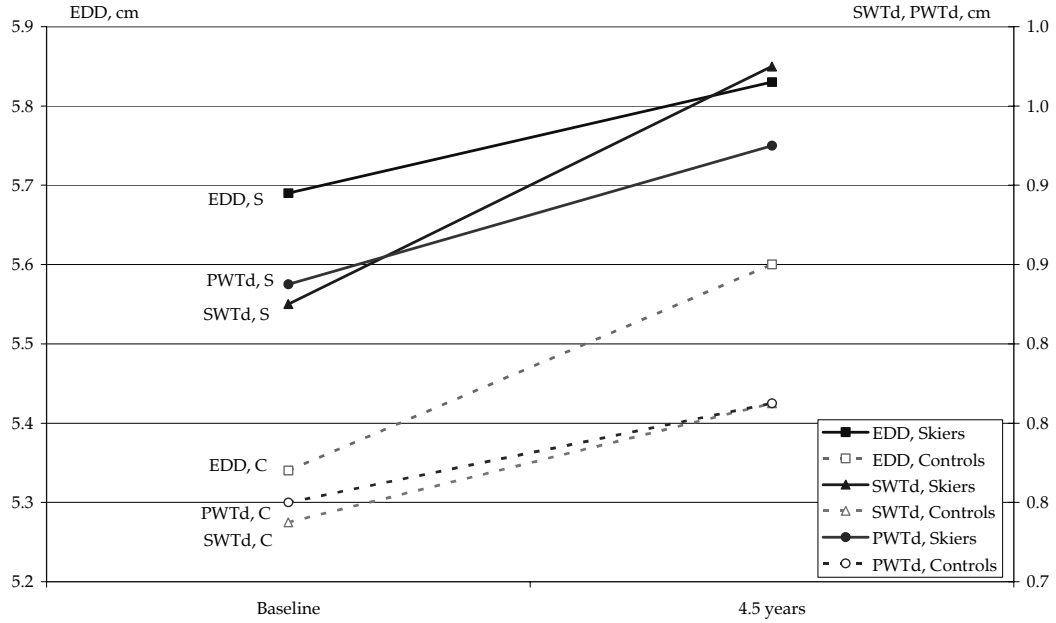

FIGURE 20 End-diastolic diameter (EDD) and septal wall (SWTd) and posterior wall (PWTd) thickness in diastole of skiers (S) and controls © at baseline and at follow-up after 4.5 years.

6.4 Limitations

The number of skiers in the 6.5-year follow-up study was large (n = 20). It is also noteworthy that the study dealt with top-class skiers, the structure and function of whose hearts and cardiovascular capacity could be examined over such a long period. Although the number of the skiers at the annual measurements was small (n = 8), they seemed, according to the results, to represent reasonably well the whole group of skiers. During the first few years of the study the participation of the skiers in the measurements was active, but during the final years only those who were competing actively in cross-country skiing continued to participate. Some of the skiers finished training altogether and were no longer interested in presenting for the measurements.

The skiers benefited from the measurements, as they were given accurate information of their treadmill test performance, e.g. aerobic and anaerobic thresholds, information which they could use in planning and carrying out their training. It was difficult to retain the control subjects in the study for four years. During the first couple of years the control subjects were easy to reach, because they presented for the measurements during school hours and from a school that was situated near the research institute. But later their interest in coming to the measurements declined, as they were little interested in their physical condition, and the test results were of no practical use to them. All the control subjects, however, gave information about their sports and the time spent on them.

The echocardiographic measurements were taken every time by the same researcher, and the same person digitalized all the echocardiograms. In this way different method of measuring and digitalizing data was avoided. The person who digitalized the echocardiograms was present at the measurements and thus was able to determine the right place in difficult echoes when later digitalizing the echocardiograms. A defect in the digitalizing phase was the fact that the digitalizing was done by only one reader. Reproducibility was, however, good when the internal reproducibility of the one reader and the reproducibility between two readers for two separate measurements of EDD, SWTd and PWTd were examined. ICC varied between the readers depending on the variable from .77 to .90, and within the reader from .88 to .98. The respective CV was 2.8 - 6.8 % and 1.4 - 3.7 %. The results are presented on pages 46 - 47. The accuracy of the digital results was considered adequate, as the resolution of the digitalizing table was 0.05 cm.

The reliability of the echocardiographic results was increased by the fact that the subjects were measured always in the same way in the same left posture, and at every measurement on the same research table. The $\dot{V}O_{2max}$ values obtained at the different measurement times were easily comparable, as the exercise patterns were the same. The reliability of the results was also increased by the fact that after the baseline measurements the skiers were measured at four, nine and thirteen weeks, during which period the echocardiographic changes were small.

It was impossible, however, to determine the total effects of training on the skiers, as not all the skiers kept a training diary, despite being asked to do so, and neither were they able to give accurate data on the volume and contents of their training when requested. Data on the whole year's training between the baseline and the follow-up after 6.5 years was obtained from 40 % skiers on kilometers, from 70 % on hours spent training, and from 55 % skiers on training times. Data on the 16 and 12 weeks of the basic training seasons (BTS1 and BTS2) before the baseline and the follow-up after 6.5 years were available on kilometers, hours and times, respectively, from 35, 75 and 60 % and from 30, 75 and 60 % of the skiers.

7 CONCLUSIONS

This 6.5-year follow-up study gave information about the effect of training on the structure and function of the heart of young endurance athletes. The results also clarified the role of $\dot{V}O_{2max}$, indicating that maximal cardiovascular capacity was due endurance training carried out over the 6.5-year period. Finally the results clarified the periods when changes occurred in the structure and function of the heart and in $\dot{V}O_{2max}$, and how these were affected by body growth and development.

1. The 16-year-old cross-country skiers could be regarded as in good physical condition already at the beginning of the study. They were well trained, as was demonstrated by their $\dot{V}O_{2max}$ results obtained in ski-walking test on a treadmill. Also the EDD of the left ventricle of the heart in the skiers, measured at the beginning of the study was already equal to that of adult endurance athletes in cross-sectional studies.
2. EDD increased significantly during the 6.5 years. The measurements taken at intervals of three and a half years and annually indicated that the increase of EDD was largest during the first three and a half years of study (ages 16 - 19), thereafter levelling off. The changes in $\dot{V}O_{2max}$ followed the same trend as those in EDD.
3. The average increase in 0.5 cm of EDD during the 6.5-year period could be considered small. There were several factors which could have contributed to this result.
    a) EDD was large already at the beginning of the study due to training and genotype.
    b) Military service was completed during the study period, which prevented the skiers from fully implementing their training programme.
    c) The training of all skiers did not progressively increase throughout the study period.
    d) Injuries caused breaks and changes in the training programme.

4. SWTd and PWTd increased significantly during the 6.5 years. The measurements taken at intervals of three years and annually indicated that the increase of the walls was largest during the last three years of the study (ages 19 - 22).
5. Different training seasons had no effect on the echocardiographic variables, although $\dot{V}O_{2max}$ measured by ski-walking test on the treadmill increased, indicating that functional changes occurred during maximal exercise between training seasons.
6. The baseline values played a role in the increase in EDD and in the size of the increase, but had no effect on wall thickness.
    a) When the skiers were grouped by size of hearts according to their baseline EDD, an interaction in EDD was found. In skiers with small hearts EDD increased more than in those with large hearts during the 6.5-year period, although not quite reaching the size of those with large hearts even at the end of the study.
    b) EDD in both groups increased during the first three and a half years. The increase continued during the last three years only in the group of skiers with small hearts.
7. The impact of size and growth
    a) EDD/BSA of the skiers did not change during the 6.5 years. Training affected the baseline level of EDD and its further dilatation, and with body growth BSA also increased significantly.
    b) The skiers and control subjects of the same age showed no significant differences in EDD, when they were followed from age 17 to age 22. EDD tended, however, to be larger in the group of skiers than in the group of control subjects at both the baseline and the follow-up after 4.5 years.
    c) The difference in EDD was not significant, e.g. one control subject had an EDD equal to the largest EDD (6.6 cm) among the skiers. Thus genes may play an important role in EDD development.
    d) The good results of the control subjects can also be explained by the fact that these were fairly active. The activity of the controls was also affected by military service done during the study, during which the volume of aerobic exercise of these subjects increased considerably.
    e) The skiers were divided into two groups according to body growth on the basis of the change in BSA. In the group with a small change in BSA biological growth had finished already at the beginning of the study, whereas in the group with a large change in BSA biological development continued during the study period. In the group where the change in BSA was large EDD increased more than in the group where the change in BSA was small. The magnitude of the change in BSA showed no interaction with wall thickness.

f) The division of the skiers into two groups according to size of hearts was also done according their baseline EDD/BSA values. An interaction was found in EDD/BSA: EDD increased more in the group with small hearts than in the group with large hearts. This increse was affected by the continuation of biological growth in the group with small hearts throughout the study period.

g) The walls of the skiers remained significantly thicker than those of the control subjects after analysis of covariance.

8. It can be concluded that dilatation in the skiers' hearts had occurred already before the baseline measurements of this study. At ages 16 to 19, further dilatation took place in the heart and the walls remained unchanged. During the next three years, at ages 19 to 22, the walls thickened, during which period the increase in EDD levelled off. These results suggest the hearts of young cross-country skiers first undergo dilatation, due to endurance training, after which, as training continues the walls begin to thicken.

9. In addition to training the increase in EDD in skiers was also caused by body size and growth. Because the walls of the control subjects did not thicken, training was responsible for the thickening of SWTd and PWTd in the skiers. Had the increase of the size of the hearts of the skiers of this study already reached the so-called "ceiling"? It is, however, probable that if training had continued and increased in both volume and intensity the dilatation of the heart and the thickening of the walls would also have increased, and these skiers, at least with respect to their $\dot{V}O_{2max}$ values, become international-level top skiers.

8 SUGGESTIONS FOR FUTURE RESEARCH

Future studies should involve younger subjects than the 16-year-old cross-country skiers in this study. They had already the EDD of a mature athlete's heart at the baseline, but the walls thickened only later (at the age of 19 to 22), even if at the end of the study they were not as thick as those reported in several studies for endurance athletes. On the basis of the present study we cannot know at what age EDD grew or weather the changes were caused by training selection for cross-country skiing, in which having a large heart is an advantage. This study did not obtain enough data about training, i.e. about how long and how strenuous training needs to be for the features typical of an athlete's heart to develop. Relatively few echocardiographic studies have been done among children, and the results have been mixed. The hearts of athletic children have grown larger and the walls have become thicker than those of inactive children (Drescher et al. 1986, Gutin et al. 1985, Medved et al. 1986, Meško et al. 1993, Rowland et al. 1987, 2002), whereas even a 3-year training programme did not increase EDD in 12-year-olds, when it was normalized by the age of skeletal structure and body weight (Telford et al. 1988). On the other hand, if training and competing had started in childhood it would have an increase in heart volume and thickening of the walls even before puberty, but in these children heart volume was after puberty similar to that of the youngsters who started training after puberty (Rost 1982). The greater cardiac index found in 9 to 13-year-old endurance runners during exercise compared to nonathletic children was the result of greater stroke volume (Rowland et al. 1998). SV_{max} was a critical factor for a high $\dot{V}O_{2max}$ in 12-year-old cyclists and the factors which affected SV at rest were important when explaining the differences between training and non-training child endurance athletes (Rowland et al. 2000). A greater SV_{max} in young endurance athletes is a sign of greater ventricle preload, i.e. diastolic ventricle volume at rest (Rowland et al. 2002).

An ideal population for such a study would be children engaged in different sports at an early stage (8 to 10 years old). Data could then be collected on the differences that different sports cause in changes in the heart. Due to the rapid development of children and adolescents measurements need to be taken

2 - 3 times a year. Examining hormonal changes would also give further information. The subject group should include both boys and girls. In order to obtain more detailed information about the development of athletes' hearts, data would also be needed about the function and changes in the heart during exercise. The measurements should be taken at all levels up to maximum load.

One important factor that should be examined in future studies is the connection between athletes' training and the structure and the changes in function of the heart. EDD is known to grow with training, and with further training $\dot{V}O_{2max}$ and $\dot{Q}$ are said to increase as the result of the growth in EDD and the walls and the decrease in ESD. The number of hours and kilometers spent on training, and training times do not give enough information about the effect of training on the development of an athlete's heart. The intensity and length of training need to be known. Also the effect of classical vs. free-style skiing, roller skiing, running, power training, games, rowing etc. training should be examined. In skiing the training done should be defined more closely e.g. how much uphill skiing has been included? The skating and double poling techniques emphasize the importance of the $\dot{V}O_{2max}$ of the upper body. The increased power training of the arms and upper body increases the role of static work in training, which in turn increases arterial pressure, affects blood flow, and preload, and causes thickening of the walls. It is important to determine the relative role of static/dynamic work in classical skiing and free-style skiing training. Athletes have to be taught to fill in their training diaries accurately. They must know when e.g. aerobic, anaerobic and maximal training is concerned. Training periods at a high altitude, competitions and success in competitions should also be marked in the training diary.

The baseline measurements of this study were taken in 1987, using the echocardiographic method employed at the time. Future measurements should use magnetic resonance imaging (MRI), which would give accurate and repeatable data on the dimensions of the heart. In calculating the M-mode of the dimensions of the heart echocardiographic data it has been assumed that the heart has a certain standard form. Moreover possible mistakes have been multiplied, because in most figures the end-diastolic dimensions of the left ventricle have either cubed or squared, possible leading to errors of measurements (Fagard et al. 1984, Troy et al. 1972, Wolfe et al. 1979, 1986). Modern ultrasound equipment has developed enormously and is very suitable in estimating heart function, tissue characterization and exercise descriptions. The only significant notable defect in ultrasound is in estimating LVM, where MRI is clearly superior. MRI is poorly adapted to dynamic measurements, as a standard and preferably slow HR is required for descriptions. MRI is a good method in tissue characterization. When MRI is used, LVM is not affected by any potential changes that training has caused in the geometry of the left ventricle. In MRI the mass of the wall of the left ventricle is calculated directly and no geometric assumptions concerning its form are needed (Milliken et al. 1988, Myerson et al. 2002, Riley-Hagan et al. 1992). There are also fewer interpretation errors in MRI, because there are no breaks similar to those caused

by the disappearance of the echo in echocardiographic research. With MRI method the results are affected neither by the limitations of the acoustic window or the position of the transducer, which may happen with the ultrasound technique (Milliken et al. 1988).

Comparisons of echocardiographic methods and MRI have produced conflicting results. According to Wernstedt et al. (2002) no significant difference in the LVM in endurance athletes, weightlifters and controls was found when calculated with the formula of Devereux et al. (1986). Scharhag et al. (2003) found, on the contrary, that when the MRI results of endurance athletes and untrained people were compared to those obtained by different echocardiographic methods, the Dickhuth et al. (1996) method was the most accurate. The Devereux et al. (1986) method, which the present study also used, overestimated the size of LVM significantly. When estimating the heart structure and function MRI is the only adequate method. It can also define the blood flow and perfusion of the heart muscle. To examine other differences in the athlete's heart isotopic methods (particularly metabolism) and e.g. the sympathetic nervous system of the heart positron emission tomography (PET) and single photon emission tomography (SPET) can also be used. Highly versatile possibilities exist for noninvasive study of the athlete's heart. Methodological improvements will generate significant data on the impact of sports on the heart in the near future.

YHTEENVETO

Tämä 6.5 vuoden seurantatutkimus antoi tietoa harjoittelun vaikutuksista nuorten kestävyysurheilijoiden sydämen rakenteeseen ja toimintaan. Tuloksista selvisi myös maksimaalista kardiovaskulaarista toimintakykyä kuvaavan maksimaalisen hapenottokyvyn ($\dot{V}O_{2max}$) kehittyminen 6.5 vuoden kestävyysharjoittelun vaikutuksesta. Tutkimustuloksista selvisi ajankohdat, jolloin sydämen rakenteessa ja toiminnassa ja $\dot{V}O_{2max}$:ssa tapahtui muutoksia, ja myös se, kuinka koehenkilöiden kasvu ja kehitys vaikuttivat tuloksiin ja mikä oli lähtötason merkitys sydämen koon kasvuun.

Tämän tutkimuksen koehenkilöjoukko koostui ikäluokkansa (15 – 17 -vuotiaat) parhaimmista miespuolisista maastohiihtäjistä (n = 38). Tutkimuksen kontrollihenkilöinä oli samanikäisiä koulupoikia (n = 18).

Koe- ja kontrollihenkilöiden $\dot{V}O_{2max}$ mitattiin juoksumatolla uupumukseen asti suoritetussa kuormitustestissä, joka tehtiin sauvakävelynä. Sydämen rakenteen ja toiminnan selville saamiseksi koe- ja kontrollihenkilöille tehtiin kaikukardiografiset mittaukset kaikukameralla. Päämuuttujina olevat sydämen vasemman kammion loppudiastolinen läpimitta (EDD = end-diastolic diameter), loppusystolinen läpimitta (ESD = end-systolic diameter), loppudiastolinen väliseinämän paksuus (SWTd = septal wall thickness in diastole) ja loppudiastolinen takaseinämän paksuus (PWTd = posterior wall thickness in diastole) mitattiin tutkittavien ollessa makuuasennossa vasemmalla kyljellään.

Tutkimuksen päätulokset:

1. Tutkimuksen koehenkilöitä voitiin jo tutkimuksen alussa pitää hyväkuntoisina 16-vuotiaina maastohiihtäjinä. He olivat hyvin harjoitelleita, joka ilmeni heidän juoksumatolla suoritetussa sauvakävelytestissä saavutetuista $\dot{V}O_{2max}$:n tuloksistaan. Myös tutkimuksen alussa mitattu EDD osoitti, että heillä oli jo yhtä suuri EDD kuin aikuisilla kestävyysurheilijoilla.
2. EDD kasvoi merkitsevästi 6.5 vuoden aikana. Kolmen vuoden välein ja vuosittain tehdyt mittaukset osoittivat, että EDD:n kasvu oli suurinta ensimmäisen 3.5 tutkimusvuoden (ikävuodet 16 – 19) aikana, jonka jälkeen EDD:n kasvu tasaantui. $\dot{V}O_{2max}$:n tulokset muuttuivat samalla tavalla samoina ajankohtina kuin EDD:n tulokset.
3. EDD:n keskimäärin 0.5 cm:n kasvua hiihtäjillä 6.5 vuoden aikana voitiin pitää pienenä. Tulokseen on voinut vaikuttaa useita tekijöitä.
    a) EDD oli suuri jo tutkimuksen alussa johtuen harjoittelusta ja perimästä.
    b) Tutkimusjakson aikana suoritettu varusmiespalvelus, joka esti hiihtäjiä täydellisesti toteuttamasta harjoitusohjelmaansa.
    c) Kaikkien hiihtäjien harjoittelu ei lisääntynyt progressiivisesti tutkimusjakson aikana.

d) Loukkaantumiset aiheuttivat taukoja ja muutoksia harjoitusohjelmaan.

4. SWTd ja PWTd kasvoivat tutkimuksen aikana merkitsevästi. Kolmen vuoden välein ja vuosittain tehdyt mittaukset osoittivat, että seinämien kasvu oli suurinta tutkimuksen kolmen viimeisen vuoden aikana (ikävuodet 19 – 22).
5. Maastohiihtäjien erilaisilla harjoituskausilla 6 - 7 kuukauden aikana ei ollut vaikutusta kaikukardiografisiin muuttujiin, vaikka juoksumatolla sauvakävelytestissä mitattu $\dot{V}O_{2max}$ parani. Tulos viittasi siihen, että toiminnallisia muutoksia tapahtui maksimikuormituksessa harjoituskausien välillä.
6. Lähtötilanteella oli merkitystä EDD:n kasvuun ja kasvun suuruuteen, mutta ei seinämäpaksuuksiin.
   a) Tutkimuksen alkumittausten EDD:n koon mukaan määritettiin pieni- ja isosydämisten hiihtäjien ryhmä. EDD kasvoi 6.5 vuoden aikana enemmän pieni- kuin isosydämisten ryhmässä, mutta pienisydämisten EDD:n koko ei tutkimuksen lopussakaan ihan saavuttanut isosydämisten EDD:n kokoa.
   b) EDD kasvoi molemmilla ryhmillä ensimmäisen kolmen ja puolen vuoden aikana. Kasvu jatkui kolmen viimeisen vuoden aikana vain pienisydämisten ryhmässä.
7. Koon ja kasvun merkitys
   a) Hiihtäjien EDD/BSA (body surface area = kehon pinta-ala) ei muuttunut 6.5 vuoden aikana. Harjoittelu vaikutti EDD:n lähtötasoon ja edelleen dilatoitumiseen ja kasvun myötä myös BSA suureni merkitsevästi.
   b) Samanikäisten hiihtäjien ja kontrollihenkilöiden EDD:ssä ei ollut merkitseviä eroja, kun heitä seurattiin neljä ja puoli vuotta ikävuodesta 17 ikävuoteen 22. EDD oli kuitenkin keskiarvoisesti hiihtäjillä suurempi sekä alkumittauksissa että 4.5 vuoden seurannan jälkeen kuin kontrolliryhmällä.
   c) Ero EDD:ssä ei ollut merkitsevä; esimerkiksi yhdellä kontrollihenkilöllä EDD oli yhtä iso kuin suurin hiihtäjän EDD. Perintötekijöillä saattoi täten olla merkittävä vaikutus EDD:n kehittymisessä.
   d) Kontrolliryhmän hyviä tuloksia voidaan selittää myös sillä tosiasialla, että ryhmä oli fyysisesti melko aktiivinen. Aktiivisuuteen vaikutti myös tutkimuksen aikana suoritettu asepalvelus, jonka aikana kontrollihenkilöillä aerobisen liikunnan määrä lisääntyi huomattavasti.
   e) Koehenkilöt jaettiin pieni- ja isokasvuisiin BSA:n muutoksen perusteella. Pienen muutoksen BSA-ryhmässä biologinen kasvu oli loppunut jo tutkimuksen alussa, kun taas suuren muutoksen BSA-ryhmässä biologinen kehitys jatkui vielä tutkimusjakson aikana. Ryhmässä, jossa BSA:n muutos oli suuri, EDD kasvoi enemmän kuin ryhmässä, jossa BSA:n muutos oli pieni. BSA:n muutoksen suuruus ei vaikuttanut seinämäpaksuuksiin.

f) Koehenkilöiden jako pieni- ja isosydämisiin tehtiin tutkimuksen alkumittausarvojen perusteella myös EDD/BSA:n tulosten mukaan. EDD suureni pienisydämisten ryhmässä enemmän kuin suurisydämisten ryhmässä. Kasvuun vaikutti se, että biologinen kasvu jatkui pienisydämisillä koko tutkimusjakson ajan.

g) Seinämät olivat hiihtäjillä merkitsevästi paksummat kuin kontrollihenkilöillä.

8. Loppuyhteenvetona voidaan todeta, että hiihtäjillä sydämen dilataatio oli tapahtunut jo ennen tämän tutkimuksen alkumittauksia. Ikävuosina 16 – 19 sydämessä tapahtui edelleen dilatoitumista ja seinämät pysyivät muuttumattomina. Seuraavien kolmen vuoden aikana, ikävuosina 19 – 22 seinämät paksuuntuivat, jona aikana EDD:n kasvu tasaantui. Näiden tulosten perusteella näyttää siltä, että nuorilla maastohiihtäjillä kestävyysharjoittelun vaikutuksesta sydämessä tapahtuu ensin dilatoitumista, jonka jälkeen harjoittelun edelleen jatkuessa seinämät alkavat paksuuntua.

9. Harjoittelun lisäksi kasvu ja kehitys vaikuttivat hiihtäjillä siihen, että EDD kasvoi. Koska kontrollihenkilöillä seinämät eivät paksuuntuneet, vaikutti harjoittelu SWTd:n ja PWTd:n paksuuntumiseen hiihtäjillä. Oliko tämän tutkimuksen hiihtäjien sydämen koon kasvu jo saavuttanut ns. ”ylärajan”? Todennäköistä kuitenkin on, että harjoittelun edelleen jatkuessa ja lisääntyessä sekä määrältään että teholtaan, myös sydämen dilatoituminen ja seinämien hypertrofioituminen lisääntyisivät, jolloin tämän tutkimuksen hiihtäjistä tulisi kansainvälistä tasoa olevia huippuhiihtäjiä myös $\dot{V}O_{2max}$:n arvoja tarkasteltaessa.

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APPENDIX 1 Exercise patterns of the skiers in treadmill test.

Running

Time (min)	Velocity ($m \cdot s^{-1}$)	Inclination (degree)	Demand ($ml \cdot kg^{-1} \cdot min^{-1}$)
3	6.3	1.0	20
6	8.1	1.0	26
9	9.9	1.0	32
12	11.7	1.0	38
15	13.5	1.0	44
18	13.5	2.4	50
21	13.5	3.8	56
24	13.5	4.7	62
27	13.5	5.6	68
30	13.5	6.5	74

Ski-walking

Time (min)	Velocity ($m \cdot s^{-1}$)	Inclination (degree)	Demand ($ml \cdot kg^{-1} \cdot min^{-1}$)
3	6.4	2.0	26
6	6.7	5.0	32
9	7.0	6.4	38
12	7.0	7.9	44
15	7.0	9.6	50
18	7.0	11.2	56
21	7.0	12.8	62
24	7.0	14.4	68
27	7.5	14.5	74
30	8.0	14.8	80

APPENDIX 2 Exercise patterns of the control subjects in ski-walking tests.

Model 1

Time (min)	Velocity ($m \cdot s^{-1}$)	Inclination (degree)	Demand ($ml \cdot kg^{-1} \cdot min^{-1}$)
3	6.0	2.3	20
6	6.0	4.2	26
9	6.5	5.3	32
12	7.0	6.4	38
15	7.0	7.9	44
18	7.0	9.6	50
21	7.0	11.2	56
24	7.0	12.8	62
27	7.0	14.4	68
30	7.5	14.5	74

Model 2

Time (min)	Velocity ($m \cdot s^{-1}$)	Inclination (degree)	Demand ($ml \cdot kg^{-1} \cdot min^{-1}$)
3	6.0	0.8	15
6	6.0	2.3	20
9	6.5	3.2	25
12	6.5	4.7	30
15	6.5	6.2	35
18	6.5	7.7	40
21	6.5	9.1	45
24	6.5	10.6	50
27	6.5	12.1	55
30	6.5	13.5	60

Model 3

Time (min)	Velocity ($m \cdot s^{-1}$)	Inclination (degree)	Demand ($ml \cdot kg^{-1} \cdot min^{-1}$)
3	6.0	0.9	16
6	6.0	2.3	20
9	6.0	3.5	24
12	6.0	4.8	28
15	6.0	6.1	32
18	6.0	7.3	36
21	6.0	8.6	40
24	6.0	9.9	44
27	6.0	11.1	48
30	6.0	13.5	52

APPENDIX 3 Training seasons of the skiers.

Training season	Duration (weeks)	Point of time
Basic training season 1 (BTS1)	16	weeks 17-32
Basic training season 2 (BTS2)	12	weeks 33-44
Pre competition season (PCS)	8	weeks 45-52
Competition season	12	weeks 1-12
Transition season	4	weeks 13-16

APPENDIX 4 End-diastolic diameter (EDD) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

EDD		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	5.32 ± 0.33						5.77 ± 0.39
6.5y3m		14	5.38 ± 0.33			5.68 ± 0.37			5.74 ± 0.42
Annual		8	5.45 ± 0.39	5.56 ± 0.40	5.72 ± 0.48	5.77 ± 0.45	5.76 ± 0.47	5.78 ± 0.38	5.83 ± 0.49
EDD-SL	Small	10	5.07 ± 0.17						5.70 ± 0.27
EDD-SL	Large	10	5.57 ± 0.25						5.85 ± 0.49
EDD/BSA-SL	Small	10	5.14 ± 0.26						5.85 ± 0.34
EDD/BSA-SL	Large	10	5.50 ± 0.30						5.69 ± 0.44
BSAChange-SL	Small	10	5.38 ± 0.42						5.65 ± 0.33
BSAChange-SL	Large	10	5.26 ± 0.22						5.89 ± 0.42
4.5y2m	Skiers	15			5.69 ± 0.35				5.83 ± 0.43
4.5y2m	Controls	10			5.34 ± 0.39				5.60 ± 0.54
			BTS1	BTS2	PCS				
Seasons		15	5.72 ± 0.38	5.77 ± 0.34	5.78 ± 0.39				

APPENDIX 5 End-systolic diameter (ESD) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

ESD		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	3.83 ± 0.38						3.90 ± 0.41
6.5y3m		14	3.88 ± 0.43			4.07 ± 0.36			3.92 ± 0.37
Annual		8	4.14 ± 0.34	3.93 ± 0.28	4.12 ± 0.26	4.21 ± 0.25	4.02 ± 0.25	4.14 ± 0.28	3.97 ± 0.21
EDD-SL	Small	10	3.58 ± 0.26						3.76 ± 0.38
EDD-SL	Large	10	4.09 ± 0.30						4.05 ± 0.40
EDD/BSA-SL	Small	10	3.71 ± 0.32						3.89 ± 0.37
EDD/BSA-SL	Large	10	3.96 ± 0.41						3.92 ± 0.46
BSAChange-SL	Small	10	3.90 ± 0.41						3.82 ± 0.35
BSAChange-SL	Large	10	3.77 ± 0.36						3.99 ± 0.46
4.5y2m	Skiers	15			4.21 ± 0.17				4.03 ± 0.20
4.5y2m	Controls	10			3.78 ± 0.29				3.78 ± 0.37

		BTS1	BTS2	PCS
Seasons	15	3.99 ± 0.34	3.99 ± 0.44	4.01 ± 0.44

APPENDIX 6 Septal wall thickness in diastole (SWTd) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

SWTd		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	0.84 ± 0.05						0.96 ± 0.12
6.5y3m		14	0.85 ± 0.05			0.85 ± 0.05			0.97 ± 0.11
Annual		8	0.86 ± 0.06	0.85 ± 0.08	0.88 ± 0.06	0.84 ± 0.06	0.93 ± 0.11	0.97 ± 0.07	0.98 ± 0.10
EDD-SL	Small	10	0.84 ± 0.06						0.98 ± 0.11
EDD-SL	Large	10	0.85 ± 0.04						0.94 ± 0.12
EDD/BSA-SL	Small	10	0.84 ± 0.05						0.97 ± 0.10
EDD/BSA-SL	Large	10	0.84 ± 0.05						0.95 ± 0.13
BSAChange-SL	Small	10	0.84 ± 0.07						0.94 ± 0.14
BSAChange-SL	Large	10	0.84 ± 0.03						0.98 ± 0.09
4.5y2m	Skiers	15			0.86 ± 0.06				0.98 ± 0.11
4.5y2m	Controls	10			0.75 ± 0.07				0.81 ± 0.08
			BTS1	BTS2	PCS				
Seasons		15	0.97 ± 0.07	0.99 ± 0.08	0.99 ± 0.08				

APPENDIX 7 Posterior wall thickness in diastole (PWTd) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S= small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

PWTd		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	0.83 ± 0.05						0.92 ± 0.08
6.5y3m		14	0.83 ± 0.05			0.86 ± 0.05			0.93 ± 0.09
Annual		8	0.85 ± 0.04	0.85 ± 0.04	0.90 ± 0.06	0.86 ± 0.04	0.90 ± 0.08	0.96 ± 0.08	0.93 ± 0.07
EDD-SL	Small	10	0.82 ± 0.05						0.89 ± 0.08
EDD-SL	Large	10	0.84 ± 0.04						0.96 ± 0.08
EDD/BSA-SL	Small	10	0.81 ± 0.05						0.89 ± 0.06
EDD/BSA-SL	Large	10	0.84 ± 0.04						0.95 ± 0.10
BSAChange-SL	Small	10	0.85 ± 0.04						0.93 ± 0.09
BSAChange-SL	Large	10	0.81 ± 0.04						0.92 ± 0.09
4.5y2m	Skiers	15			0.87 ± 0.05				0.94 ± 0.08
4.5y2m	Controls	10			0.76 ± 0.05				0.81 ± 0.10
			BTS1	BTS2	PCS				
Seasons		15	0.96 ± 0.08	0.95 ± 0.10	0.94 ± 0.10				

APPENDIX 8 Stroke dimension (SD) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

SD		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	1.39 ± 0.09						1.49 ± 0.11
6.5y3m		14	1.39 ± 0.11			1.40 ± 0.08			1.47 ± 0.11
Annual		8	1.33 ± 0.39	1.44 ± 0.10	1.41 ± 0.05	1.39 ± 0.05	1.44 ± 0.04	1.41 ± 0.07	1.46 ± 0.05
EDD-SL	Small	10	1.42 ± 0.10						1.52 ± 0.09
EDD-SL	Large	10	1.37 ± 0.08						1.45 ± 0.13
EDD/BSA-SL	Small	10	1.39 ± 0.09						1.51 ± 0.10
EDD/BSA-SL	Large	10	1.40 ± 0.09						1.46 ± 0.13
BSAChange-SL	Small	10	1.39 ± 0.06						1.49 ± 0.12
BSAChange-SL	Large	10	1.40 ± 0.12						1.49 ± 0.12
4.5y2m	Skiers	15			1.36 ± 0.06				1.44 ± 0.08
4.5y2m	Controls	10			1.42 ± 0.12				1.48 ± 0.08
			BTS1	BTS2	PCS				
Seasons		15	1.43 ± 0.10	1.45 ± 0.13	1.44 ± 0.10				

APPENDIX 9 End-diastolic volume (EDV) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

EDV		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	137.4 ± 20.0						165.6 ± 26.3
6.5y3m		14	140.9 ± 20.6			159.4 ± 23.8			164.0 ± 28.1
Annual		8	145.2 ± 24.3	152.3 ± 26.0	162.8 ± 30.5	165.8 ± 28.7	165.3 ± 32.1	166.2 ± 24.5	170.2 ± 33.2
EDD-SL	Small	10	122.5 ± 9.5						160.3 ± 17.0
EDD-SL	Large	10	152.2 ± 16.3						171.0 ± 33.2
EDD/BSA-SL	Small	10	126.5 ± 14.7						170.6 ± 23.4
EDD/BSA-SL	Large	10	148.2 ± 19.1						160.7 ± 29.2
BSAChange-SL	Small	10	141.2 ± 25.4						157.4 ± 21.8
BSAChange-SL	Large	10	133.5 ± 12.9						173.9 ± 28.8
4.5y2m	Skiers	15			160.3 ± 21.9				169.8 ± 29.3
4.5y2m	Controls	10			138.8 ± 23.9				155.3 ± 34.9
			BTS1	BTS2	PCS				
Seasons		15	162.3 ± 25.0	165.5 ± 22.8	166.2 ± 25.9				

APPENDIX 10 End-systolic volume (ESV) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

ESV		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	64.2 ± 15.3						67.1 ± 16.2
6.5y3m		14	66.4 ± 17.2			73.5 ± 14.6			67.5 ± 14.2
Annual		8	76.5 ± 14.7	67.4 ± 11.7	75.5 ± 11.6	79.3 ± 11.3	71.1 ± 11.0	76.5 ± 12.4	68.9 ± 8.6
EDD-SL	Small	10	54.0 ± 9.7						61.2 ± 14.3
EDD-SL	Large	10	74.3 ± 13.0						73.0 ± 16.6
EDD/BSA-SL	Small	10	59.1 ± 12.7						66.4 ± 14.4
EDD/BSA-SL	Large	10	69.3 ± 16.5						67.9 ± 18.7
BSAChange-SL	Small	10	66.8 ± 16.8						63.5 ± 13.6
BSAChange-SL	Large	10	61.6 ± 13.9						70.7 ± 18.5
4.5y2m	Skiers	15			79.0 ± 7.8				71.6 ± 8.6
4.5y2m	Controls	10			61.7 ± 11.6				62.0 ± 14.5
			BTS1	BTS2	PCS				
Seasons		15	70.3 ± 14.0	70.7 ± 17.4	71.7 ± 17.8				

APPENDIX 11 Stroke volume (SV) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

SV		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	73.2 ± 11.9						98.5 ± 19.1
6.5y3m		14	74.5 ± 12.4			85.9 ± 15.3			96.5 ± 22.4
Annual		8	72.0 ± 12.6	90.6 ± 22.8	91.6 ± 13.6	90.5 ± 13.1	94.7 ± 21.0	91.8 ± 9.3	97.7 ± 19.3
EDD-SL	Small	10	68.5 ± 10.7						99.1 ± 7.1
EDD-SL	Large	10	77.9 ± 11.6						98.0 ± 26.8
EDD/BSA-SL	Small	10	67.4 ± 12.6						104.2 ± 17.9
EDD/BSA-SL	Large	10	79.0 ± 8.0						92.8 ± 19.5
BSAChange-SL	Small	10	74.5 ± 10.8						93.8 ± 18.4
BSAChange-SL	Large	10	71.9 ± 13.3						103.2 ± 19.5
4.5y2m	Skiers	15			82.7 ± 14.3				97.3 ± 23.5
4.5y2m	Controls	10			77.1 ± 22.5				93.2 ± 22.9
			BTS1	BTS2	PCS				
Seasons		15	88.8 ± 18.0	91.6 ± 14.7	92.2 ± 13.3				

APPENDIX 12 Left ventricular mass (LVM) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

LVM		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	158.9 ± 21.7						214.1 ± 36.1
6.5y3m		14	163.3 ± 22.1			183.7 ± 22.4			213.9 ± 33.7
Annual		8	171.1 ± 21.1	175.7 ± 23.4	195.3 ± 24.8	187.4 ± 25.7	205.0 ± 27.6	220.9 ± 31.6	221.4 ± 42.1
EDD-SL	Small	10	144.1 ± 13.3						207.3 ± 27.7
EDD-SL	Large	10	173.6 ± 18.4						220.9 ± 43.3
EDD/BSA-SL	Small	10	147.5 ± 15.1						216.9 ± 39.1
EDD/BSA-SL	Large	10	170.3 ± 21.9						211.3 ± 34.7
BSAChange-SL	Small	10	164.5 ± 27.3						204.0 ± 32.1
BSAChange-SL	Large	10	153.3 ± 13.7						224.4 ± 38.6
4.5y2m	Skiers	15			186.7 ± 19.4				223.2 ± 38.0
4.5y2m	Controls	10			141.1 ± 27.1				166.4 ± 27.4

			BTS1	BTS2	PCS				
Seasons		15	216.6 ± 29.1	220.8 ± 23.0	220.3 ± 32.8				

APPENDIX 13 Fractional shortening (FS) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 =basic training season 1 and 2, PCS=pre competition season during 5th year).

FS		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	28.0 ± 4.5						32.3 ± 5.1
6.5y3m		14	27.9 ± 5.2			28.4 ± 4.0			31.7 ± 5.0
Annual		8	24.8 ± 2.3	30.5 ± 5.1	29.0 ± 2.5	27.9 ± 2.7	30.3 ± 1.7	28.9 ± 3.4	31.4 ± 2.4
EDD-SL	Small	10	29.4 ± 4.8						34.1 ± 4.0
EDD-SL	Large	10	26.6 ± 3.9						30.6 ± 5.7
EDD/BSA-SL	Small	10	27.8 ± 4.8						33.5 ± 4.6
EDD/BSA-SL	Large	10	28.2 ± 4.4						31.2 ± 5.6
BSAChange-SL	Small	10	27.7 ± 3.0						32.3 ± 5.3
BSAChange-SL	Large	10	28.3 ± 5.6						32.4 ± 5.2
4.5y2m	Skiers	15			26.4 ± 3.3				30.5 ± 3.9
4.5y2m	Controls	10			29.1 ± 5.7				32.4 ± 3.6
			BTS1	BTS2	PCS				
Seasons		15	29.7 ± 4.9	30.4 ± 5.9	30.3 ± 4.5				

APPENDIX 14 Hypertrophy index (HI) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

HI		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	0.32 ± 0.02						0.33 ± 0.04
6.5y3m		14	0.31 ± 0.02			0.30 ± 0.03			0.33 ± 0.05
Annual		8	0.32 ± 0.03	0.31 ± 0.03	0.31 ± 0.04	0.30 ± 0.03	0.32 ± 0.05	0.34 ± 0.03	0.33 ± 0.04
EDD-SL	Small	10	0.33 ± 0.02						0.33 ± 0.04
EDD-SL	Large	10	0.30 ± 0.02						0.33 ± 0.04
EDD/BSA-SL	Small	10	0.32 ± 0.02						0.32 ± 0.02
EDD/BSA-SL	Large	10	0.31 ± 0.02						0.34 ± 0.05
BSAChange-SL	Small	10	0.32 ± 0.03						0.33 ± 0.04
BSAChange-SL	Large	10	0.32 ± 0.01						0.33 ± 0.04
4.5y2m	Skiers	15			0.30 ± 0.02				0.33 ± 0.04
4.5y2m	Controls	10			0.28 ± 0.02				0.29 ± 0.05
			BTS1	BTS2	PCS				
Seasons		15	0.34 ± 0.03	0.34 ± 0.04	0.34 ± 0.04				

APPENDIX 15 Wall stress index (WS) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S= small, L = large), 4.5y2m (4.5 years, 2 measurements, S= skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

WS		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	37.7 ± 5.2						42.7 ± 6.2
6.5y3m		14	37.9 ± 5.7			40.2 ± 6.3			43.4 ± 6.9
Annual		8	39.8 ± 5.8	38.6 ± 4.6	43.2 ± 6.2	39.8 ± 5.3	43.2 ± 5.7	45.2 ± 4.6	42.3 ± 3.8
EDD-SL	Small	10	40.0 ± 6.0						42.3 ± 7.0
EDD-SL	Large	10	35.4 ± 3.1						43.2 ± 5.7
EDD/BSA-SL	Small	10	38.7 ± 5.8						41.4 ± 3.4
EDD/BSA-SL	Large	10	36.7 ± 4.5						44.0 ± 8.1
BSAChange-SL	Small	10	39.3 ± 5.6						43.6 ± 5.4
BSAChange-SL	Large	10	36.2 ± 4.5						41.8 ± 7.1
4.5y2m	Skiers	15			35.9 ± 3.9				42.2 ± 5.5
4.5y2m	Controls	10			35.0 ± 5.7				39.1 ± 8.9
			BTS1	BTS2	PCS				
Seasons		15	45.8 ± 6.2	44.7 ± 7.2	44.0 ± 8.8				

APPENDIX 16 Index of contractility (IC) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

IC		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	2.00 ± 0.58						2.10 ± 0.59
6.5y3m		14	1.99 ± 0.64			1.88 ± 0.57			2.07 ± 0.57
Annual		8	1.67 ± 0.46	1.87 ± 0.40	1.80 ± 0.28	1.67 ± 0.33	1.96 ± 0.30	1.80 ± 0.32	1.94 ± 0.23
EDD-SL	Small	10	2.37 ± 0.53						2.32 ± 0.62
EDD-SL	Large	10	1.64 ± 0.35						1.89 ± 0.49
EDD/BSA-SL	Small	10	2.16 ± 0.50						2.13 ± 0.46
EDD/BSA-SL	Large	10	1.85 ± 0.63						2.08 ± 0.71
BSAChange-SL	Small	10	1.98 ± 0.59						2.18 ± 0.51
BSAChange-SL	Large	10	2.02 ± 0.59						2.03 ± 0.67
4.5y2m	Skiers	15			1.50 ± 0.22				1.86 ± 0.20
4.5y2m	Controls	10			2.04 ± 0.38				2.26 ± 0.65
			BTS1	BTS2	PCS				
Seasons		15	2.00 ± 0.52	2.06 ± 0.72	1.99 ± 0.70				

APPENDIX 17 Maximal oxygen uptake ($\dot{V}O_{2max}$, $l \cdot min^{-1}$) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

$l \cdot min^{-1}$		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	4.5 ± 0.4						5.1 ± 0.6
6.5y3m		14	4.4 ± 0.3			5.0 ± 0.6			5.1 ± 0.6
Annual		8	4.5 ± 0.5	4.8 ± 0.7	4.9 ± 0.8	5.0 ± 0.8	5.3 ± 0.8	5.2 ± 0.7	5.1 ± 0.8
EDD-SL	Small	10	4.4 ± 0.3						5.1 ± 0.5
EDD-SL	Large	10	4.6 ± 0.5						5.1 ± 0.8
EDD/BSA-SL	Small	10	4.6 ± 0.4						5.3 ± 0.6
EDD/BSA-SL	Large	10	4.5 ± 0.4						4.9 ± 0.6
BSAChange-SL	Small	10	4.5 ± 0.3						4.7 ± 0.5
BSAChange-SL	Large	10	4.6 ± 0.5						5.5 ± 0.5
4.5y2m	Skiers	15			5.1 ± 0.6				5.2 ± 0.7
4.5y2m	Controls	10			3.4 ± 0.4				3.8 ± 0.6
			BTS1	BTS2	PCS				
Seasons		15	5.1 ± 0.6	5.3 ± 0.5	5.3 ± 0.5				

APPENDIX 18 Maximal oxygen uptake ($\dot{V}O_{2max}$, $ml \cdot kg^{-1} \cdot min^{-1}$) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S = small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

$ml \cdot kg^{-1} \cdot min^{-1}$		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	68.2 ± 3.0						69.2 ± 6.2
6.5y3m		14	67.4 ± 3.1			67.7 ± 2.4			68.9 ± 5.6
Annual		8	67.0 ± 2.7	67.8 ± 3.6	66.5 ± 4.0	67.1 ± 1.9	71.5 ± 5.4	70.1 ± 4.9	67.9 ± 5.2
EDD-SL	Small	10	68.4 ± 3.5						70.9 ± 6.1
EDD-SL	Large	10	67.9 ± 2.4						67.6 ± 6.2
EDD/BSA-SL	Small	10	67.8 ± 3.2						69.6 ± 7.0
EDD/BSA-SL	Large	10	68.6 ± 2.8						68.9 ± 5.7
BSAChange-SL	Small	10	67.3 ± 2.6						66.4 ± 7.5
BSAChange-SL	Large	10	69.0 ± 3.2						72.0 ± 3.0
4.5y2m	Skiers	15			70.8 ± 1.8				69.5 ± 6.2
4.5y2m	Controls	10			52.7 ± 3.7				52.8 ± 7.0
			BTS1	BTS2	PCS				
Seasons		15	68.4 ± 4.3	72.9 ± 3.9	72.9 ± 3.5				

APPENDIX 19 Maximal oxygen uptake ($\dot{V}O_{2max}$, $ml \cdot kg^{-2/3} \cdot min^{-1}$) during the study. 6.5y2m (6.5 years, 2 measurements), 6.5y3m (6.5 years, 3 measurements), Annual (6.5 years, 7 measurements), EDD-SL (S = small, L = large), EDD/BSA-SL (S = small, L = large), BSAChange-SL (S= small, L = large), 4.5y2m (4.5 years, 2 measurements, S = skiers, C = controls), Seasons (BTS1, BTS2 = basic training season 1 and 2, PCS = pre competition season during 5th year).

$ml \cdot kg^{-2/3} \cdot min^{-1}$		N	Baseline	1.5 years	2.5 years	3.5 years	4.5 years	5.5 years	6.5 years
6.5y2m		20	275.6 ± 12.4						290.1 ± 26.8
6.5y3m		14	271.4 ± 12.0			283.1 ± 15.1			288.1 ± 24.6
Annual		8	272.6 ± 13.3	280.0 ± 21.5	278.6 ± 20.4	281.8 ± 18.3	300.1 ± 26.7	295.0 ± 23.6	285.1 ± 27.9
EDD-SL	Small	10	274.6 ± 13.3						294.3 ± 24.7
EDD-SL	Large	10	276.7 ± 12.2						285.8 ± 29.5
EDD/BSA-SL	Small	10	274.9 ± 10.7						286.0 ± 25.6
EDD/BSA-SL	Large	10	274.9 ± 10.7						286.0 ± 25.6
BSAChange-SL	Small	10	273.0 ± 11.4						275.7 ± 29.2
BSAChange-SL	Large	10	278.2 ± 13.5						304.5 ± 14.1
4.5y2m	Skiers	15			293.6 ± 14.3				292.1 ± 27.3
4.5y2m	Controls	10			211.7 ± 15.3				218.9 ± 30.2
			BTS1	BTS2	PCS				
Seasons		15	286.9 ± 21.8	304.2 ± 15.5	304.6 ± 14.9				

APPENDIX 20

DEVELOPMENT OF THE ATHLETE'S HEART DURING THREE YEAR'S TRAINING IN YOUNG CROSS-COUNTRY SKIERS

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This study aimed to investigate the development of the athlete's heart in 18 male cross-country skiers (15.5±0.7 yrs) during three years when their training volume increased gradually. Intermediate measurements were done after two years when a control group (n=15) was included in the study. Resting echocardiograms were recorded in left lateral position and exercise echocardiograms during semisupine bicycle exercise at 100 W. ANOVA showed increases in VO_{2max} ($p<0.001$) and in resting EDD ($p<0.001$), ESD ($p<0.001$) and MWTd ($p<0.001$). Greatest changes occured during the first two years in all variables, e.g. EDD increased from 5.41±0.29 cm to 5.74±0.35 cm after two years and to 5.77±0.35 cm after three years, and MWTd from 0.83±0.03 cm to 0.88±0.04 cm and to 0.86±0.04 cm respectively. Exercise EDD increased similarly from 5.41±0.36 cm to 5.79±0.33 cm and to 5.85±0.26 cm ($p<0.001$). Exercise wall stress also increased from 43.0±3.6 mmHg to 46.7±4.6 mmHg and to 46.5±4.3 mmHg ($p<0.05$) respectively. The control group had significantly smaller EDD and MWTd at rest and during exercise than skiers both after two and three years. In skiers the correlation between VO_{2max} and EDD at rest increased from r=.45 ($p<0.05$) to r=.52($p<0.01$) after two years and to r=.62 ($p<0.01$) after three years. These longitudinal results suggest that the development of the athlete's heart may include both dilatation and wall thickness changes.

APPENDIX 21

ECHOCARDIOGRAPHIC CHANGES DURING TWO SKIING SEASONS IN YOUNG CROSS-COUNTRY SKIERS

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The purpose of this study was to investigate the differences and changes in echocardiographic dimensions during two skiing seasons in young (17.0 ± 0.8 yrs) Finnish male cross-country skiers (n=17). During two successive years the first measurements were done in the autumn (A1 and A2) after dry land training seasons and the second measurements in the winter (W1 and W2) at the end of cross-country skiing training and competition seasons. Resting echocardiograms were recorded in left lateral position and training data were calculated from training records of the skiers. During 12 weeks preceding A1 and A2 the volume of training was similar during both years (10.5 ± 2.6 vs. 10.9 ± 8.0 hours per week, respectively). During the skiing training and competition season (12 weeks) the volume of training was significantly higher during the second year (5.9 ± 1.6 vs 8.1 ± 2.8 hours per week, $p<0.01$, respectively). The volume of training decreased from A1 to W1 ($p<0.001$) but the difference between A2 and W2 was not significant. During the first year end-diastolic diameter (EDD) and end-systolic diameter (ESD) increased from A1 to W1: EDD from 5.57 ± 0.32 cm to 5.73 ± 0.36 cm ($p<0.001$) and ESD from 3.91 ± 0.23 cm to 4.21 ± 0.21 cm ($p<0.001$). During the second year EDD increased from 5.74 ± 0.36 cm to 5.80 ± 0.34 cm ($p<0.05$) and ESD from 4.19 ± 0.21 cm to 4.29 ± 0.17 cm ($p<0.05$). There was also a difference in EDD between A1 and A2 ($p<0.05$) and W1 and W2 ($p<0.05$) and in ESD between A1 and A2 ($p<0.001$). EDD/BSA ($p<0.01$) and ESD/BSA ($p<0.001$) increased from A1 to W1 and ESD/BSA was also greater in the winter than in the autumn during the second year ($p<0.05$). Mean wall thickness increased from A1 to W1 (0.86 ± 0.06 cm vs. 0.89 ± 0.05 cm, $p<0.01$, respectively) but decreased from A2 to W2 (0.90 ± 0.05 vs. 0.86 ± 0.05, $p<0.05$, respectively). Wall stress was unchanged during the first year but decreased during the second skiing season ($p<0.001$). Fractional shortening was significantly ($p<0.001$) smaller in W1 than in A1. It is concluded that cross-country skiing training and competition season may increase the dimensions of the heart despite of the same or even smaller volume of training compared with the preceding dry land training season. The higher volume of skiing training during the second year further increased the end-diastolic diameter of the heart. It is suggested that combined arm and leg exercise during skiing together with intensive uphill and downhill skiing during competitions explain the echocardiographic changes.

APPENDIX 22

THE INFLUENCE OF THE INITIAL HEART SIZE ON THE DEVELOPMENT OF THE ATHLETE'S HEART

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The purpose of this study was to analyze the influence of the initial heart size on the development of the athlete's heart. Young male cross-country skiers (n=21, age 15.7 yrs, height 177.7 cm, weight 65.5 kg) were divided into two groups having SMALL (<5.40 cm, n=11) or LARGE (□5.40 cm, n=10) end-diastolic diameter (EDD) in the beginning of the study. Resting echocardiograms were recorded in left lateral position also after two and four years. The groups didn't differ from each other in BSA, maximum oxygen uptake or training volume during the study. ANOVA showed a statistically significant ($p<0.001$) difference in EDD between the groups and between the measurements. EDD increased as much in both groups; in SMALL from 5.16± 0.13 cm to 5.52±0.30 cm after two years and to 5.56±0.16 cm after four years and in LARGE from 5.67±0.34 cm to 5.91±0.34 cm and to 5.99±0.49 cm respectively. Mean wall thickness (MWT) increased significantly ($p<0.01$) and similarly in both groups; from 0.83±0.05 cm to 0.88±0.06 cm and to 0.90±0.07 cm in SMALL and from 0.87±0.04 cm to 0.89±0.06 cm and to 0.93±0.09 cm in LARGE. End-systolic diameter (ESD) was smaller in SMALL than in LARGE and there were differences between the groups ($p<0.05$). In conclusion, the initial heart size didn't influence the development of the heart and the initial differences in EDD remained the same after four years.

APPENDIX 23

DEVELOPMENT OF THE ATHLETE'S HEART DURING FIVE YEARS' TRAINING IN YOUNG ATHLETES

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The purpose of this study was to investigate the development of the athlete's heart. Young male cross-country skiers (n=9, 16.1 ± 0.4 years) and a control group (n=9, 16.9 ± 0.4 years) were measured three times during five years. Resting echocardiograms were recorded in left lateral position and exercise echocardiograms during semisupine bicycle exercise at 50 W. The results showed differences between the groups at rest in end-diastolic diameter (EDD) ($p<0.05$), mean wall thickness (MWT) ($p<0.001$), total diameter (TD) ($p<0.001$), TD in exercise ($p<0.05$) and VO_{2max} ($p<0.001$) and between the measurements in MWT at rest ($p<0.001$) and in TD in exercise ($p<0.05$). TD at rest increased both in the skiers (from 7.34 ± 0.50 cm to 7.80 ± 0.49 cm, $p<0.01$) and in the control group (from 6.84 $\pm$ 0.48 cm to 7.17 $\pm$ 0.54 cm, $p<0.05$) during five years and the difference between the groups was greatest at the end of the study ($p<0.01$). The increase in resting MWT was more significant in the skiers (from 0.86 ± 0.06 cm to 0.97 ± 0.07 cm, $p<0.001$) than in the control group (from 0.75 $\pm$ 0.06 cm to 0.83 $\pm$ 0.05 cm, $p<0.05$). At the age of 21 the echocardiographic dimensions in the control subjects almost reached the initial values of the skiers. It is concluded that dilatation and wall thickness changes may occur after the puberty both in the control subjects and in the athletes.

APPENDIX 24

MAXIMUM OXYGEN UPTAKE AND THE DEVELOPMENT OF THE ATHLETE'S HEART

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The purpose of this study was to analyze the influence of the maximum oxygen uptake (VO_{2max}) on the development of the athlete's heart. Young (age 15.6±0.7 yrs) male cross-country skiers were divided into two groups having high (H) (≥ 68 $ml \cdot kg^{-1} \cdot min^{-1}$, n=8) or low (L) (≤ 67 $ml \cdot kg^{-1} \cdot min^{-1}$, n=7) VO_{2max} in the beginning of the study. VO_{2max} was measured on the treadmill and resting echocardiograms were recorded in left lateral position in the beginning of the study and after three and five years. No differences were observed in the training of the groups during the study. MANOVA showed significant group difference ($p<0.05$) and interaction ($p<0.05$) in VO_{2max} and significant training effect in left ventricular end-diastolic diameter (EDD) ($p<0.01$) and in left ventricular mean wall thickness (MWT) ($p<0.001$): (mean ± SD)

		0 year	3 years	5 years
VO_{2max}	H	70.8±2.0	68.5±2.6	71.4±2.6
($ml \cdot kg^{-1} \cdot min^{-1}$)	L	65.0±1.4	67.0±2.2	70.3±5.9
EDD	H	5.44±0.34	5.80±0.38	5.84±0.45
(cm)	L	5.34±0.33	5.59±0.35	5.74±0.34
MWT	H	0.85±0.04	0.87±0.06	0.98±0.08
(cm)	L	0.83±0.05	0.85±0.03	0.93±0.08

Training effect was also observed in total diameter, left ventricular mass, fractional shortening, hypertrophic index, end-diastolic volume and stroke volume ($p<0.001$-0.01). No correlation was observed between VO_{2max} and echocardiographic variables. In conclusion, the echocardiographic dimensions and the changes in the dimensions were similar in athletes having high or low initial maximum oxygen uptake.

APPENDIX 25

TRAINING AND DETRAINING EFFECTS ON THE ATHLETE'S HEART DURING FIVE YEARS' ECHOCARDIOGRAPHIC STUDY

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The purpose of this study was to investigate the development of the athlete's heart in 30 male (16.6±1.2 y.) cross-country skiers during five years. During the period 18 skiers (S) increased their training volume while 12 skiers (D) finished or decreased their training volume 2-3 years after the beginning of the study. A control group (C) consisted of 11 physically active non-athletes (16.9±0.4 y.). MANOVA showed group difference ($p<0.001$) and interaction ($p<0.01$) in maximum oxygen uptake (S: from 281±19 to 286±22 $ml \cdot min^{-1} \cdot kg^{-2/3}$, D: from 279±17 to 256±20 $ml \cdot min^{-1} \cdot kg^{-2/3}$, C: from 216±20 to 222±30 $ml \cdot min^{-1} \cdot kg^{-2/3}$). Left ventricular end-diastolic diameter (EDD), mean wall thickness (MWT) and left ventricular mass (LVM) were calculated from resting echocardiograms recorded in left lateral position in the beginning of the study and after five years.

	EDD (cm)	(a)	MWT (mm)	(a, b)	LVM (g)	(a, b, c)
	pre	post	pre	post	pre	post
S	5.4±0.4	5.8±0.4	84±0.4	95±1.1	209±32	281±48
D	5.4±0.6	5.7±0.5	84±0.7	91±0.9	208±47	250±53
C	5.3±0.4	5.6±0.5	75±0.5	81±0.8	178±33	210±33

a=change ($p<0.001$), b=group difference ($p<0.01$), c=interaction ($p<0.05$)
We concluded that the effects of progressively increased training volume were demonstrated in maximal oxygen uptake and in LVM. Detraining induced minor changes in heart dimensions and LVM.

APPENDIX 26

DEVELOPMENT OF THE ATHLETE'S HEART DURING SIX YEARS' ECHOCARDIOGRAPHIC STUDY

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The purpose of this study was to investigate the development of the athlete's heart in male cross-country skiers (15.6 ± 0.4 years, n = 13) during six years. Intermediate measurements were done after two and four years. After two years a control group (16.9 ± 0.2 years, n = 7) was included in the study. Resting echocardiograms were recorded in left lateral position and echocardiograms were digitized according to the recommendations of the American Society of Echocardiography. Maximal oxygen uptake (VO_{2max}) was measured on the treadmill. After two, four and six years' training the skiers demonstrated changes in left ventricular end-diastolic diameter (EDD) from 5.33 ± 0.31 cm to 5.65 ± 0.42 cm to 5.75 ± 0.45 cm to 5.80 ± 0.48 cm (ANOVA, $p<0.01$), mean wall thickness (MWT) from 0.85 ± 0.04 cm to 0.88 ± 0.06 cm to 0.92 ± 0.08 cm to 0.99 ± 0.08 cm ($p<0.01$), left ventricular mass (LVM) from 206.8 ± 28.1 g to 240.7 ± 29.1 g to 261.1 ± 40.1 g to 291.0 ± 44.8 g ($p<0.001$), stroke volume (SV) from 118.1 ± 17.2 ml to 138.5 ± 22.7 ml to 148.8 ± 29.7 ml to 154.7 ± 35.4 ml ($p<0.01$) and VO_{2max} from 4.5 ± 0.5 $l \cdot min^{-1}$ to 5.0 ± 0.6 $l \cdot min^{-1}$ to 5.4 ± 0.5 $l \cdot min^{-1}$ to 5.3 ± 0.7 $l \cdot min^{-1}$, respectively. During the last four years MANOVA showed differences between the measurements and the groups in MWT ($p<0.001$), LVM ($p<0.001$) and VO_{2max} ($l \cdot min^{-1}$) ($p<0.001$) and between the measurements in SV ($p<0.01$). No significant interactions were found although echocardiographic parameters did not change in the control group between 19 and 21 years of age. In the skiers EDD correlated with VO_{2max} ($l \cdot min^{-1}$) in the beginning of the study ($r = .60$, $p<0.05$), after four years ($r = .72$, $p<0.01$) and at the end of the study ($r = .76$, $p<0.01$). SV correlated with VO_{2max} ($l \cdot min^{-1}$) ($r = .66$ - $.79$, $p<0.05$ - 0.001) in every measurement. This longitudinal study showed that endurance training for six years resulted in the athlete's heart in male cross-country skiers and the development of the athlete's heart included dilatation, wall thickness changes and functional changes.

APPENDIX 27

INFUENCE OF ENDURANCE TRAINING FOR SIX YEARS ON THE HEART SIZE AND MAXIMAL OXYGEN UPTAKE OF YOUNG ENDURANCE ATHLETES

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INTRODUCTION

Maximal oxygen uptake (VO_{2max}) of young elite cross-country skiers increases with age and training by 1-3 $ml \cdot kg^{-1} \cdot min^{-1}$ per year (Rusko 1992). Heart size and end-diastolic diameter of left ventricle (EDD) increase with endurance training, and wall thickness may also be increased (Keul et al. 1982, Pelliccia et al. 1991, Rusko 1992). The purpose of this study was to examine the changes in heart size and function and in VO_{2max} of young athletes during endurance training for six years.

METHODS

Fifteen national level male cross-country skiers (age 15.7±0.6 years) participated in this 6-year study. At the beginning and at the end of the study resting echocardiograms were recorded in left lateral position. VO_{2max} was determined on a treadmill. On the 5th year, measurements were done during three training seasons: basic training season 1 and 2 (BTS1, BTS2) and pre competition season (PCS).

RESULTS

VO_{2max} increased significantly during the 6-year period and also between the seasons. EDD, septal (SWTd) and posterior (PWTd) wall thickness in diastole, left ventricular mass (LVM) and stroke volume (SV) increased during six years ($p<0.001$) but there were no statistical differences between different seasons (Table 1).

Table 1. Results of VO_{2max} and echocardiographic variables (* $p<0.05$, ** $p<0.01$, *** $p<0.001$).

	0. year	6. year	t-test	BTS1	BTS2	PCS	Anova
l/min	4.5±0.4	5.2±0.4	***	5.1±0.6	5.3±0.5	5.3±0.5	**
ml/kg/min	68.6±2.9	71.6±4.8	*	68.4±4.3	72.9±3.9	72.9±3.5	***
EDD (cm)	5.33±0.31	5.81±0.45	***	5.72±0.38	5.77±0.34	5.78±0.39	n.s.
SWTd (cm)	0.85±0.05	1.00±0.10	***	0.97±0.07	0.99±0.08	0.99±0.08	n.s.
PWTd (cm)	0.83±0.05	0.94±0.09	***	0.96±0.08	0.95±0.10	0.94±0.10	n.s.
LVM (g)	161.1±22.1	225.5±34.3	***	216.6±29.1	220.8±23.0	220.3±32.8	n.s.
SV (ml)	73.4±10.3	95.5±16.5	***	88.8±18.0	91.6±14.7	92.2±13.3	n.s.

DISCUSSION

The results confirm the findings of cross-sectional studies that both structural and functional changes occur in the heart with age and training. The minor changes in echocardiographic variables during the 5th and 6th year suggest that the upper limit of the development of the heart size was approaching. However, the changes in VO_{2max} during the 5th year's training seasons suggest that functional changes during maximal exercise can still occur.

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APPENDIX 28

Athlete's Heart and Maximal Oxygen Uptake of Young Endurance Athletes: Annual Changes during Six Years

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Introduction

Heart size and left ventricular end-diastolic diameter (EDD) increase with endurance training, and elite endurance athletes may also have thickened walls of left ventricle (Henriksen et al. 1997, Pelliccia et al. 1991). Maximal oxygen uptake (VO_{2max}) increases with age and training 1-3 ml · kg^{-1} · min^{-1} per year in 15-20 years elite cross-country skiers and up to 22 years of age in international level skiers if training volume and intensity increases (Rusko 1992). The purpose of this study was to examine the longitudinal changes in the heart size and VO_{2max} of young endurance athletes during six years.

Methods

Eight national level male cross-country skiers (age16.0 ± 0.9 years) participated in this 6-year study. Resting echocardiograms were recorded in left lateral position annually at the end of the basic training period of cross-country skiers and analysed according to the recommendations of the American Society of Echocardiography. VO_{2max} was also determined annually during progressive ski walking test with ski poles until exhaustion on a treadmill. Training data of the skiers were calculated from training records. Friedman test was used as the non-parametric test of repeated measurements.

Results

VO_{2max} increased during six years: from 4.5 ± 0.5 to 5.1 ± 0.8 l ·min^{-1} ($p<0.01$), from 67.0 ± 2.7 to 67.9 ± 5.2 ml ·kg^{-1} ·min^{-1} ($p<0.05$) and from 272.6 ± 13.3 to 285.1 ± 27.9 ml ·$kg^{-2/3}$ · min^{-1} ($p<0.01$). Training volume ($p<0.05$) and training hours ($p<0.01$) increased during the study. EDD tended to increase (n.s.) during six years, septal wall thickness in diastole (SWTd) increased ($p<0.001$) and posterior wall thickness in diastole (PWTd) increased ($p<0.05$) (Table 1). Left ventricular mass grew from 171.1 ± 24.1 g to 187.4 ± 25.7 g after three years and to 221.4 ± 42.1 g ($p<0.001$) after six years and stroke volume from 72.0 ± 12.6 ml to 90.5 ± 13.1 g and to 97.7 ± 19.3 ml ($p<0.05$), correspondingly.

Table 1. EDD, SWTd and PWTd during the study. Mean ± SD.

	0. year	1. year	2. year	3. year	4. year	5. year	6. year	Non-par test
EDD (cm)	5.45 ± 0.39	5.56 ± 0.40	5.72 ± 0.48	5.77 ± 0.45	5.76 ± 0.47	5.78 ± 0.38	5.83 ± 0.49	n.s.
SWTd (cm)	0.86 ± 0.06	0.85 ± 0.08	0.88 ± 0.06	0.84 ± 0.06	0.93 ± 0.11	0.97 ± 0.07	0.98 ± 0.10	p <0.001
PWTd (cm)	0.85 ± 0.04	0.85 ± 0.04	0.90 ± 0.06	0.86 ± 0.04	0.90 ± 0.08	0.96 ± 0.08	0.93 ± 0.07	p <0.05

Discussion

Annual results showed that the first step in the development of the heart in young endurance athletes was a dilatation of the left ventricle, which happened at the age of 18 - 19 years, and during the last three years EDD was unchanged. During the first three years the walls were unchanged but during the second step at the age of 20 - 22 years, the walls thickened. The results of VO_{2max} were at its highest after four years and after that VO_{2max} began to decrease. Because there was no dilatation during the last three years and body surface area was also unchanged we concluded that training volume and training intensity were too low to increase dilatation of the heart.

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