Aerobic Capacity of Competitive Ice Hockey Players 10-15 Years Old

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Abstract Oxygen uptakes ($\dot{V}_{O_2}$) recorded at anaerobic threshold and at the end of a maximal exercise ($\dot{V}_{O_{2max}}$) and their relation to left ventricular function were analyzed in 11 young ice hockey players during an incremental exercise on a bicycle ergometer. The children, highly trained, participated annually during 6 years (from the age of 10-15 years) in laboratory tests. The maturative status of the subjects was evaluated from peak height velocity (PHV). Heart rate was recorded by electrocardiogram. Oxygen uptake, CO_2 production, respiratory frequency, pulmonary ventilation ($\dot{V}_E$) were recorded at rest and every 30 s during exercise through a Rudolph valve connected to a calibrated oxycon gas analyser. The anaerobic threshold was determined by a non-invasive method from pulmonary ventilation curves. Left ventricular volumes at end-systole and end-diastole were obtained, at rest, by M mode echocardiography. Results showed that both $\dot{V}_{O_2}$ at anaerobic threshold and $\dot{V}_{O_{2max}}$ were positively correlated with body mass or with age of PHV. The increments were constant from year to year. At anaerobic threshold, the ratio $\dot{V}_{O_2}/\dot{V}_{O_{2max}}$ was independent of maturative age. Similar findings were observed when considering $\dot{V}_E$ except after the years of PHV where there was a remarkable increase in pulmonary ventilation. The results indicate that the growth of each cardiorespiratory component is optimized with body size increase in order to keep constant the aerobic response to exercise. As judged by the explained variance of the different linear regression analyses between resting left ventricular dimensions and $\dot{V}_{O_2}$, cardiac volume was of minimal importance in determining $\dot{V}_{O_2}$. In the postpubertal period, stroke volume accounted for 26.7% of $\dot{V}_{O_2}$ at anaerobic threshold and 30.0% of $\dot{V}_{O_{2max}}$. This suggests that local changes occurring at muscular level are of paramount importance in determining the aerobic capacity of highly trained boys.

Received on January 28, 1994; Accepted on May 9, 1994

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Key words: longitudinal study, anaerobic threshold, boys, maximum oxygen uptake, stroke volume.

Aerobic power partly depends on gas transport capacity and on cardiac size and function.

Changes in maximal oxygen uptake ($\dot{V}_{O_{2\max}}$) are frequently used to assess the effects of growth on the performance and the development of the gas transport system in children. Most studies of the interaction between exercise and growth in children have dealt with $\dot{V}_{O_{2\max}}$ changes although this parameter only indicates the maximal gas transport capacity. Kobayashi *et al.* [1] reported that the largest increase in $\dot{V}_{O_{2\max}}$ was associated with the period of most rapid growth. However, other studies [2, 3] did not find any change in $\dot{V}_{O_{2\max}}$. Likewise, Cooper *et al.* [3] and Paterson *et al.* [4] demonstrated that anaerobic threshold, an important indicator of performance, was not related to the period of greatest growth. The anaerobic threshold indicating the upper limit of aerobic metabolism is sensitive to exercise training [5–7]. In adults, Davis *et al.* [5] reported that after 9 weeks of endurance training, the anaerobic threshold increased of 44% while $\dot{V}_{O_{2\max}}$ only increased 15%. This threshold can be determined by a non-invasive method taking into account the different response patterns of the pulmonary ventilation and the analysis of expired flow gases [8, 9]. This method, inducing minimal discomfort, can be easily used with children.

The functional capacity of gas transport also depends on the cardiac output. Except for a few experiments [2, 10, 11], little attention has been given to changes in cardiac output during longitudinal design over a wide range of ages. Cunningham *et al.* [2] reported that, in exercising boys, stroke volume reflected $\dot{V}_{O_2}$ increase at all maturative ages except during the period of growth spurt. Cardiac output is generally assessed by the CO₂ rebreathing method. To our knowledge, only a few studies have evaluated the cardiac development of children using echocardiography even though this technique permits accurate measurements of cardiac structures, particularly separate calculation of ventricular volumes at end-systole and end-diastole providing an approach of the cardiac function through Franck Starling's law. Blinkie *et al.* [11], measuring the internal dimensions of the left ventricle of untrained boys at rest by M mode echocardiography, showed that the cardiac dimensions did not play a major role in determining maximal gas transport capacity when the subjects ran on a treadmill.

The purpose of the present study was to examine aerobic capacity of highly trained children during a longitudinal design (boys 10–15 years) in connection with cardiac function. Respiratory parameters have been recorded at anaerobic threshold and at the end of a maximal exercise performed on a bicycle ergometer. In addition, left ventricular parameters have been measured by the M echocardiographic method to assess the contribution of selected cardiac dimensions to $\dot{V}_{O_2}$ changes.

METHODS

Experimental design. Eleven boys, highly motivated, participated annually over a period of 6 years (from the age of 10 to 15 years) in laboratory tests to determine their physical aptitude. All children (Table 1) were trained ice hockey players. They were all members of the same team. Their training programs included at least 2 h/session, four times a week. This study was approved by Ethical Commission.

For the standardization of maturative status among subjects, pre- and post-pubertal periods were determined from peak height velocity (PHV) [1, 12]. For PHV determination, the body height was plotted against chronological age for each subject. The midpoint of the interval with maximum positive slope was defined as the age of PHV. This determination was completed by clinical observations.

Anaerobic threshold (AT) as defined Kindermann *et al.* [9] was determined by a non-invasive method. The ventilatory threshold point was automatically detected from linear regressions fitted on to pulmonary ventilation curves by a least square mathematical method and can be identified by the second inflexion point of non-linear increases in the curves of $\dot{V}_E$. The disruption points of $\dot{V}_{CO_2}$, respiratory exchange ratio, equivalents O_2 and CO_2 were also taken into account.

All subjects performed an incremental exercise on a bicycle ergometer (Rey-Thomas Gauthier) with the pedalling frequency fixed at 60 rpm. After fifteen minutes of resting, the work load was increased by 30 W every minute. Exercise was discontinued on exhaustion. Verbal encouragements were given towards the end of maximal exercise. The cycle was constructed in such a way that the rate of work was independent of the cycling frequency. The subjects were wearing shorts, T-shirt and tennis shoes. All exercise tests were performed in the morning and 2 h after a light breakfast in the laboratory (air temperature between 19 and 22°C, relative humidity = 40–60%).

The following physiological measures were monitored at 30-s intervals during exercise. Heart rate from electrocardiogram was continuously recorded on a cardioscope (ITS 06-CGR). Oxygen uptake ($\dot{V}_{O_2}$), CO_2 production ($\dot{V}_{CO_2}$), pulmonary ventilation ($\dot{V}_E$), and respiratory frequency were measured with the subjects breathing through a Rudolph valve connected to a calibrated oxycon IV (Mijnhart-MSR, The Netherlands). Analysers were calibrated with known concentrations of

Table 1. Physical characteristics of subjects.

	Age (year)					
	10	11	12	13	14	15
Wt (kg)	32.9±5.9	36.7±6.8	39.3±8.0	43.7±8.4	50.3±8.8	59.8±9.3
Ht (m)	1.39±0.06	1.45±0.06	1.48±0.07	1.58±0.09	1.64±0.08	1.70±0.08
AD (m ²)	1.13±0.15	1.22±0.16	1.28±0.17	1.41±0.10	1.53±0.16	1.69±0.06

Values are means ± SD. Wt, body mass; Ht, height; AD, body surface area.

gas mixture before each run. Tidal volume, respiratory exchange ratio, equivalent O_2 , equivalent CO_2 were calculated from an on-line computer.

M mode echocardiography was obtained before exercise using a phased-array ultrasonograph (HP 77020) with a 3.5–5 MHz transducer. All measurements were made with the subjects in the dorsal decubitus position. Concomitant electrocardiogram recordings allowed to exclude cardiac cycles with arrhythmias. Left ventricular end-diastolic and end-systolic diameters were measured from standard views according to the recommendations of the American Society of Echocardiography. Because of technical reasons and the quality of projection views, analyses were performed on data obtained from 29 and 17 examinations in pre- and post-pubertal periods, respectively. The views were thus selected to avoid errors and to have good accuracy of information.

Left ventricular volume at end-diastole (LVVD) and end-systole (LVVS) were derived from the formula of Teichholz *et al.* [13]. Stroke volume (SV) was calculated by difference between LVVD and LVVS.

The left ventricular ejection fraction was calculated (SV/LVVD). This is an index of left ventricular performance describing changes in myocardial contractility.

Posterior wall thickness was measured at end-diastole, as the distance between the posterior wall endocardial surface and the pericardial space.

Septal thickness was measured, at end-diastole, as the distance between right and left endocardial surface of the interventricular septal wall.

These measures were representative of the changes of the left ventricular mass.

Calculations and statistical analyses. Linear regressions were computed to assess the relative importance of body mass and of maturative ages (relative to PHV) to the changes in aerobic power.

Allometric equations between energy metabolism ($\dot{V}_{O_{2AT}}$; $\dot{V}_{O_{2max}}$) and body mass (Wt) have been calculated:

$$\dot{V}_{O_{2AT}}(\text{or } \dot{V}_{O_{2max}}) = \alpha Wt^b.$$

As reported by Heusner [14], the mass component (scaling factor b) reflects the energetic cost induced by morphological and structure adjustments at the cellular level. For mature organisms which tend to remain homomorphic, b is equal to 2/3. During the phase of growth involving organizational changes in the organism, the mass component becomes greater than 2/3.

The significance of the regression equations (slope value against 0) was tested by analysis of variance. The degrees of freedom of F -values were 1 and $n-2$ (n = number of observations). The explained variance of each regression equation (r^2) was expressed in percent.

t -tests were used to compare difference between slope values of the regression equations.

$p < 0.05$ was accepted as significant level. When regression equations were not significant, the values were means $\pm$ SD.

RESULTS

The mean age for maximal peak height velocity was 12.8 ± 0.5 years and its instantaneous value was $9.3 \pm 3.0 \text{ cm year}^{-1}$.

Maximal exercise

The relationships between $\dot{V}_{O_2}$ and $\dot{V}_E$ recorded at the end of maximal exercise and body mass are shown in Fig. 1. Individual data are also plotted against the age of peak height velocity (PHV). Data obtained from the different linear regressions are presented in Table 2.

$\dot{V}_{O_2 \text{ max}}$ was closely correlated with body mass in pre- ($F_{1,34}=37.91$; $p<0.001$) and postpubescent boys ($F_{1,28}=71.13$; $p<0.001$). The relationships did not differ between the two groups of children ($p>0.50$). For pooled data the relationship between $\dot{V}_{O_2 \text{ max}}$ and body mass explained 81% of the observed variance; the slope value was $0.054/\text{min}^{-1} \text{ kg}^{-1}$.

The body mass scaling factors for $\dot{V}_{O_2 \text{ max}}$ are reported in Table 3. For the

Fig. 1. Oxygen uptake ($\dot{V}_{O_2 \text{ max}}$) and pulmonary ventilation ($\dot{V}_E \text{ max}$) recorded at the end of the maximal exercise as a function of body mass (Wt) and of age of peak height velocity (PHV). Open (○) and full (●) circles indicated pre- and postpubertal data, respectively.

Table 2. Linear regression analyses between $\dot{V}_{O_{2\max}}$, $\dot{V}_{O_{2AT}}$ ($l \min^{-1}$), $\dot{V}_{E\max}$ and $\dot{V}_{EAT}$ ($l \min^{-1}$) and body mass (Wt, kg) and maturative ages relative to peak height velocity (PHV) in the pre- and postpubertal periods.

The explained variance r^2 is expressed in %.

		Prepubertal		Postpubertal		All data	
		Wt	PHV	Wt	PHV	Wt	PHV
$\dot{V}_{O_{2\max}}$	α	0.044	0.309	0.050	0.433	0.054	0.266
	Y intercept	0.546	2.825	0.491	2.346	0.241	2.701
	r^2	52.7	49.6	71.8	32.1	0.809	66.9
$\dot{V}_{E\max}$	α	1.205	10.530	1.455	18.420	1.908	10.610
	Y intercept	21.725	88.230	29.496	71.390	0.508	87.410
	r^2	30.6	45.6	60.4	60.8	72.6	78.9
$\dot{V}_{O_{2AT}}$	α	0.036	0.247	0.041	0.295	0.044	0.207
	Y intercept	0.396	2.243	0.317	1.946	0.159	2.138
	r^2	43.7	39.4	73.6	23.6	78.2	61.7
$\dot{V}_{EAT}$	α	0.676	5.367	0.949	8.481	0.986	5.109
	Y intercept	19.599	55.730	12.636	47.690	9.407	54.680
	r^2	19.3	23.8	54.3	31.1	60.9	57.3

Table 3. Mass coefficients (α) and scaling factors (b) as a function of body mass in prepubertal, postpubertal and all children.

Group		α	b	r^2
$\dot{V}_{O_{2AT}}$	Prepubertal	0.0933	0.807	41.1
	Postpubertal	0.0744	0.885	71.6
	All children	0.0586	0.941	73.6
$\dot{V}_{O_{2\max}}$	Prepubertal	0.1250	0.791	51.0
	Postpubertal	0.1154	0.833	69.9
	All children	0.0809	0.918	78.3

$\dot{V}_{O_{2AT}}$, oxygen uptake at anaerobic threshold; $\dot{V}_{O_{2\max}}$, maximum oxygen uptake. All regression equations are highly significant ($p < 0.001$). The explained variance (r^2) was expressed in %

whole group, the scaling factor was 0.92 (0.79 and 0.83 before and after puberty, respectively).

$\dot{V}_E$ increased significantly ($p < 0.001$) with body mass in the pre- ($F_{1,34} = 14.99$) and postpubertal periods ($F_{1,28} = 42.67$). These relationships were independent of the maturative status. For pooled data, the slope value was $1.908 l \min^{-1} kg^{-1}$.

In both periods, there was no significant relationship between body mass and the maximal values of heart rate or respiratory frequency. The maximal heart rate value was of 196.2 ± 6.1 beat $\min^{-1}$ before puberty and 192.9 ± 8.9 beat $\min^{-1}$ after. For respiratory rates, the values were of 53.8 ± 11.2 and 56.6 ± 9.9 breath $\min^{-1}$

before and after puberty, respectively.

The increases in $\dot{V}_{O_2}$ at maturative relative to PHV showed a pattern similar to that obtained with body mass. There was a significant increase ($p \leq 0.001$) in $\dot{V}_{O_2\max}$ across maturative ages reaching $0.309 \text{ ml min}^{-1} \text{ years}^{-1}$ before PHV ($F_{1,34}=33.48$) and $0.433 \text{ ml min}^{-1} \text{ years}^{-1}$ after ($F_{1,28}=13.21$). The slope difference was not significant.

$\dot{V}_{E\max}$ increases ($p < 0.001$) in pre- ($F_{1,34}=28.46$) and postpubertal period ($F_{1,28}=43.39$). This increase was accentuated during the period following PHV ($t_{64}=8.70$; $p < 0.001$).

Anaerobic threshold

$\dot{V}_{O_2\text{AT}}$ increased significantly ($p < 0.001$) with body mass in pre- ($F_{1,34}=26.38$) and postpubescent boys ($F_{1,28}=78.22$) (Fig. 2). The slopes of the regression equations (Table 2) did not differ between the two periods.

When all data were grouped, the slope was $0.044 \text{ l min}^{-1} \text{ kg}^{-1}$. The slopes of the relationships $\dot{V}_{O_2\text{AT}}$ vs body mass were not significantly different from those obtained from $\dot{V}_{O_2\max}$ data.

Fig. 2. Oxygen uptake ($\dot{V}_{O_2\text{AT}}$) and pulmonary ventilation ($\dot{V}_{E\text{AT}}$) recorded at the anaerobic threshold as a function of body mass (Wt) and of age of peak height velocity (PHV). Open (○) and full (●) circles indicated pre- and postpubertal data, respectively.

Fig. 3. Ratio between oxygen uptake ($\dot{V}_{O_{2AT}}/\dot{V}_{O_{2max}}$), pulmonary ventilation ($\dot{V}_{E_{AT}}/\dot{V}_{E_{max}}$) at anaerobic threshold and maximal values measured at the end of exercise as a function of body mass and of age of peak height velocity (PHV). Open (○) and full (●) circles indicated pre- and postpubertal data, respectively.

The scaling factors for $\dot{V}_{O_{2AT}}$ (Table 3) for the group as a whole was 0.94 (0.81 and 0.88 before and after puberty, respectively).

$\dot{V}_{O_{2AT}}/\dot{V}_{O_{2max}}$ ratio was independent of body mass (Fig. 3). The mean value of the ratio reached $79.3 \pm 5.1\%$ (79.1 ± 5.5 and $79.5 \pm 4.6\%$ in the pre- and postpubertal periods, respectively).

$\dot{V}_{E_{AT}}$ recorded at anaerobic threshold increased with body mass, independently of the maturative status of the children. $\dot{V}_{E_{AT}}$ was closely related to body mass in prepubertal period ($F_{1,34}=8.16$; $p=0.007$) and postpubertal period ($F_{1,28}=33.28$; $p<0.001$).

The difference in the slope between pre- and postpubescent children did not reach statistical significance ($p>0.20$). When data were grouped, the slope value reached $0.990 \text{ l min}^{-1} \text{ kg}^{-1}$.

When $\dot{V}_{E_{AT}}$ measured at the anaerobic threshold was expressed as a percentage of $\dot{V}_{E_{max}}$, the ratio did not depend on body mass and never accounted for more than 1% of the observed variance in each group (8% for all subjects).

Neither heart rate nor respiratory frequency measured at the anaerobic threshold were significantly related to body mass. Heart rate mean values were of

174.9 ± 15.7 beat min^{-1} before puberty and 177.5 ± 10.8 beat min^{-1} after. Respiratory rates were of 39.4 ± 8.5 and 35.7 ± 7.6 breath min^{-1} before and after puberty, respectively.

The relationship between $\dot{V}_{\text{O}_{2\text{AT}}}$ and the maturative ages (PHV) revealed that oxygen uptake recorded at anaerobic threshold increased before ($F_{1,34}=22.15$; $p < 0.001$) and after PHV ($F_{1,28}=8.64$; $p=0.006$). This increase was independent of the maturative ages of the children. Similar results were obtained for $\dot{V}_{\text{EAT}}$ which increased before ($F_{1,34}=10.61$; $p=0.003$) and after PHV ($F_{1,28}=12.65$; $p=0.001$) with the most rapid increase in the years following this marker ($t_{64}=2.68$; $p=0.01$). The ratio $\dot{V}_{\text{O}_{2\text{AT}}}/\dot{V}_{\text{O}_{2\text{max}}}$ or $\dot{V}_{\text{EAT}}/\dot{V}_{\text{Emax}}$ was independent of maturative ages.

Echocardiographic data

The relationships between left ventricular dimensions and body mass or age of PHV are shown in Fig. 4. For prepubescent children, LVVD tended to increase with body mass ($0.836 \text{ Wt} + 64.074$, $r^2=13.2\%$; $F_{1,17}=4.11$; $p=0.053$) while for LVVS this was not observed ($0.375 \text{ Wt} + 17.340$, $r^2=8.2\%$; $F_{1,27}=2.42$; $p=0.131$). In contrast, after puberty, there was a positive relation with LVVD ($1.572 \text{ Wt} +$

Fig. 4. Left ventricular volumes measured at end-systole (LVVS) and at end-diastole (LVVD) as a function of body mass (Wt) and of age of peak height velocity (PHV). Open (○) and full (●) circles indicated pre- and postpubertal data, respectively.

29.580, $r^2=58.9\%$; $F_{1,15}=21.52$; $p<0.001$) while for LVVS the relation was indicative ($0.546 \text{ Wt} + 9.860$, $r^2=19.3\%$; $F_{1,15}=3.598$; $p=0.078$).

For late maturers, the end-diastolic volume increase was larger (slope value: 1.57 ml kg^{-1}) than end-systolic one (0.55 ml kg^{-1}). The difference features that pubescent children mainly increased their stroke volume by increment of the end-diastolic volume.

Compared with reference values calculated from the formula of Henry *et al.* [15] taking into account the body mass and the age of the children, it appears that training increases LVVD in the prepubertal period (94.13 ± 16.21 vs 73.39 ± 8.11 ml; $t_{28}=6.28$; $p<0.001$) and in the postpubertal period (109.02 ± 15.36 vs 91.98 ± 10.97 ml, $t_{16}=3.38$; $p=0.004$). This was also observed for LVVS in prepuberty (30.81 ± 9.21 vs 25.16 ± 2.92 ml, $t_{28}=3.24$; $p=0.004$) and postpuberty (37.44 ± 9.32 vs 31.61 ± 3.88 ml, $t_{16}=2.47$; $p=0.025$).

The left ventricular ejection fraction was not significantly related to body mass and did not differ across the different maturative status ($38.3 \pm 4.6\%$ in prepuberty and $36.5 \pm 4.2\%$ in postpuberty).

The posterior wall thickness was not significantly correlated with body mass. The mean values were of 5.95 ± 0.97 mm before puberty and 7.94 ± 2.03 mm after. In the prepubertal period, septal thickness was of 6.60 ± 0.79 mm. For post-pubescent children, there was a positive relationship with body mass ($0.120 \text{ Wt} + 1.967$; $r^2=53.4\%$). When all data were grouped, the relationships became significant for posterior wall ($r^2=19.4\%$; $p<0.01$) and intraventricular septum ($r^2=24.7\%$; $p<0.001$).

In the pre- or postpubertal period, left ventricular volumes were not significantly correlated with maturative age relative to PHV. Significant correlations were only found for pooled data (LVVS = $1.598 \text{ PHV} + 34.479$, $r^2=12.9\%$; $F_{1,44}=6.54$; $p=0.014$; LVDD = $3.095 \text{ PHV} + 101.65$, $r^2=14.9\%$; $F_{1,44}=7.71$; $p=0.008$).

Oxygen uptake and cardiac dimensions

The results obtained from regression analyses between the dependent variables ($\dot{V}_{\text{O}_2\text{AT}}$ and $\dot{V}_{\text{O}_2\text{max}}$) and the left ventricular volumes are summarized in Table 4. There were poor correlations between $\dot{V}_{\text{O}_2}$ and the changes in cardiac volumes. Significant correlations were found between $\dot{V}_{\text{O}_2\text{max}}$ and left ventricular volumes at end-diastole in the prepubertal and postpubertal periods. At anaerobic threshold, the relationship was significant in prepubertal period but did not reach a significant level after. At end-systole, the relationship $\dot{V}_{\text{O}_2\text{max}}$ vs -LVVS was only indicative in prepubertal period.

At anaerobic threshold and at the end of the maximal exercise, the stroke volume accounted for 26.7 and 30.0% of the explained variance for oxygen uptake. This effect was only significant in the postpubertal period. Before puberty, the relationship did not reach statistical significance.

Table 4. Linear regression analyses between $\dot{V}_{O_{2\max}}$, $\dot{V}_{O_{2AT}}$, and the different resting left ventricular dimensions in pre- and postpubertal periods.

	Prepubertal					Postpubertal				
	α	Y-intercept	r^2	$F_{1,27}$	p	α	Y-intercept	r^2	$F_{1,15}$	p
LVVD $\dot{V}_{O_{2AT}}$ $\dot{V}_{O_{2\max}}$	0.046 0.051	0.437 0.253	19.3 16.5	6.47 5.35	0.017* 0.029*	0.058 0.076	0.377 0.572	22.7 30.2	4.40 6.48	0.053 (a) 0.022*
LVVS $\dot{V}_{O_{2AT}}$ $\dot{V}_{O_{2\max}}$	0.033 0.047	0.721 0.735	9.5 13.5	2.84 4.21	0.104 0.050 (a)	0.023 0.039	1.727 1.900	3.8 8.3	0.59 1.37	0.454 0.261
SV $\dot{V}_{O_{2AT}}$ $\dot{V}_{O_{2\max}}$	0.010 0.009	0.993 1.479	13.0 7.0	4.04 2.03	0.054 (a) 0.166	0.017 0.021	1.191 1.600	26.7 30.0	5.47 6.434	0.034* 0.023*

LVVD, left ventricular volume at end-diastole; LVVS, left ventricular volume at end-systole; SV, stroke volume. Slope values (α) are expressed in $l O_2 \min^{-1} ml^{-1}$. * $p < 0.05$. (a): $0.05 \leq p \leq 0.01$. The explained variance r^2 is expressed in %.

DISCUSSION

Aerobic capacity. The results of this longitudinal study show that changes in oxygen uptake were not different, in effect, when related to body mass rather than yearly intervals from the age of PHV. There is a constant increase of $\dot{V}_{O_{2\max}}$ ($0.054 \text{ l min}^{-1} \text{ kg}^{-1}$) irrespective of the maturative age of children engaged in competitive sports. Similar values have been reported by Fergusson *et al.* [16] for adult Canadian ice hockey players ($0.055 \text{ l min}^{-1} \text{ kg}^{-1}$). The functional capacity of the young trained children appears to be identical to that of highly trained mature athletes involved in the same sports when training programs are of sufficient duration and intensity. Comparisons with data of the literature indicate that this value is higher than that found for untrained children.

Using a cross-sectional design, Cooper *et al.* [3] found a linear $\dot{V}_{O_{2\max}}$ increase by $27.3 \text{ ml min}^{-1} \text{ kg}^{-1}$ for untrained children participating only in normal physical education programs at school. Yoshida *et al.* [17] showed on very young boys that training failed to increase $\dot{V}_{O_{2\max}}$, whereas Mirwald *et al.* [12] and Bar-Or [18] reported that prior to puberty aerobic training was less effective. This difference might be explained by the fact that, in the present study, the children were very active athletes and that training should be very intensive to produce a significant difference in $\dot{V}_{O_{2\max}}$ between the pre- and postpubertal periods. Ekblom [19] and Daniels and Oldridge [20] also reported that $\dot{V}_{O_{2\max}}$ was not significantly improved when the pretraining physical fitness of children was high.

From the present longitudinal study inducing long-term training, it is not possible to draw firm conclusions on the relative contribution of the growth and of the trainability of the children since there was no control group of untrained boys. Although the duration of the training program was strictly controlled it is not possible to assume that the intensity of the physical activities was kept constant. However, the fact that the ratio of $\dot{V}_{O_{2AT}}$ to $\dot{V}_{O_{2\max}}$ did not change during growth might suggest that, in our experimental condition, the level of physical fitness induced by training remained unchanged since it is well-known that this ratio increases in adult [5].

At the anaerobic threshold, oxygen uptake is highly correlated to body mass. On the contrary, the ratio $\dot{V}_{O_{2AT}}/\dot{V}_{O_{2\max}}$ does not depend on the maturative status. The mean value reaches $79.3 \pm 5.1\%$ and is close to that reported by Van Den Eynde *et al.* [21] showing a decline from 79% at 11–12 years old to 70% at the age of 13. Gaisl and Buchberger [22] also found comparable value of oxygen uptake at anaerobic threshold in children ($84\% \dot{V}_{O_{2\max}}$). As pointed out by Cooper *et al.* [3], our data suggest that the growth of each component of the cardiorespiratory responses to exercise is so integrated that the temporal coupling among them is preserved at an optimized value early in life despite the overall change in body mass.

For pulmonary ventilation, the year-to-year increment is accentuated in the years following PHV. This increase is mainly accomplished by an increment of

tidal volume, the breathing frequency remaining constant before and after puberty at the anaerobic threshold and at the end of exercise.

Cardiac dimensions and aerobic capacity. The myocardial contractility remained constant throughout the maturative age intervals. The large values of thicknesses of the posterior wall and of the interventricular septum reflect that the myocardial mass tends to be greater in postpubescent than in prepubescent children. This finding is reinforced by the fact that posterior wall and septal thicknesses were correlated with body mass when all data were grouped. Similar observations have been previously reported by Blimkie *et al.* [11] who found a positive relationship between left ventricular mass and body mass in children.

Echocardiographic measures showed that well-training children exhibited increases of left ventricular volumes above reference values attributable to body mass and age.

For postpubescent trained boys, the role of the increase in stroke volume in determining oxygen uptake at anaerobic threshold and at maximal exercise is relatively modest. The contribution of heart size in $\dot{V}_{O_2}$ revealed by regression analysis, showed that, after puberty, the stroke volume accounted for 26.7 to 30.0% of the explained variance. This is mainly ascribed to the distensibility of the ventricle. The modest contribution of stroke volume can be explained from the observations of Eriksson *et al.* [23] showing that this parameter kept up maximal values for exercise performed at 35% $\dot{V}_{O_{2max}}$, that is lower than $\dot{V}_{O_{2AT}}$ levels reached in the present study; further increases of cardiac output depending only on heart rate increments. This hypothesis is based on the assumption that the mechanisms regulating the left ventricular function at rest are identical to those intervening during exercise. As reported by Rerych *et al.* [24], a common mechanism was responsible for changes in cardiac function induced by training, in resting, as well as in exercising adults. Using radionuclide angiography, these authors demonstrated that, at rest and exercise, the stroke volume depended on the increases in pulmonary and total body blood volumes, which augment the ventricular preload.

The best correlation between $\dot{V}_{O_2}$ and left ventricular volume was observed at end-diastole. Stroke volume increases might result from an improvement of venous blood return and/or from an increase in total blood flow. Blimkie *et al.* [11] indicated that the left ventricular end-diastolic volume measured at rest was the primary factor that differentiated stroke volume of children with high and low maximal gas transport capacity. The present data suggest that, for trained children, the ability of the muscles to consume oxygen could be limited by heart function, especially during the prepubertal period. Other factors can work to extract more oxygen. The findings obtained from allometric relationships indicate that the scaling factors for $\dot{V}_{O_{2max}}$ and $\dot{V}_{O_{2AT}}$ are higher than the theoretical value of 0.67 in pre- and postpubertal period. This has been previously reported by others investigators [3, 24–28] in growing organisms whereas for mature untrained male and female subjects, Nevill *et al.* [29] found power function parameters of 0.63 and 0.72. This difference might reflect structural changes at the muscular level which

could increase the peak force generated per unit cross-sectional area of muscle. The increase and the distribution of the amount of blood to active skeletal muscles, tissues or organs [27], the increased concentration of oxidative enzymes [30] are probably more important factors than cardiac ones in the modifications of the anaerobic capacity.

In conclusion, the results of the present study show that the growth of each cardiorespiratory component is optimized during maturation in order to keep constant the aerobic response to exercise. The enhanced resting left ventricular functions does not seem to be the major factor explaining the ability of the muscles to consume oxygen. This suggests that local changes in structure and function occurring at the muscular level are of paramount importance and should be taken into consideration.

This study was supported by the Regional Council of Picardy.

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