

## SESSION I: Paper 1

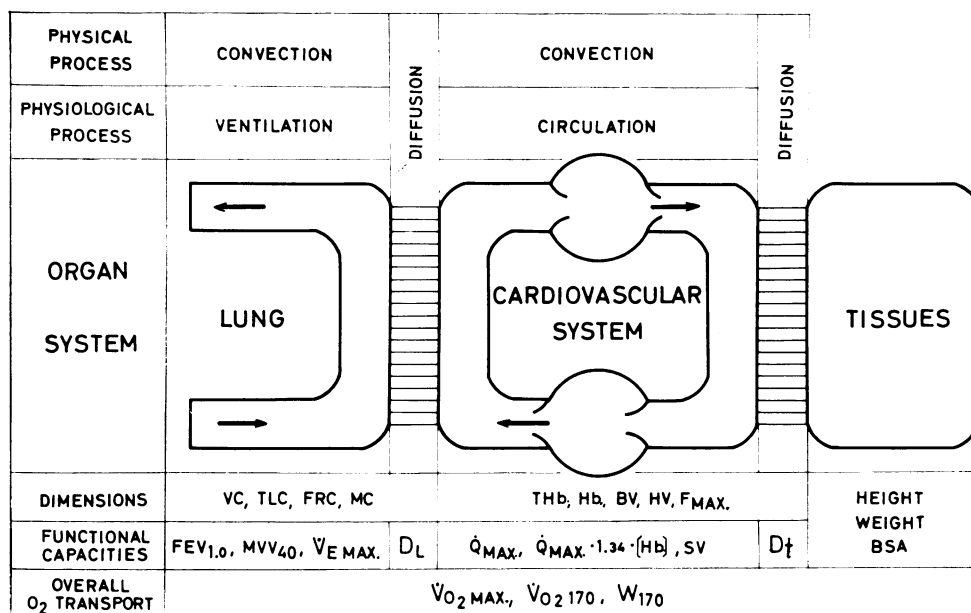
# Cardiorespiratory Determinants of Cardiovascular Fitness

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**C**ARDIOVASCULAR fitness will in this presentation be defined as the oxygen-forwarding capacity of the cardiorespiratory system. It thus has absolute dimensions ( $\text{ml. STPD. min}^{-1}$ ). Another alternative would be to define fitness as the observed transport capacity in relation to a predicted normal value, i.e. with relative dimensions. However, the choice of definition will not affect the basic ideas in this presentation.

fitness as defined above will depend on the net transport capacity for oxygen of this transport system and can be measured as the *maximal oxygen uptake* ( $\dot{V}O_{2 \text{ max.}}$ ).

The present discussion can therefore be limited to an analysis of the determinants of  $\dot{V}O_{2 \text{ max.}}$  in *healthy* subjects (Table I). These determinants can be divided into two groups: (i) *dimensional factors* and (ii) *functional capacities*.<sup>6-8</sup>



**Fig. 1.**—Schematic illustration of the oxygen-transport system with measures of dimensions and functional capacities of the various components. VC = vital capacity, TLC = total lung capacity, FRC = functional residual capacity, MC = pulmonary midcapacity, THb. = total hemoglobin, Hb. = hemoglobin concentration, BV = blood volume, HV = supine heart volume, F<sub>max.</sub> = maximal heart rate, FEV<sub>1.0</sub> = forced expiratory volume in one second, MVV<sub>40</sub> = maximal ventilatory volume estimated at 40 breaths per minute,  $\dot{V}_E \text{ max.}$  = minute ventilation during determination of  $\dot{V}O_{2 \text{ max.}}$ ,  $D_L$  = diffusing capacity of the lungs,  $\dot{Q}_{\text{max.}}$  = maximal cardiac output, SV = stroke volume during determination of  $\dot{Q}_{\text{max.}}$ ,  $D_t$  = diffusing capacity of the tissues,  $\dot{V}O_{2 \text{ max.}}$  = maximal oxygen uptake,  $\dot{V}O_{2 \text{ 170}}$  and  $W_{170}$  = oxygen uptake and rate of work at a heart rate of 170 beats per minute.

Oxygen is forwarded from the surrounding atmosphere to the site of oxidative metabolism—the mitochondria—by two convective systems, pulmonary ventilation and blood circulation, and two diffusing systems—the alveolar-capillary and tissue-capillary cell systems (Fig. 1). Physical

By *dimensional factors* is meant the influence of the size of the organs that compose this transport line, i.e. the size of the lungs, the size of the diffusing surface, the size of the pulmonary capillary bed, the size of the vascular system, the size of the heart, the maximal heart rate and the concentration of hemoglobin in the blood. All these factors constitute the dimensional prerequisites for O<sub>2</sub> transport.

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This work was supported by grants from the Swedish National Association against Heart and Lung Diseases.



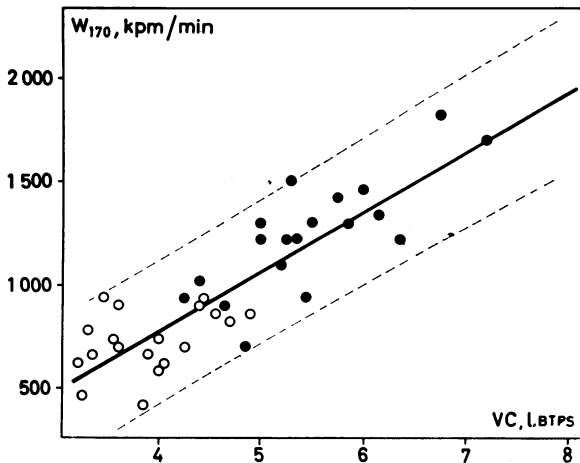


Fig. 2.—Rate of work at a heart rate of 170 beats per minute ( $W_{170}$ , kpm/min) in relation to vital capacity (VC l. BTPS). Full line represents least squares regression:  $W_{170} = 286.8 (VC) - 366.4$ ,  $SD = 171.9$ ,  $n = 38$ . Broken lines represent 95% confidence limits. Filled circles represent men, open circles women.

The dimensions of the lungs can be described by the *vital capacity (VC)*, *total lung capacity (TLC)* or *functional residual capacity (FRC)*. The dimensions of the cardiovascular system can be described by *blood volume*, *total hemoglobin*, *hemoglobin concentration* and *roentgenological heart volume*.

It can be shown<sup>6, 7</sup> that the oxygen-forwarding capacity is highly correlated to all these different dimensional measures. In the material used to illustrate these relationships,<sup>7</sup> the rate of work at a heart rate of 170 beats per min ( $W_{170}$  kpm/min) is highly correlated to  $\dot{V}_{O_2 \max}$  l./min, the

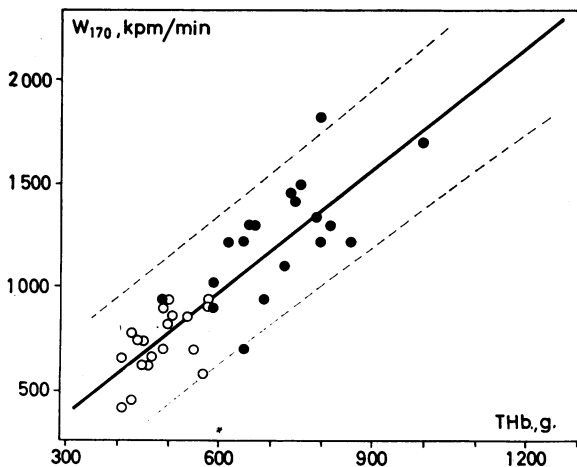


Fig. 3.—Rate of work at a heart rate of 170 beats per minute ( $W_{170}$ , kpm/min) in relation to total hemoglobin in grams. Symbols and subjects as in Fig. 2. Regression equation:  $W_{170} = 1.974 (THb.) - 204.1$ ,  $S.D. = 177.8$ ,  $n = 38$ .

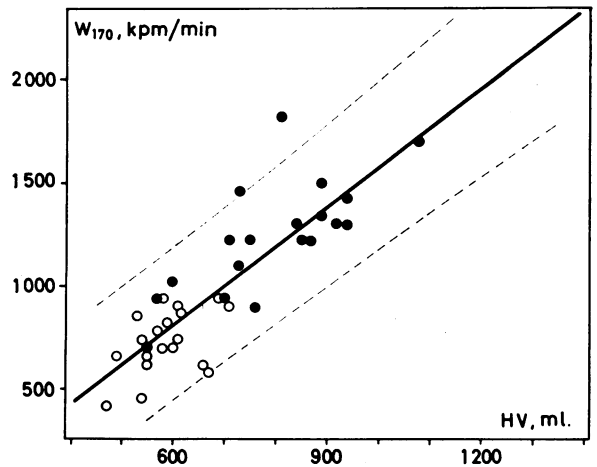


Fig. 4.—Rate of work at heart rate of 170 beats per minute ( $W_{170}$ , kpm/min) in relation to heart volume (HV, ml.). Symbols and subjects as in Fig. 2, subjects in supine position. Regression equation:  $W_{170} = 1.901 (HV) - 328.3$ ,  $S.D. = 187.0$ ,  $n = 38$ .

linear regression being described by the equation:  $W_{170} = 396 (\dot{V}_{O_2 \max.}) - 176$ ,  $S.D. = 125$  kpm/min,  $r = 0.93$ .

The correlation matrix for these variables is shown in Table II. Fig. 2 illustrates the high correlation between  $W_{170}$  and vital capacity; Fig. 3, the correlation between  $W_{170}$  and total hemoglobin; and Fig. 4, the correlation between  $W_{170}$  and heart volume.

Each of the described components of the  $O_2$  transport system also has a *functional transport capacity*. This functional capacity is related to the dimensions of the component and to its optimal function. The functional capacity of the ventilatory system can be described by the *maximal voluntary ventilation (MVV)*, or the *ventilation during determination of  $\dot{V}_{O_2 \max.}$* . The transport capacity of the alveolo-capillary diffusing system can be described by the *diffusing capacity* of the lungs for carbon dioxide ( $D_{L, CO}$ ). The functional capacity of the cardiovascular system can be described by the *maximal cardiac output* and the *stroke volume (SV)* that can be maintained during maximal work. The oxygen transport capacity is highly correlated to the functional capacities of the different components of the cardiorespiratory system. The different measures of overall oxygen transport are all highly correlated to  $FEV_{1.0}$ ,  $MVV_{40}$  and  $\dot{V}_E \max.$ , to  $D_{L, CO}$ <sup>8</sup> (Fig. 5), to stroke volume and maximal cardiac output.

A large oxygen-transport capacity obviously requires an optimal combination of dimensions and functional capacities of the different components of the cardiovascular system. The dimensional factor is probably the main determinant of the *interindividual variations* while the

TABLE II.—CORRELATION COEFFICIENTS FOR 22 VARIABLES RELATED TO STATIC DIMENSIONS AND FUNCTIONAL CAPACITIES OF THE LUNGS AND CARDIOVASCULAR SYSTEM, TO BODY SIZE AND TO OVERALL OXYGEN TRANSPORT. SYMBOLS AS IN TABLE I.

Variables	Lung										Cardiovascular system										Overall O <sub>2</sub> transport				
	Static dimensions					Functional capacities					Diff- sion	Static dimensions					Functional capacities					Tissues			
	VC	TLC	FRC	MC	FEV <sub>1.0</sub>	FEV <sub>1.0</sub>	MVV <sub>40</sub>	V <sub>G</sub> max	D <sub>LCO</sub> ≥ 120	THb.		Hb.	BV	HV	SV	Q <sub>max</sub>	Q <sub>max</sub>	I.S <sub>4</sub> Hb.	F <sub>max</sub>	Height	Weight	BSA	V̇O <sub>2</sub> max	V̇O <sub>2</sub> 170	W 170
VC, l.	.981	.888	.903	.807	.766	.823	.824	.586	.874	.800	.717	.700	.758	-.273	.834	.875	.928	.876	.726	.781	.775	.681	.858	.867	.887
TLC, l.	.981	.943	.854	.791	.779	.790	.753	.583	.869	.834	.704	.692	.753	-.296	.842	.861	.922	.875	.685	.687	.708	.871	.853	.850	.759
FRC, l.	.888	.943	.995	.718	.714	.735	.878	.485	.798	.774	.591	.583	.639	-.282	.891	.752	.837	.800	.882	.685	.698	.813	.803	.786	.821
MC, l.	.903	.995	.995	.718	.717	.766	.717	.485	.820	.793	.604	.593	.637	-.327	.893	.767	.848	.827	.893	.685	.688	.813	.841	.821	.754
FEV <sub>1.0</sub> , l.	.796	.718	.710	.882	.804	.660	.804	.743	.795	.773	.613	.618	.716	-.050	.726	.781	.775	.815	.726	.781	.775	.681	.711	.711	.711
MVV <sub>40</sub> , l./min.	.807	.822	.757	.763	.882	.732	.864	.797	.731	.780	.716	.720	.824	-.101	.685	.687	.708	.871	.685	.687	.708	.729	.729	.759	.759
V <sub>G</sub> max, l./min.	.766	.779	.714	.904	.864	.663	.904	.763	.688	.740	.653	.648	.742	-.190	.682	.685	.698	.813	.682	.685	.698	.703	.754	.754	.754
D <sub>LCO</sub> ≥ 120, ml./min./mm.Hg.	.823	.790	.735	.660	.732	.663	.663	.586	.793	.738	.646	.605	.676	-.401	.708	.728	.745	.881	.708	.745	.745	.910	.922	.922	.922
THb., g.	.924	.878	.891	.850	.821	.850	.785	.660	.959	.926	.687	.661	.745	-.423	.861	.852	.882	.919	.861	.852	.882	.919	.916	.913	.913
Hb., g./100 ml.	.596	.593	.485	.743	.797	.586	.763	.586	.520	.586	.446	.432	.602	-.152	.465	.460	.466	.680	.465	.460	.466	.680	.541	.612	.612
BV, l.	.874	.869	.820	.795	.731	.688	.793	.520	.898	.898	.627	.596	.659	-.437	.809	.836	.855	.880	.809	.836	.855	.880	.898	.886	.886
HV, ml.	.800	.834	.774	.793	.780	.740	.738	.586	.898	.898	.733	.709	.774	-.488	.762	.801	.810	.803	.762	.801	.810	.877	.829	.829	.829
Stroke vol., ml.	.717	.704	.591	.604	.613	.646	.653	.446	.627	.733	.627	.993	.975	-.357	.683	.686	.703	.701	.683	.686	.703	.659	.649	.649	.649
Q <sub>max</sub> , l./min.	.700	.692	.583	.618	.720	.648	.648	.432	.596	.709	.983	.979	.979	-.306	.684	.681	.700	.676	.684	.681	.700	.676	.616	.606	.606
Q <sub>max</sub> , l.34. Hb.	.758	.753	.639	.647	.716	.824	.742	.602	.659	.774	.975	.979	.979	-.302	.714	.718	.734	.763	.714	.718	.734	.763	.680	.686	.686
F <sub>max</sub> , beats/min.	-.273	-.296	-.282	-.050	-.101	-.190	-.190	-.152	-.437	-.488	-.357	-.306	-.302	-.302	-.246	-.159	-.209	-.373	-.246	-.159	-.209	-.373	-.564	-.514	-.514
Height, cm.	.934	.842	.893	.726	.685	.708	.682	.465	.809	.762	.683	.684	.714	-.246	.906	.906	.970	.779	.906	.970	.970	.779	.762	.750	.750
Weight, kg.	.875	.861	.752	.781	.687	.728	.685	.460	.836	.801	.686	.681	.718	-.159	.906	.981	.981	.809	.906	.981	.981	.809	.776	.762	.762
BSA, m <sup>2</sup>	.928	.922	.848	.775	.708	.745	.698	.466	.855	.810	.703	.700	.734	-.209	.981	.981	.981	.824	.981	.981	.981	.824	.798	.782	.782
V <sub>O2</sub> max, l./min.	.876	.875	.800	.827	.815	.881	.813	.680	.880	.903	.701	.676	.763	-.373	.779	.809	.824	.943	.779	.809	.824	.943	.930	.930	.930
V <sub>O2</sub> 170, l./min.	.858	.853	.803	.841	.691	.910	.703	.541	.898	.877	.659	.616	.680	-.564	.762	.776	.798	.943	.762	.776	.798	.943	.976	.976	.976
W 170, kg/min.	.867	.850	.786	.821	.711	.922	.754	.612	.886	.829	.649	.606	.686	-.514	.750	.762	.782	.980	.750	.762	.782	.980	.976	.976	.976

n = 20 f = 18 Value of r for P of 0.05 = 0.444, for P of 0.01 = 0.561, for P of 0.001 = 0.679.

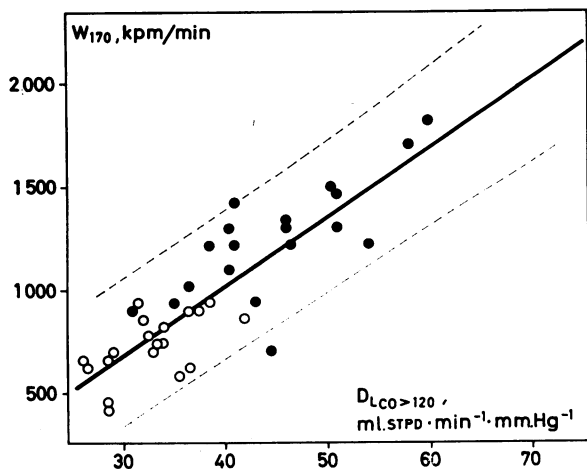


Fig. 5.—Rate of work at heart rate of 170 beats per minute ( $W_{170}$ , kpm/min) in relation to the steady state diffusing capacity of the lungs for CO during exercise ( $D_{L, CO}$ , ml. STPD/min/mm. Hg). Symbols and subjects as in Fig. 2. Regression equation:

$$W_{170} = 33.4 (D_{L, CO}) - 316.7, \\ SD = 177.3, n = 38.$$

functional factor is the one which accounts for the *intra-individual* day-to-day variations. The component in the oxygen-transport system which causes the highest intra-individual variation of fitness is probably the cardiovascular system. There are two functional mechanisms by which the oxygen forwarding capacity can be influenced. Consider the Fick equation for oxygen:

$\dot{V}O_2 = \dot{Q} \times AVD = F \times SV \times (C_{a, O_2} - C_{\bar{v}, O_2})$   
where  $\dot{V}O_2$  = oxygen uptake,  $\dot{Q}$  = cardiac output, AVD = arterio-mixed venous oxygen difference, F = heart rate, SV = stroke volume of the heart,  $C_{a, O_2}$  = arterial  $O_2$  content and  $C_{\bar{v}, O_2}$  = mixed venous  $O_2$  content.

During submaximal work,  $\dot{Q}$  and F vary as linear functions of  $\dot{V}O_2$ , and AVD and SV consequently vary as hyperbolas. The slope for the linear regression of  $\dot{Q}$  on  $\dot{V}O_2$  varies very little between individuals while the intercept may vary markedly, with a S.D. of 1 l./min in normal subjects.<sup>5</sup> The average intercept in the normal population is said to represent a *normokinetic* circulation; if the intercept is higher than normal the circulation is called *hyperkinetic* and if lower—*hypokinetic* (Fig. 6). In a situation with a hyperkinetic circulation, oxygen is thus forwarded with a larger cardiac output than under normal circumstances.

A limitation of the oxygen-forwarding capacity of the cardiovascular system can be due (i) to an incapacity to maintain the stroke volume during exercise, (ii) to an inability to increase heart rate, or (iii) to a lower than normal AVD, which implies that a larger than normal amount of blood is used for a given oxygen transport.

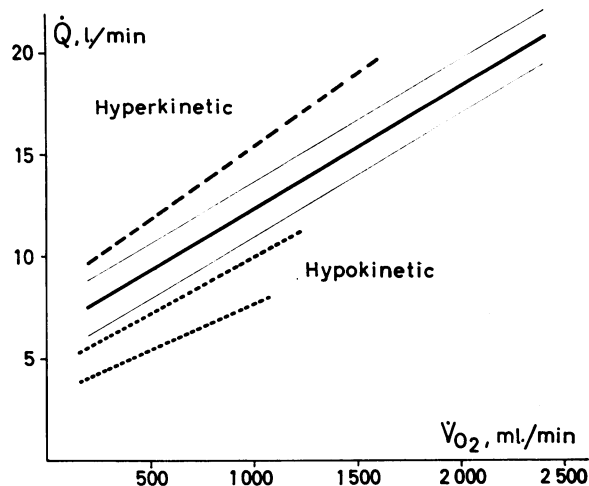


Fig. 6.—Cardiac output ( $\dot{Q}$ , l./min) in relation to oxygen uptake ( $\dot{V}O_2$ , ml./min). Solid lines indicate the normal relationship  $\pm$  SD, broken lines the relationship in subjects with vasoregulatory asthenia and dotted lines the relationship in subjects with mitral stenosis.

The *stroke volume* is determined by the *dimensions* of the heart, the *filling* and *emptying* condition of the two ventricles. The influence of the dimensional factor—the size of the heart—is obvious. The filling of the ventricles is determined by (i) the *amount of energy* (potential and kinetic) that is *available* when the A-V valves open at the end of systole, (ii) the *time available* for filling, and (iii) the *elastic and resistive properties of the ventricular myocardium*. With a normal myocardium and an adequate amount of filling energy available, the ventricles fill mainly during the rapid filling phase of diastole and the filling process is relatively insensitive to marked variations in heart rate. If the energy available is decreased (for instance, owing to a shift in the distribution of the blood within the capacitance vessels), the ventricles fill during a longer part of diastole. The filling process is then sensitive to a shortening in diastole, which may result in a smaller than normal SV.

From the above it is obvious that a low  $O_2$ -forwarding capacity can be caused by the following cardiovascular factors:

- (1) Small dimensions of the heart.
- (2) Low filling energy available to the ventricles.
- (3) Hyperkinetic circulation.

All these conditions are accompanied by a higher than normal heart rate during submaximal work (Table III).

*Variations in the filling energy* are seen as the result of orthostatic blood shifts within the capacitance vessels, commonly observed at rest in the erect position. Such orthostatic shifts are often seen also during exercise, especially in patients who live a sedentary life or who have been lying

TABLE III.—HEART RATE (F) AT A CONSTANT WORK LOAD (W), IN SITTING AND SUPINE POSITION, ECG DURING EXERCISE, Q -  $\dot{V}_{O_2}$  INTERCEPT AND STROKE VOLUME, IN THREE TYPES OF IMPAIRED OXYGEN TRANSPORT—PATIENTS WITH DIFFERENT TYPES OF "FUNCTIONAL HEART DISEASE"

Variable	Small dimensions	Impaired filling	Hyperkinetic circulation
$W_F$ sitting	Decreased	Decreased	Decreased
$W_F$ supine	Decreased	Normal or slight decreased	Decreased
ECG during exercise	Normal	Normal	Evidence of sympathetic activity on ECG
Q - $\dot{V}_{O_2}$ - intercept	Normal or low	Normal or low	High
Stroke volume	Low	Low	Normal

in bed for a long period of time. These patients exercise with a lower heart rate for a given sub-maximal work load in the supine than in the sitting position. This mechanism is also probably a common cause of part of the asthenia that is seen so often in convalescent patients. Orthostatic blood shifts during exercise have also been reported in patients with lung disease, varicose veins in the legs, and congenital absence of the venous valves. In patients where physical training can be applied, these blood shifts can be eliminated rather rapidly with physical training. In patients with organic lesions or defects causing the blood shift within the capacitance vessels, the treatment should be aimed at the primary cause, e.g. the varicose veins.

A *hyperkinetic circulation* as a cause of low oxygen-forwarding capacity is seen in vasoregulator asthenia, VA (Ref. 4 and Fig. 6). The cause of this overperfusion is unknown, but it is believed to be due to an increased sympathetic activity. It has been eliminated by  $\beta$ -blocking

agents,<sup>2</sup> chlorisondamine chloride (Ecolid), and systematic physical training. A hyperkinetic circulation has also been observed in well-trained athletes<sup>1</sup> and in patients with beri-beri,<sup>10</sup> thyrotoxicosis,<sup>10</sup> the "dumping syndrome",<sup>3</sup> and anemia.<sup>9</sup>

Which link in the oxygen transport line limits the transport capacity in normal subjects? From the above it is obvious that a limitation can occur in the dimensional prerequisites of the transport line—small lungs, a small blood volume or a small heart, or in the functional capacities of the different components. Judging from studies of the blood gases during exhausting work, ventilation and diffusion seldom limit the  $O_2$ -transport capacity. This leaves the functional capacity of the cardiovascular system, i.e. the capacity to maintain a large cardiac output that is adequately distributed through the body.

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## Commentaries

Commentary: R. J. SHEPHARD, *Toronto, Ontario*

THE maximal oxygen intake is widely accepted as one index of cardiorespiratory fitness. Dr. Beeckmans and I have recently developed a simple equation which permits an assessment of the relative contributions of various cardiac and respiratory variables to this aspect of endurance fitness.

The maximal oxygen uptake ( $\dot{V}_{O_2}$ ) is described in terms of a transfer coefficient ( $\dot{U}_{O_2}$ ) and an appropriate concentration gradient from inspired gas ( $C_{I, O_2}$ ) to the active tissues ( $C_{t, O_2}$ ):

$$(1) \quad \dot{V}_{O_2} = \dot{U}_{O_2} (C_{I, O_2} - C_{t, O_2})$$

The transfer coefficient ( $\dot{U}_{O_2}$ ) has the dimensions of a conductance, and is conveniently expressed in units of l./min.

By a few simple algebraic manipulations, both the overall conductance and the corresponding concentration gradient may be partitioned into four series components representing respectively maximum alveolar ventilation ( $\dot{V}_A$ ) the interaction between maximum pulmonary diffusion

and blood transport  $\left( \frac{1}{\lambda \dot{Q}} \left[ \frac{B}{1-B} \right] \right)$ , blood transport  $\left( \frac{1}{\lambda \dot{Q}} \right)$ , and the interaction between tissue diffusion and blood transport  $\left( \frac{1}{\lambda \dot{Q}} \left[ \frac{K}{1-K} \right] \right)$ :