# Ventilatory Threshold During a Graded Exercise Test in Male Youths with Cerebral Palsy and Spastic Paresis of Lower Limbs

*by*

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*A group of 16 male youth with spastic diplegy of lower extremities were examined. Their mean age was 18.5 years, body height 166.5 ± 6,6 cm, and body mass 57.4 ± 7,7 kg. They were subjected to a graded cycle ergometer arm exercise until exhaustion. The following indices of the circulatory and respiratory systems at maximum intensity and at the ventilatory threshold (VT) were recorded: percentage difference in the inhaled-exhaled oxygen ($t\%O_2$) and carbon dioxide ($t\%CO_2$), lung ventilation ($V_E$), oxygen uptake ($VO_2$), carbon dioxide output ($VCO_2$), respiratory quotient (RQ) as well as the respiratory equivalent of oxygen and carbon dioxide ($V_E \cdot VO_2^{-1}$ and $V_E \cdot VO_2^{-1}$), indices of acid-base balance.*

*Aerobic capacity of the subjects studied, expressed as the maximum oxygen uptake ($VO_{2max}$), amounted to 2.42 l · min$^{-1}$ or 42.6 ml · kg$^{-1}$ · min$^{-1}$. At maximum work intensity the load was 158.4 W(2.78 W kg$^{-1}$) and the maximum heart rate - 184 beats · min$^{-1}$. The load at the VT threshold was 96.6 W(1.71 W · kg$^{-1}$), which required 1,71 l · min$^{-1}$ (30.1 ml · kg$^{-1}$ · min$^{-1}$) of oxygen uptake. The threshold heart rate was 157 beats · min$^{-1}$. The percentage value of oxygen uptake at VT was to 69.5% of $VO_{2max}$ and 89.6% of maximum heart rate.*

***Key words:*** *Cerebral palsy, ventilatory threshold, exercise, aerobic capacity*

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## *Introduction*

The course of adaptation to exercise is an extremely complex phenomenon that cannot be reflected by a single index even as universal as $VO_{2max}$. Numerous observations point to the fact that the capacity to perform prolonged work is determined by the mode of transition from aerobic metabolism to the anaerobic glycolysis. Some problems related to metabolic thresholds, which concern the efficacy of training, were studied not only in healthy children and youth (Ballarin *et al.* 1989; Balardi *et al.*1989; Davis 1985; Gaisl and Buchberger 1980; Haffor and Catledge - Kirk 1988; Reybrouck *et al.*1982), but also in handicapped subjects (Bhambhani *et al.*1993; Klimek-Piskorz and Piskorz 1997; Piskorz and Klimek-Piskorz 1994, 1998). Reybrouck *et al.* (1985) who viewed this problem from the angle of developmental changes in a broader age range (6 – 18 years) demonstrated that the ventilation threshold, expressed in terms of $VO_{2max}$, was highest in the youngest group (70%), only slightly lower in children 11 – 12 years old (66 - 68 % of $VO_{2max}$), and was steadily decreasing thereafter. Bar-Or (1983) as well as Inbar and Bar-Or (1986) reported that anaerobic capacity was influenced in children and youth not only by the developmental changes but also by differences in the proportions of muscle fibres.

No reliable data, based on objective ventilation criteria, on the magnitude of the ventilatory threshold (VT) in children suffering from dyskinesis of cerebral origin could be found in the available literature. Thus, the aim of this study was to determine the level of VT not only in relation to $VO_{2max}$ and $HR_{max}$, but also as a function of load, which may be of practical value in the rehabilitation process.

## *Material and Methods*

The study was conducted on 16 male subjects aged 18 – 19 years (mean body height 166.5 ± 6,6 cm, mean body mass 57.4 ± 7,7 kg), attending a secondary vocational boarding school. The subjects and their parents were informed about the objectives of the study and possible risk involved. They were told they could stop the exercise at any time. During the medical examinations, none of them was disqualified from the exercise on cardio-vascular, respiratory, neurological, or orthopaedic grounds. For the last six months they attended school and their rehabilitation classes regularly. All of them had spastic paresis, particularly pronounced in lower limbs (spastic diplegy) and associated with the characteristic adductive flexure of their thighs in the hip joints, flexion contracture in the knee joints, and equinal contracture of feet. The gait with knees bent with a tendency to crossing thighs was the most frequent dysfunction. The pel-

vic anteverion was compensated by an increased lumbar lordosis and chest kyphosis. The stabilisation of the head brought about the raising of the shoulders, which in case of increased chest kyphosis restricted chest mobility and the range of movement of upper limbs. Also a tendency for grasping with pronation of the hand and forearm was observed.

All subjects performed a graded exercise in the sitting position on a cycle ergometer (Monark 824 E, Sweden), adapted for arm work and optimised ergonomically. Following an initial load of 37.5 W lasting 5 min, the load was increased every minute by 15 W (Piskorz and Klimek-Piskorz 1994; 1998). The exercise was continued until exhaustion. The stabilization of $VO_2$ in the last stage of exercise was the criterion of accepting it as the value of $VO_{2max}$. The increments of $VO_2$ in the last minute were compared to oxygen uptake at lower workloads as suggested by Kemper and Verschur (1983). Respiratory quotient ($R>1$) and heart rate exceeding 200 beats per minute (bpm) served as auxiliary criteria.

Respiratory variables were recorded at rest, during the warm-up and graded exercise, as well as during 3 min post-exercise recovery, by using a spiroergotest device (Medikro, Finland), at 30 s intervals. Heart rate was monitored by means of a Sport-Tester (Polar-Electronic, Finland). The following variables were recorded throughout the test: pulmonary ventilation ($V_E$), oxygen uptake ($VO_2$) and carbon dioxide elimination ($VCO_2$), percentage of oxygen ($t\%O_2$) and carbon dioxide ($t\%CO_2$). From these, the ratio of pulmonary ventilation to oxygen uptake ($V_E \cdot VO_2^{-1}$) or carbon dioxide elimination ($V_E \cdot VCO_2$) were computed. From changes in the maximum $t\%CO_2$ minimum $V_E \cdot VCO_2^{-1}$, and spurts in $V_E$ and $VCO_2$, the anaerobic threshold was determined according to Wasserman *et al.* (1986).

At rest and during the third minute of recovery, blood samples were drawn from the fingertips for the following assays: concentration of hydrogen ions ($H^+$), base excess (BE); all by means of Ciba Corning 238 device), plasma lactate concentration (La) – by enzymatic commercial kits (Lactate-PAP, BioMérieux, France) and Specol 11 colorimeter (Germany).

## *Results*

Pre-exercise pulmonary ventilation ($V_E$) amounted to 18.7 l·min$^{-1}$ and increased to 31.6 l·min$^{-1}$ following a 5 min warm-up at a constant load of 37.5 W. The ventilation increments were inversely related to resting values. Following warm-up, the pulmonary ventilation was proportional to work intensity ranging from 37.5 to 97.5 W (table 1). The subjects attained the anaerobic ventilation threshold equal to 49.6 l·min$^{-1}$ at a load of 96.6 W. Passing that threshold was

associated with an increase in pulmonary ventilation by 13 $l \cdot min^{-1}$ and that increase rose steadily, attaining a mean value of 91.5 $l \cdot min^{-1}$ during maximum effort (see table 2).

**Table 1**

*Values of pulmonary ventilation ($V_E$), oxygen uptake ($VO_2$), carbon dioxide elimination ($VCO_2$) and respiratory quotient (R) at various workloads, recorded in male youth with spastic paresis of lower limbs (n = 16)*

| Load [W] | $VO_2$ [$l \cdot min^{-1}$] | | $VO_2$ [$ml \cdot kg^{-1} \cdot min^{-1}$] | | $V_E$ [$l \cdot min^{-1}$] | | R | | $VCO_2$ [$l \cdot min^{-1}$] | |
|---|---|---|---|---|---|---|---|---|---|---|
| | $\overline{x}$ | SD | $\overline{x}$ | SD | $\overline{x}$ | SD | $\overline{x}$ | SD | $\overline{x}$ | SD |
| 37.50 | 1.05 | 0.13 | 18.49 | 3.01 | 31.64 | 4.70 | 0.82 | 0.10 | 0.86 | 0.11 |
| 52.50 | 1.15 | 0.12 | 20.24 | 3.23 | 33.73 | 5.19 | 0.81 | 0.09 | 0.93 | 0.13 |
| 67.50 | 1.32 | 0.19 | 23.26 | 4.25 | 38.25 | 5.78 | 0.84 | 0.09 | 1.10 | 0.15 |
| 82.50 | 1.59 | 0.19 | 28.10 | 4.96 | 45.74 | 7.37 | 0.87 | 0.01 | 1.38 | 0.18 |
| 97.50 | 1.72 | 0.23 | 30.36 | 5.75 | 51.81 | 8.31 | 0.91 | 0.08 | 1.55 | 0.27 |
| 112.50 | 1.97 | 0.25 | 34.90 | 6.66 | 64.10 | 11.73 | 0.69 | 0.09 | 1.89 | 0.26 |
| 127.50 | 2.14 | 0.25 | 37.59 | 5.87 | 73.78 | 10.72 | 0.99 | 0.09 | 2.09 | 0.20 |
| 142.50 | 2.30 | 0.31 | 40.14 | 5.98 | 84.94 | 11.68 | 1.02 | 0.09 | 2.31 | 0.22 |
| 157.50 | 2.46 | 0.31 | 43.06 | 8.16 | 96.73 | 13.28 | 1.02 | 0.09 | 2.51 | 0.24 |
| 172.50 | 2.47 | 0.31 | 38.83 | 5.45 | 100.73 | 11.40 | 1.03 | 0.70 | 2.55 | 0.21 |

**Table 2**

*Threshold and maximum values of the selected physiological indices in male youth with spastic paresis of lower limbs (n = 16)*

| Variables | VT | | MAX | |
|---|---|---|---|---|
| | $\overline{x}$ | SD | $\overline{x}$ | SD |
| Load (W) | 96.6 | 12.8 | 158.4 | 28.7 |
| ($W \cdot kg^{-1}$) | 1.71 | 0.34 | 2.78 | 0.50 |
| Work (kJ) | 27.4 | 4.7 | 61.2 | 22.2 |
| ($kJ \cdot kg^{-1}$) | 0.48 | 0.10 | 1.08 | 0.33 |
| $V_E$ ($l \cdot min^{-1}$) | 49.6 | 7.6 | 91.5 | 14.6 |
| $VO_2$ ($l \cdot min^{-1}$) | 1.71 | 0.22 | 2.42 | 0.32 |
| ($ml \cdot min \cdot kg^{-1}$) | 30.1 | 5.3 | 42.6 | 7.0 |
| at VT ($\%VO_{2max}$) | 70.7 | 11.1 | - | - |
| $VCO_2$ ($l \cdot min^{-1}$) | 1.5 | 0.21 | 2.39 | 0.23 |
| HR (heart beats $\cdot min^{-1}$) | 157 | 14 | 184 | 13 |
| at VT ($\% HR_{max}$) | 85.3 | 7.9 | - | - |

Pre-exercise oxygen uptake (VO$_2$) amounted to 0.51 l · min$^{-1}$, increased to 1.05 l · min$^{-1}$ towards the end of the 5 min warm-up, and then increased steadily (table 1) after having passed the VT threshold at 1.71 l VO$_2$. At the last minute of the exercise, the increments were smaller and the VO$_2$ value amounted to a mean value of 2.42 l · min$^{-1}$ (table 2).

Mean resting carbon dioxide output (VCO$_2$) was 0.45 l · min$^{-1}$. It increased to 0.86 l · min$^{-1}$ during the warm-up, ± 0.11 l · min$^{-1}$ and was proportional to VO$_2$ (table 1) throughout the exercise, attaining a th014shold value of 1.5 l · min$^{-1}$. Above the anaerobic threshold, VCO$_2$, and V$_E$, rose disproportionally attaining a mean value of 2.39 l · min$^{-1}$ (see table 2).

Respiratory quotient (R) paralleled power output and attained a mean value of 1.0 during maximal effort (table 1).
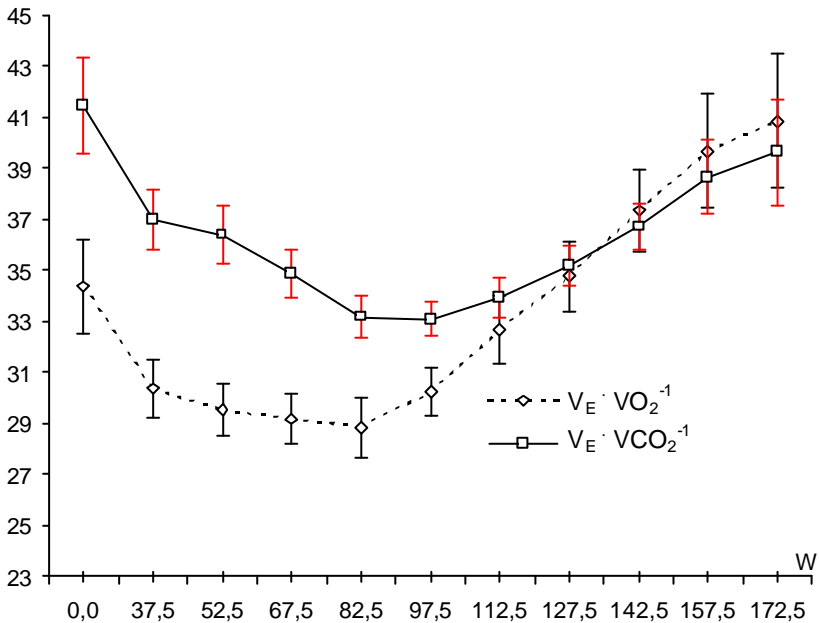


**Fig. 1.** *Changes in pulmonary ventilation-to-oxygen consumption ratio and - to-carbon dioxide elimination ratio*

Respiratory equivalents of oxygen (V$_E$·VO$_2$$^{-1}$) and of carbon dioxide (V$_E$·VCO$_2$$^{-1}$) were relatively constant (fig. 1). Mean resting value of the first ratio amounted to 34.4 and decreased to 30.4 during work at a constant load (fig. 1).

The utilisation of pulmonary ventilation for the elimination of carbon dioxide (V$_E$·VCO$_2$$^{-1}$) changed only slightly from 41.4 at rest to 37.0 towards the end of

warm-up (see fig 1). Since oxygen uptake ($VO_2$) and carbon dioxide elimination ($VCO_2$) increased proportionally to the load, yet faster than ventilation ($V_E$), both $V_E \cdot VCO_2^{-1}$ and $V_E \cdot VCO_2^{-1}$ decreased to respective threshold values (29.5 and 32.6) over the range of loads. Above that threshold, the VT was reflected in the increase of $V_E \cdot VO_2^{-1}$, which reached an average value of 38.1 during maximum effort, exceeding only slightly the resting value. The value of $V_E \cdot VCO_2^{-1}$ also increased and reached its mean value of 38.3, not exceeding the resting one.

The inhalation-exhalation difference in oxygen ($t\%O_2$) and carbon dioxide ($t\%CO_2$) contents increased together with work intensity. Resting values of $t\%O_2$ and $t\% CO_2$ were 3.26 and 2.90, respectively. At the end of warm-up, these values reached $4.07 \pm 0.58$ and $3.40 \pm 0.45$, respectively. The corresponding threshold values were $4.29 \pm 0.33$ and $3.82 \pm 0.33$. At maximum intensity, the values were $3.33 \pm 0.47$ and $3.28 \pm 0.33$ (fig. 2).
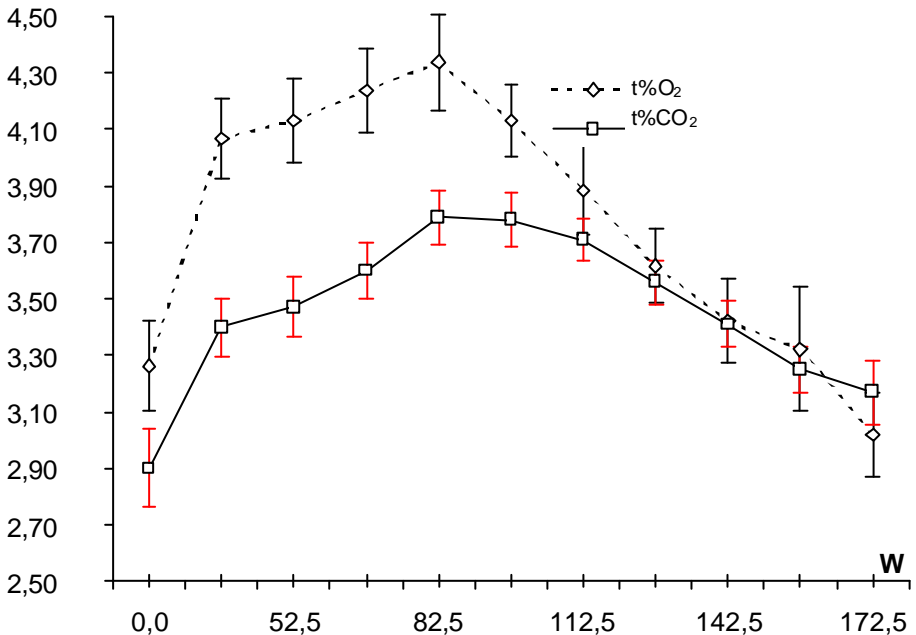


***Fig. 2.*** *Percentage differences in oxygen ($t\%O_2$) and in carbon dioxide ($t\%CO2$) content between the inhaled and exhaled air*

Individual values of acid-base balance indices were within normal limits (see table 3). Mean pre-exercise value of H concentration in blood was 44.7 nmol $\cdot$ l$^{-1}$, ranging from 42.7 to 49 nmol $\cdot$ l$^{-1}$, increasing to 58.6 nmol·l$^{-1}$ (range: 47.9 - 72.4 nmol $\cdot$ l$^{-1}$) post-exercise. This was accompanied by decreases in base excess (BE) from -2.7 mmol $\cdot$ l$^{-1}$, ranging from -1.2 to -4.6 mmol $\cdot$ l$^{-1}$ at rest to -9.3 – -20.6

post-exercise. Mean post-exercise lactate concentration was 9.5 mmol $\cdot$ l$^{-1}$ (range: 6.7 – 14.5 mmol $\cdot$ l$^{-1}$).

**Table 3**

*Biochemical variables measured at rest and after a graded exercise performed until exhaustion by male youth with spastic paresis of lower limbs (n = 16)*

| Variables | Rest | | 3 min | |
|---|---|---|---|---|
| | $\overline{x}$ | SD | $\overline{x}$ | SD |
| H$^{+}$ (nmol $\cdot$ l$^{-1}$) | 44.7 | 1.6 | 58.6 | 5.9 |
| BE (mmol $\cdot$ l$^{-1}$) | -2.7 | 0.9 | -14.2 | 2.9 |
| La (mmol $\cdot$ l$^{-1}$) | 1.7 | 0.6 | 9.5 | 2.6 |

## *Discussion*

The increase in lactate concentration at VT brings about an intensified buffering action and, in consequence, a displacement of $CO_2$ from plasma; the partial pressure of $CO_2$ increases which results in hyperventilation (Skinner and Mc Lellan 1980; Wasserman 1984; Wasserman *et al.* 1986). The pressure of carbon dioxide (pCO$_2$) and the percentage difference in $CO_2$ content between the inhaled and exhaled air remain constant, whereas the percentage of $O_2$ decreases. Parallel increases in $V_E$ and $VCO_2$ at and above the threshold level together with constant $V_E \cdot VCO_2^{-1}$ and $CO_2$ pressure, and increased $V_E \cdot VO_2^{-1}$ and $O_2$ pressure, are referred to as isocaping buffering (Wasserman 1984).

Further increase in intensity brings about a fast accumulation of hydrogen ions followed by an increase in ventilation faster than expected from the production of carbon dioxide (Wasserman 1984).

The respiratory compensation of metabolic acidosis is reflected in increases in respiratory equivalents $V_E \cdot VO_2^{-1}$ and $V_E \cdot VCO_2^{-1}$. At peak effort, the former slightly exceeds the resting value, the latter one is close to it.

Threshold HR values seem to be relatively low, especially in light of reports that the VT in healthy children occurs at 160 – 180 bpm (Skinner and McLellan, 1980) or at about 180 bpm (Kindermann *et al.*, 1979). Such a high heart rate threshold reflects exercise intensity at which lactate concentration equals 4 mmol $\cdot$ l$^{-1}$ (Wasserman 1984). In the light of our data (Piskorz and Klimek-Piskorz 1994, Klimek-Piskorz and Piskorz 1997) and reports of other authors (Van der Woude *et al.* 1977; Bhambhani *et al.* 1993; Keyser *et al.* 1993), VT in disabled people is associated with about 150 - 165 bpm or 85% of HR$_{max}$. Oxygen uptake at the threshold load was 1.71 l $\cdot$ min$^{-1}$ or 30.1 $\pm$ 5.3 ml $\cdot$ min$^{-1} \cdot$kg$^{-1}$ or 70% VO$_2$max. Similar values were reported by other authors (Aunola and Rusko 1984;

Gaesser *et al.*1984; Bhambhani and Singh 1985; Davis 1985; Henritze *et al.*1985; Reybrouck *et al.*1985; Emons and van Baak 1993; Keyser *et al.*1993), who regard 60 - 70% of $VO_{2max}$ to be typical for non competitive athletes.

Mean threshold load amounted to $96.6 \pm 12.8$ W or $1.71 \pm 0.34$ W $\cdot$ kg$^{-1}$ which corresponded to a moderate level.

In subjects with spastic paresis of lower limbs, undergoing laboratory exercise, especially a maximum one, upper limbs are usually engaged. Maximum oxygen uptake recorded in arm exercise is by about 20 % lower than in leg exercise (Piskorz and Klimek-Piskorz 1998) due to a smaller mass of muscles involved. McArdle and Magel (1970), and Boileau *et al.* (1977) point to the fact that muscle tension during a high resistance cycle ergometer exercise may, to some extent, reduce the blood flow, thus engaging the anaerobic mechanism to a greater degree. This view is supported by higher values of RQ in equivalent workouts on a treadmill and cycle ergometer. Also, the hierarchy of recruitment of various types of muscle fibres is of importance as it was demonstrated that an increasing power output recruited more Type II fibres (Sargeant and Beelen 1993). According to Coyle *et al.* (1992), Type I fibres are characterised by a higher mechanical efficiency, i.e. lower oxygen uptake at a given power output than Type II fibres. Thus, a larger number of Type II fibres are engaged, they contract with greater power and with greater contribution of anaerobic processes. According to Essen *et al.* (1975), Bar-Or *et al.* (1980) and Tesch (1980), those muscle fibres are predisposed to derive energy mainly from anaerobic metabolism due to the high activity of such enzymes as phosphorylase, phosphofructokinase, and lactate dehydrogenase. It should be emphasised that the tolerance of acid-base balance disturbances increases up to the age of 18 (Bar-Or 1983; Inbar and Bar-Or 1986). Nevertheless, muscle efficiency of disabled youth deteriorates and they achieve smaller work effects at the expense of considerable homeostasis disturbances, as shown by high concentrations of lactate following maximal exercise.

## *Conclusions*

Defining the VT threshold not only in terms of $VO_{2max}$ and $HR_{max}$ but also as a function of the load applied (W $\cdot$ kg$^{-1}$), may be of practical value when designing programmes aimed at specific adaptation of youth with spastic paresis of lower limbs to the increasing demands posed by their school and professional environments.

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