Eur Respir J
1993, 6, 237–247

The large lungs of elite swimmers: an increased alveolar number?

J. Armour*, P.M. Donnelly**, P.T.P. Bye*

The large lungs of elite swimmers: an increased alveolar number? J. Armour, P.M. Donnelly, P.T.P. Bye.

ABSTRACT: In order to obtain further insight into the mechanisms relating to the large lung volumes of swimmers, tests of mechanical lung function, including lung distensibility (K) and elastic recoil, pulmonary diffusion capacity, and respiratory mouth pressures, together with anthropometric data (height, weight, body surface area, chest width, depth and surface area), were compared in eight elite male swimmers, eight elite male long distance athletes and eight control subjects. The differences in training profiles of each group were also examined.

There was no significant difference in height between the subjects, but the swimmers were younger than both the runners and controls, and both the swimmers and controls were heavier than the runners. Of all the training variables, only the mean total distance in kilometres covered per week was significantly greater in the runners. Whether based on: (a) adolescent predicted values; or (b) adult male predicted values, swimmers had significantly increased total lung capacity ((a) 145±22%, (mean±SD) (b) 128±15%); vital capacity ((a) 146±24%, (b) 124±15%); and inspiratory capacity ((a) 155±33%, (b) 138±29%), but this was not found in the other two groups. Swimmers also had the largest chest surface area and chest width. Forced expiratory volume in one second (FEV_1) was largest in the swimmers ((b) 122±17%) and FEV_t as a percentage of forced vital capacity (FEV_1/FVC)% was similar for the three groups. Pulmonary diffusing capacity (D_{LCO}) was also highest in the swimmers (117±18%). All of the other indices of lung function, including pulmonary distensibility (K), elastic recoil and diffusion coefficient (K_{CO}), were similar.

These findings suggest that swimmers may have achieved greater lung volumes than either runners or control subjects, not because of greater inspiratory muscle strength, or differences in height, fat free mass, alveolar distensibility, age at start of training or sternal length or chest depth, but by developing physically wider chests, containing an increased number of alveoli, rather than alveoli of increased size. However, in this cross-sectional study, hereditary factors cannot be ruled out, although we believe them to be less likely.

Eur Respir J., 1993, 6, 237–247.

* Institute of Respiratory Medicine, Royal Prince Alfred Hospital, Camperdown, NSW, Australia.
** University of Sydney, Sydney, NSW, Australia.

Correspondence: P.M. Donnelly
Institute of Respiratory Medicine
Royal Prince Alfred Hospital
Camperdown NSW 2050
Australia

Keywords: Alveolar distensibility
chest enlargement
diffusion coefficient
growth hormone
lung growth
respiratory mouth pressures
swimmers' lungs

Received: November 5 1991
Accepted after revision September 12 1992

Lung volume is fairly well predicted on the basis of age, height and weight, but lung volumes which are greater than predicted have been repeatedly observed in swimmers [1]. This characteristic of swimmers has, largely, been attributed to genetic endowment [2], or to increased values for inspiratory mouth pressure [3] implying that swimmers can distend their lungs more than non-swimmers. However, longitudinal studies have suggested that swimming itself may be responsible for the increased lung size [4–6], and recent reports of normal inspiratory mouth pressure in swimmers, have suggested that the large lung volume found in swimmers is not due to increased inspiratory muscle strength [7].

Increased lung size in association with normal lung mechanics can occur with environmental or hormonal stress, including swimming [7], exposure to high altitude [8], hypoxia [9] and in subjects with high levels of circulating growth hormone [10], but the mechanism(s) for these findings has not been fully elucidated. The aim of this study was to examine tests of gas exchange and mechanical lung function (in particular pulmonary diffusing capacity, lung distensibility and respiratory mouth pressures) in swimmers, in order to determine whether the larger lung size was likely to have been achieved through a process of alveolar hypertrophy or by an increase in alveolar number.

Subjects

Subjects were male national level competitors (eight swimmers, seven long distance runners and one marathon walker) and were chosen for suitability by the state coaching director in each sport. Eight control subjects were not, and had not been, involved in any sort of intensive athletic training previously. All subjects were nonsmokers, and none had any history of recurrent respiratory illness, such as asthma or chronic cough. Details of the athletes training schedule, years of training, age at start of training and average distances per week were recorded. Subjects were not considered eligible for the study if they had experienced a training break of greater than 6 months during their athletic career. They were informed of the experimental requirements prior to attendance and signed consent forms on arrival for testing. They were made aware that they could discontinue the testing at any time.

Methods

Tests/facilities and equipment

The following tests were conducted by the same investigator on all subjects using the same equipment for the duration of the study:

Height, weight and body composition. Fat free mass (FFM) was determined using skinfold measurements as outlined by TELFORD *et al.* [11] and DURNIN and WOMERSLEY [12]. Body surface area (BSA) was determined using the Dubois Body Surface Chart (Boothby & Sandiford cited by FOX and MATHEWS [13]. Body density was calculated from skinfold measurements as outlined by SLOAN and DE WEIR [14]. Fat free mass was calculated as percentage lean mass: FFM = body weight (kg) × (100 - % body fat)/100.

Thoracic wall dimensions. These dimensions were external chest measurements, using the technique outlined by SCHRADER *et al.* [15]. One investigator performed all measurements which included: 1) sternal length; 2) thoracic width measured at the level of the xyphoid process; and 3) thoracic depth, *i.e.* the anteroposterior diameter measured in a horizontal plane at the junction of the manubrium and sternum.

Measurements 2 and 3 were performed at full lung inflation (total lung capacity (TLC)), at the end of normal expiration (functional residual capacity (FRC)), and at the end of a maximal expiration (residual volume (RV)). All measurements were made at least in triplicate, *i.e.* until the differences between corresponding recordings were less than 1.0 cm. Average values were used for further analyses. Chest surface area (CSA) (cm^2) was calculated using the formula:

$$CSA = \pi (r_1^2+r_2^2) + \pi (r_1+r_2) \sqrt{h^2 + (r_2-r_1)^2}$$

where h is the length of the sternum, r_1 half the thoracic depth and r_2 half the thoracic width. Chest surface area was computed for the three levels of lung inflation.

Spirometry. The forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were measured using a Model S Vitalograph wedge bellows spirometer. Repeat manoeuvres were performed until at least two recordings reproducible to 50 ml were obtained, from which the best effort was recorded and corrected to body temperature and pressure saturated with water vapour (BTPS).

Flow volume curves. Peak expiratory flow rate (PEFR) and forced expiratory flow when 50% of vital capacity has been exhaled (FEF_{50}) were measured using a Medscience wedge spirometer, with internal correction of volumes to BTPS. Signals of volume and flow rate were recorded on a memory oscilloscope and photographed for permanent record. The test was repeated until two reproducible flow volume curves were obtained (shape and volume), from which the best values were recorded.

Lung volumes. FRC, inspiratory capacity (IC), and relaxed vital capacity (VC) were measured in a Gould 2800 pressure body plethysmograph, after internal temperature stabilization had occurred. Immediately after measurement of thoracic gas volume the subjects inspired maximally, and the inspired volume was added to the thoracic gas volume to obtain TLC. RV was calculated from the difference between TLC and VC. Measurements were computed from the mean FRC with the largest VC and mean IC taken from 3–4 measurements of each parameter.

Maximum respiratory mouth pressures. Maximum inspiratory mouth pressure at RV (MIP_{RV}) and maximum expiratory mouth pressure at TLC (MEP_{TLC}) were recorded, using a hand-held pressure gauge calibrated to 0–350 cmH_2O. The gauge mouthpiece had a 1 mm air leak to prevent glottic closure during testing. Subjects were instructed to inhale and exhale with the glottis open and not to use their buccal cavity. Measurements of mouth pressure were repeated multiple times until values reproducible to 5 cmH_2O, held for at least one second, were obtained. Respiratory muscle force was calculated by multiplying respiratory mouth pressure by chest surface area at TLC and RV:

$$\text{Force} = P\ (kPa) \times CSA\ (m^2)$$

Alveolar distensibility. Static pressure-volume (P-V) data were generated during 8–12 interrupted deflations from TLC to FRC, with the subject seated in an Emerson volume plethysmograph. Transpulmonary pressure was measured with a one metre oesophageal balloon catheter (balloon length 10 cm, gas volume 0.5 ml) and a Hewlett Packard differential pressure transducer 267B. Several tidal volumes were recorded

to establish baseline volume before asking subjects to inspire fully to TLC. Transpulmonary pressure was measured during a maximum inspiratory effort, maintained for approximately one second at full inflation. The mean of the four highest values was recorded. After measurement of TLC and maximal elastic recoil, subjects were asked to relax against the occluded mouthpiece, which allowed pressure and volume just below TLC to be recorded with sufficient data points to provide an entire fitted curve.

Static deflation of the TLC to 50% below TLC was obtained over 1–2 s duration for each interruption, with lung deflation occurring as a result of passive elastic recoil. Up to five P-V curves, each with 7–10 data points, were pooled to produce a final curve. Curves that deviated significantly from the mean because of oesophageal spasm were excluded.

An exponential function of the form V = A-Be-Kp, where V is lung volume, P is static elastic recoil pressure and A, B and K are constants, was fitted to the P-V data from TLC to a lower volume limit not less than 50% of TLC (*e.g.* 50.8±3.9% TLC) (COLEBATCH and co-workers [16, 17]) and was analysed by computer. The exponential constant K describes the shape of the pressure volume curve independent of TLC. The constant A is the volume asymptote, and B is the difference between A and the volume at a zero recoil pressure. The distribution of the original P-V data about the derived curve was quantified by the ratio of residual variance to the total variance for volume (mean residual variance±SD = 2.2±1.3%). The ratio A/TLC% was 101.7±2.3% near TLC, indicating a good fit.

Single breath diffusing capacity. The diffusing capacity of the lungs (DLCO) at rest was measured by the single breath method of OGILVIE *et al.* [18], performed in duplicate after a five minute interval, using a Hewlett-Packard single breath diffusion system (HP4704A). The diffusion coefficient (KCO) was calculated as mean DLCO (standard temperature and pressure, dry (STPD)) divided by mean alveolar volume (BTPS) according to CRAPO and MORRIS [19]. Diffusion capacities were not corrected for haemoglobin levels.

Statistical calculations

One-way analysis of variance was conducted on all variables to determine the significance of difference between the three groups. Three *post hoc* tests were calculated, using the pooled variance to determine where the difference lay.

These tests were the Scheffe, Newman-Keuls and Bonferroni. A probability level of $p<0.05$ was considered to be statistically significant.

Correlation matrices were also determined for all variables and a p level <0.05 was considered to be significant. Thus, unless otherwise noted in the tables, significant differences were at the $p<0.05$ level.

Predicted values. The spirometric data of CRAPO and co-workers [20], POLGAR and PROMADHAT [21], the lung volume data of CRAPO and co-workers [22], POLGAR and PROMADHAT [21], the data of COTES [23] for PEFR and of CRAPO and MORRIS [19] for DLCO and KCO were used to calculate predicted values. The normal values for K and elastic recoil reported by COLEBATCH *et al.* [17] were used. Normal values for respiratory mouth pressures were from WILSON *et al.* [24]. Results for all subjects were expressed in absolute units and as percentages of the values predicted on the basis of age and height.

Results

Anthropometric - lung size correlations

Table 1 shows the age, physical characteristics, FFM, % body fat, BSA and body density for the three groups. The swimmers were significantly younger and had greater FFM and BSA than the runners. Both swimmers and controls were significantly heavier than the runners. There was no difference in height between the groups. The controls had a significantly larger % body fat than the runners and significantly smaller body density than both the runners and swimmers.

Table 1. – Anthropometric characteristics

	Group 1 Swimmers n=8	Group 2 Runners n=8	Group 3 Controls n=8	Significant differences between groups
Age yrs	18 (2.4)	24 (3.2)	22 (4.8)	1<2
Height cm	182 (9.9)	182 (5.6)	178 (4.9)	NS
Weight kg	83 (15.7)	66 (4.7)	80 (10)	1>2<3
FFM kg	72 (11.8)	60 (4.1)	67 (5.8)	1>2
% body fat	12.5 (2.6)	9.0 (1.0)	15.7 (5.1)	3>2
BSA cm^2	2.06 (0.2)	1.86 (0.1)	1.99 (0.13)	1>2
Body density	1.08 (0.01)	1.09 (0.00)	1.07 (0.02)	1>3; 2>3

Data are presented as mean and SD in parentheses. FFM: fat free mass; BSA: body surface area; NS: nonsignificant.

Table 2. – Correlations matrix for vital capacity and physical characteristics

	Vital capacity			
	All	Swimmers	Runners	Controls
Height	0.67#	0.74*	0.31	0.86#
Weight	0.71#	0.81*	0.54	0.59
Age	-0.05	0.64	-0.19	-0.53
Fat	0.22	0.51	-0.28	0.17
FFM	0.77#	0.84#	0.60	0.73*
BD	-0.26	-0.67	0.41	-0.26
BSA	0.74#	0.82*	0.49	0.60
CSA at TLC	0.76#	0.94#	0.27	0.69
CSA at FRC	0.76#	0.9#	0.05	0.67
CSA at RV	0.76#	0.82*	0.59	0.74*

*: $p<0.05$; #: $p<0.01$; CSA: chest surface area; FFM: fat free mass; BD: body density; BSA: body surface area; TLC: total lung capacity; FRC: functional residual capacity; RV: residual volume.

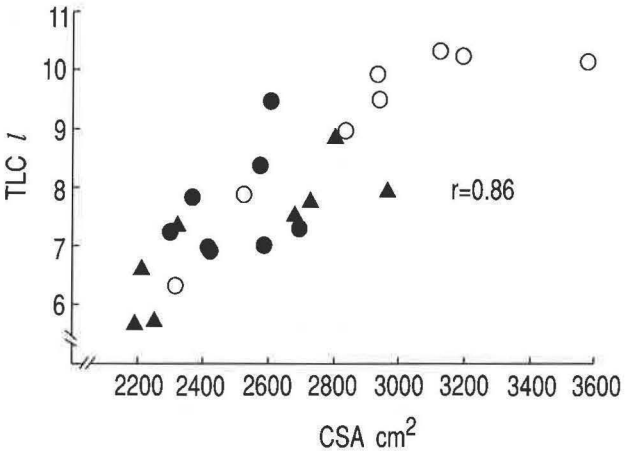

Fig. 1. – Chest surface area (CSA) measured at total lung capacity (TLC) in eight elite swimmers (○), eight elite runners (●) and eight control subjects (▲), plotted against their TLC measured by body plethysmography.

The relationship between VC and physical characteristics for all subjects and the correlations within groups is given in the table 2. Height, weight, FFM, BSA and CSA at TLC, FRC and RV were found to be significantly correlated with VC, when all group results were examined. When the VC was correlated with FFM, CSA at TLC and FRC, the coefficient was highly significant for the swimmers but not for either the runners or controls. The individual points of the relationship between the calculated CSA at TLC and the measured TLC for all subjects are given in figure 1. There is a close correlation between TLC and CSA with the large lungs of swimmers exhibiting the greatest CSA.

Swimmers were found to have significantly larger CSA at all lung volumes compared to runners (table 3). The swimmers also had a significantly greater chest width at TLC than the runners but there was no significant difference between sternal length or chest depth at TLC in any of the groups.

Table 3. – Thoracic wall dimensions, respiratory mouth pressures and respiratory force in swimmers, runners and control subjects

	Group 1 Swimmers n=8	Group 2 Runners n=8	Group 3 Controls n=8	Significant differences between groups
CSA at TLC cm^2	2924 (389)	2493 (136)	2629 (337)	1>2
CSA at FRC cm^2	2650 (384)	2252 (166)	2396 (306)	1>2
CSA at RV cm^2	2725 (390)	2316 (151)	2459 (264)	1>2
Sternal length cm	19.5 (2.7)	18 (1.9)	18 (2.5)	NS
Chest width at TLC cm	32. 4 (1.7)	31.1 (1.3)	31.9 (1.8)	1>2
Chest depth at TLC cm	19.1 (1.5)	17.6 (1.5)	18.6 (1.4)	NS
MEP_{TLC} kpa	17.9 (2.4)	17.1 (4.4)	18.4 (2.6)	NS
% pred	117 (5)	108 (26)	118 (16)	NS
MIP_{RV} kPa	14.4 (2.4)	14.1 (6.4)	14.3 (3.6)	NS
% pred	90 (17)	81 (41)	90 (22)	NS
Expiratory muscle force at TLC $kPa \cdot m^{-2}$	5350 (1190)	4338 (1029)	4942 (1212)	NS
Inspiratory muscle force at RV $kPa \cdot m^{-2}$	4006 (1177)	3335 (1680)	3590 (854)	NS

Data are present as mean and SD in parenthesis. MEP_{TLC}: maximum expiratory mouth pressure at TLC; MIP_{RV}: maximum inspiratory mouth pressure at RV. For further abbreviations see legend to table 2.

Table 4. – Training variables

	Swimmers	Runners	F value
Age at start of training yrs	11.0 (2)	12.0 (3)	3.47
Duration of training yrs	6.5 (1.9)	10.8 (5.2)	4.65
Mean training time·week^{-1} h	24.0 (10)	16.0 (53)	3.99
Mean distance·session^{-1} km	6.5 (1.1)	9.9 (4.3)	4.72
Mean total distance·week^{-1} km	69.4 (22.1)	114.0 (3.9)	8.08*

*: $p<0.01$.

Table 5. – Subdivisions of lung volume and spirometric measurements in swimmers, runners and control subjects

			Group 1 Swimmers n=8	Group 2 Runners n=8	Group 3 Controls n=8	Significant difference between groups
VC	*l*		7.26 (1.2)	5.87 (0.7)	5.70 (0.8)	1>2; 1>3
	% pred	(a)	146 (24)			
		(b)	124 (15)	102 (10)	103 (10)	1>2; 1>3
TLC	*l*		9.22 (1.5)	7.66 (0.9)	7.27 (1.0)	1>2; 1>3
	% pred	(a)	145 (22)			
		(b)	128 (15)	107 (13)	107 (7)	1>2; 1>3
RV	*l*		1.96 (0.4)	1.79 (0.5)	1.57 (0.3)	1>3; 2>3
	% pred	(a)	151 (37)			
		(b)	132 (26)	113 (31)	107 (16)	NS
RV/TLC	%		21.3 (2.6)	23.3 (4.9)	21.6 (3.7)	NS
	% pred		109 (13)	108 (23)	104 (14)	NS
FRC	*l*		4.05 (0.6)	3.97 (0.8)	3.14 (0.5)	1>3; 2>3
	% pred	(a)	137 (23)			
		(b)	117 (16)	116 (24)	98 (12)	1>3; 2>3
IC	*l*		5.17 (1.1)	3.70 (0.4)	4.13 (0.8)	1>2; 1>3
	% pred	(a)	155 (33)			
		(b)	138 (29)			
FVC	*l*		7.20 (1.2)	5.89 (0.8)	5.55 (0.6)	1>2; 1>3
	% pred	(a)	149 (23)			
		(b)	122 (20)	106 (10)	112 (17)	1>2; 1>3
FEV_1	*l*		5.98 (1.0)	5.09 (0.6)	4.55 (0.6)	1>3
	% pred	(a)	131 (16)			
		(b)	122 (17)	107 (12)	101 (19)	1>3
FEV_1/FVC			83.0 (7.5)	86.5 (3.2)	81.9 (5.4)	NS
	% pred		99 (8)	104 (4)	98 (6)	NS
FEF_{50}	$l \cdot s^{-1}$		6.95 (1.5)	5.79 (1.1)	5.7 (1.4)	NS
	% pred	(b)	105 (23)	91 (19)	91 (22)	NS
PEFR	$l \cdot s^{-1}$		11.69 (1.2)	10.55 (1.8)	10.67 (1.3)	NS
	% pred		116 (11)	106 (18)	109 (18)	NS

Data are presented as mean and SD in parenthesis. All subdivisions of lung volume for swimmers are presented: (a) as % predicted for an 18 yr old adolescent male; and (b) as % predicted for a 20 yr old adult male. This was done in order to allow for the possibility of there being a difference between physical development and chronological age. VC: vital capacity; IC: inspiratory capacity: FVC: forced vital capacity; FEV_1: forced expiratory volume in one second; FEF_{50}: forced expiratory flow when 50% of VC has been exhaled; PEFR: peak expiratory flow rate. For further abbreviations see legend to table 2.

Training variables. The differences in training variables between the athletes and swimmers are listed in table 4. The only variable which was significantly different between groups was that of mean total distance covered per week, with the runners covering 45 km more than the swimmers. However, the quantitative significance of this variable between athletes and swimmers is debatable. There was no difference in the age at start of training, years of training, training time per week, or distance per session.

Table 6. – Diffusion capacity, diffusion coefficient, alveolar distensibility and elastic recoil of swimmers, runners and controls

		Group 1 Swimmers n=8	Group 2 Runners n=8	Group 3 Controls n=8	Significant differences between groups
DLCO	mmol·min^{-1}·kPa^{-1}	17.7 (2.8)	14.9 (1.6)	13.2 (1.9)	1>2; 1>3
	% pred	117 (18)	102 (13)	93 (11)	1>3
KCO	mmol·min^{-1}·kPa^{-1}·l^{-1}	2.00 (0.32)	1.93 (0.21)	1.86 (0.15)	NS
	% pred	93 (15)	92 (11)	88 (8)	NS
K	kPa^{-1}	1.12 (0.20)	1.02 (0.20)	1.11 (0.20)	NS
	% pred	97 (20)	86 (16)	93 (18)	NS
Pel_{100}	kPa	4.31 (0.76)	4.75 (1.63)	4.02 (0.95)	NS
	% pred	102 (17)	124 (62)	93 (17)	NS
Pel_{90}	kPa	2.15 (0.25)	2.35 (0.82)	2.12 (0.52)	NS
	% pred	117 (15)	132 (42)	117 (26)	NS
Pel_{60}	kPa	0.95 (0.12)	0.99 (0.27)	0.99 (0.30)	NS
	% pred	118	137	126	NS

Data are presented as mean and SD in parenthesis. DLCO: diffusion capacity of the lung for carbon monoxide at rest; KCO: carbon monoxide diffusion coefficient (DLCO STPD/alveolar volume (VA) BTPS). K: alveolar distensibility; Pel_{100}: elastic recoil at 100% TLC; Pel_{90}: elastic recoil at 90% TLC ; Pel_{60}: elastic recoil at 60% TLC; TLC: total lung capacity; STPD: standard temperature and pressure, dry; BTPS: body temperature and pressure, saturated; NS: nonsignificant.

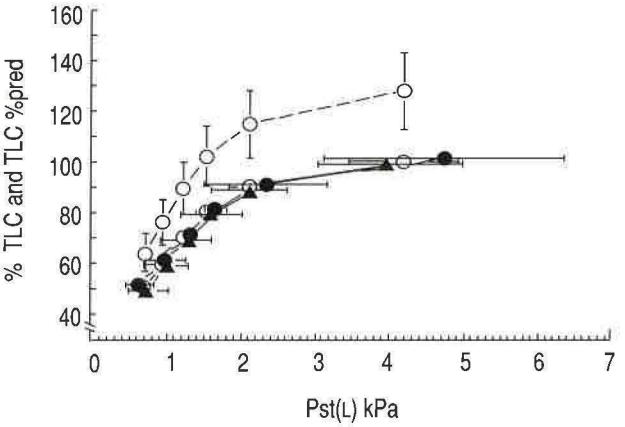

Fig. 2. – Static expiratory pressure-volume curves for eight elite swimmers (○—○), eight elite long distance runners (●—●) and eight control subjects (▲—▲), volume expressed as percentage measured total lung capacity (TLC). Volume is expressed as percentage predicted total lung capacity (TLC %pred) for swimmers only (○- - -○). Pst(L): static recoil pressure of the lung. Each bar represents±1 SD.

Spirometry and lung volumes. Mean subdivisions of lung volumes and spirometric function are listed in table 5. The swimmers (Group 1) had significantly larger values for nearly all subdivisions of lung volume, both in absolute volumes and in % predicted, when compared to both runners (Group 2) and controls (Group 3), except for RV% predicted, RV/TLC ratio and RV/TLC ratio % of predicted value. These differences persisted whether adolescent (a) or adult (b) predicted values were used. The FRC and RV of swimmers was not significantly different from the FRC and RV found in the runners.

The swimmers were found to have significantly larger FVC, both as absolute and as % predicted, than either runners or controls (table 5). Swimmers were also found to have significantly larger FEV_1, both as absolute and as % predicted, than controls. There was no significant difference in the FEV_1/FVC ratio, FEF_{50} and PEFR, both as absolute and as % predicted, between the groups.

Respiratory mouth pressures. There was no significant difference in inspiratory or expiratory respiratory mouth pressures or forces at TLC or RV between the groups (table 3).

Gas exchange and lung mechanics. Diffusing capacity, diffusion coefficient, alveolar distensibility and elastic recoil are shown in table 6. The swimmers had the highest values for DLCO, being higher than both the runners and controls. However, when DLCO was expressed per unit of lung volume (KCO) there was no difference between the groups. Mean values for K were similar for all three groups, indicating that all groups had a similar degree of alveolar distensibility. The shape of the pressure-volume curves and the elastic recoil at 100, 90 and 60% TLC were not significantly different and thus correlated with our finding of similar alveolar distensibilities for the three groups studied (fig. 2).

Discussion

Many investigators have reported lung capacity to be higher in champion swimmers than in other athletes and in the non-swimming population. Longitudinal studies of child, adolescent and young adult swimmers have shown an enhancement of lung volumes that cannot be ascribed to normal growth or development [5, 6, 25]. It has been suggested that increased

respiratory muscle strength [5], or perhaps alveolar expansion or hyperplasia [5] are responsible for these changes in lung volume but the mechanism for this increase in lung size has not been elucidated. To this end, measurement of the alveolar distensibility of the lungs in swimmers was undertaken as part of a detailed analysis of mechanical lung function. Physiological predictors such as height, fat free mass and respiratory mouth pressures were not found to explain the difference in lung size between swimmers, athletes and controls. In this study in swimmers, the increased lung volumes were not due to an increase in alveolar distensibility and may, therefore, have been due to an increased alveolar number in association with their physically larger chests.

The technique of determining alveolar distensibility by applying an exponential function to a static compliance curve was first proposed by SALAZAR and KNOWLES [26], and further elaborated by PENGELLY [27] and COLEBATCH *et al.* [16]. The exponential constant "K" has been shown to be related to the mean linear intercept (L_M), a morphometric estimate of the mean size of peripheral airspaces at maximal inflation, in rats, cats and dogs [28], and in humans, including normal subjects, smokers [29] and those with emphysema [30]. In the present study, there was no significant difference in alveolar distensibility between swimmers, runners and controls, suggesting either: 1) alveolar multiplication and not hypertrophy as the growth mechanism to account for the larger lungs of swimmers; 2) alveolar hypertrophy, not detectable through the measurement of pulmonary distensibility; or 3) the swimmers were endowed with the potential for a high number of alveoli from early childhood, enabling them to develop big lungs, although we think this proposal is less likely. Detailed discussion on the relationship between chest size, alveolar distensibility, and alveolar size and number is covered in an earlier publication [31].

For some time it was accepted that the number of alveoli in the human lung was fairly constant by the eighth year at 296×10^6 [32]. Research by ANGUS and THURLBECK [33], however, suggested that the number was more variable. Their results showed a range of 212×10^6 to 605×10^6 alveoli, and that the number of alveoli correlated with body length or lung volume, although there was a wide scatter of data. ANGUS and THURLBECK [33] had admitted the difficulties in counting alveoli and the relative superiority and ease in measuring L_M, the parameter upon which the interpretation of alveolar distensibility (K) in the present paper is based. They concluded that the precise age at which alveolar growth was constant should be regarded as an open issue.

ZELTNER and BURRI [34] described two phases of human lung growth. Phase I, lasting from birth to 18 months, was characterized by substantial structural remodelling, due to bulk alveolar formation and restructuring of septal morphology. By 1.5 yrs most septa show the adult structure. It appears that the lung entering the II growth phase represents a miniaturized version of the adult lung. Zeltner and Burri commented that they had no data as to the age at which lung growth stops but that, in view of the linear relationship between lung volume and body mass, it made sense to assume that normal lung growth is going to end when body growth stops. In the immediate postnatal period, alveolar formation may prevail, whereas later on, increases in surface area may be due to septal growth appearing in sections such as lengthening of the septa or deepening of the alveoli. In relation to experiments on lung regeneration, BURRI *et al.* [35] suggested that it could indeed be shown that an increase in surface to volume ratio (S:V) of airspaces could be achieved by the latter mechanism alone, without the requirement of new alveolar formation. How this explanation equates with our measurement of normal alveolar size is conjectural. This mechanism [35], however plausible, refers to a model and gives no actual data on the surface to volume ratio (or L_M) of the lungs themselves. The model also showed that an initial decrease in the S:V after distension of the lungs might be followed by an increase in S:V owing to growth of septal tissue. Again, the model is plausible but it is difficult to believe that growth of septal tissue would completely abolish the decrease in S:V ratio produced by the initial distension.

Recent evidence suggests that lung growth in young adult males can continue into their early twenties, despite cessation of somatic growth [36], and there is also some morphological evidence that alveolar proliferation can occur in the adult lung. BOYDEN [37] refers to the formation of ductular alveoli in an adult lung being observed histologically by HAYEK [38]. In his book, Hayek shows a picture of spherical alveoli in the first respiratory bronchiole of an adult lung. Three different types of alveoli are shown in the same bronchiole; the first wholly lined by cuboidal epithelium, the second partly lined, and the third wholly lined by alveolar epithelium. Boyden accepted Hayek's hypothesis, that this may be a new way of producing alveoli.

In swimming, there is a large amount of upper body work. The stress of excessive upper body muscle contraction on the bones comprising the thoracic cavity may be a stimulus for growth of the chest wall. The swimmers in this study had a significantly larger chest surface area at TLC, FRC and RV than the runners. There was also a highly significant correlation between chest surface area and VC at all lung volumes. In this regard, the increased TLC found in Caucasians as opposed to Indians has been found to be related to the longer and wider chests of Caucasians [31]. The exact mechanism which causes growth of the chest wall is unknown, though it is clear that swimmers have a larger chest surface area to accompany their larger lungs. The association between increased growth hormone levels, increased thoracic size, and increased lung volume with normal mechanical properties in acromegaly, may provide a useful model for the study of lung growth in the adult lung [10, 39, 40].

This study did confirm the findings of other researchers, that the maximal inspiratory mouth pressures measured in swimmers did not explain their increased lung size [7]. However, during swimming, swimmers may go repeatedly to TLC during regular training and this may lead to an increased ability to contract their inspiratory muscles to shorter minimal lengths, without concomitant increases in maximal inspiratory mouth pressures [41, 7]. However, if this effect alone accounted for swimmers' larger VC one would not anticipate an increase in the FRC seen in our swimmers, which is consistent with the possibility that swim training *per se* enhances lung growth. A previous study from this laboratory showed that Caucasians with higher values for MIP_{RV} had values for alveolar distensibility similar to Indians and Chinese subjects with lower values for MIP_{RV} [31]. This implies that an increased ability to distend the thoracic cage does not necessarily lead to an increased distensibility of the alveoli.

Another of the stressors involved in swimming is hypoxia. In a recent study, it was found that in natives of high altitude there is an increase in chest dimensions and vital capacity, which is thought to be related to the hypoxic conditions experienced at altitude [8]. Both YAMAMOTO *et al.* [42] and STAGER *et al.* [43] measured arterial desaturation with controlled frequency breathing during simulated swimming exercise. Although the extent of periodic arterial desaturation during simulated swimming seems difficult to compare with permanent hypoxia at high altitude, a periodic hypoxic stimulus may be just as effective. However the degree of arterial desaturation required or even necessary to stimulate lung growth is unknown.

Synchronized swimmers perform strenuous underwater exercises during prolonged breathholds, and have been shown to have increased lung volumes, blunted hypoxic ventilatory responses, prolonged normoxic breathholding times and marked bradycardia during apnoea [44]. In this context, it has been found that exercising during hypoxia leads to increased serum growth hormone (GH) levels [39], and that the two are related [45]. Thus, considering the hypoxic nature of swimming and the enhanced growth hormone release with arm exercise [46], it is very possible that intensive swim training over the adolescent growth spurt, is capable of eliciting a lung growth response of alveolar multiplication in response to an enlarging chest cavity.

In two "adult onset" conditions such as acromegaly [10] and hypothyroidism [40], the excessive serum growth hormone levels in acromegaly have caused proportional increases in chest wall and lung size [10], whereas in hypopituitarism with decreased growth hormone, lung size is small [40, 47]. In both conditions, elastic recoil and lung compliance have been found to be normal, suggesting normal alveolar distensibility. DE TROYER *et al.* [40] suggested that these findings indicate an influence of GH on lung volume in adult man, and that the loss of GH secretion is likely to be responsible for the restrictive ventilatory impairment associated with hypopituitarism. Unpublished data from this laboratory (in preparation) have shown pulmonary distensibility (K) to be normal in a large group of nonsmoking acromegalic subjects with large lungs and increased serum GH levels. In the case of acromegaly, serum GH levels have been found to be similar to those found in untrained normal male subjects during exercise [39, 46].

MOSTYN *et al.* [48] suggested that champion swimmers have a high diffusing capacity, as they must transfer large amounts of oxygen across the lung when the alveolar oxygen tension (Pao_2) has fallen to low levels. The swimmers in this study had a significantly increased diffusing capacity at rest, *i.e.* 117% predicted, which was similar to the 113% predicted found by other researchers [49]. However, in this study, when DLCO was expressed relative to alveolar volume (KCO) there was no significant difference between the groups. This would suggest that the pulmonary capillary blood volumes in each of our three study groups was proportional to their alveolar volumes at rest, and supports the hypothesis that the increased alveolar volume in swimmers is due to an increase in alveolar number and not to an increased alveolar size. Similar values for KCO in swimmers and non-athletes have been reported by ANDREW *et al.* [25].

One would assume that the age at which swim training began would also be important in terms of the lungs' growth response. In an important longitudinal study of young swimmers and control subjects (8–15 yrs old) BLOOMFIELD *et al.* [4] found significant increases in FEV_1 and FVC only in the swimmers. This became apparent by pubescent stage 2 (12 yrs) in boys and by pubescent stage 4 (13 yrs) in girls. The increased lung capacity was greater in the males than females. A similar finding has been observed in acromegalic males [10, 50]. There is some evidence to suggest that testosterone, in conjunction with GH, is necessary to stimulate bodily growth [51] and this may apply to the lungs also. One study has shown that significant increases in lung volumes are possible in 19 yr old females after 12 weeks of swim training [5]. In the current study there was no significant difference in the age at initial training between runners and swimmers, implying that swim training *per se* is an important factor in lung growth, although the low numbers studied may have influenced this finding.

In adolescent males, it is sometimes difficult to choose representative predicted values for lung volumes, because of the difference between physical maturity and chronological age. However, even when adult [20], instead of childrens' [21], predicted values were substituted in our young swimmers, the mean vital capacity was reduced but still remained significantly above normal at 128% predicted. This was similar to the mean vital capacity of 131% predicted, previously reported in 10 elite adult male swimmers [52]. Thus, the finding of large volumes in our swimmers was real and not merely due to the application of inappropriate predicted values.

Our interpretation of the finding of normal expiratory flow rates in the swimmers in this study is at variance with the interpretations of BRODY *et al.* [53] and BRODY and VACCARO [54]. In our swimmers, we found that both airways and parenchyma were proportionately enlarged, implying that both airways and parenchyma participated in the "growth spurt". It might be argued, however, that since airways form in early foetal life, it would seem reasonable to assume that in our swimmers, the airways and parenchyma maintained their normal relationship because both large airways and lungs were genetically determined. There are several possibilities to explain our finding:

1) Airway calibre (PEFR, FEV_1) has been shown to increase after training in adolescent athletes but not in mature athletes (*e.g.* rowers) [55]. We have observed the maximal expiratory flow volume envelope of adolescent boys to increase significantly after submaximal work on a bicycle ergometer (unpublished observations). Perhaps, persistent training in young athletes can result in a permanent increase in airway calibre.

2) The higher FEV_1/FVC ratio observed by BRODY *et al*, [53] in their lowland Peruvian natives, as distinct from their ethnically similar highland group, may be more a reflection of delayed growth (including the lung) as a result of the lower protein reserves [56] and perhaps a lower FFM [57] in the lowland group. This may have caused the lowland group to have a smaller parenchymal mass emptying into airways of normal calibre [58], resulting in the very high FEV_1/FVC ratios reported. Also, low protein reserves could result in the lowland group having lower MEP_{TLC} than the highland group, with resultant reduction in FVC and increased FEV/FVC ratio. On the other hand, the highland cohort had a more normal FEV_1/FVC ratio, similar to our swimmers, perhaps reflecting parallel airway and parenchymal adaptation to high altitude, rather than parenchymal adaptation alone. In a recent study [31], we reported lung volumes and chest dimensions in Chinese men closely approximating Caucasian values, and speculated that the next generation of Chinese will have similar lung capacities to Caucasians, probably as a result of better nutrition and more exercise.

3) CLANTON *et al.* [5] reported increased TLC, FRC, VC and FEV_1 in 19 yr old girl swimmers after 12 weeks training but not in a control group. They demonstrated that all of the changes in FEV_1 could be attributed to the increase in TLC and that the time constant for lung emptying was approximately the same for both groups.

4) The similar FEV_1/FVC ratios found in both acromegalic and hypopituitarism patients implies that parallel changes in both parenchyma and airways can occur in the adult state with hormonal stress [40], and that GH levels may be important in maintaining lung growth throughout life.

There were significant differences between the groups in terms of chest surface area and width, diffusing capacity and alveolar volume. Without a longitudinal study, it is impossible in the present study to determine whether these changes in lung volumes can be attributed to inheritance or training, as it may be that individuals will only become elite swimmers if they have the potential to grow or recruit extra alveoli through swimming, thus making the sport self selective. However, this seems unlikely, since it is quite obvious that most competitive swimmers at all levels possess large chests and, therefore, probably large lungs. Secondly, longitudinal studies have demonstrated accelerated increase in FEV_1, VC [4] and TLC [5] in adolescent swimmers, whilst a control group experienced normal lung growth. Thirdly, the study of NESS *et al.* [59], failed to show differences in static lung capacities (VC, FRC, TLC) between "selected to train" girl swimmers, those unselected to train, and a control group with no interest in swimming. Overall, similar negative findings in relation to lung size were observed amongst the childrens' parents.

Conclusion

This study has confirmed that swimmers possess significantly larger lungs than both controls and runners. These larger lungs could not be attributed to changes in height, fat free mass, maximal respiratory mouth pressures, alveolar distensibility, age at start of training, years of training, training time per week, distance per session, sternal length, or chest depth at TLC, between groups. What has been established is that swimmers have the same alveolar distensibility as runners and controls and may have achieved greater lung volumes by increasing the number of alveoli, rather than the size. This finding may have important implications for children with lung disease or following pneumonectomy. With further longitudinal studies, using a larger number of subjects, together with assessment of swimming exercise performance, growth hormone response and alveolar distensibility (K), it is hoped that the mechanisms behind the increase in total lung size can be identified. Morphological and morphometric studies of the lungs of swimmers, high altitude natives and acromegalic subjects are required.

Acknowledgements: The authors wish to acknowledge B. Lee for processing the manuscript, J. Reynolds for preparing the figures, S. Anderson and J. Colebatch for their helpful comments.

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