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Estimation of the bronchodilatory effect of deep inhalation after a free run in children

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ABSTRACT: The bronchomotor effects of a deep inhalation (DI) may provide relevant information about the mechanisms of exercise-induced airway obstruction in children and may be assessed by respiratory conductance (*G*rs) measured using the forced oscillation technique. The aims of the present study were to assess the effect of DI on *G*rs after exercise in relationship to the lung function response to exercise.

_G_rs at 12 Hz using a head generator and spirometric data were measured in 62 children suspected of asthma before and 5 min after a 6-min free run.

After exercise, *G*rs was significantly increased by DI in 38 subjects, who also showed larger *G*rs and forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) responses to exercise than the 24 nonresponders. Stepwise regression indicated significant correlation between the response of *G*rs to DI and both *G*rs and FEV_1/FVC responses to exercise.

The data are consistent with exercise-induced bronchoconstriction being reversed by deep inhalation.

KEYWORDS: Childhood asthma, exercise-induced bronchial obstruction, lung function measurements, respiratory impedance

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Exercise is a major cause of acute airway obstruction in asthmatic children [1], and exercise-induced airway obstruction (EIAO) a specific indicator of active asthma disease [2]. The forced oscillation technique (FOT) is increasingly being used in children as it is noninvasive and requires little active cooperation [3]. Respiratory resistance (*R*rs) or its reciprocal, respiratory conductance (*G*rs), derived from the measured respiratory impedance (*Z*rs), has the potential to describe the bronchial response to exercise. However, few data are available regarding the *G*rs response to exercise in children in the routine laboratory. *G*rs measured at frequencies above a few Hertz is thought to express airway conductance to flow [4–6], and, in describing this response to exercise, it is important to ensure that the observed decrease in *G*rs after exercise truly reflects a decrease in airway calibre at the bronchial level. Since the 1980s, the effects of deep breaths on airway mechanics have been the object of intensive research [7, 8], and have potentially important applications in the lung function laboratory [9]. The current consensus is that pharmacologically induced bronchoconstriction can be reversed by a deep inhalation (DI) as a result of the stretching of those airways subjected to parenchymal tethering. As a corollary, a significant increase in *G*rs after a DI could be taken as evidence for the relief of EIAO at this level. There are only few studies on the bronchomotor effects of DI in EIAO [10, 11], and their correspondence with the *G*rs response to exercise has not been established. When using a single excitation frequency, the FOT offers a fairly simple way of tracking *G*rs during breathing, thereby providing an estimate of the effect of DI on airway calibre [5, 11], provided the breath-by-breath variability of *G*rs is taken into account.

The aims of the present study were to assess the *G*rs response to exercise in relation to the effects of DI on airway calibre after exercise in children suspected of asthma. The effects of exercise were also monitored spirometrically. The hypothesis is that positive responses to exercise are associated with a significant increase in *G*rs after the DI.

MATERIAL AND METHODS

Subjects

Children aged 7–16 yrs (n=62; 39 male) were referred to the Laboratoire d'Explorations Fonctionnelles Pédiatriques (Hôpital d'Enfants, Vandoeuvre les Nancy, France) for lung function testing and evaluation of their airway response to exercise. Their baseline forced expiratory volume in one second (FEV_1) was $\geqslant 75\%$ of the predicted value [12]. The population was selected on the basis of the child's ability to complete the free running test and participate in the measurements

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of lung function, including a successful DI manoeuvre as described below. Forty-five children had doctor-diagnosed asthma, six recurrent cough and 11 dyspnoea on exertion. In 44 children, respiratory complaints were triggered or increased by exercise. Bronchodilator therapy was discontinued >12 h prior to the study, including in nine children on long-acting β_2-agonists. Twenty-three children were on inhaled steroids: budesonide $\leqslant$400 µg or fluticasone $\leqslant$200 µg daily in those aged <12 yrs, and budesonide $\leqslant$800 µg or fluticasone $\leqslant$500 µg daily in those aged >12 yrs. Five children were on montelukast 5 mg daily. The characteristics of the subjects and their baseline lung function data are reported in table 1. The protocol was approved by the ethical committee of the Centre Hospitalier Universitaire de Nancy (Vandoeuvre les Nancy, France), explained to the children and their parents, and informed consent obtained prior to exercise.

Measurements

Respiratory conductance

*Z*rs was measured using the head generator technique [13] in order to minimise upper airway wall motion. The measuring system (Pulmosfor; SEFAM, Vandoeuvre les Nancy, France) is in conformity with the recommendations issued by a European Respiratory Society task force [3]. The children wore a nose-clip and breathed through a mouthpiece connected to a Fleisch No. 1 pneumotachograph (Metabo, Epalinges, Switzerland). Sinusoidal pressure variation was applied at 12 Hz in the head generator.

Pressure and flow signals were low-pass filtered at 32 Hz using analogue filters and digitised at a sampling rate of 96 Hz. The breathing component in the signals was eliminated using a fourth order Butterworth high-pass filter with a corner frequency of 6 Hz. The Fourier coefficients (real and imaginary parts) of pressure (Re*P*, Im*P*) and flow (Re*V′*, Im*V′*) were computed and *R*rs calculated oscillation cycle by oscillation cycle according to NAVAJAS *et al*. [14]:

$$R_{rs}=(\mathrm{Re}P\cdot\mathrm{Re}V'+\mathrm{Im}P\cdot\mathrm{Im}V')/(\mathrm{Re}V'^2+\mathrm{Im}V'^2) \quad (1)$$

Twelve *R*rs measurements were thus provided every second. A filtering procedure was included in order to detect spurious data associated with rapid flow transients, a low signal-to-noise ratio or glottis closure [15]. Airflow, tidal volume (*V*T) and *R*rs were displayed immediately after each acquisition in order to allow visual inspection and selection of the data that were stored on disk. *G*rs was computed from the reciprocal of *R*rs during inspiration and averaged breath by breath. Therefore, in what follows, *G*rs refers to inspiration.

TABLE 1 Physical characteristics and baseline lung function of the children

Age yrs	11±3 (7–16)
Height m	1.45±0.15 (1.20–1.73)
FVC % pred	104±10 (85–141)
FEV1 % pred	102±11 (77–122)

Data are presented as mean±SD (range). FVC: forced vital capacity; FEV1: forced expiratory volume in one second; % pred: percentage of the predicted value.

TABLE 2 Respiratory conductance (*G*rs) and spirometric data before and after exercise

	Grs L·s^{-1}·hPa^{-1}	FEV1 L	FVC L	FEV1/FVC %
Baseline	0.213±0.067	2.41±0.69	2.79±0.86	87±6
Exercise	0.158±0.073#	2.15±0.71#	2.69±0.87¶	81±9#

Data are presented as mean±SD. FEV1: forced expiratory volume in one second; FVC: forced vital capacity. #: $p<0.0001$ *versus* baseline; ¶: $p=0.009$.

The children were first familiarised with the equipment and instructed to breathe calmly and regularly and to take a deep breath on demand. Once this was learnt, the children breathed quietly for 1 min through the pneumotachograph, avoiding taking a DI while *V*T was continuously displayed on the computer screen. *G*rs was then measured for 2 min as follows. 1) During the first minute, *G*rs was obtained during tidal breathing and used to assess variability and response to exercise. 2) During the second minute, after four to six tidal breaths, the children performed a DI and then resumed normal breathing. The initial end-point set for validating the DI was an inspired volume of $\geqslant$40% pred forced vital capacity (FVC).

Spirometry

Measurements of FVC and FEV1 were made by a trained and experienced technician using an electronic flow meter (Masterscope; Erich Jaeger, Würtzburg, Germany). The forced expiratory manoeuvre was explained to the children and trials were performed with the help of computer animation programs and repeated until at least two curves displaying an early rise to peak flow followed by a regular decrease throughout expiration were obtained, with FVCs within 5% of each other. This was usually obtained within five trials. The best curve was selected as the one with the highest sum FVC+FEV1.

Protocol

*G*rs was invariably measured prior to performing the spirometry. Duplicate measurements using each technique were

TABLE 3 Change (Δ) in lung function and response to deep inhalation after exercise# in children with negative and positive response to exercise by forced expiratory volume in one second (FEV1) or respiratory conductance (*G*rs)

	FEV1		Grs	
	Negative	Positive	Negative	Positive
Subjects n	26	36	27	35
ΔFEV1 %	-2±3	-17±13	-3±4	-17±14
ΔGrs %	-10±16	-37±19	-6±12	-41±15
Grs,DI	1.29±0.21	1.49±0.34¶	1.27±0.22	1.51±0.33+

Data are presented as mean±SD, unless otherwise stated. *G*rs,DI: post- to pre-deep inhalation (DI) *G*rs ratio. #: defined as positive when FEV1 or *G*rs decreases by at least 2 within-subject SDs (see Methods section); ¶: $p=0.008$; +: $p=0.002$.

made 10–15 min apart at baseline and are referred to as measurements A and B. The children were then asked to run outdoors for 6 min or until they perceived dyspnoea or chest tightness or started to cough. A fast running rate was encouraged until completion. The measurements were repeated 5 min following cessation of exercise.

Data analysis

Repeatability and response to exercise

Repeatability was expressed using the within-subject SD (SDw), *i.e.* the population SD of the differences between measurements A and B divided by the square root of 2 [16].

The response to exercise was expressed in terms of number of SDws (nSDw): the difference between exercise and baseline divided by SDw, where baseline is the mean of A and B. Responses to exercise were analysed by FEV1 and *G*rs and considered positive when the relevant nSDw was <-2. The responses to exercise were also expressed as percentage change from baseline.

*G*rs breath-by-breath variability

A parameter indicative of the spontaneous breath-by-breath variability was obtained that could be easily compared to that used in assessing the effect of DI (see below). The recording without DI after exercise was used to calculate the ratios of two successive breaths, *i.e.* for n breaths (n *G*rs): $G\mathrm{rs}_2/G\mathrm{rs}_1$, $G\mathrm{rs}_3/G\mathrm{rs}_2$, . . . $G\mathrm{rs}_n/G\mathrm{rs}_{n-1}$. The upper limit of the 95% confidence interval (CI) of these n values ($G\mathrm{rs}_{,95}$) was calculated for each recording and the whole population mean was computed ($G\mathrm{rs}_{,95,\mathrm{mean}}$).

*G*rs response to deep inhalation

The *G*rs response to DI was estimated as the *G*rs ratio of each breath following DI to the mean of the three or four breaths before DI. The maximal response to DI was defined as the largest ratio within the first three breaths immediately after the DI ($G\mathrm{rs}_{,\mathrm{DI}}$). A child was considered a DI responder when its $G\mathrm{rs}_{,\mathrm{DI}}$ was $>G\mathrm{rs}_{,95,\mathrm{mean}}$. In those DI responders, the duration of the effect of DI (t_{DI}) was also estimated as the time necessary for the ratio to return to within the pre-DI range.

Statistics

Statistical analyses were performed using ANOVA, simple regression and stepwise multiple regression analysis. Data are expressed as mean±SD, 95% CI or range. A difference was considered significant at $p<0.05$.

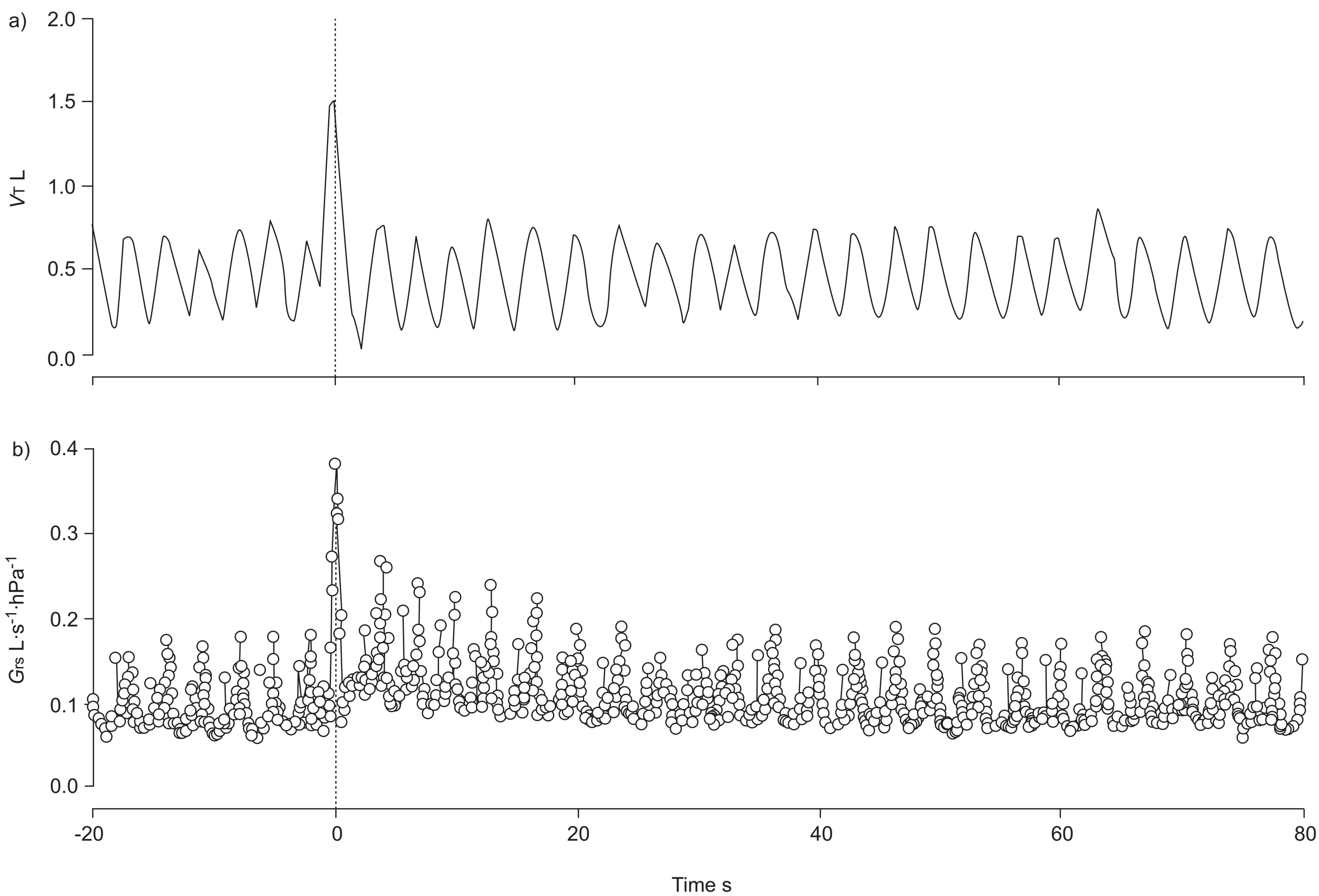

FIGURE 1. Bronchodilatory effect of deep inhalation after exercise; a) tidal volume (*V*T); and b) respiratory conductance (*G*rs). Within-breath variations in *G*rs are mainly related to tidal flow.

RESULTS

Repeatability of lung function and response to exercise data

The repeatability of measurements of *G*rs, FEV1, FVC and FEV1/FVC were characterised by SDws of 0.019 $L \cdot s^{-1} \cdot hPa^{-1}$, 0.06 L, 0.07 L and 2.8%, respectively. The corresponding mean coefficients of variation were 9, 3, 3 and 3%, respectively. Exercise was associated with a significant decrease in *G*rs, FEV1 and FEV1/FVC ($p<0.0001$) and FVC ($p<0.009$; table 2). Thirty-five children showed a decrease in *G*rs of <-2SDw and 36 a decrease in FEV1 of <-2SDw. Responses to exercise of *G*rs and FEV1 were concordant in 51 children, positive by *G*rs and negative by FEV1 in five, and negative by *G*rs and positive by FEV1 in six. *G*rs and FEV1 percentage changes from baseline after exercise are reported in table 3 in children classified as exercise responder and nonresponders by nSDw for *G*rs (nSDw,*G*rs) or FEV1 (nSDw,FEV1).

*G*rs *variability and response to deep inhalation*

*G*rs spontaneous within-breath variability after exercise was expressed as a *G*rs,95 of 1.13–1.52 (*G*rs,95,mean=1.26). The DI after exercise was at least 40% pred FVC in all but six children, in whom it ranged 30–40% pred FVC. The data in these children were nonetheless kept for analysis since they showed full cooperation at baseline, where DI amplitude, expressed as a percentage of either measured or predicted vital capacity, was ≥40%. Indeed, in the whole group, a significant decrease in DI amplitude was observed after exercise (1.44±0.44 L) compared to baseline (1.62±0.48 L, $p<0.0001$). An example of response to DI after exercise is shown in figure 1, showing a clear impact on airways mechanics. The *G*rs,DI was significantly greater when the response to exercise was positive by either FEV1 ($p=0.008$) or *G*rs ($p=0.002$; table 3). Overall, there were 24 DI nonresponders (*G*rs,DI=1.13±0.08) and 38 DI responders (*G*rs,DI=1.58±0.26). Responses to exercise by nSDw,*G*rs ($p=0.001$) and nSDw,FEV1/FVC ($p=0.004$) but not nSDw,FEV1 ($p=0.17$) or nSDw,FVC ($p=0.81$) were found to be significantly greater in DI responders than in DI nonresponders (fig. 2). The same trend was found with the percentage change in FEV1/FVC ($p=0.004$) and *G*rs ($p<0.0001$). In addition, there was also a borderline difference in FEV1 ($p=0.05$; table 4). The regression analysis indicated significant correlation between *G*rs,DI on the one hand and FEV1, FEV1/FVC and *G*rs responses to exercise expressed as nSDw or change from baseline on the other, as illustrated in figure 3. Stepwise regression analysis was performed in order to assess the relative contribution of each parameter of the response to exercise as determinant of *G*rs,DI. The latter was entered as the dependent variable and FVC, FEV1, FEV1/FVC and *G*rs responses to exercise, as either nSDw or percentage change, as independent variables. It was found that the only factors significantly associated with *G*rs,DI were *G*rs and FEV1/FVC responses to exercise, whether expressed as nSDw or percentage change. The partial correlation coefficients obtained with the stepwise regression are listed in table 5.

*t*DI in the 38 DI responders ranged 3–92 s (21±19 s) and was found to be independent of any parameter of the response to exercise by stepwise regression.

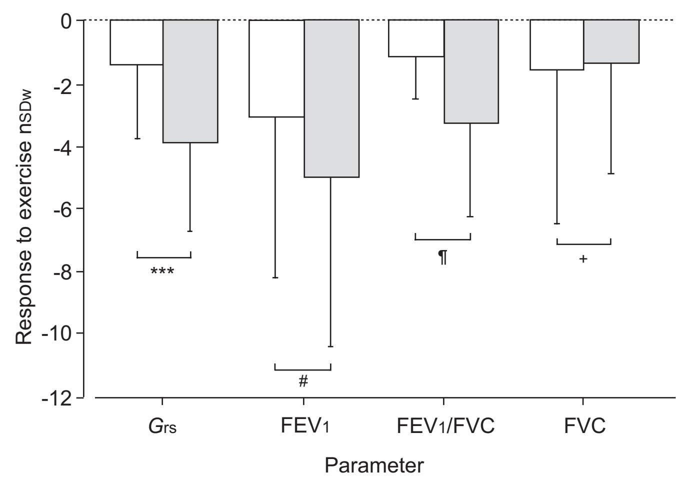

FIGURE 2. Spirometric and respiratory conductance (*G*rs) responses to exercise expressed as number of within-subject SDs (nSDw, see Methods section) in deep inhalation (DI) responders (■, n=38) and DI nonresponders (□, n=24). DI responders show a significantly greater response to exercise by *G*rs and forced expiratory volume in one second/forced vital capacity (FEV1/FVC) but not by FEV1 or FVC. ***: $p=0.001$; #: $p=0.17$; ¶: $p=0.004$; +: $p=0.81$.

DISCUSSION

To the best of the present authors' knowledge, this is the first study identifying bronchodilation by DI after exercise in children based on *G*rs breath-by-breath variability and demonstrating the association of this effect with the *G*rs response to exercise.

*R*rs or *G*rs measured using the FOT is increasingly used as an index of airway dimensions during breathing [4–6], and represents a potentially powerful tool for evaluating the bronchomotor effects of DI. Two significant physiological sources of *G*rs variability, however, should be minimised in order to specifically address this effect. Respiratory flow is a well-known determinant of *G*rs and it is important that ventilation should remain stable and regular, a condition that was required in the present study to measure *G*rs before and after the DI. It was also established, in a preliminary study, that

TABLE 4 Change# (Δ) in spirometric data and respiratory conductance (*G*rs) after exercise in children with negative and positive *G*rs responses to deep inhalation

	Negative	Positive
Subjects n	24	38
ΔFVC %	-3±10	-4±10
ΔFEV1 %	-7±11	-13±13*
ΔFEV1/FVC %	-3±4	-9±9¶
ΔGrs %	-12±19	-35±20+

Data are presented as mean±SD, unless otherwise stated. FVC: forced vital capacity; FEV1: forced expiratory volume in one second. #: percentage change from baseline for FEV1, FVC and *G*rs, and difference between exercise and baseline for FEV1/FVC; *: $p=0.05$; ¶: $p=0.004$; +: $p<0.0001$.

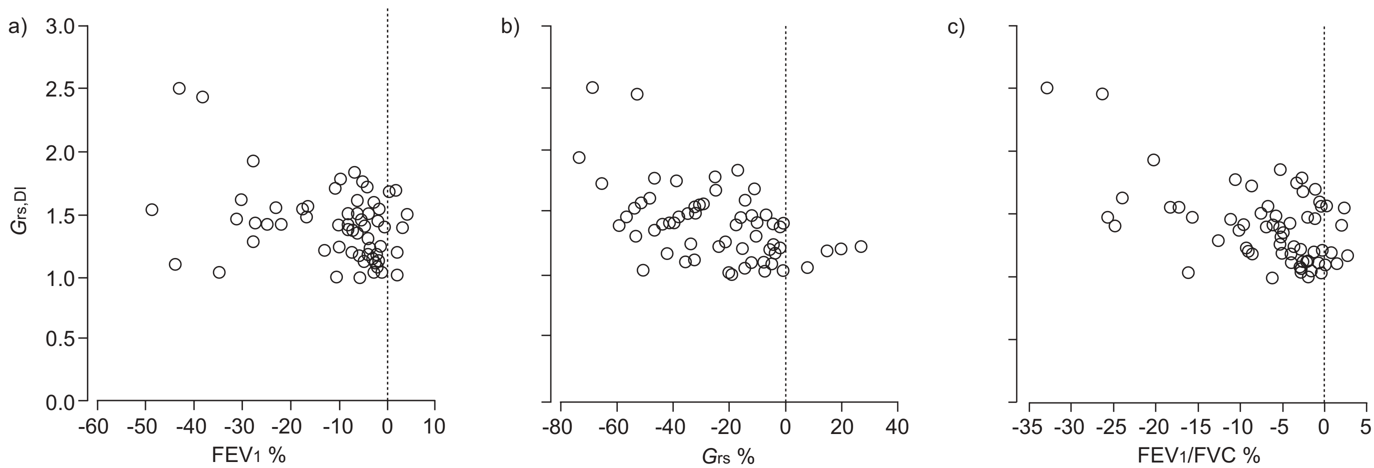

FIGURE 3. Significant correlations are disclosed between the respiratory conductance (*G*rs) response to deep inhalation (*G*rs,DI) after exercise and the airway response to exercise expressed as percentage change from baseline in: a) forced expiratory volume in one second (FEV1); and b) *G*rs; and c) difference between exercise and baseline in FEV1/forced vital capacity (FVC). ······: no change. a) $p=0.002$; b) $p<0.0001$; and c) $p<0.0001$).

ventilation was close to baseline 5 min after exercise, at the time the measurement was made [11]. An alternative is the measurement of end-inspiratory and end-expiratory values to eliminate the flow dependence of *R*rs [5, 17]. Upper airway calibre changes during breathing and the tendency for the laryngeal folds to close during expiration probably account for larger *R*rs variability compared to that found during inspiration in children [18]. Since the current primary interest was to estimate the change in intrathoracic airway dimensions after the DI, it was decided to focus on the *G*rs during inspiration. *G*rs spontaneous variability was expressed in a way that could easily be compared with the *G*rs,DI. For instance, a *G*rs,95,mean of 1.26 indicates that the upper limit of the 95% CI for between-breath spontaneous variability corresponds to a 26% increase in *G*rs. Therefore, a *G*rs,DI of ⩾1.26 was taken as an indicator of a significant increase in *G*rs by DI, *i.e.* unlikely to be accounted for by spontaneous variation. There are only a limited number of studies in the literature reporting on breath-by-breath computation of *G*rs or *R*rs variability. When estimating end-inspiratory and end-expiratory *R*rs in children, TRÜBEL and BANIKOL [17] reported a coefficient of variation of ~10%, which would correspond to an upper limit of the 95% CI of 20%, slightly lower than in the current study, perhaps explained by the fact that only end-inspiratory values were used. Variability is usually expressed within a set of three to five measurements or between two separate occasions. A coefficient of variation of 8–15% is representative of most studies in children [3], and the 9% mean coefficient of variation for *G*rs found in the present study is in keeping with such reports. Assessing a response to challenge using a measure which considers baseline variability, *i.e.* nSDw, has advantages in situations in which spontaneous variation may be large, such as in children [19] and asthmatics [17, 20]. Furthermore, it allows meaningful comparison among methods that measure parameters with different units and variability. A drawback is that a significant response may be disclosed below the conventional clinically significant threshold. A number of children classified as exercise responders in the current study exhibited a drop in FEV1 smaller than the recommended 12% decrease from baseline [21]. Since the primary aim of the present study was to assess the response to exercise in relation to *G*rs,DI, it appeared meaningful to address a population exhibiting all amplitudes of responses to exercise. Establishing a clinical diagnosis of EIAO based on parameter variability certainly requires further validation.

TABLE 5 Partial correlation coefficients of types of response to exercise *versus* airway response to deep inhalation after exercise

	FVC	FEV1	FEV1/FVC	*G*rs
nSDw	-0.008	-0.351	-0.530#	-0.444#
Δ	-0.038	-0.387	-0.530#	-0.519#

FVC: forced vital capacity; FEV1: forced expiratory volume in one second; *G*rs: respiratory conductance; nSDw: number of within-subject SDs; Δ: difference (percentage change from baseline for FEV1, FVC and *G*rs, and difference between exercise and baseline for FEV1/FVC). #: significant effect on response to deep inhalation by multiple linear regression analysis.

An increase in absolute lung volume, an important determinant of airway resistance, could occur after a DI and thus contribute to increase *G*rs [22]. Although absolute lung volume was not measured during the manoeuvre, the tracking of *V*T, as illustrated in figure 1, did not suggest much alteration in end-expiratory level after the DI. The change in lung volume was limited to full inspiration, in contrast to the alteration in airway calibre that lasted well beyond the DI. In subjects that show critical dependence on positive pressure for the maintenance of their lung volume, a passive insufflation to 38 hPa has been shown to be associated with a mean increase in functional residual capacity of ~10% [23]. This change could be associated with significant effect on airway dimensions in a context of low lung volume. However, the significant decrease in DI amplitude after exercise from baseline is indicative of some degree of lung distension, a condition previously documented during EIAO [24].

The most likely explanation of the *G*rs increase after DI relates to the mechanical interaction between lung parenchyma and conducting airway wall subjected to parenchymal tethering [7]. In adult subjects challenged with methacholine, the bronchoconstriction can be transiently reversed by DI as long as lung parenchyma hysteresis is not increased. Conversely, subjects showing increased lung hysteresis, such as that resulting from peripheral airway contraction, do not show much bronchodilation after the DI [25, 26]. The effect of DI on airway calibre may be accounted for by a decrease in smooth muscle tension and airway wall elastance. This suggestion comes from *in vitro* experiments on contracted tracheal smooth muscle subjected to short oscillations, where a sudden increase in amplitude of the oscillation was associated with a dramatic decrease in smooth muscle stiffness and increase in hysteresivity that indicated a stretch-induced decrease in the dynamics of the actin–myosin interaction [27].

The association of positive *G*rs responses to exercise and significant *G*rs,DI is suggestive of the reversal of exercise-induced conducting airway constriction because, above a few Hertz, *G*rs is a good indicator of the dimensions of these airways [28]. The current observations are thus in keeping with the mechanisms initiating EIAO. Cooling and dehydration are induced by the hyperventilation of exercise primarily in those airways most exposed to thermal and evaporative losses [29, 30], and are thought to initiate mediator release, which promotes airway obstruction in the corresponding part of the airway tree [29]. Bronchoconstriction in the conducting airways, but not in the peripheral lung, results in differential hysteresis and may therefore be transiently reversed after DI [25, 26].

Discrepancies between *G*rs and FEV1 responses to exercise are likely to occur because the maximal airway distension required by the forced expiratory manoeuvre may attenuate the degree of bronchial obstruction detected by FEV1 compared to *G*rs. Nevertheless, the agreement between FEV1 and *G*rs was surprisingly good since responses to exercise were discordant in 11 children, being negative by FEV1 and positive by *G*rs in only five. Although the *G*rs,DI was significantly larger in exercise responders than in nonresponders by both FEV1 and *G*rs, the most significantly different lung function parameters between DI responders and nonresponders were the *G*rs and FEV1/FVC responses to exercise. Furthermore, only the latter two were shown to be significantly correlated to the *G*rs,DI by stepwise regression. A recent interpretation of alterations in spirometric results induced by methacholine challenge has suggested that a decrease in FEV1/FVC is indicative of airway narrowing in the conducting airways [7, 31, 32], whereas a decrease in FEV1 and FVC, and therefore no change in FEV1/FVC, is thought to reflect an increase in residual volume and gas trapping associated with airway closure [31, 33]. Gas trapping has been demonstrated during severe EIAO [24], *i.e.* those responses extending from the conducting into the more peripheral airways [29], increasing peripheral lung as well as conducting airway hysteresis. Relative hysteresis is therefore unchanged, resulting in only a minimal airway response to DI [26]. In a previous study of a different group of children in whom definite EIAO had been documented by a decrease in FEV1 of $\geqslant$15%, it was found that the *G*rs response to DI correlated negatively with the FVC response to exercise, *i.e.* the children with a large decrease in FVC showed only a minor *G*rs response to DI [11].

An important issue in studying the airway effects of DI is that the induced bronchodilation is significant and prolonged in control adults but mild and transient or even absent in asthmatics [7–9], possibly because of the characteristics of the airway smooth muscle contractile apparatus in asthma [8]. Such evidence on bronchodilation by DI after methacholine challenge may be difficult to translate to EIAO because the condition is unlikely to occur in healthy subjects. The current evidence indicates that children with EIAO may readily exhibit bronchodilation by DI. Diagnosis of asthma throughout childhood encompasses more heterogeneous conditions than at adult age; from the many early childhood wheezers, a comparatively smaller fraction will be identified as asthmatic at adolescence [34]. Moreover, some children in the current study were on steroid or montelukast therapy, which prevented further analysis of the determinants of *G*rs,DI. Adult asthmatics receiving methacholine were reported to show not only lesser bronchodilation but also a faster rate of renarrowing after DI compared with controls [5]. The rate of renarrowing disclosed in the present study was found to be unrelated to the response to exercise, possibly because it was estimated in a rather crude way. A more detailed analysis of the latter parameter during EIAO would deserve further investigation.

It is concluded that significant bronchodilation may be demonstrated after deep inhalation by respiratory conductance measurements in children with exercise-induced airway obstruction. The effect is consistent with the mechanisms of airway obstruction and the mechanical interaction between lung and conducting airways. The identification of a positive response of respiratory conductance to deep inhalation after exercise may thus help in improving the detection of exercise-induced bronchoconstriction in children. Whether or not the pattern of airway response to deep inhalation after exercise-induced airway obstruction permits the identification of different forms of asthma in children is an interesting speculation that deserves further investigation.

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