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*CORRESPONDENCE*

# Evaluation of impulse oscillation system: comparison with forced oscillation technique and body plethysmography

*To the Editor:*

We read with interest the study by HELLINCKX *et al.* [1], comparing the techniques of impulse oscillation, forced oscillation and body plethysmography. The impulse oscillation system (IOS) was recently introduced by Jaeger (IOS Enrich Jaeger, Hoechberg, Germany) and its clinical potential is due to its relatively easy acquisition of data. Subjects perform tidal breathing in the upright position without any forced expiratory manoeuvres. This method could therefore potentially acquire broad usage, especially among patients with poor coordination. However, our limited experience with IOS suggests that its wider utilization will be problematic. Conversely, body-box plethysmography is an established method that allows for the measurement of airways resistance and spirometry and has been universally accepted as the gold standard in the clinical assessment of lung function.

There is very little data available on the comparison and, more specifically, assessment of the reproducibility and sensitivity of IOS, body-box plethysmography and spirometry. We have recently performed spirometry, IOS and body-box plethysmography in nine (six female) stable asthmatic patients aged mean±SE 40±4.3 yrs. The patients underwent spirometry, IOS and body-box plethysmography on two separate days, 1-week apart. Each test was performed before and 30 min after inhalation of 12 μg Eformoterol fumarate (Oxis®, Turbohaler®, AstraZeneca) or 50 μg Salmeterol (Serevent, Accuhaler®, Allen and Hanburys) in a randomized, double-blind, double-dummy, crossover manner. Preinhalation and change from baseline (Δ) results for forced expiratory volume in one second ($FEV_1$), respiratory system resistance measured at 5 Htz ($R_{rs5}$) using IOS and total specific airway compliance ($sG_{aw}$) using body-box plethysmography were compared using Jaeger equipment. This allowed for the assessment of the reproducibility of each technique in recording baseline measurements in the individual subjects, and the sensitivity of each method to detect change in airway measurements in response to bronchodilators. The results showed that reproducibility between the baseline testing on the 2 days was $FEV_1 > R_{rs5} > sG_{aw}$ with Pearson correlation coefficient (r) values of 0.95, 0.75 and 0.40, respectively. Using the Δ response, the comparative sensitivity was $R_{rs5} > FEV_1 > sG_{aw}$ with mean values of -8.3%, 7.5% and 2.5% for Eformoterol and -11.5%, 5.6% and 7.2% for salmeterol. Our data revealed that IOS and body-box plethysmography did not display the same degree of reproducibility as spirometry. However, the IOS seemed to record greater changes from the baseline in the respiratory resistance in response to the bronchodilators.

We agree with HELLINCKX *et al.* [1] that the measurements obtained with impulse oscillation system cannot be interchangeable with that of body-box plethysmography or spirometry. It is also our opinion that the reliability of the impulse oscillation system needs to be confirmed, with particular effort made to provide standardized guidelines on its usage. At present, the impulse oscillation system could be used to assess respiratory resistance in clinical research experiments. However, it is not clear whether respiratory resistance measured with the impulse oscillation system is mutually interchangeable with airway resistance assessed by body-box plethysmography. We believe that further studies are warranted before this technique could replace traditional spirometry as the gold standard research and clinical tool.

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*From the authors:*

We thank J.A. Kastelik and colleagues for their interest in our paper [1] and we would like to respond to some of their comments. First of all, it was not our purpose to examine whether the impulse oscillation system (IOS) "could replace traditional spirometry as the gold standard research and clinical tool", but simply to compare its results with those of the classical pseudorandom noise forced oscillation technique and body plethysmographic airway resistance.

Secondly, we would like to point out that the finding of J.A. KASTELIK and colleagues that "the IOS records greater changes from baseline after bronchodilatation than forced expiratory volume in one second ($FEV_1$) or specific airway conductance" does not necessarily imply that the former is more sensitive.

Indeed, it has been clearly established that for the comparison of the bronchodilator response by different lung function tests, one has to take several issues into consideration [2–6], such as: 1) the type of expression of the response (*e.g.* absolute *versus* relative changes, which have opposite effects on the values of spirometry and of resistance measurements); 2) the thresholds of a positive response, which depend on the intraindividual coefficients of variation for the particular lung function tests (and as this is smaller for spirometry than for resistance measurements, consequently the threshold is smaller for the former). In variance with the results cited by J.A. Kastelik and colleagues, NIELSEN and BISGAARD [7] recently showed that, in asthmatic children, specific airway resistance provides a better discriminative power for bronchodilator response than IOS or the interruptor technique. It may still be worthwhile considering the results of several tests (*e.g.* $FEV_1$ and measurement of airway resistance) as this may provide additional information.

Finally, we fully agree with J.A. Kastelik and colleagues that although the impulse oscillation allows a variety of data to be obtained in noncooperative subjects, especially young children, "the reliability of IOS needs to be confirmed with particular effort made to provide standardized guidelines in its usage". We would like to add that it is also necessary to assess the accuracy of equipment and data handling (*e.g.* criteria for technical reliability of data) and to evaluate the applicability of the implemented simple model simulating mechanics of the central and peripheral parts of the respiratory system.

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# Total energy expenditure in children with obstructive sleep apnoea syndrome

*To the Editor:*

I found the recent article by BLAND *et al.* [1] extremely interesting. The authors evaluated total energy expenditure in children with obstructive sleep apnoea syndrome (OSAS) and found no difference between OSAS and controls. They therefore concluded that the previously reported increases in sleeping energy expenditure, in both children and adults with OSAS, were probably compensated for by a decrease in waking energy expenditure. However, I think there are several methodological problems with the study that were not addressed.

The most surprising finding of the study was that OSAS did not resolve in the patients following tonsillectomy and adenoidectomy. In fact, the apnoea index actually increased slightly postoperatively. This occurred despite the fact that none of the patients were morbidly obese or had a craniofacial syndrome. Presumably, none of the patients had neurological disease or other causes of OSAS besides adenotonsillar hypertrophy. This lack of improvement following surgery is in total contradiction with the literature on childhood OSAS. SUEN *et al.* [2] re-evaluated 26 children following surgery. All of the children had an improvement in the apnoea index, and only the very severe cases (respiratory disturbance index (RDI) >19) had an RDI >5 postoperatively. FRANK *et al.* [3] showed that the total number of obstructive apnoeas in a cohort of children undergoing tonsillectomy and adenoidectomy fell from 194 to 7. WIET *et al.* [4] showed a mean decrease in the apnoea/hypopnoea index from $23 \cdot h^{-1}$ to $6 \cdot h^{-1}$ in uncomplicated patients postoperatively. ZUCCONI *et al.* [5], NISHIMURA *et al.* [6] and NIEMINEN *et al.* [7] all showed a 95–100% cure rate with surgery. The current study by BLAND *et al.* [1] is the only study I could find that did not show a highly-significant improvement in OSAS in otherwise healthy children following tonsillectomy and adenoidectomy. This suggests that there were technical problems with

either the surgery or the polysomnography, or that there were some other, undisclosed abnormalities with the patient cohort.

In addition, few data were provided regarding the controls. In particular, was any screening technique used to ensure that the controls did not have obstructive sleep apnoea syndrome? This is important as obstructive sleep apnoea syndrome is common in the paediatric population (with a prevalence of ~2% [8–10]).

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# Nasal continuous positive airway pressure for sleep apnoea following stroke

*To the Editor:*

The papers by SANDBERG *et al.* [1] and WESSENDORF *et al.* [2] in the October 2001 issue of the *European Respiratory Journal* provide interesting preliminary data on the use of continuous positive airway pressure (CPAP) in patients with obstructive sleep apnoea (OSA) after stroke. This approach is a logical extension of several published studies showing a high prevalence of sleep disordered breathing in such patients. We are, however, concerned that the conclusions of these studies are overstated and that the likely benefits of CPAP treatment in such patients are being overestimated.

SANDBERG *et al.* [1] in a randomized controlled study showed a significant improvement in symptoms of depression in convalescent patients with OSA receiving CPAP treatment. However, compliance with treatment was relatively poor, with only half of the treated patients using CPAP for >4 h per night, even while in hospital. The better compliers were less confused and had better cognition and less depression. There was no improvement in cognitive function or activities of daily living after 4 weeks of treatment. No placebo control was attempted, a point of particular importance as in nonstroke patients with OSA, both active and sham CPAP produce similar improvements in symptoms of depression [3]. Furthermore, the magnitude of the improvement in depression reported by SANDBERG *et al.* [1] was less than that found with placebo using the same depression scale in a study of pharmacological treatment of depression after stroke [4].

WESSENDORF *et al.* [2] performed an uncontrolled study in highly selected patients during rehabilitation, an average of 8 weeks after stroke. It is particularly worth noting that the patients had an average age of only 60.9 yrs, which contrasts with ~75 yrs in the typical stroke population admitted to hospital [5]. In addition, the level of disability in the patients studied was mild (average Barthel index 15.4/20). WESSENDORF *et al.* [2] defined acceptance of CPAP treatment as continued use during hospital admission and agreement to continue at home, but no quantitative compliance data were given. In a subgroup of patients, the response to CPAP was assessed by a simple visual analogue scale of well-being, which showed greater improvement in those accepting CPAP treatment than in those who declined to continue. Since the study was uncontrolled, it is clear that a placebo effect

cannot be excluded and it should also be noted that visual analogue scales are unreliable in patients after stroke [6].

Our own experience with attempting to initiate CPAP treatment in patients with sleep apnoea post-stroke, with a view to subsequently undertaking a randomized controlled trial of CPAP for OSA following stroke, contrasts markedly with these two studies. We evaluated the tolerability of CPAP between 2 and 6 weeks poststroke, using a ResMed Autoset® (Sydney, Australia) system. Patients were selected after a diagnostic study showing either an apnoea/hypopnoea index (AHI) >20 or an AHI >10 together with daytime sleepiness. After studying 15 patients, the study was discontinued because the patients were unable to tolerate the treatment. In terms of age (mean 74 yrs) and disability (median Barthel score 7/20), our patients were a typical, unselected, inpatient, acute-stroke population. Of the 15 recruited, one withdrew when shown the equipment and a second after a short daytime trial because of discomfort. Only three of the remaining 13 completed a titration night and all declined further CPAP therapy. At this point it was decided that further recruitment was not justified because of lack of success and poor tolerance of the treatment. Our patients were studied earlier after stroke than those of WESSENDORF *et al.* [2] and the majority in an acute-stroke unit rather than a rehabilitation department in which the staff were experienced in regular initiation of CPAP treatment. We would suggest, however, that if CPAP treatment were shown to be beneficial to OSA following acute stroke, an acute-stroke unit is the setting in which it would need to be initiated.

WESSENDORF *et al.* [2] concluded that nasal continuous positive airway pressure is an "important therapeutic option" in stroke patients with obstructive sleep apnoea and state that "stroke patients with obstructive sleep apnoea can be treated effectively with nasal continuous positive airway pressure and show a similar improvement and primary acceptance to obstructive sleep apnoea patients without stroke". We suggest that these statements require major qualification. A more appropriate conclusion would be that it is feasible to treat some younger patients following mild stroke with nasal continuous positive airway pressure, but to assess whether this produces useful clinical benefit requires a randomized placebo-controlled study.

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*From the authors:*

We agree that placebo-controlled studies are necessary in order to evaluate the effect of nasal continuous positive airways pressure (nCPAP) on patients with acute stroke. How to carry out a placebo nCPAP treatment study was, however, not obvious at the time our study was initiated. We also agree with J. Harbison and colleagues that tolerance to nCPAP in stroke patients is lower than for patients who visit our sleep laboratory for treatment of snoring and severe sleep apnoea. We find it reasonable that tolerance to nCPAP is lower among stroke victims than for a subject who has attended for snoring and sleep apnoea and experienced a new life without daytime sleepiness by the simple means of using an nCPAP.

J. Harbison and colleagues report an almost zero tolerance for nCPAP. In complete contrast, 50% of our patients tolerated nCPAP during the 1-month study period. MILANOVA *et al.* [1] reported successful titration in 65% of their patients in an acute stroke unit using the same equipment (ResMed AutoSet system®) as J. Harbison and colleagues. MILANOVA *et al.* [1] also reported that >50% of the stroke patients were discharged from hospital with nCPAP treatment. They also concluded that "CPAP-treatment in the acute stage of stroke is feasible and short-term compliance is fair". Delirium and low cognitive level were the two factors that explained poor tolerance to nCPAP in our study.

J. Harbison and colleagues refer to a study by YU *et al.* [2], and claim that "both active and sham CPAP produce similar improvements in depressive symptoms in patients without stroke". YU *et al.* [2] concluded that "the effect of CPAP treatment on mood symptoms in apnoeic patients could be a placebo effect". The study by YU *et al.* [2] included only a very brief placebo treatment period (1 week), which is too short a time to evaluate any antidepressive effect. It is well known from placebo-controlled pharmacological studies that initially (during the first few weeks) there are significant placebo effects when treating poststroke depression [3]. The diagnosis of depression, after stroke, is also difficult to make which might lead to an over diagnosis of at least less severe depression. YU *et al.* [2] also conclude that nCPAP treatment may be effective in improving mood

states but perhaps only in patients who have severe depressive symptoms secondary to sleep apnoea.

Extensive neuropsychological investigations of sleep-apnoea patients without stroke have shown that these patients show a whole range of neuropsychological deficits, including depressive symptoms [4–7]. Numerous well-constructed studies, some randomized, some placebo-controlled and one crossover trial have shown significant benefit from nCPAP treatment in mild, moderate and severe sleep apnoea with regard to mental flexibility, daytime sleepiness and daytime function, cognitive function, depression scores, psychosocial wellbeing, health status and quality of life in patients without stroke. nCPAP treatment effects have been found after a mean of only 2.8 h use per night [4, 6, 8–12]. Some of these findings are also supported by a Cochrane Collaboration report which showed that treatment with nCPAP significantly improved several quality of life and depression measures compared to placebo in patients without stroke [13].

The studies we refer to above show conflicting results compared to the article referred to by J. Harbison and colleagues, namely that nCPAP treatment is effective at diminishing or abolishing neuropsychological symptoms, including depressive symptoms, in patients without stroke. We are convinced that our results reflect the mood status of stroke patients with sleep apnoea and we are looking forward to future randomized, placebo-controlled nCPAP treatment studies of stroke patients with sleep apnoea, which we are convinced will confirm our results.

The design of the statistical analysis of the four outcome variables was intention-to-treat. Despite the fact that 50% of the patients did not comply with nCPAP treatment, the treatment effect in those who did comply was much greater than for the whole group. When we analysed the treatment effects on those who complied with nCPAP treatment, not only did depressive symptoms improve much more than the average for the whole group, but there was also a significant improvement in activities of daily-living ability compared to the control group.

The scientific evidence today for both physiological and psychological benefit from nasal continuous positive airway pressure treatment in patients without stroke is indisputable. One might expect that an early nasal continuous positive airway pressure treatment of sleep apnoea after stroke would be beneficial for the patient's recovery and further rehabilitation. Future studies may provide an answer to that question.

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*From the authors:*

We agree entirely with the concluding statement made by J. Harbison and colleagues that randomized controlled trials are necessary to evaluate the role of treatment with sleep-disordered breathing in patients with stroke, and such a study has been commenced.

The mean age of 74 yrs quoted by J. Harbison

and colleagues seems to reflect an elderly stroke population. In a recent study, the mean age of stroke patients in a large hospital database in Germany was 66±14.1 yrs [1]. The mean age of our patients (61±10 yrs) was the same as that in a prevalence study we performed on 147 randomly selected patients, in rehabilitation after their first ever stroke [2]. To date, the mean age has ranged from 59 [3] to 72 [4] yrs in other large prevalence studies.

The achievement of 0% compliance by J. Harbison and colleagues is surprisingly low and very different from our experience. The reported differences could have been due to the use of different techniques or the large variations in the population, including age, degree of disability, and time since stroke. Our study showed that acceptance of nasal continuous positive airway pressure (nCPAP) depended on functional disability and the presence of aphasia, *i.e.* compliance in more severely affected patients is poorer. Age *per se* appeared to be a minor additional factor. To the best of our knowledge, only one study in the acute stroke setting has been published [5]. MILANOVA *et al.* [5] successfully completed titration with the AutoSet® (ResMed, Sydney, Australia) in 26 of 40 patients with acute stroke, and discharged 22 with continuous positive airway pressure (CPAP) from hospital.

Our visual analogue scale (VAS) was designed as a (rather simple) global measurement of well-being. This was not the first use of a VAS in stroke patients [6]. We agree with J. Harbison and colleagues that a difference in the degree of aphasia between CPAP and non-CPAP users could have caused a random or systematic difference in how the patients filled out the VAS at baseline. However, we reported a larger improvement in VAS in the users *versus* nonusers. The only way that the within-subject degree of disability could cause a change in VAS with therapy is if the within-subject degree of disability really did change with therapy.

As previously stated in our paper, we think that appropriate neuropsychological tests are necessary to evaluate possible treatment effects on neuropsychological function.

In view of the known physiological benefits of nasal continuous positive airway pressure in the treatment of obstructive sleep apnoea, actual physiological benefit is inevitable. The only reasons for withholding nasal continuous positive airway pressure in the treatment of obstructive sleep apnoea in stroke were the assumptions that the therapy could not be initiated and that the subjects would feel no subjective benefit and therefore refuse treatment. Our study has shown that both these reasons for withholding nasal continuous positive airway pressure and denying the patient the physiological benefits are unfounded. It still remains to be shown whether the subjective benefit is in excess of a placebo in a randomized controlled trial.

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