Accuracy of Nasal Cannula Pressure Recordings for Assessment of Ventilation during Sleep

ROBERT THURNHEER, XIAOBIN XIE, and KONRAD E. BLOCH

Department of Internal Medicine, University Hospital of Bruderholz, Basel and Pulmonary Division, Department of Internal Medicine, University Hospital of Zürich, Switzerland

Nasal prong pressure monitoring (P_{NOSE}) is utilized to assess ventilation during sleep. However, it has not been rigorously validated against the gold standard of face-mask pneumotachography ($\dot{V}_{FM}$). Therefore, we compared P_{NOSE} with $\dot{V}_{FM}$ in 20 patients with suspected sleep apnea during nocturnal polysomnography, and analyzed factors affecting accuracy of P_{NOSE}-derived variables. Patients rated their nasal obstruction on a visual analog scale. Mean ± SE apnea/hypopnea index (AHI) by $\dot{V}_{FM}$ was 24.0 ± 5.1 h^{-1}. The bias (mean difference) and limits of agreement (± 2 SD) of AHI derived from P_{NOSE}, and square root–transformed P_{NOSE}, a measure proposed as a surrogate of airflow, were +3.9 (± 4.6), and −0.9 (± 9.0) h^{-1}. Subjective scores of nasal obstruction before polysomnographies did not herald inaccuracy of AHI from P_{NOSE}. Square root–transformed P_{NOSE} closely tracked pneumotachographic airflow over 10 breaths (r^2 among signals 0.88 to 0.96) but the relationship among these signals was highly variable if comparisons were extended over an entire night. Compared with face-mask pneumotachography, nasal pressure monitoring provides accurate AHI for clinical purposes even in patients perceiving nasal obstruction. Square-root transformation provides near linear nasal pressure/airflow relationships over a short time but is not essential for estimation of AHI.

Keywords: nasal prong pressure transducer; sleep apnea; polysomnography; physiologic monitoring; inspiratory flow limitation

The diagnosis of sleep-related breathing disorders relies on a typical history and is confirmed by a sleep study to objectively document the presence and severity of sleep-related respiratory disturbances. As quantitative measurement of ventilation by a flowmeter attached to a face mask is inconvenient, less obtrusive means such as oral-nasal thermistors and chest wall motion sensors are commonly used (1). However, these methods cannot reliably quantify airflow for detection of hypopnea. As the physiological consequences of apnea and hypopnea are similar, quantitative rather than qualitative methods for monitoring respiration during sleep are desired (1).

A promising technique for estimation of ventilation during sleep is based on analysis of the pressure signal derived from nasal prongs (2). Several validation studies for nasal pressure–derived apnea/hypopnea index (AHI) used thermistors and chest wall motion sensors as reference methods (3–7). These data are difficult to interpret since the reference standard did not allow quantitative estimation of ventilation. Nasal pressure recordings were also compared with airflow measured by a flowmeter attached to a nasal mask (8, 9), but in these studies, the potential influence of oral breathing on accuracy of nasal pressure–derived estimation of ventilation could not be assessed.

Monserrat and colleagues (2) proposed a simple method of correcting for the nonlinear nasal pressure/airflow relationship. These investigators demonstrated that the square root–transformed nasal pressures signal closely tracked nasal airflow in seated healthy subjects over a few breaths and in a model simulation (10). Whether nasal pressure quantitatively reflects ventilation over longer time periods and in supine patients during sleep has not been reported.

To more rigorously evaluate nasal pressure monitoring as a simple means to quantify ventilation during sleep, we performed comparisons with the gold standard for measurement of ventilation, i.e., face-mask pneumotachography during polysomnography in patients with suspected sleep-disordered breathing. Our purpose was to assess accuracy and evaluate factors influencing accuracy of apnea/hypopnea detection by nasal prong pressure transducers. In particular, we intended to investigate whether analysis of the square root–transformed as opposed to the nasal pressure raw signal improved detection of respiratory events, and whether impaired nasal breathing (presumably caused by a greater prevalence of oral breathing under such circumstances) was associated with reduced accuracy of apnea/hypopnea detection by nasal pressure monitoring. Finally, we compared nasal pressure–derived AHI with the AHI as defined in epidemiologic studies on adverse health effects of sleep disordered breathing (11) where respiratory event definitions included criteria of both breathing amplitude (assessed by nasal pressure and inductive plethysmography) and oxygenation (by pulse oximetry).

METHODS

Patients

Twenty patients (17 male, 3 female, mean age, 52 yr [range, 33 to 73 yr]; mean body mass index, 27.3 kg/m^2 [range, 20.3 to 50.5 kg/m^2]) referred for evaluation of suspected sleep apnea consented to participate in the study, which was approved by the Hospital Ethics Committee (Methods are detailed in an online supplement).

Measurements

Patients estimated impairment of nasal breathing on a visual analog scale. Nasal resistance was measured with rhinomanometry (12).

Polysomnographies included derivations of EEG, EOG, EMG, ECG, pulse oximetry, calibrated respiratory inductive plethysmography (13), and body position. Nasal cannulas were fitted and taped to the skin. Their tubing was connected to a differential pressure transducer referenced to face-mask pressure. A face mask with a flowmeter attached to its air inlet was strapped onto the face. Respiratory signals were digitally sampled at 50 Hz with 12 bit resolution.

Data Analysis

Apnea/hypopnea scoring. Apneas/hypopneas were defined as a clear amplitude reduction of a "measure of breathing" to < 50% of baseline for ⩾ 10 s, according to the American Academy of Sleep Medi-

(*Received in original form February 26, 2001; accepted in final form July 8, 2001*)

Supported by: Zürich Lung League.

Correspondence and requests for reprints should be addressed to Konrad E. Bloch, M.D., Pulmonary Division, Dept. of Internal Medicine, University Hospital of Zürich, Rämistrasse 100 CH-8091, Zürich, Switzerland. E-mail: pneubloc@usz.unizh.ch

This article has an online data supplement, which is accessible from this issue's table of contents online at www.atsjournals.org

Am J Respir Crit Care Med Vol 164. pp 1914–1919, 2001
DOI: 10.1164/rccm2102104
Internet address: www.atsjournals.org

cine Task Force (1). Baseline was defined as mean amplitude of stable breathing and oxygenation over the previous 2 min, or, if breathing pattern was unstable, the mean of the three largest breaths during the previous 2 min.

The following "measures of breathing" were scored individually by separate review of successive 2.7-min epochs on a computer video screen: Nasal pressure (PNOSE), square root–transformed nasal pressure ($\dot{V}$NOSE) (2), summed rib cage plus abdominal volume from calibrated inductive plethysmography (VolRIP), time derivative of the latter ($\dot{V}$RIP, i.e., RIP-derived "flow") (14), airflow from flowmeter ($\dot{V}$FM). Signals of the inductive plethysmograph (rib cage, abdomen, sum), and nasal pressure were also scored together, with priority on apnea/hypopnea criteria by inductive plethysmography in case of discrepancies.

Assuming $\dot{V}$FM ≅ square root–transformed PNOSE (2), overdetection of hypopnea by PNOSE was expected (10) if the same criterion for amplitude reduction as that for $\dot{V}$FM (< 0.5 times baseline) was applied. To account for this, PNOSE was also scored with an amplitude reduction criterion of $< 0.5^2$, i.e., < 25% of baseline.

Furthermore, apneas/hypopneas were scored according to Peppard and colleagues (11) by combined analysis of PNOSE, inductive plethysmography, and pulse oximetry. Apnea/hypopnea was defined as absence of any deflection of PNOSE ⩾ 10 s, or as any discernible reduction in VolRIP ⩾ 10 s associated with ⩾ 4% oxygen desaturation (11).

Recordings were scored independently by two observers. Means of corresponding individual apnea/hypopnea indices (AHI) were compared among methods.

Estimation of ventilation by nasal pressure monitoring. Short-term correlation among $\dot{V}$NOSE and $\dot{V}$FM was evaluated by computing proportionality coefficients among the two signals (50 Hz time series) over 10 successive inspirations (KI) and expirations (KE). Stability of correlations of $\dot{V}$NOSE with $\dot{V}$FM over the course of the night was assessed by computing mean KI and KE over four 2-min epochs, in the evening, after turning the lights off, at the beginning of the second, third, and fourth quarters of the night.

Statistics

Agreement among AHI by different methods was assessed according to Bland and Altman (15). Intraclass correlation among epoch-by-epoch apnea/hypopnea scores by different methods was determined by Cohen's kappa statistics. KI and KE at successive time points were compared by analysis of variance. Statistical significance was assumed at $p < 0.05$.

RESULTS

Sleep Data

Mean ± SE recording time was 421 ± 11 min, mean total sleep time was 283 ± 16 min, and mean sleep latency was 21 ± 4 min. All patients entered stages III or IV NREM and REM sleep. Sleep efficiency was 68 ± 4%.

Detection of Apnea/Hypopnea by the Different Measurement Techniques

The patients had a wide range of AHI (from 1.3 to 71.5 h^{-1}, mean ± SE 24.0 ± 4.5 h^{-1}) by flowmeter ($\dot{V}$FM) (Figure 1). Compared with $\dot{V}$FM, the AHI were slightly but statistically significantly overestimated by PNOSE, and the inductive plethysmographic volume signal (VolRIP), and by the combined analysis of PNOSE with inductive plethysmographic rib cage, abdomen, and sum volume signals (VolRIP-RCRIP-ABRIP) (Table 1). The surrogates of flow obtained by square root transformation of nasal pressure ($\dot{V}$NOSE), and by differentiating the inductive plethysmographic volume signal ($\dot{V}$RIP) provided estimates of AHI without significant bias relative to $\dot{V}$FM (Table 1). If the criterion for hypopnea detection by PNOSE was defined as an amplitude reduction to < 25% (rather than to < 50%) of baseline, then the bias of the AHI versus that from $\dot{V}$FM was not statistically different from zero or from corresponding values derived from $\dot{V}$NOSE and $\dot{V}$RIP (Table 1).

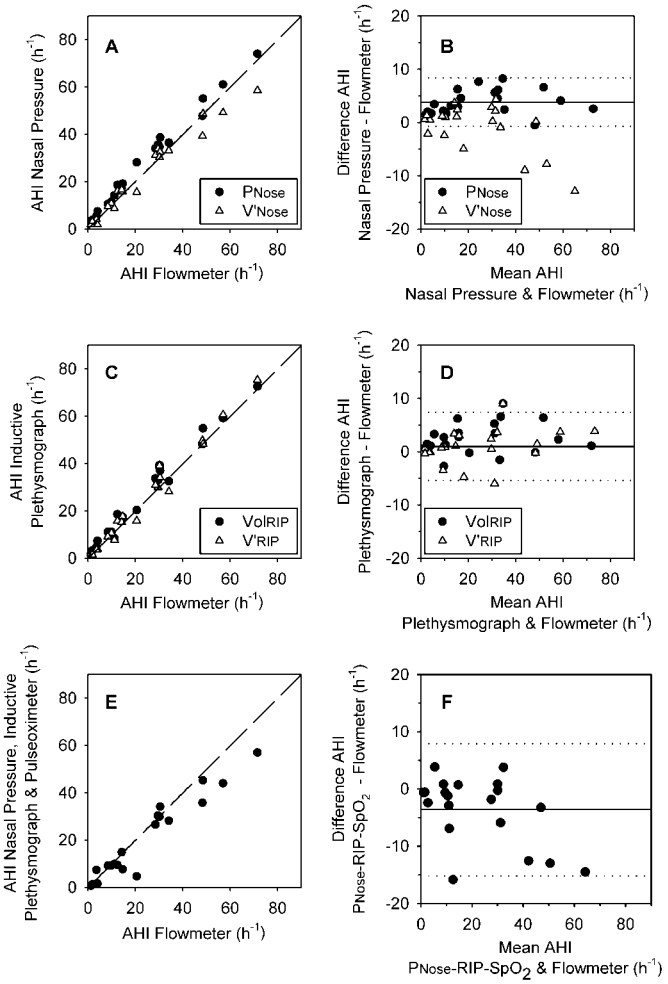

Figure 1. Identity plots and plots of differences versus mean apnea/hypopnea indices (AHI) derived by nasal pressure transducer, respiratory inductive plethysmography, and flowmeter attached to a face mask. Symbols = individual AHI, dashed lines = lines of identity, solid lines = mean differences (bias), dotted lines = bias ± 2 SD (limits of agreement). (Panels *A* and *B*) AHI from nasal pressure raw (PNOSE) and square root–transformed ($\dot{V}$NOSE) signals compared with AHI by flowmeter. Bias and limits of agreement are displayed for PNOSE only. Differences in AHI by $\dot{V}$NOSE and flowmeter (Panel *B*, *triangles*) were negatively correlated with the mean AHI ($r = -0.58$, $p < 0.01$). (Panels *C* and *D*) AHI from respiratory inductive plethysmographic sum volume signal (VolRIP) and time derivative of the latter ($\dot{V}$RIP, a measure reflecting flow), compared with AHI by flowmeter. Bias and limits of agreement are displayed for $\dot{V}$RIP only. (Panels *E* and *F*) AHI from combined analysis of nasal pressure (PNOSE), inductive plethysmographic signals (RIP = rib cage, abdominal, and sum volume), and pulse oximetry (Sp_{O_2}), compared with AHI by flowmeter. Differences in AHI (Panel *F*) were negatively correlated with the mean AHI ($r = -0.51$, $p = 0.02$).

It is illustrated in Figure 1 (Panel B) that the differences between AHI by $\dot{V}$NOSE and $\dot{V}$FM were negatively correlated with their mean AHI (Pearson's $r = -0.58$, $p < 0.01$), i.e., AHI by $\dot{V}$FM was progressively overestimated by $\dot{V}$NOSE with increasing AHI. Definition of apnea/hypopnea based on analysis of nasal pressure, inductive plethysmograph, and pulse oximetry, according to Peppard and colleagues (11), provided AHI that were systematically lower than corresponding AHI from all other methods (Table 1). In addition, the differences in relation to the AHI by $\dot{V}$FM were negatively correlated with the corresponding mean AHI (Pearson's $r = -0.51$, $p = 0.02$) (Figure 1, Panel F).

TABLE 1. AGREEMENT OF APNEA/HYPOPNEA SCORES BY VARIOUS MEASUREMENT TECHNIQUES WITH THAT FROM FLOWMETER*

Evaluated Method for Apnea/Hypopnea Estimation†	Apnea/Hypopnea Indices: Bias (h^{-1})	Apnea/Hypopnea Indices: Limits of Agreement bias ± 2 SD (h^{-1})	Apnea/Hypopnea Indices: Mean Deviation ± SD (h^{-1})	Coefficients of Intraclass Correlation (κ) among Epoch-by-Epoch Apnea/Hypopnea Scores by Different Methods (means ± SE)**
P_{NOSE}	3.9§	−0.8 to 8.5	3.9 ± 2.2	0.88 ± 0.02
P_{NOSE}(25%)	−0.8‖	−8 to 9.6	3.1 ± 3.2	0.86 ± 0.02
$\dot{V}_{NOSE}$	−0.9‖	−9.9 to 8.1	3.1 ± 3.3	0.78 ± 0.03
Vol_{RIP}	2.6‡	−3.3 to 8.6	3.1 ± 2.4	0.83 ± 0.03
$\dot{V}_{RIP}$	1.0	−5.6 to 7.6	2.5 ± 2.3	0.77 ± 0.04
P_{NOSE}-Vol_{RIP}-RC_{RIP}-AB_{RIP}	2.9‡	−5.7 to 11.5	4.3 ± 2.8	0.84 ± 0.03
P_{NOSE}-RIP-Sp_{O_2}	−3.6†§¶	−15.5 to 8.2	4.6 ± 5.1	0.82 ± 0.03

* The analysis was based on the average of the apnea/hypopnea scores obtained independently by two observers for each of the eight methods in the sleep studies of 20 patients. The reference method was the flowmeter.

† The evaluated methods were P_{NOSE}, P_{NOSE}(25%), $\dot{V}_{NOSE}$: nasal pressure raw signal, nasal pressure raw signal scored with hypopnea threshold < 25% baseline (*see* text), square root–transformed nasal pressure; Vol_{RIP}, $\dot{V}_{RIP}$: inductive plethysmographic sum volume signal and its time derivative; P_{NOSE}-Vol_{RIP}-RC_{RIP}-AB_{RIP}: nasal pressure and inductive plethysmographic rib cage, abdominal, and sum volume signals; P_{NOSE}-RIP-Sp_{O_2}: nasal pressure, inductive plethysmograph, and pulse oximetry (*see* reference 11); Bias: mean difference in apnea/hypopnea index by evaluated minus reference method; mean deviation: mean difference in apnea/hypopnea index by evaluated minus reference method, irrespective of algebraic sign.

‡ $p < 0.05$.

§ $p < 0.005$ for comparisons of bias versus flowmeter.

¶ $p < 0.005$ for comparisons of bias versus all other methods.

‖ $p < 0.005$ for comparisons of bias versus P_{NOSE}, Vol_{RIP}, P_{NOSE}-Vol_{RIP}-RC_{RIP}-AB_{RIP}, P_{NOSE}-RIP-Sp_{O_2}.

** Cohen kappa intraclass correlation coefficients (κ) were computed for a total of 1,890 epochs of 2.7 min duration from the 20 sleep studies; p = NS for comparisons among methods.

The means of absolute deviations (mean differences without respect to the algebraic sign) of AHI by the various evaluated methods from the AHI by $\dot{V}_{FM}$ were not statistically different, suggesting a similar precision in estimation of the AHI (Table 1). The slightly wider limits of agreement for the AHI derived from $\dot{V}_{NOSE}$ versus that from P_{NOSE} was related to the systematic overestimation of AHI by $\dot{V}_{NOSE}$ at higher AHI values (i.e., to the negative correlation of differences among AHI by $\dot{V}_{NOSE}$ and $\dot{V}_{FM}$ with their mean; Figure 1, Panel B).

If the criterion for the case definition of sleep apnea syndrome was set at an AHI > 5 h^{-1} by $\dot{V}_{FM}$, all subjects would have been correctly classified by $\dot{V}_{NOSE}$, but there would have been two false positives by P_{NOSE}. At a criterion level of > 15 h^{-1} by $\dot{V}_{FM}$, 13 instead of 10 patients would have been identified by both $\dot{V}_{NOSE}$ and P_{NOSE} (sensitivity, 100%; specificity, 70%). There were no false negative classifications at any of the two criterion levels, neither with P_{NOSE} nor with $\dot{V}_{NOSE}$. If P_{NOSE} was scored with a hypopnea amplitude reduction criterion of < 25% baseline, to compensate for the nonlinear relationship to $\dot{V}_{FM}$, all subjects were correctly classified for a sleep apnea syndrome criterion value of AHI > 5 h^{-1}, i.e., these results were identical to those from scoring $\dot{V}_{NOSE}$ (with hypopnea defined by amplitude reduction to < 50% baseline).

If the apnea/hypopnea definition by Peppard and colleagues (11) was taken as the reference standard, mean deviations of AHI by P_{NOSE} exceeded corresponding values from $\dot{V}_{NOSE}$, $\dot{V}_{RIP}$, and $\dot{V}_{FM}$, suggesting a greater precision of the latter three methods in prediction of the AHI according to Peppard and colleagues (11) (these data are provided in Table E1 of the online supplement).

Cohen kappa intraclass correlation coefficients among epoch-by-epoch apnea/hypopnea scores by the various methods suggested that 77 to 88% of the variation in the AHI by P_{NOSE}, $\dot{V}_{NOSE}$, Vol_{RIP}, $\dot{V}_{RIP}$ was related to variation in the AHI by the reference standard, and only 12 to 23% to random variation (Table 1).

Correlation of Nasal Obstruction with Accuracy of Apnea/Hypopnea Detection by Nasal Pressure Monitoring

The mean ± SE subjective estimates of nasal breathing impairment by the 20 patients on a visual analog scale in the evening before beginning of the sleep study was 58 ± 5%. The observed range was 4% to 90% on a scale extending from 0%, not impaired, to 100%, completely obstructed. These scores did not correlate with differences of nasal pressure–derived AHI minus that by the flowmeter (AHI P_{NOSE} minus $\dot{V}_{FM}$ versus visual analog scores: Pearson's r = 0.17, p = NS).

In 10 consecutive patients, mean ± SE inspiratory nasal resistance measured at 150 Pa in the evening before the sleep studies was 0.56 ± 0.09 Pa/s/cm^{-3}. The corresponding value in the morning was 0.45 ± 0.18 (p = NS for comparison versus value in the evening). There was no significant correlation between subjectively perceived nasal obstruction and measured nasal resistances in the evening and morning (n = 10, Pearson's r = 0.14 and 0.16, respectively, p = NS). Nasal resistances (mean values from evening and morning measurements) were not correlated with differences between AHI by nasal pressure ($\dot{V}_{NOSE}$) and flowmeter (n = 10, Pearson's r = −0.09, p = NS).

Estimation of Ventilation by Nasal Pressure Monitoring

In five patients, comparisons of the square root–transformed nasal pressure signal with that from the flowmeter over short time periods, i.e., 10 consecutive breaths, revealed close correlation, with a mean value ± SE of the coefficient of determination among the two signals of $r^2 = 0.94 \pm 0.03$ (range, 0.93 to 0.96) during inspiration, and $r^2 = 0.93 \pm 0.01$ (range, 0.88 to 0.96) during expiration. There were only minor breath-by-breath variations of inspiratory and expiratory proportionality coefficients (K_I, K_E) (*see* Table E2 of the online supplement). An example is shown in Figure 2 of a representative recording of $\dot{V}_{FM}$ and $\dot{V}_{NOSE}$ from the beginning of a recording session, after turning the lights off. The time series and identity plots (Figure 2, Panels A and B) reveal near perfect tracking of $\dot{V}_{FM}$ by $\dot{V}_{NOSE}$.

Changes in K_I and K_E over the course of an entire night were also analyzed. To this end, K_I and K_E over a 2-min epoch in the evening, immediately after turning the lights off, were calculated for each of the 20 patients and designated as individual baseline for the inspiratory and expiratory proportionality coefficients. Subsequent K_I and K_E over 2-min epochs at the beginning of the second, third, and fourth quarters of the night revealed major individual deviations from baseline, but

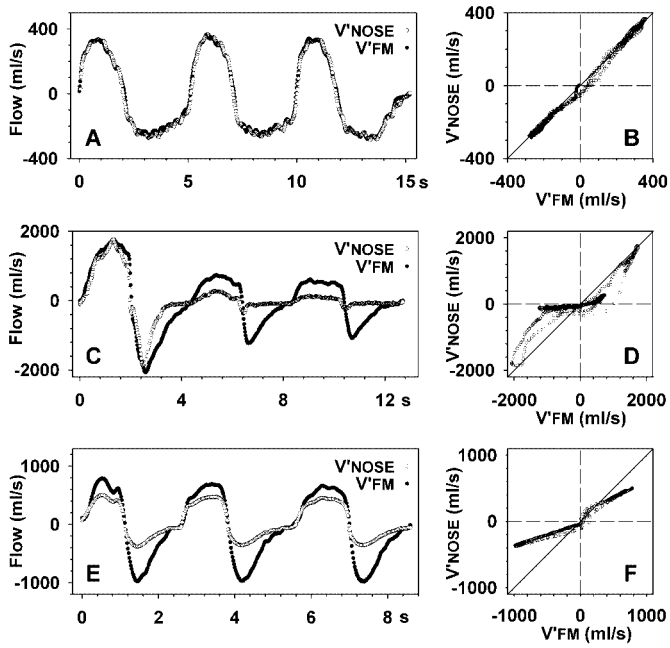

Figure 2. Representative time series (Panels *A, C, E*), and identity plots (Panels *B, D, F*) of airflow estimated by flowmeter ($\dot{V}_{FM}$) and square root–transformed nasal pressure ($\dot{V}_{NOSE}$). Panels *A* and *B* correspond to the beginning of a recording session. $\dot{V}_{NOSE}$ closely tracks $\dot{V}_{FM}$, and, accordingly, the relationship between the two signals is nearly linear. The recordings displayed in Panels *C, D*, and *E, F* were obtained during a later stage of the session. Panels *C* and *D* correspond to transition from exclusively nasal (first breath) to oral-nasal ventilation (second and third breaths) as visually confirmed. Partial mouth breathing resulted in major reduction of $\dot{V}_{NOSE}$ amplitude. In Panels *E* and *F* the amplitude and shape (time course) of $\dot{V}_{NOSE}$ deviates from that of $\dot{V}_{FM}$, although visual inspection confirmed exclusive nasal breathing and no obvious displacement of nasal prongs. During inspiration the relationship between the two signals is clearly alinear.

the group median values did not change significantly (Table 2). The amount of the deviations of inspiratory proportionality coefficients from baseline values (i.e., the absolute difference, irrespective of algebraic sign, of K_I minus corresponding baseline values) was positively correlated with elapsed time from beginning of the study (Spearman's rank order correlation R = 0.31, $p < 0.01$). For expiration, the corresponding correlation of deviations of K_E with elapsed time was not statistically significant (Spearman's rank order correlation R = 0.23, p = NS).

In Figure 2 (Panels C–F), recordings obtained in the course of prolonged monitoring are depicted. In these examples, the relationship between $\dot{V}_{FM}$ and $\dot{V}_{NOSE}$ deviated significantly from linearity. Visual observation revealed that this was related to transition from exclusive nasal to oral-nasal breathing in the example displayed in Panels C and D of Figure 2. However, in another example (Figure 2, Panels E and F), amplitude and shape of the time series of $\dot{V}_{NOSE}$ and $\dot{V}_{FM}$ differed clearly, although neither mouth breathing nor any displacement of nasal progs or face mask was obvious on visual inspection.

The performance of the time derivative of the calibrated sum volume signal of the inductive plethysmograph ($\dot{V}_{RIP}$) in reflecting airflow was also evaluated by computing proportionality coefficients among $\dot{V}_{RIP}$ and $\dot{V}_{FM}$ (these results are summarized in Table E3 of the online supplement). Group medians of K_I and K_E for $\dot{V}_{RIP}$ remained stable over the course of the night, but individual values varied to a similar degree as noted for $\dot{V}_{NOSE}$ (Table 2).

DISCUSSION

Several previous studies have compared AHI derived from the nasal pressure raw or "linearized" signal with either thermistor and chest wall motion recordings (3 to 7) or with nasal mask pneumotachography (8, 9). The reported bias of nasal pressure–derived AHI ranged from $-9.6\ h^{-1}$ (8) to $+4.6\ h^{-1}$ (3), and limits of agreement (i.e., ± 2 SD of the bias) from ± 9 h^{-1} (9) to as much as ± 33 h^{-1} (8). These results may have been biased by the qualitative nature of the reference methods or by partial mouth breathing, respectively.

To more rigorously define the accuracy of nasal-pressure monitoring for estimation of apnea/hypopnea, we compared this technique with face-mask pneumotachography, a gold standard for quantitative measurement of ventilation that is not affected by mouth breathing. The apnea/hypopnea definition proposed by the American Academy of Sleep Medicine Task Force (1), i.e., a clear amplitude decrease (to < 50%) from stable baseline in the 2 min preceding an event, or from the mean amplitude of the three largest breaths in the 2 min preceding an event, if breathing pattern was unstable, and an event duration of ⩾ 10 s, was applied.

We found fair agreement of nasal pressure–derived AHI with that from the flowmeter, as well as with corresponding values from calibrated inductive plethysmography (Table 1). The

TABLE 2. OVERNIGHT COMPARISON OF SQUARE ROOT–TRANSFORMED NASAL PRESSURE AND AIRFLOW BY FLOWMETER*

	Proportionality Coefficients among $\dot{V}_{NOSE}$ and $\dot{V}_{FM}$			
	Inspiration: K_I (Epoch1, after lights off) = 100%		Expiration: K_E (Epoch 1, after lights off) = 100%	
Epoch	K_I (Epochs 2 to 4) in % K_I (Epoch 1) medians (quartile ranges)	Deviation of K_I (Epochs 2 to 4) from K_I (Epoch 1) in % K_I (Epoch 1) medians	K_E (Epochs 2 to 4) in % K_E (Epoch 1) medians (quartile ranges)	Deviation of K_E (Epochs 2 to 4) from K_E (Epoch 1) in % K_I (Epoch 1) medians
2nd quarter of night	99 (82 to 111)	15	87 (45 to 104)	38
3rd quarter of night	97 (72 to 129)	28	87 (28 to 153)	56
4th quarter of night	102 (78 to 148)	41†	63 (4 to 129)	68
Epochs 2 to 4	99 (78 to 128)	24	81 (35 to 129)	56

* n = 20 patients. As data were not normally distributed values are summarized by medians and quartiles.

Inspiratory (K_I) and expiratory (K_E) proportionality coefficients among 50 Hz time series of square root–transformed nasal pressure ($\dot{V}_{NOSE}$) and airflow by flowmeter ($\dot{V}_{FM}$) were calculated for four epochs of 2-min duration. Epoch 1 was immediately after lights off, Epochs 2, 3, and 4 at the beginning of the 2nd, 3rd, and 4th quarter of the night.

Values for K_I and K_E for Epochs 2 to 4 are expressed in percent of corresponding value for Epoch 1. Deviations correspond to absolute differences, irrespective of algebraic sign, of K_I and K_E (Epochs 2 to 4) from values of Epoch 1, expressed in percent of values for Epoch 1.

p = NS for all comparisons among medians of K_I and K_E at corresponding times.

† $p < 0.05$ versus median deviation of K_I during 2nd and 3rd quarter by analysis of variance.

latter was included in the evaluation to provide comparisons to this commonly employed nonobtrusive respiratory monitoring technique that is not influenced by the oral-nasal route of breathing. The nasal pressure and plethysmographic raw signals, PNOSE and VOLRIP, systematically, although slightly, overestimated the flowmeter derived AHI by a mean of 3.9 h^{-1} and 2.6 h^{-1}, respectively (Table 1). In contrast, the transformed signals ($\dot{V}$NOSE and $\dot{V}$RIP) provided AHI without significant bias. The higher apnea/hypopnea scores from PNOSE compared with those from $\dot{V}$NOSE are expected from the mathematical relationship between the two signals, which became increasingly effective in patients with greater prevalence of apnea/hypopnea (Figure 1, Panel B). To avoid overestimation of the AHI by PNOSE, the amplitude reduction criterion for hypopnea can be lowered to $< 25\%$ (i.e., to < 0.25, which is equal to $< 0.5^2$ of baseline). This provides AHI identical to those obtained by scoring $\dot{V}$NOSE with hypopnea defined as an amplitude reduction to $< 50\%$ baseline (Table 1) (*See also* Figure E1 in the online supplement). More generally, the AHI for $\dot{V}$NOSE can be derived from PNOSE simply by applying a hypopnea threshold equal to the squared value (expressed as a fraction of 1) of the one for $\dot{V}$NOSE. This could be easily implemented in software for automatic event scoring.

The number of hypopnea overestimated by PNOSE relative to $\dot{V}$NOSE depends on the prevalence of events of ≥ 10 s duration, with an amplitude reduction in PNOSE between the hypopnea criterion (C, expressed as a fraction of 1) and the squared value of the hypopnea criterion (C^2). If the prevalence of events within this range of amplitude reduction was relatively constant among patients, then the AHI derived from PNOSE and $\dot{V}$NOSE had a constant relationship. This is suggested by a close correlation between the AHI by $\dot{V}$NOSE and PNOSE (*See* Figure E1 of the online supplement). Therefore, if square root transformation of PNOSE is not available, the AHI by $\dot{V}$NOSE may be predicted from AHI by PNOSE according to the prediction equation ($AHI[\dot{V}NOSE] = -0.25 + 0.84 * AHI[PNOSE]$; $r^2 = 0.97$, $p < 0.0001$). Application of a correction factor of 0.84 is another acceptable way to correct the overestimation of the AHI by PNOSE.

With regard to the AHI, a mean index of respiratory disturbances over an entire sleep study, analysis of measures reflecting changes in lung volume (VOLRIP) and airflow ($\dot{V}$RIP, $\dot{V}$FM) provided similar results. Nevertheless, the physical and physiological significances of VOLRIP and $\dot{V}$RIP (or $\dot{V}$FM) are quite different, and the ratio of peak flow amplitude to tidal volume may vary depending on the shape (i.e., the time course) of the flow contour, in particular during inspiratory flow limitation. Related characteristics can even be utilized to infer presence of inspiratory flow limitation from inductive plethysmography waveforms (14).

In terms of precision in predicting the AHI by the flowmeter, PNOSE, $\dot{V}$NOSE, VOLRIP, and $\dot{V}$RIP seem to be equivalent as the mean absolute deviation from AHI by the flowmeter did not statistically differ among these methods (Table 1). The range within limits of agreement was wider for the square root–transformed nasal pressure than for the corresponding raw signal (the limits of agreement were bias $\pm$ 9.0 h^{-1} for $\dot{V}$NOSE, and bias $\pm$ 4.6 h^{-1} for PNOSE) (Table 1). This was related to a systematic trend for increasing overestimation of flowmeter-derived AHI by $\dot{V}$NOSE at higher values (Figure 1, Panel B).

The various evaluated methods (PNOSE, $\dot{V}$NOSE, VOLRIP, and $\dot{V}$RIP) also performed similarly well in apnea/hypopnea detection when comparisons to the flowmeter were made on an epoch-by-epoch basis. Between 77% and 88% of the variations in their apnea/hypopnea scores were related to variation in scores by the flowmeter (Table 1).

Results from apnea/hypopnea scoring according to Peppard and colleagues (11) demonstrate that including oxygen desaturation of $\geq 4\%$ into the event definition results in systematically lower AHI than when only flow amplitude criteria are considered (Table 1) (*See also* Table E1 in the online supplement). Our data provide a basis for conversion of AHI scored according to criteria validated by correlation with long-term outcome, i.e., the development of hypertension, with AHI based on quantitative measurement of ventilation by the gold standard of face-mask pneumotachography (i.e., by adding the bias of +3.6 h^{-1}). This may be of some help in the interpretation of mean AHI in groups of patients studied with one or the other technique. In an individual patient, however, simple algebraic conversions of AHI among reference standards is not appropriate because of the variability in AHI estimation by available methods. The major impact of various apnea/hypopnea definitions on the resulting AHI has been demonstrated recently (16).

The lack of significant correlations among subjectively perceived impairment of nasal breathing or objectively measured nasal resistance with deviation of AHI by nasal pressure from that by the flowmeter suggests that neither subjective nor objective nasal obstruction heralds inaccuracy of nasal pressure monitoring for estimation of the AHI. Relating amplitude reduction for definition of hypopnea to a local baseline over 2 min preceding an event may reduce the influence of changes in the nasal pressure/airflow relationship because of changes in nasal patency or oral ventilation.

We were able to reproduce close tracking of flowmeter-derived airflow by the square root–transformed nasal pressure signal over short time periods (Figure 2, Panels A and B), as reported in seated healthy subjects (2) and in a model simulation (10). However, we found highly variable proportionality coefficients among $\dot{V}$NOSE and $\dot{V}$FM if comparisons were extended over several hours (Table 2). Even in the absence of oral breathing or nasal cannula displacement, as verified by visual observation, shifts in proportionality coefficients were common over time (Figure 2, Panels E and F). Therefore, nasal pressure recordings as currently performed do not quantitatively reflect changes in airflow over more than very short time periods. Nevertheless, detection of inspiratory flow limitation events from the shape of the nasal pressure curve, an important application of the technique, does not seem to depend on quantitative tracking of airflow amplitude by the raw or linearized nasal pressure signal (10).

In conclusion, our data indicate that in terms of apnea/hypopnea detection nasal pressure monitoring compares favorably with the gold standard of face-mask pneumotachography and with respiratory inductive plethysmography, even in patients with partial nasal obstruction. Subjective and measured impairment of nasal breathing does not correlate with inaccuracy of nasal pressure–derived AHI. Square root transformation may linearize the nasal pressure/airflow relationship over short time periods, but it is not essential for improving accuracy of apnea/hypopnea scoring compared with analysis of the nasal pressure raw signal.

References

1. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. *Sleep* 1999;22:667–689.
2. Montserrat JM, Farré R, Ballester E, Felez MA, Pastó M, Navajas D. Evaluation of nasal prongs for estimating nasal airflow. *Am J Respir Crit Care Med* 1997;155:211–215.
3. Gugger M. Comparison of ResMed AutoSet (version 3.03) with poly-

somnography in the diagnosis of the sleep apnoea/hypopnoea syndrome. *Eur Respir J* 1997;10:587–591.
4. Bradley PA, Mortimore IL, Douglas NJ. Comparison of polysomnography with ResCare Autoset in the diagnosis of the sleep apnoea/hypopnoea syndrome. *Thorax* 1995;50:1201–1203.
5. Kiely JL, Delahunty C, Matthews S, McNicholas WT. Comparison of a limited computerized diagnostic system (ResCare Autoset) with polysomnography in the diagnosis of obstructive sleep apnoea syndrome. *Eur Respir J* 1996;9:2360–2364.
6. Rees K, Wraith PK, Berthon-Jones M, Douglas NJ. Detection of apnoeas, hypopnoeas and arousals by the AutoSet in the sleep apnoea/hypopnoea syndrome. *Eur Respir J* 1998;12:764–769.
7. Hernandez L, Ballester E, Farre R, Badia JR, Lobelo R, Navajas D, Montserrat JM. Performance of nasal prongs in sleep studies: spectrum of flow-related events. *Chest* 2001;119:442–450.
8. Mayer P, Meurice JC, Philip-Joet F, Cornette A, Rakotonanahary D, Meslier N, Pepin JL, Levy P, Veale D. Simultaneous laboratory-based comparison of ResMed Autoset with polysomnography in the diagnosis of sleep apnoea/hypopnoea syndrome. *Eur Respir J* 1998;12:770–775.
9. Fleury B, Rakotonanahary D, Hausser-Hauw C, Lebeau B, Guilleminault C. A laboratory validation study of the diagnostic mode of the Autoset system for sleep-related respiratory disorders (published erratum appears in *Sleep* 1996 19:601). *Sleep* 1996;19:502–505.
10. Farre R, Rigau J, Montserrat JM, Ballester E, Navajas D. Relevance of linearizing nasal prongs for assessing hypopneas and flow limitation during sleep. *Am J Respir Crit Care Med* 2001;163:494–497.
11. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med* 2000;342:1378–1384.
12. Panagou P, Loukides S, Tsipra S, Syrigou K, Anastasakis C, Kalogeropoulos N. Evaluation of nasal patency: comparison of patient and clinician assessments with rhinomanometry. *Acta Otolaryngol* 1998;118:847–851.
13. Bloch KE, Li Y, Sackner MA, Russi EW. Breathing patterns during sleep disruptive snoring. *Eur Respir J* 1997;10:576–586.
14. Kaplan V, Zhang JN, Russi EW, Bloch KE. Detection of inspiratory flow limitation during sleep by computer assisted respiratory inductive plethysmography. *Eur Respir J* 2000;15:570–578.
15. Bland MJ, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;1:307–310.
16. Redline S, Kapur VK, Sanders MH, Quan SF, Gottlieb DJ, Rappoport DM, Bonekat WH, Smith PL, Kiley JP, Iber C. Effects of varying approaches for identifying respiratory disurbances on sleep apnea assessment. *Am J Respir Crit Care Med* 2000;161:369–374.