

Finger plethysmography—a method for monitoring finger blood flow during sleep disordered breathing

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Abstract

We investigated a non-invasive measurement for changes in finger blood flow during wakefulness and sleep. Changes in finger blood flow, reflected by pulse wave amplitude (PWA) derived from finger plethysmography, were compared with changes in forearm vascular flow assessed by venous occlusion plethysmography after intra-arterial infusion of norepinephrine (NE), phentolamine, and isoproterenol ($n = 15, 15, 14$ subjects, respectively). Moreover, PWA was assessed during obstructive breathing during sleep ($n = 8$ patients). Vasoconstriction in the finger (PWA) was stronger than that obtained in the forearm vascular bed in the higher NE dose range both without (ANOVA, $P = 0.002$) and with concomitant phentolamine-induced α -receptor blockade ($P = 0.02$). Isoproterenol increased forearm blood flow but did not induce flow changes detectable by the finger plethysmographic technique ($P < 0.001$). PWA was significantly reduced during arousal from obstructive sleep apnea ($-37.5 \pm 16.1\%$, $P = 0.002$, $n = 6$ patients), suggesting vasoconstriction in the digital vascular bed. PWA derived from finger plethysmography allows continuous, non-invasive measurement of changes in finger blood flow during wakefulness and sleep. However, as may be expected from the anatomical and functional differences between the finger and forearm vascular beds and demonstrated by the lack of response to β_2 -receptor stimulation, PWA does not mimic forearm vascular flow characteristics. Thus, finger plethysmography may be a useful complement to current vascular research techniques, in particular to monitor sympathetic influences on skin blood flow in the finger.

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1. Introduction

Cutaneous circulation has to fulfil a number of different tasks: nutrition of tissue, reservoir of

blood volume for the systemic circulation, control of blood pressure, and the dissipation/conservation of body heat (Abramson, 1972). There is evidence generated by anatomical, physiological, and pharmacological studies that skin sympathetic activity, mediated via postganglionic pathways, plays a major role in regulation of cutaneous blood flow (Vissing, 1997). Beside the sympathetic nervous system output cutaneous finger blood flow is altered by systemic responses like changes

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in stroke volume, cardiac output, peripheral resistance, particularly in the arterioles, as well as by superimposed humoral and metabolic activation (Abramson, 1972).

The assessment of changes in blood flow and resistance of the peripheral circulation is complex due to the beat-by-beat changes in the flow/volume dynamics in the human cardiovascular system. Available methods for the assessment of changes in peripheral vascular resistance and flow are limited by reduced practicability, accuracy, or do not provide a continuous measurement signal. For instance, forearm flow measurement by venous occlusion plethysmography (Whitney, 1953) requires repeated inflation of a cuff placed around the upper limb and, thus, entails a limiting upper sampling frequency. Moreover, this technique is unsuitable for vascular studies during sleep, because cuff inflation frequently induces an arousal response. The pulse contour method (Thomas, 1978), based on mathematical modelling of the pulse wave form, is still hampered by limited accuracy and reliability due to specific changes of the wave form caused by peripheral pulse wave amplification (Bos et al., 1995). Techniques directly assessing sympathetic activity, e.g. recording of muscle sympathetic nerve activity, are invasive and cumbersome and may, in the sleeping subject, disturb sleep quality.

Sleep disordered breathing is characterised by frequent hypoxic episodes with subsequent arousals. Both mechanisms are known to activate the sympathetic system (Hedner et al., 1988). Different methods like heart rate analysis, pulse transit time calculations, and peripheral sympathetic nerve recordings have been used as diagnostic tools to assess autonomic responses to apneas and hypopneas during sleep. Another potential method may be finger plethysmography that is a non-invasive method to assess changes in finger blood flow. Pulse wave amplitude (PWA) is the most frequently used parameter obtained by finger plethysmography. PWA is directly and positively correlated to finger blood flow (Burch, 1954).

The hypothesis of this study was that finger plethysmography detects pharmacologically induced changes in finger blood flow, in particular changes induced by stimulation and blockade of

vascular α -receptors. Due to the anatomic structure of the finger we expected that alterations of vascular tone following sympathetic activation or inhibition might be reflected by changes of PWA. Moreover, we were interested in potential differences between responses of PWA and forearm flow measurements obtained by venous occlusion plethysmography. Finally, we hypothesised that physiologic sympathetic activation by obstructive sleep disordered breathing may alter the PWA signal.

PWA and forearm blood flow (venous occlusion plethysmography) were analysed during intra-arterial infusion of the α -receptor agonist norepinephrine (NE) with and without concomitant infusion of the α -receptor blocker phentolamine. Moreover, we investigated the effect of β_2 -receptor stimulation with isoproterenol on the PWA and forearm flow signals. Finally, to demonstrate the ability of finger plethysmography to continuously monitor vascular tone, PWA responses to obstructive breathing and concomitant arousal events in patients with obstructive sleep apnea were recorded and analysed.

2. Methods

2.1. *Protocols and subjects*

The study was performed in accordance with the principles outlined in the Declaration of Helsinki for research including human subjects. A written consent was obtained from all participants after oral and written information prior to the start of the investigation. The study protocol was evaluated and approved by the Research Ethics Committee of the Medical faculty, Gothenburg University.

In a previous protocol, forearm blood flow was assessed by venous occlusion plethysmography during intra-arterial infusion of NE, phentolamine, or isoproterenol (Grote et al., 2000). In the current study, PWA (plethysmography) was measured in parallel with forearm blood flow in 15 subjects (mean age 46.7 ± 5.1 years (\pm Standard Deviation (S.D.)), BMI 27.7 ± 2.1 kg/m² (S.D.), systolic/diastolic blood pressure $124.9 \pm 12.3/$

67.3 ± 7.7 mmHg (S.D.) and heart rate 59.5 ± 8.1 bpm (S.D.)). All subjects were free of cardiovascular or metabolic disease and did not take any vasoactive drug (Protocol 1).

Moreover, eight consecutive patients (one female, mean age 47 ± 3 years (S.D.), mean BMI 28.5 ± 3.4 kg/m² (S.D.), mean Respiratory Disturbance Index 39.5 ± 14.7 events/h (S.D.)), who had been referred to the sleep laboratory, were investigated during obstructive sleep disordered breathing (Protocol 2).

2.2. Description of the finger plethysmographic device

The finger plethysmograph consisted of a 30 ml cylinder (Terumo, Leuven, Belgium). The finger (digits II, III, or IV) was fixed up to the middle part of the proximal phalanx in the cylinder. The space between skin and cylinder wall was sealed for air tightness by a circular tape placed around the proximal phalanx. The air volume around the finger was approximately 14 ml. The open end of the cylinder was connected to a pressure transducer (pnb Medizintechnik, Kirchsesen, Germany) via a plastic tubing. The pressure signal was amplified by a Wheatstone-bridge and an in-house signal amplifier (Peura and Webster, 1978) and graphically displayed with a pen recorder (BD 101-6, Kipp & Zonen, Delft, Holland). The analogue output port (DC signal) allowed data export for further digital processing and analysis during the sleep studies.

2.3. Application of the finger plethysmographic technique

2.3.1. Protocol 1: forearm blood flow measurement

The forearm venous occlusion technique has previously been described in detail (Carlson et al., 1996). In short, a catheter was placed into the brachial artery of the non-dominant arm. A mercury-in-silastic strain gauge was placed around the widest part of the forearm and connected to an electronically calibrated plethysmograph (Elektromedicin AB, Kungsbacka, Sweden). Venous occlusion was achieved by inflation of a cuff placed proximate of the elbow. The finger plethysmo-

graphic signal was obtained from the same side as the forearm flow measurement.

Basal blood flow was quantified using 4 measurements without infusion. Thereafter, NE (Apoteksbolaget, Sweden) was infused intra-arterially to achieve consecutive active doses of 7.4, 31, 120, 472 and 1420 pmol/100 ml forearm-volume/min during 4 min at each dosage step. Flow was measured at four occasions during the last minute of each concentration step. After 30 min rest a constant infusion of phentolamine (Rogitine[®], Ciba Geigy, Basel, Switzerland) (dosage 5.3 µmol/100 ml forearm-volume/min), an α-adrenergic receptor antagonist acting on α₁- and α₂-receptors, was started, and the flow response to NE infusion protocol was repeated. After an additional 60 min rest the β₂-receptor agonist isoproterenol (Apoteksbolaget), was infused at 4.7, 9.5, 28.4, 71.0 pmol/100 ml forearm-volume/min, for 4 min at each dosage step.

2.3.2. Protocol 2: vascular responsiveness during sleep disordered breathing

Sleep recordings were performed during an overnight session at the hospital sleep laboratory equipped with standard polysomnographic montage and the EMBLA[®] polysomnography system (Flaga, Reykjavik, Iceland). Moreover, blood pressure was continuously assessed with the Finapres[®] system (Model 2300, Ohmeda, USA). The PWA signal was recorded concomitantly by finger plethysmography. Both the Finapres[®] and the PWA signals were derived from the non-dominant arm. The signals were digitised, amplified and evaluated using sleep-staging software (Somnologica[®]) (Flaga, Iceland). Conventional criteria for the scoring of sleep (Rechtschaffen and Kales, 1968) and breathing disorders (Anonymous, 1999) were used for sleep staging and classification of apneas.

2.4. Data analysis

Analysis was performed with a SPSS 7.5 for WINDOWS software package (SPSS, Illinois, USA). Throughout this article, data are reported as mean ± S.D. In the figures, means ± 95% confidence intervals (CI) are shown. *P*-values of *P* =

0.05 or less were considered as statistically significant.

Forearm blood flow was determined by geometrical analysis of at least four flow curves each lasting at least 10 sec. The mean value was taken for further evaluation. The PWA was determined manually from the original tracing. PWA was defined as the difference between the peak and the nadir of one pulse waveform and expressed in millivolt (mV).

2.4.1. Finger plethysmography during pharmacological stimulation (Protocol 1)

The percent changes in blood flow from baseline and the percent changes in PWA from baseline during infusion of NE ($n=12$), phentolamine together with NE ($n=14$), or isoproterenol ($n=12$) were compared using analysis of variance (ANOVA) for repeated measurements. 'Dosage' (e.g. NE 7.4–1420 pmol/dl/min) was treated as factor one and 'method' (forearm flow measurement and finger plethysmography) as factor two. Interaction between factor one and factor two was tested for significance as part of the ANOVA analysis. The two-sample *t*-test was used to compare the effect of phentolamine infusion between the two methods. Three subjects did not tolerate the highest dose of NE due to a profound vasoconstrictive response. In addition, the PWA signal was partly not valid during isoproterenol infusion in three subjects and during NE infusion together with phentolamine infusion in one subject. Therefore, ANOVA analysis was performed in 12, 12, and 14 subjects, respectively.

2.4.2. Sleep disordered breathing (Protocol 2)

End expiratory PWA, systolic and diastolic blood pressures, oxygen saturation and heart rate were assessed during obstructive sleep disordered breathing in eight patients. Obstructive apneas were evaluated in six patients (2–6 events per patient) and obstructive hypopneas in seven patients (2–5 events per patient). Data were obtained at the start, the middle, and the end of the breathing event, as well as at the start (first breath), the middle (third breath) and the end (fifth breath) of the arousal/hyperventilation period. PWA measurements at the different time

points were averaged for each type of breathing disorder (apnea or hypopnea) in each patient. The mean percent change in PWA from the end of the breathing event to the middle of the hyperventilation period was computed again for each type of breathing disorder in each patient. Significance was tested with the one sample *t*-test.

3. Results

3.1. Documentation of the finger plethysmographic method

NE infusion reduced both forearm vascular flow and PWA. (Fig. 1, $P < 0.001$ for dosage, $P = 0.55$ for method). The reduction of PWA by finger plethysmography was stronger in the high NE concentration range compared with the change in forearm blood flow (interaction factor 'dosage' and 'method', $P = 0.002$). During α -receptor blockade with phentolamine, overall increase in PWA was smaller than that in forearm flow (61.4 ± 60 and $135.2 \pm 77\%$, $P < 0.01$, $n = 15$), although both PWA and forearm flow were still reduced by increasing NE doses (Fig. 2, $P < 0.001$ for dosage, $P = 0.73$ for method). Moreover, the decrease in PWA was still significantly stronger at higher NE concentrations when compared with the decrease in forearm blood flow ($P = 0.02$ for interaction between dosage and method). Isoproterenol did not induce any identifiable change in the finger plethysmographic signal but increased forearm flow in a dose-dependent manner (Fig. 3, ANOVA interaction factor $P < 0.001$, factor 'method' $P < 0.001$).

3.2. Obstructive sleep disordered breathing

Average PWA increased early during apneas and hypopneas and reached its maximum between middle and late phases of the event. During the subsequent hyperventilation period there was a prominent decrease in PWA (Fig. 4). The mean reduction in PWA during apneas and hypopneas was $-37.5 \pm 16.1\%$ ($P = 0.002$, $n = 6$), and $-27.0 \pm 14.4\%$ ($P = 0.003$, $n = 7$), respectively (Fig. 5). The particular response pattern was consistent

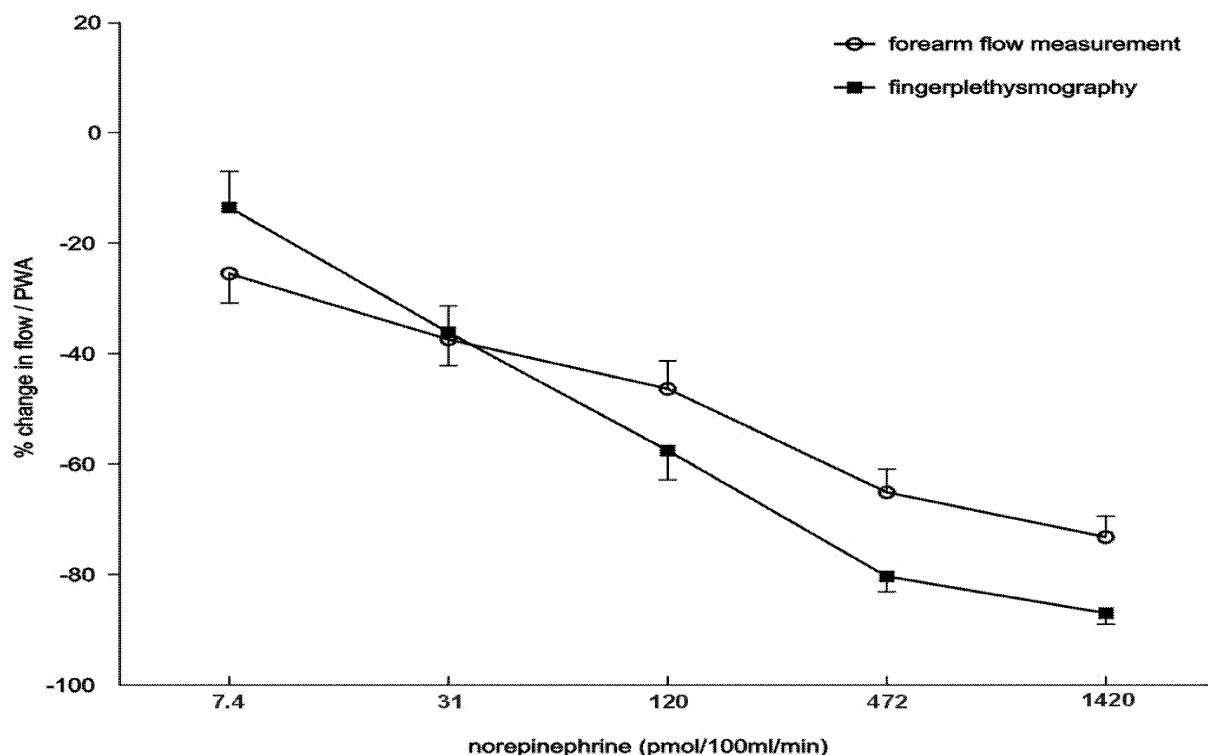


Fig. 1. Reduction of forearm vascular flow (open circles) and PWA (filled squares) by finger plethysmography (% change from baseline) during NE infusion (increasing dose steps). Shown are means \pm 95% CI in 15 subjects (highest NE dose 12 subjects).

for within-patient and between-patients comparisons. Obstructive events with arousal caused a less pronounced reduction of PWA during REM sleep compared with during NREM sleep (-36 ± 63.5 and -86.0 ± 73.6 mV, $P = 0.004$, $n = 7$). Visually, peaks of systolic blood pressure during the hyperventilation phase appeared to coincide with PWA nadirs (Fig. 4). The magnitude of systolic blood pressure increase correlated negatively and not significantly with the reduction of PWA ($r = -0.64$, $P = 0.17$, $n = 6$).

4. Discussion

Determination of PWA by finger plethysmography provides a tool for continuous non-invasive assessment of changes in digital blood flow. PWA was highly sensitive to pharmacological stimulation of vascular α -receptor in the finger vascular

bed. In addition, arousal following upper airway obstruction during sleep markedly reduced PWA. However, there was no PWA response to a marked forearm blood flow increase induced by vascular β_2 -receptor stimulation.

4.1. The finger plethysmographic technique

Due to their large skin surface extremities play an important role in body heat conservation and dissipation by modulating skin blood flow in these regions (Burch, 1954). Digital blood flow varies between approximately 0.5 and 90 ml/100 cm³ tissue within the same subject (Burton, 1939) and is mainly (to 95%) determined by skin blood flow (Porter and Swain, 1986). Moreover, constriction and dilation of cutaneous vessels of the finger have been suggested to be predominantly modulated by sympathetic vasoconstrictor tone (Doupe et al., 1939; Abramson, 1972).

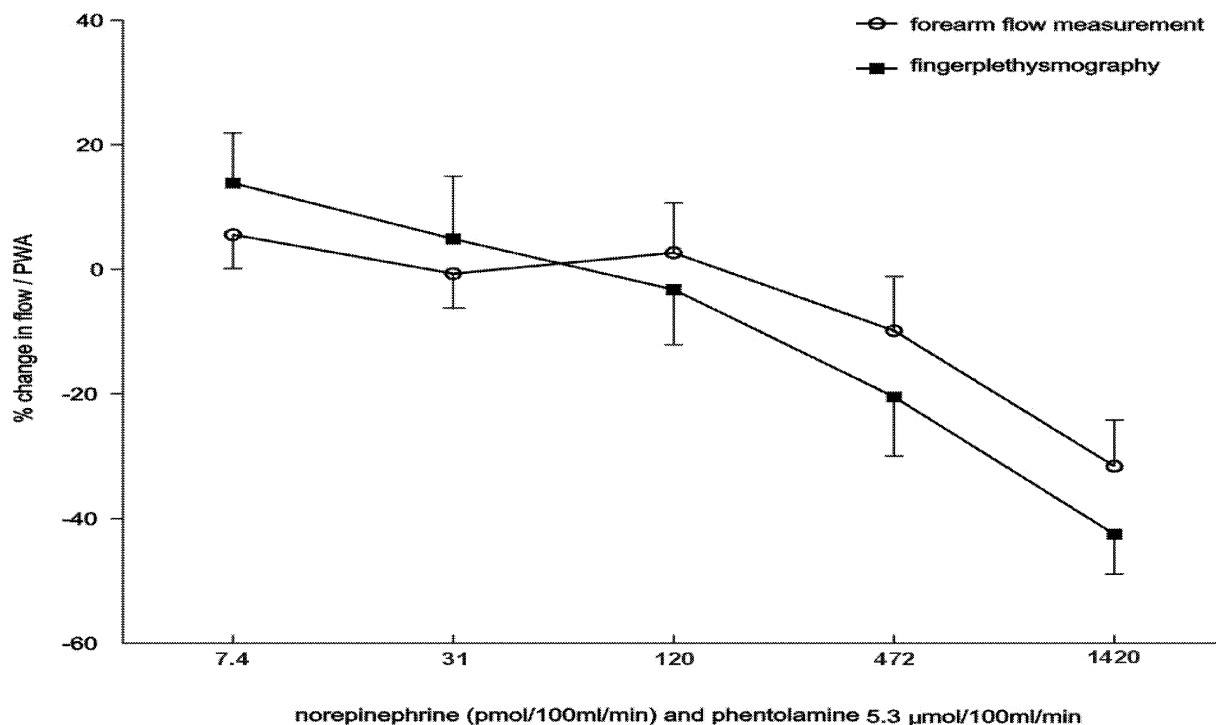


Fig. 2. Reduction of forearm vascular flow (open circles) and PWA (filled squares) by finger plethysmography (% change from baseline) during NE infusion (increasing dose steps) and ongoing phentolamine administration ($5.3 \mu\text{mol}/100 \text{ ml}$ forearm-volume/min). Baseline (0-point of y-axis) was defined after 10 min of phentolamine infusion when the NE-infusion protocol was started. Shown are means \pm 95% CI in 14 subjects as relative changes from the above mentioned baseline value.

Finger plethysmography, a technique first described decades ago (Burch, 1954), uses a cylinder or a strain gauge applied to the distal phalanx of one finger for detection of beat by beat changes in pulsatile blood volume of the finger. Studies comparing pulse wave finger plethysmography and laser Doppler measurements, thermography, or forearm flow measurement have demonstrated good agreement in the determination of dynamic changes between PWA and digital blood flow (Burch, 1954). Temperature changes (Doupe et al., 1939; Rieckert and Closs, 1968; Saumet et al., 1986b), smoking (Saumet et al., 1986a), breathing manoeuvres (Doupe et al., 1939), pain or acoustic stimuli (Burton, 1939), as well as local/systemic drug application (Burch, 1986; Schnelle et al., 1981) have been shown to modify finger blood flow and PWA. The analysis of PWA is validated and easy to perform (Burton, 1939).

4.2. Limitations of the PWA analysis

Application of finger plethysmography requires some limiting considerations. Firstly, PWA is determined by a number of hemodynamic factors including arterial inflow, venous outflow, cardiac stroke volume, venous return to the heart as well as alterations of autonomic neural control (Burch, 1954). Moreover, the position of the finger relative to the heart level (Burch, 1954), arm and hand movements (e.g. in sleep studies after arousal), and pre-constriction of finger arteries (e.g. low surrounding temperature, excitement, and stress), may affect the signal. Finally, the agreement between baseline digital blood flow and PWA has been reported to vary to a great extent between subjects (Zweifler et al., 1967). Therefore, only within-subject changes in PWA during a limited time interval were evaluated in the current study.

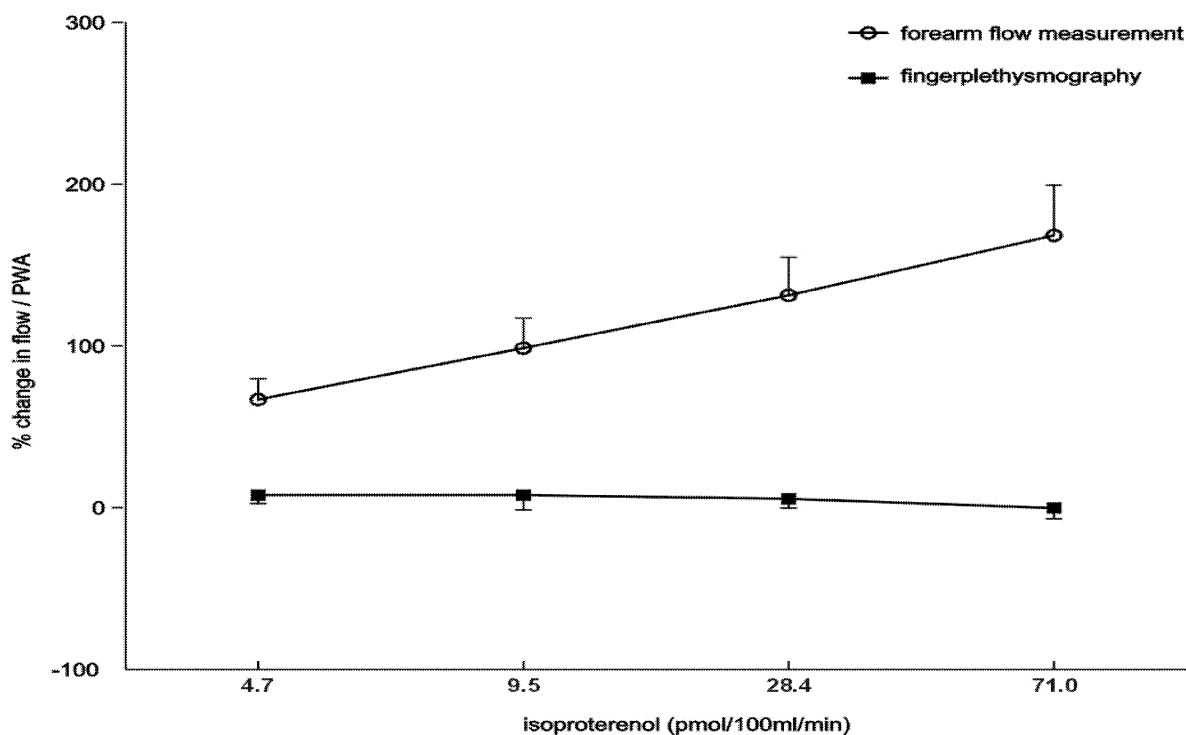


Fig. 3. Change in forearm vascular flow (open circles) and PWA (filled squares) by finger plethysmography (% change from baseline) during isoproterenol infusion at four different dose steps. Shown are means \pm 95% CI in 12–14 subjects.

Henriksen and Sejrsen (1976) reported approximately 25–35% reduction of cutaneous, subcutaneous and muscular blood flow during occlusion of venous flow with cuff pressures exceeding 40 mmHg. This local reflex mechanism, which may prevent oedema by balancing the ratio of pre- to post-capillary pressure, was attenuated in sympathectomized patients. In the current experimental setting, the finger plethysmographic cylinder was applied to the finger in an airtight manner, and a venous congestion may have occurred distal to this circular occlusion. We did observe a discrete oedema in several fingers after the measurement. However, an adaptation period of 15 min took place before start of the experimental procedure and we did not record any consistent decline in PWA after initiation of the measurement. Although we cannot exclude that an auto-circulatory reflex may have affected our measurement of PWA, it is unlikely that the relative change in PWA was biased to any major extent.

Furthermore, phentolamine-induced flow increase, a potential cause of oedema, did not cause an observable disagreement between forearm flow and finger plethysmography. Finally, volume changes in the finger plethysmographic chamber were accompanied by small changes in blood pressure, which may have dampened/enhanced the PWA at high/low digital flow, respectively. Part of the non-linear relationship between α -receptor stimulation by NE and the PWA response (Figs. 1 and 2) may be explainable by this phenomenon.

Other limitations of the device have to be mentioned. (1) Our results show that PWA is not just a substitute measure of forearm blood flow changes, since there are profound differences between the forearm and the finger vascular beds (Abramson, 1972; Vissing, 1997). (2) PWA obtained by finger plethysmography depends on stroke volume but it has not been used to predict changes in cardiac output. (3) Although blood

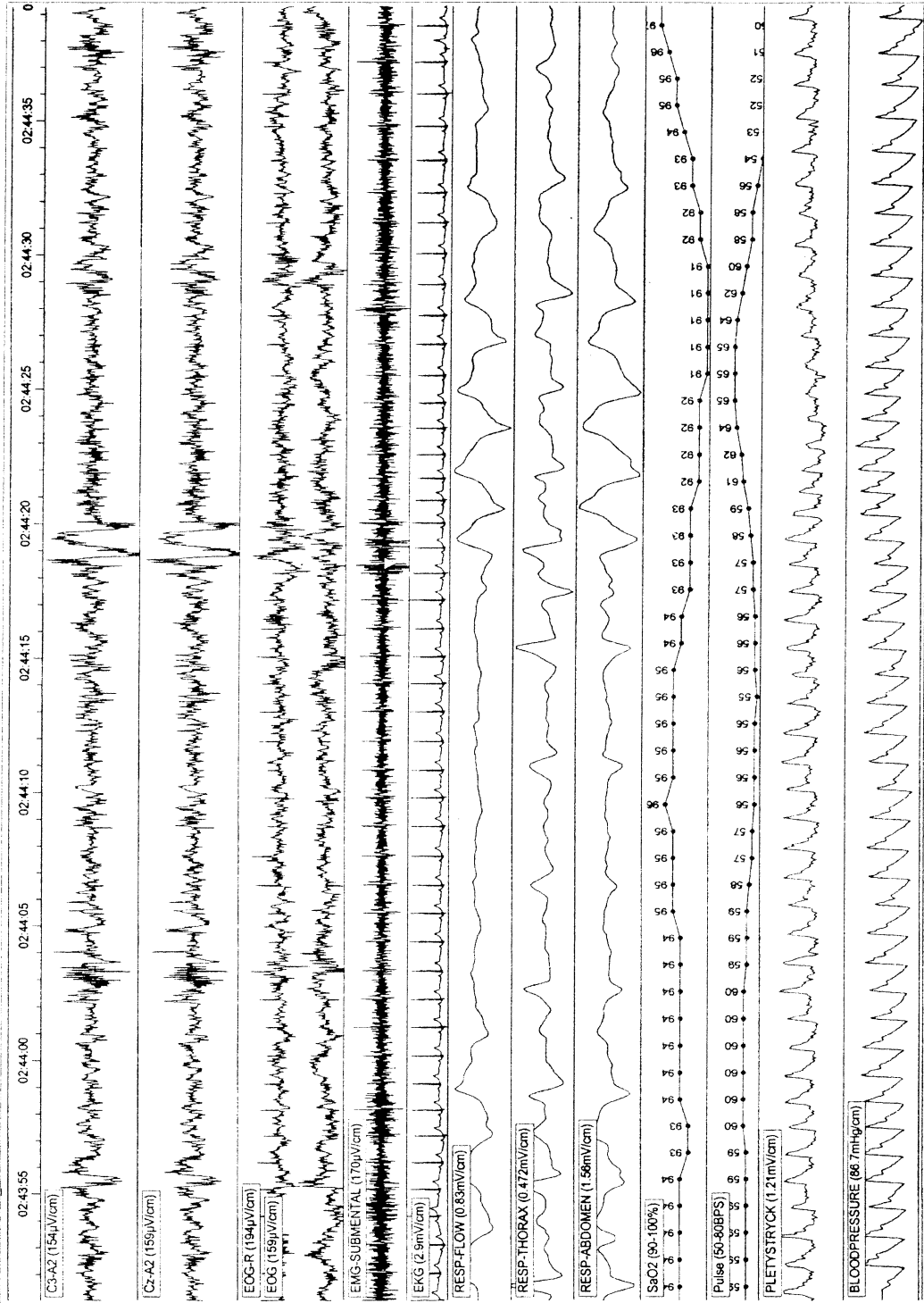


Fig. 4

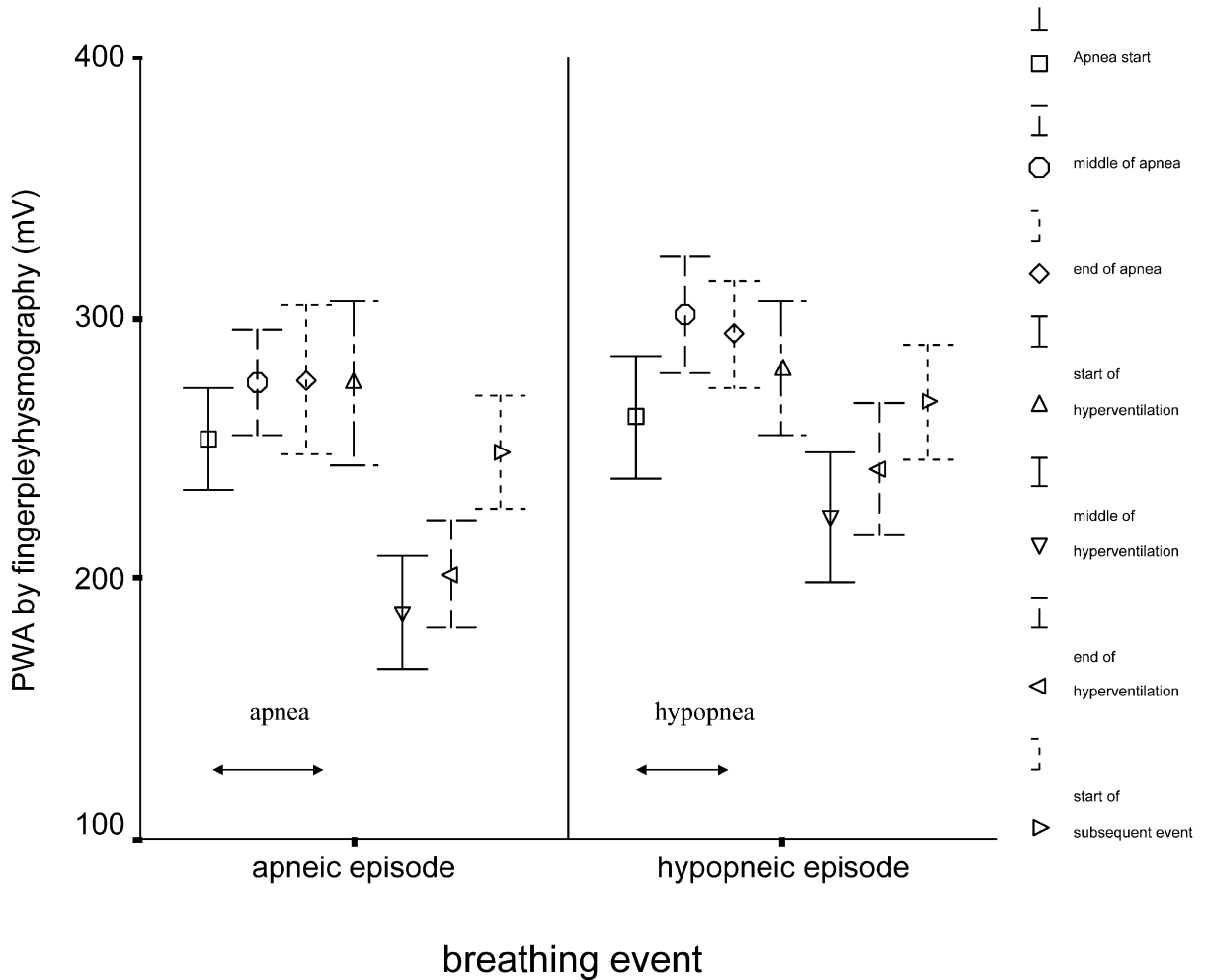


Fig. 5. PWA during sleep disordered breathing events (start/middle/end of event) and during subsequent arousal (start/middle/end of hyperventilation and start of next breathing event) in obstructive apnea ($n = 6$ patients, 39 events), and obstructive hypopnea ($n = 7$ patients, 26 events). Shown are means \pm 95% CI.

pressure and digital blood flow may be negatively correlated during certain reflexes or stimulations, PWA analysis is not a reliable tool to assess changes in blood pressure as demonstrated by the results in our study.

In addition, there are a number of other physiological stimuli, which may cause a significant change in PWA assessed by finger plethysmography, which were not assessed in this study. Doupe et al. (1939) used a different plethysmo-

Fig. 4. Polysomnographic recording during NREM sleep stage II with electroencephalogram (C3-A2 and Cz-A2), electrooculogram (EOG R and EOG), electromyogram (EMG submental), electrocardiogram (EKG), thermistor (Resp Flow), thoracic (Resp Thorax) and abdominal belt (Resp Abdomen), pulseoximetry (SaO₂ and Pulse), digital plethysmography (Plethystryck), fingerphotoplethysmography by Finapres (Blood Pressure) and the time scale (h:min:sec) at the top of the figure. The PWA obtained by digital plethysmography varied during the obstructive apnea (no visible flow in the Rep Flow signal) and was prominently reduced during arousal and hyperventilation. Blood pressure increased during arousal and hyperventilation.

graphic technique compared with our study. The authors described a marked reduction of PWA after deep inspiration during wakefulness, cold exposure, and a mental arithmetic test. Reduced PWA was accompanied by increased digital blood pressure and a reduction of finger blood flow.

4.3. Documentation of the finger plethysmographic method during pharmacological modification of vascular tone

Venous plethysmography is a validated tool for the assessment of blood flow in the forearm vascular bed (Whitney, 1953). NE markedly reduced both forearm blood flow and PWA. This response in the finger vascular bed may in part be explained by an increase in vascular resistance in the forearm and hand. However, we observed that the relative response of PWA to NE was more pronounced than the forearm flow response. This may reflect a higher relative density of α -receptors in digital artery smooth muscle cells. Indeed, marked vasoconstriction and blood pressure elevation in the finger were produced in the absence of altered brachial artery pressure (Doupe et al., 1939). In contrast, stimulation of β_2 -receptors with isoproterenol did not change the PWA signal. Thus, while vasomotor tone in fingers and the hand appears mainly regulated by stimulation of α -receptors but not β_2 -receptors, forearm vascular regulation may be governed by both types of receptors and, thus, active vasoconstrictory and vasodilatory processes.

Phentolamine increased both forearm blood flow and finger PWA. However, the relative augmentation of PWA was smaller when compared with forearm blood flow. There are several possible explanations for this finding. Firstly, this observation may reflect the differences in basal vascular tone in the two vascular beds of the forearm and the finger (Abramson, 1972; Vissing, 1997). Secondly, due to local reflex mechanisms (Henriksen and Sejrsen, 1976) or other reasons, PWA may not be linearly related to finger blood flow in the higher flow range. Thirdly, the results reflect systemic effects of NE and secondary to those changes in finger blood flow.

Another important finding was the fact that isoproterenol (β_2 -receptor agonist)-induced vasodilation in the forearm was not detectable by PWA analysis in the finger. An early study by Barcroft and Swan (1953) found no initial flow increase in the hand and the fingers during epinephrine infusion, while forearm blood flow increased profoundly. At the time, however, these authors did not have access to modern receptor pharmacology to explain their findings. Subsequent investigations have provided additional evidence that β_2 -receptors have no functional role in modulating sympathetic tone in the hand and the finger (Cohen and Coffman, 1981).

4.4. Documentation of the finger plethysmographic method during obstructive sleep disordered breathing

PWA increased during the initial phase of the obstructive breathing event, suggesting vasodilation. Thus, our observation may be explainable by hypoxia-induced release of vasodilatory mediators and/or withdrawal of constrictive factors (Leuenberger et al., 1999). Another possible explanation is a return to baseline values after the sharp decline caused by the preceding arousal. In fact, PWA values comparable to quiet breathing were not reached until the end of apnea. Recent work (Schnall et al., 1999) using a pressure variable finger plethysmograph did not report this apparent dilatory response during the initial phase of obstructive apneas and hypopneas. The muscle sympathetic nerve traffic recorded from the peroneal nerve, often referred to as a marker of vasoconstriction, increases continuously during obstructive apneas to abruptly decrease after cortical arousal (Hedner et al., 1988). This discrepancy in the pattern between muscle sympathetic nerve activity and PWA may reflect a differential sympathetic outflow to the muscle and cutaneous vascular beds (Vissing, 1997; Abramson, 1972). Moreover, other vasoactive modulators such as angiotensin II, endothelin-1, nitric oxide, or adenosine may have influenced vascular tone differentially in the finger and the forearm vascular beds during obstructed breathing.

The post apnea hyperventilation period was associated with a marked decrease in PWA (Schnall et al., 1999) in conjunction with a marked pressor response. Several different mechanisms may contribute to this effect: first, cortical arousal is associated with bursts of sympathetic activity, both neural and humoral (Baust et al., 1968; Abramson, 1972; Vissing, 1997) leading to vasoconstriction of finger arteries and/or skin vasculature. Second, apnea and hypopnea induced hypoxia may have stimulated central chemoreceptors which are known to activate cutaneous sympathetic nerve fibers (Vissing, 1997) and this pathway may at least in part explain the sympathetic activation. Third, sympathetic acceleration of heart rate results in decreased diastolic time and actual stroke volume, both of which may reduce PWA. Fourth, deep inspiration during hyperventilation per se is known to increase peripheral vascular tone which may contribute to a decrease in PWA (Bolton et al., 1936).

A recent study investigated the effect of arousal from sleep on digital blood flow assessed by a pressure-variable finger plethysmography (O'Donnell et al., 2002). The presence of an EEG-verified arousal after an obstructive breathing event during sleep was associated with a significant stronger reduction in pulse volume amplitude (1.000–0.767, arbitrary units) in the finger when compared with an obstructive breathing of same duration without an arousal (1.000–0.923, arbitrary units, $P < 0.001$). However, our study did not assess the relative importance of the different mechanisms discussed above. Thus, further mechanistic studies are warranted to answer these questions.

A previous study on sleep stage related differences in PWA (Lavie et al., 2000) found reduced signal amplitudes during REM sleep. This difference between PWA during undisturbed NREM and REM sleep was less obvious in the present study. However, we observed an attenuated PWA response after obstructive breathing events during REM sleep when compared with NREM sleep. It is possible that high baseline sympathetic activity associated with normal REM sleep may have prevented further sympathetic activation during disordered breathing in the sense of a ceiling effect. Alternatively, mechanisms counterbalancing vaso-

constriction, e.g. α -receptor down regulation (Grote et al., 2000), may be more influential in REM sleep than in NREM sleep.

Besides obstructed breathing, other phenomena disturbing sleep, such as periodic leg movements with and without cortical arousal or unexplained EEG-arousal, have been reported to reduce digital blood flow (Pillar et al., 2002). Thus, the PWA response may reflect a relatively uniform pattern of arousal related sympathetic activation, which includes, besides a number of other mechanisms, a constriction in the skin and possibly muscle vascular beds. Further studies are warranted to identify the relative contribution of these factors to arousal and apnea related hemodynamic changes. Finger plethysmography offers a simple tool to continuously monitor changes in digital blood flow that may reflect at least in part sympathetic activation of the skin vasculature.

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