

# A Novel Method for the Detection of Apnea and Hypopnea Events in Respiration Signals

Péter Várady, *Student Member, IEEE*, Tamás Micsik, Sándor Benedek, and Zoltán Benyó\*

**Abstract**—The monitoring of breathing dynamics is an essential diagnostic tool in various clinical environments, such as sleep diagnostics, intensive care and neonatal monitoring. This paper introduces an innovative signal classification method that is capable of on-line detection of the presence or absence of normal breathing. Four different artificial neural networks are presented for the recognition of three different patterns in the respiration signals (normal breathing, hypopnea, and apnea). Two networks process the normalized respiration signals directly, while another two use sophisticatedly preprocessed signals. The development of the networks was based on training sets from the polysomnographic records of nine different patients. The detection performance of the networks was tested and compared by using up to 8000 untrained breathing patterns from 16 different patients. The networks which classified the preprocessed respiration signals produced an average detection performance of over 90%. In the light of the moderate computational power used, the presented method is not only viable in clinical polysomnographs and respiration monitors, but also in portable devices.

**Index Terms**—Classification, neural networks, polysomnography, respiration monitoring, sleep apnea.

## I. INTRODUCTION

### A. Medical Background

**D**ISTURBANCE of the normal breathing process can cause the development of severe metabolic, organic, central nervous, and physical disorders. Respiration monitoring allows the continuous measurement and analysis of breathing dynamics and, thus, the detection of various disorders. There are a number of breathing disorders, but *sleep apnea syndrome* (SAS) is probably the most common amongst them. Almost 5% of the total human population suffers from it, and its occurrence increases up to 30% in the population of males over 70 years old in the industrially developed lands [1].

The Greek word *apnea* means: without breathing. An episode of *apnea* occurs if someone's breathing ceases for a certain amount of time, by definition: if the magnitude of the respiration movements are decreased for at least 10 s to less than 5% of the physiological values [1], [2]. A mild version of apnea is

*hypopnea*, where the movements are decreased below half the normal values. The occurrence of sleep apnea episodes might be physiological; they would usually be regarded as being pathological only if more than 5 episodes of apnea occur per sleeping hour [1], [3]. The origin of apnea can be *central* (CA), caused by the lack of central moto-neural respiration drive, or can be *obstructive* (OA), caused by the occlusion of the upper airways.

The blood oxygen saturation falls during apnea, because no gas exchange can take place. This reaches clinical significance if the blood oxygen saturation decreases below 95% of the saturation level before the episode of apnea and this lasts for more than 10 s. The desaturation event activates the sympathetic nervous systems. This results in increasing heart rate and blood pressure, which can stress and possibly injure aspects of the cardiovascular system. Disturbances in hormone levels caused by the sympathetic activation can result in the long-term development of metabolic disorders such as insulin resistance, diabetes mellitus, and obesity [4]. In addition, a micro-arousal happens during sleep that is related to the resolution of the apnea. Since these micro-arousals happen at each episode of apnea, the physiological structure of sleep becomes fragmented. All these effects may combine and lead to progressive and serious change, even to incurable damage to numerous parts of the human organism.

### B. State-of-the-Art Apnea Diagnosis

Today the only reliable diagnostic method for the detection of SAS is the *polysomnographic* (PSG) assay which is a multichannel signal record measured during the whole sleeping process. The standard diagnostic nocturnal PSG consists of the following vital parameters [5]: electroencephalogram (EEG), electro-oculogram (EOG), electromyogram (EMG), nasal airflow (NAF), abdominal and/or thoracic movements, body position, snore microphone, electrocardiogram (ECG), and blood oxygen saturation (SaO<sub>2</sub>). A limited-channel version of PSG is also frequently used for apnea screening, especially in portable devices, including only the following signal channels: NAF, abdominal and/or thoracic movements, SaO<sub>2</sub>, and heart rate [6].

The diagnosis of SAS has several standardized methods and steps, including the detection of apnea and hypopnea, the determination of their type (CA/OA), and the calculation of the respiration disorder index (RDI), i.e., the number of apnea and hypopnea events per sleeping hour [1]. According to current accepted clinical criteria the episodes of apnea and hypopnea are detected in the respiration signals, while the arousals detected with EEG and the desaturation episodes in the SaO<sub>2</sub> signal provide supportive evidence [5], [7].

Manuscript received July 4, 2001; revised April 5, 2002. This work was supported by the Hungarian National Research Fund under Grant OTKA T029830. Asterisk indicates corresponding author.

P. Várady and T. Micsik are with the Laboratory of Medical Informatics, Department of Control Engineering and Information Technology, Budapest University of Technology and Economics, H-1117 Budapest, Hungary.

S. Benedek is with the Institute for Electric Power Research, H-1016 Budapest, Hungary.

\*Z. Benyó is with the Laboratory of Medical Informatics, Department of Control Engineering and Information Technology, Budapest University of Technology and Economics, Pázmány s. 1/d., H-1117 Budapest, Hungary (e-mail: benyo@iit.bme.hu).

Publisher Item Identifier 10.1109/TBME.2002.802009.

Computer support is indispensable for the proper evaluation of the PSG records. There are numerous methods existing for the off-line analysis of respiration signals stored in PSG records [8]–[14]. Usually, the vendor of the PSG device provides its own signal analyzing software package. The applied methods are mostly based on time domain algorithms, measuring the amplitude and frequency of breathing. Existing tools can detect apnea with 80%–90% specificity and sensitivity. The precision in the case of hypopnea detection is somewhat lower [15].

Current compact portable PSG devices can perform stand-alone signal analysis and provide apnea indexes. These are very useful instruments since they can be mounted on the patient, who can take it home and sleep with it in a home environment.

This paper presents an innovative method for the on-line detection of apnea and hypopnea events using only the respiration signals. The developed method can be applied in the field of PSG processing and respiration monitoring and is, hence, capable of:

- robust, on-line detection of apnea and hypopnea events with high specificity and sensitivity;
- detection without using patient specific information;
- moderate computational time (implementation in portable devices).

Note: the determination of apnea type, i.e., CA versus OA is not addressed in this paper.

## II. METHODS

### A. Signal Records

We used the signal records of the MIT-BIH reference PSG database that is freely available for research purposes from PhysioNet [16]. The database consists of 18 PSG records having lengths from 1 hour up to 7 hr (4 hr on average). For the purpose of this study we selected 16 records which were all taken from different patients.

The selected records contain different vital parameters: all records contain a NAF signal measured by a thermistor, a single-channel ECG and EEG signal and a blood pressure signal. Nine records include an additional thoracic or abdominal excursion signal (RIP) measured by inductance plethysmography. Only five records include the  $\text{SaO}_2$  signal measured by pulse-oxymetry.

### B. Design Considerations

The presented apnea detection system is based only on the respiration signals (NAF and/or RIP) which carry the primary evidence of apnea and hypopnea. The developed method is capable of determining three different patterns in the respiration time series which correspond to the medical terminology:

- normal breathing (to be referred to as N): a periodic function with slightly fluctuating magnitude and frequency;
- hypopnea (to be referred to as H): abnormal breathing with attenuating or decreased amplitude (respiration effort less than half of normal breathing) for a duration of more than 10 s;

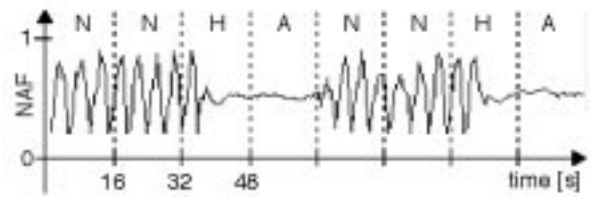


Fig. 1. Respiration patterns in a normalized NAF signal using a 16-s classification time window.

- apnea (to be referred to as A): lack of NAF (respiration effort less than 5% of normal breathing) for a duration of more than 10 s.

The on-line operation requires that only small sections of the respiration time series can be used for detection. The respiration patterns in the consecutive sections can be determined by an appropriate classification time window. According to the definition of apnea, the length of the window should be at least 10 s in order to detect apnea within a single time window. We decided to use a longer window size, since respiration signals may contain short apnea and hypopnea episodes (lasting only several seconds) which are not pathological, but could disturb classification stability. The selected window size was 16 s in order to get an integer number of samples after the signal preprocessing. Using a longer window would unnecessarily increase the system's complexity. It would also be difficult to detect periods of apnea shorter than the time window, and to distinguish between consecutive episodes of apnea and hypopnea which are usually very common in PSG records.

The selected time window is stepped along the respiration signals, resulting in a series of respiration pattern categories consisting of N, H, and A (see Fig. 1). It must be noted that the event detection is delayed by a period equal to the window time, as a consequence of windowing. The resolution of the detection is determined by the window step size which equals the window time if no window overlapping occurs. A finer resolution is possible if window overlapping is used. When the total lengths of the apnea and hypopnea periods are calculated, the consecutive A and H events must be joined, respectively.

Many authors have pointed out the nonlinearity of cardio-respiratory signals [17], and used different types of artificial neural networks (ANNs) for their processing [18], [19]. We propose four different ANNs for time-domain respiration pattern classification. Two networks directly process the normalized respiration signals, while the other two use some sophisticatedly derived signals as network input. The entire signal flowchart in the case of two networks is depicted in Fig. 2.

### C. Signal Preprocessing

The productivity of an ANN depends heavily on the proper preprocessing of its input signals. The selected PSG records were sampled at 250 Hz with 12-bit resolution. Due to practical considerations, the sampling rate was decreased to 25 Hz using a simple moving average filter.

The baseline and the gain of an  $s$  respiration signal are specific to the applied sensor and they can change during long-term

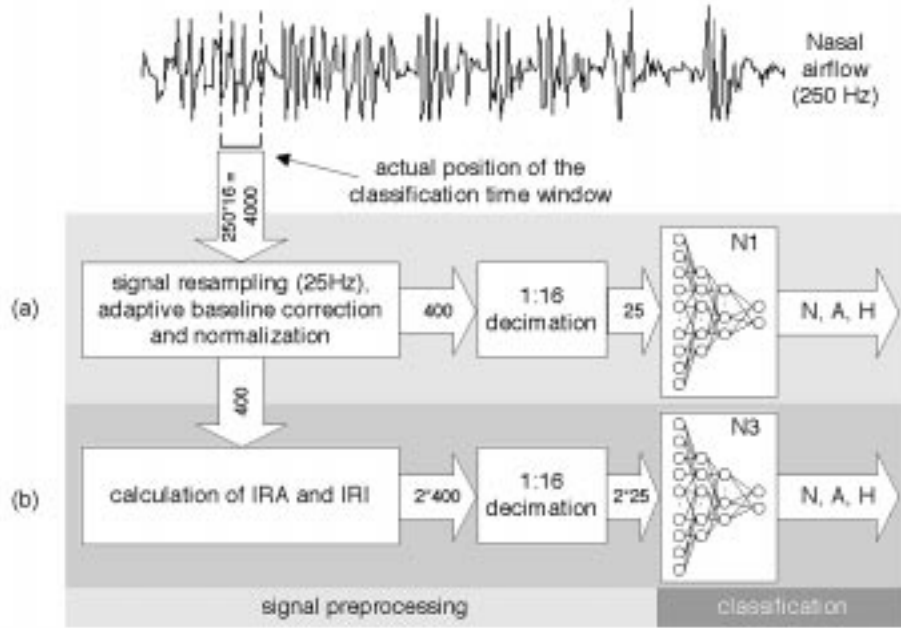


Fig. 2. Neural-network-based detection of apnea and hypopnea events using (a) the time series of the NAF signal or (b) the derived IRA and IRI signals. The numbers in the arrows show the signal dimensions in the selected classification time window.

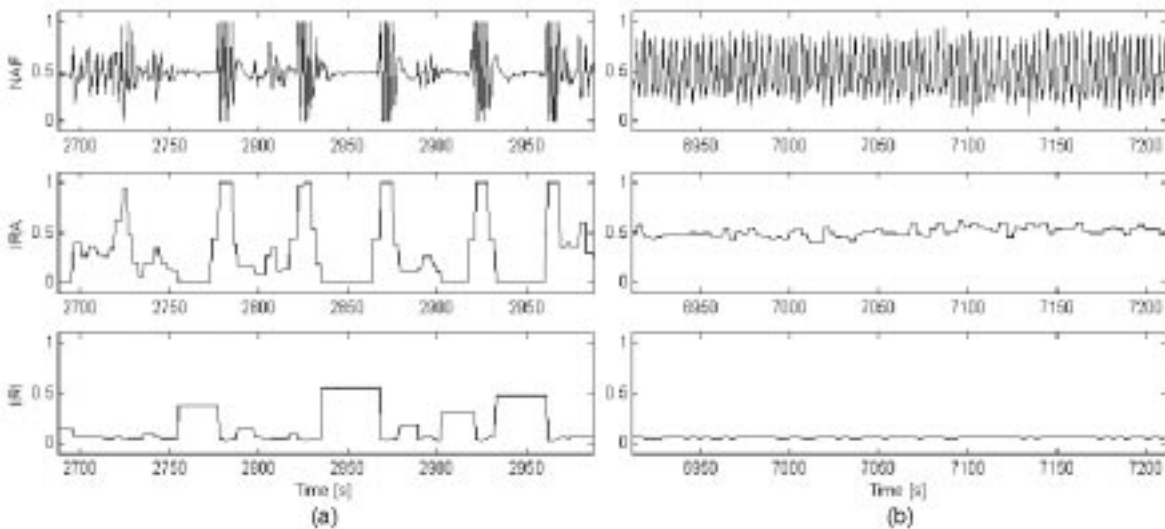


Fig. 3. NAF and the derived IRA and IRI signals in two excerpts of record SLP03 containing (a) apnea events and (b) normal breathing.

measurements because of sensor or patient movements. Therefore, an adaptive signal normalization was used where the  $d_n$  baseline correction and the  $f_n$  scale factor for the  $n$ th processing window were determined by the formulas

$$d_n = d_{n-1} - \frac{1}{4} \left( d_{n-1} - \frac{\max(s) + \min(s)}{2} \right)$$

and

$$f_n = \min \left\{ f_{n-1} - \frac{1}{8} (f_{n-1} - \max(s) + \min(s)), k \right\}$$

where the limit value  $k = 100, \dots, 500$  is specific to the sensor.

The initial factors were  $f_0 = 2 \cdot k$  and  $d_0 = 2048$ . The  $r$  samples of the normalized signal were obtained by adjusting each sample of the  $s$  original signal within the  $n$ th window

$$r = \frac{s - d_n}{f_n} + \frac{1}{2}.$$

Finally, values remaining outside the range  $[0, 1]$  due to improper factor adaptation were truncated.

An instantaneous respiration amplitude (IRA) and an instantaneous respiration interval (IRI) signal were derived from both of the normalized  $r$  respiration signals (NAF and RIP). Both the IRA and IRI signals were normalized and spanned over the continuous time (see Fig. 3). In the calculation of IRI we assumed that the respiration interval times lie in the range  $[0s, 60s]$ . The

TABLE I  
TOPOLOGIES OF THE PROPOSED ANNS

Ref.	Input Signals (dimension)	# of units in the ANN (input – hidden – hidden – output)
N1	NAF (25)	25 $\hat{n}$ 6 $\hat{n}$ 4 $\hat{n}$ 2
N2*	NAF (25) RIP (25)	50 $\hat{n}$ 10 $\hat{n}$ 4 $\hat{n}$ 2
N3	IRA from NAF (25) IRI from NAF (25)	50 $\hat{n}$ 10 $\hat{n}$ 4 $\hat{n}$ 2
N4*	IRA from NAF (25) IRA from RIP (25) IRI from RIP (25)	100 $\hat{n}$ 20 $\hat{n}$ 5 $\hat{n}$ 2

\*For records with two respiration signals (NAF, RIP)

derived IRA and IRI signals were used as inputs in the case of two ANNs (see N3 and N4 in Table I).

The calculation method of the IRA/IRI signals is shown in Fig. 4, where  $f(a, b) := c$  denotes that the  $f$  signal gets the value  $c$  over the closed interval  $[a, b]$ . At first, the individual breaths are located in the  $r$  signal, using the local minimum and maximum signal amplitudes at positions  $p_{\min}$  and  $p_{\max}$  which are relative to the current position ( $p_{\text{curr}}$ ). The  $i$  time interval of a breath corresponds to the difference between  $p_{\min}$  and  $p_{\max}$  scaled with the  $f_s$  sampling rate, while the  $a$  amplitude of the breath is given by the signal value difference in these positions. If the interval is smaller than  $t_b = 4s$  or the amplitude lies below  $a_{\min} = 0.1$ , the breath is discarded. This provides protection against signal noise. If no breath occurs over a given threshold time ( $t_a = 8s$ ), IRA is set to zero around the current breath's location. This emphasizes the apnea events.

After obtaining the IRA and IRI signals, both the normalized original and the derived signals were resampled at 1.5625 Hz using an antialiasing filter. Therefore, the selected classification time window of 16 s consisted of only 25 sample points (see Fig. 2).

#### D. Classification and Network Implementation

We designed and implemented four different feed-forward ANNs, with the topologies as summarized in Table I.

According to medical practice, the presence of apnea (both OA and CA) is clearly evident by the analysis of the NAF signal. Thus, the single-signal networks (N1 and N3) use only NAF or its derived signals. The two other networks (N2 and N4) were designed to use both the NAF and the RIP signals. During the training of these networks, we used specific target values in order to consider NAF as the main evidence of apnea and hypopnea.

The networks were implemented in Matlab, using the Neural Network Toolbox [20]. For the purpose of network training we chose nine records and selected 60 fragments from each of them, each with the length of one time window (20 pieces of N, H, and A fragments, each consisting of 25 sample points). The excerpts were taken both from the original and the derived IRA and IRI signals. The total number of training patterns was 540 (60 patterns from each of the nine selected training records).

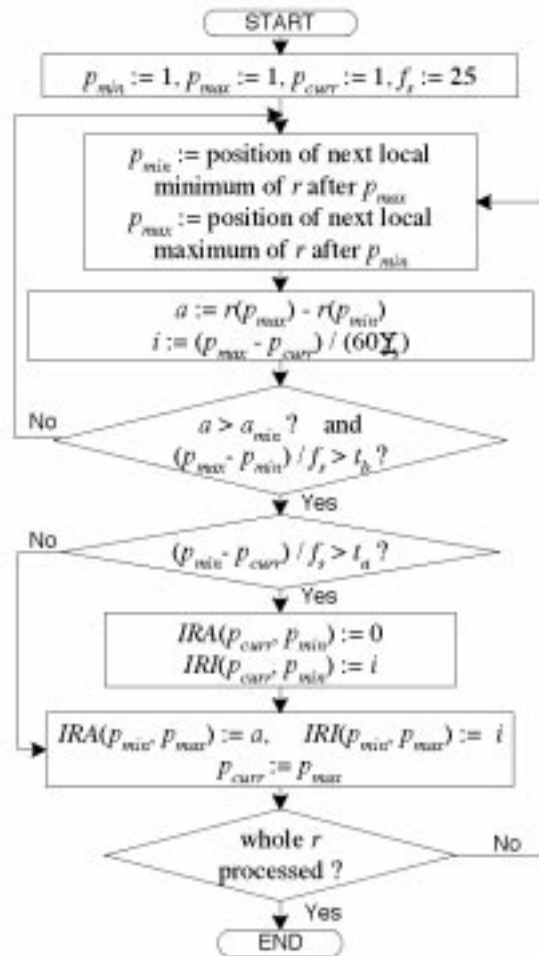


Fig. 4. Flowchart of the proposed IRA/IRI algorithm. For the meaning of the parameters, see the text.

The training vectors of the networks were derived according to the input signal structure outlined in Table I.

The network outputs were chosen as binary values, coding the respiration patterns corresponding to the actual input training pattern (N, H, A, and X—unknown). Both the hidden and the output units had sigmoid-type activation functions and each ANN was trained by the back-propagation algorithm with gradient descent and momentum [20]. The targeted mean squared error (MSE) was  $10^{-3}$  which was reached in the case of each ANN in less than 1000 epochs.

### III. RESULTS

For the test and evaluation of the networks' performance, continuous sections were selected from the 16 PSG records, each having a total length of 500 time windows (about 133 min). The networks N1 and N3 were tested with all 8000 patterns. In the case of N2 and N4, 4500 test patterns were used, since only nine records contained both the NAF and the RIP signal. The selected test record sections contained no training patterns.

Fig. 5 shows three 5-min-long excerpts of record SLP16. The classification regions are separated by vertical grid lines and the results produced by N3 are displayed in the top area of each classification region.

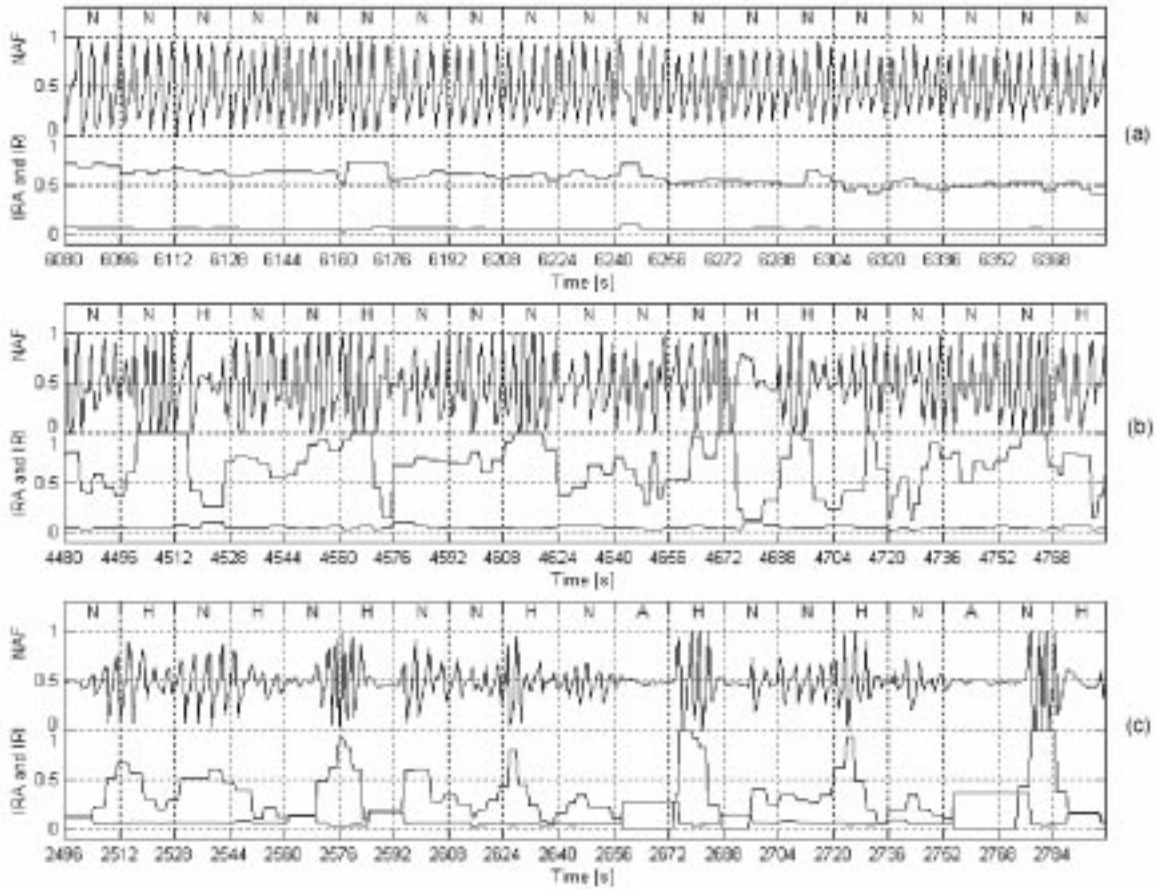


Fig. 5. Respiration patterns detected by N3 in three sections of record SLP16 containing (a) normal breathing, (b) episodes of hypopnea, and (c) episodes of apnea.

TABLE II  
CLASSIFICATION PERFORMANCE

Ref.	$N_N$	$N_H$	$N_A$	$n_N$	$n_H$	$n_A$	$\tilde{n}_N$	$\tilde{n}_H$	$\tilde{n}_A$	$P_N$	$P_H$	$P_A$	$S_N$	$S_H$	$S_A$
N1	5438	1471	1091	3290	887	710	1876	540	413	60.5	60.3	65.1	65.5	63.3	62.1
N2	3051	853	596	1898	552	406	1001	283	209	62.2	64.7	68.1	67.2	66.8	64.9
<b>N3</b>	<b>5438</b>	<b>1471</b>	<b>1091</b>	<b>5112</b>	<b>1339</b>	<b>968</b>	<b>87</b>	<b>313</b>	<b>33</b>	<b>94.0</b>	<b>91.0</b>	<b>88.7</b>	<b>98.4</b>	<b>78.7</b>	<b>97.0</b>
N4	3051	853	596	2871	767	530	55	170	26	94.1	89.9	88.9	98.2	80.1	95.6

The evaluation of the classification's performance requires untrained reference data consisting of patterns belonging to known classes. The applied PSG records originally included some signal annotations, however, they were entered only at the onset of an event (e.g. apnea, hypopnea, and arousal). The end of the events and the ranges with normal breathing were not annotated at all. Therefore, the performance of the networks was evaluated with the help of a physician practicing in the field of respiratory medicine. We constructed a program that displayed 20 consecutive time windows separated by grid lines, including the sleep stage and apnea annotations of the original records. The physician entered the class of the windows (N, H, or A) manually in each of the selected test sections as a consensus of the original annotations and his experience.

The evaluation took place by comparing the physician's results with the results produced by the ANNs. Table II contains the final test results where

$N_x$  number of patterns in category  $x$ ;

$n_x$  number of patterns classified correctly to category  $x$ ;

$\tilde{n}_x$  number of patterns and classified false to category.

The percentage  $P_x$  specificity and  $S_x$  sensitivity of the classification performance were defined as

$$P_x = \frac{n_x}{N_x} \cdot 100[\%] \text{ and}$$

$$S_x = \left(1 - \frac{\tilde{n}_x}{N_x}\right) \cdot 100[\%].$$

The best results were produced by the highlighted N3 network.

#### IV. DISCUSSION AND CONCLUSION

The presented neural network based apnea and hypopnea detection methods were based only on the processing of respira-

tion signals. At the start of the study we decided to incorporate not only respiration signals but also cardiovascular signals into the apnea detection system. Heart rate and blood pressure exhibit a well-defined behavior during sleep apnea: a slight decrease after the apnea's onset and rapid increment before its offset [2]. Unfortunately, these effects are superimposed on other cardiovascular phenomena (e.g., fluctuations due to the vascular baroreflex and autoregulation mechanisms, effects of respiration sinus arrhythmia).

Another problem is that the cardiovascular changes caused by apnea are delayed by many seconds and their exact characteristic is strongly dependent on the actual haemodynamic state of the patient. Although the SaO<sub>2</sub> signal carries the secondary evidence of apnea (desaturation episodes) [7], these changes are also typically delayed by ten or more seconds after the onset of the apnea. All these issues make the incorporation of the cardiovascular signals in an on-line apnea detection method rather problematic.

The original respiration signals used directly as network input produced poor classification results (see N1 and N2 in Table II). The cause of this unsatisfactory performance may lie in the nature of the respiration signals: even two consecutive breaths from the same patient can have different waveforms, amplitudes, and length. Additionally, the exact breathing waveform depends on the applied sensors and differ between patients of different gender and age. This results in a dispersed training set which makes the patterns for ANN training unsuitable.

The preprocessing of the original respiration signals allowed the production of signals which are more suitable for ANN training. The IRA and IRI signals calculated by the proposed algorithm can represent the whole breathing process in a way which is adequately characteristic for the detection of respiration dynamics patterns. The derived signals are robust in the face of sensor and patient-specific details of the original waveform. N3 used derived signals only from the NAF.

The use of an additional respiration signal (RIP) in the case of N4 did not result in a radical improvement of the classification performance. We explain this as a result of the strong correlation between the original NAF and RIP signals in many places. The use of the RIP signal is essential when the determination of the apnea type is also required. In the case of CA, no respiration movements occur, while during OA there is a respiration effort which tries to overcome the airway obstruction. As apnea typing was not within the scope of this study, the primary focus was concentrated on the analysis of the NAF signal.

We must underline that robustness is a key issue in the viability of an apnea detection system. We considered two pivotal points: robustness against noise and classification without any patient-specific information. Noise is handled in two ways in our system: The adaptive signal normalization eliminates baseline errors (e.g. wandering, sensor saturation, and signal losses). The resampling with low-pass antialiasing filtering and the minimum and maximum detection method of the applied IRA/IRI algorithm filters out unwanted signal spikes. The issue of avoiding the use of patient-specific information is handled by the proper derivation of the ANN input signals.

The overall classification performance achieved by N3 is a promising result. The proposed method for the preprocessing of

the respiration signals together with N3 can serve as a basis of an on-line respiration monitoring system. The moderate computational power required by the method allows for implementation in portable devices.

Although the presented system is suitable for robust classification of apnea and hypopnea events, a clinical validation study based on a larger signal database is still needed.

Our future research will focus on the incorporation of the heart rate and especially the SaO<sub>2</sub> signal which would result in a more robust detection system.

#### ACKNOWLEDGMENT

The authors would like to thank R. Horváth, an expert in respiratory medicine with the Sleep Laboratory of the National Kórányi Institute, Budapest, Hungary, for the help provided for this study.

#### REFERENCES

- [1] P. Köves, Ed., *Obstructive Sleep Apnea Syndrome*. Budapest, Hungary: Springer Verlag.
- [2] C. Guilleminault and M. Partinen, *Obstructive Sleep Apnea Syndrome, Clinical Diagnosis & Treatment*, New York: Raven, 1992.
- [3] J. Krieger, "Obstructive sleep apnea: Clinical manifestations and pathophysiology," in *Handbook of Sleep Disorders*. New York: Marcel Dekker, 1990, ch. 13.
- [4] S. Kyzer and I. Charuzi, "Obstructive sleep apnea in the obese," *World J. Surg.*, vol. 22, pp. 998–1001, 1998.
- [5] Standards of Practice Committee of the American Sleep Disorders Association, "Practice parameters for the indications for polysomnography and related procedures," *Sleep*, vol. 20, pp. 406–422, 1997.
- [6] R. Ferber, R. Millman, and M. Coppola *et al.*, "Portable recording in the assessment of obstructive sleep apnea," *Sleep*, vol. 17, pp. 378–392, 1994.
- [7] "The Chicago criteria for measurements, definitions, and severity of sleep related breathing disorders in adults," in Proc. Association of Professional Sleep Societies Conf., New Orleans, LA, June 20, 1998.
- [8] M. Kermit, A. J. Eide, T. Lindblad, and K. Waldemark, "Treatment of obstructive sleep apnea syndrome by monitoring patient airflow signals," *Pattern Recogn. Letters*, vol. 21, pp. 277–281, 2000.
- [9] B. A. Phillips, M. I. Anstead, and D. J. Gottlieb, "Monitoring sleep and breathing: Methodology. Part I: Monitoring breathing," *Clin. Chest Med.*, vol. 19, no. 1, pp. 203–212, 1998.
- [10] J. R. Stradling, "New approaches to monitoring sleep-related breathing disorders," *Sleep*, vol. 18, no. 3, pp. 77–84, 1996.
- [11] B. H. Taha *et al.*, "Automated detection and classification of sleep-disordered breathing from conventional polysomnography data," *Sleep*, vol. 20, no. 11, pp. 991–1001, 1997.
- [12] A. Bartolo, B. D. Clymer, J. A. Golish, and R. C. Burgess, "The polysomnogram assay: A method to represent the overnight polysomnogram in a condensed format," *Comput. Biomed. Res.*, vol. 33, no. 2, pp. 110–125, 2000.
- [13] R. K. Millard, "Inductive plethysmography components analysis and improved noninvasive postoperative apnoea monitoring," *Physiol. Meas.*, vol. 20, no. 2, pp. 175–186, 1999.
- [14] P. Várady, L. Szilágyi, and L. Nagy, "On-line detection of sleep apnea during critical care monitoring," in Proc. World Congr. 2000 Medicine, Physics, and Biomed. Eng., Chicago, 2000, PAPER TU-FXH-115.
- [15] S. A. Clark, C. R. Wilson, M. Satoh, D. Pegelow, and J. A. Dempsey, "Assessment of inspiratory flow limitation invasively and noninvasively during sleep," *AmER. J. Respir. Crit. Care Med.*, vol. 158, no. 3, pp. 713–722, 1998.
- [16] G. Goldberger *et al.*, "Physiobank, physiobook, and physionet: Components of a new research resource for complex physiologic signals," *Circulation*, vol. 101, no. 23, pp. 215–220, 2000.
- [17] L. A. Aguirre, V. C. Barros, and A. V. P. Souza, "Nonlinear multivariable modeling and analysis of sleep apnea time series," *Comput. Biol. Med.*, vol. 29, pp. 207–228, 1999.
- [18] J. Bock and D. A. Gough, "Toward prediction of physiological state signals in sleep apnea," *IEEE Trans. Biomed. Eng.*, vol. 45, pp. 1332–1341, NOV. 1998.

- [19] N. Stamatias, D. Parthimos, and T. M. Griffith, "Forecasting chaotic cardiovascular time series with an adaptive slope multilayer perceptron neural network," *IEEE Trans. Biomed. Eng.*, vol. 46, pp. 1441–1453, Dec. 1999.
- [20] H. Demuth and M. Beale, *Neural Network Toolbox for use With Matlab. User's Guide, Version 3.0.* Natick, MA: MathWorks Inc., 1998.



**Péter Várady** (S'98) received the M.Sc. degree in electrical engineering at the Technical University of Budapest, Budapest, Hungary, in 1997. He joined the Department of Control Engineering and Information Technology at the same university where he is working towards the Ph.D. degree in biomedical engineering.

His main research areas are biosignal processing and medical informatics. He has published more than 30 technical papers.



**Tamás Micsik** completed medical studies in 1996. He received the degree in biomedical engineering from the Technical University of Budapest, Budapest, Hungary, in 2000. He is working towards the Ph.D. degree at the university.

His main research topics are medical informatics, molecular biology, DNA-chips, genetics, and genomics.



**Sándor Benedek** received the M.Sc. degree in mechanical and in electrical engineering from the Technical University of Budapest, Budapest, Hungary, in 1965 and 1971, respectively. He received the D.Sc. degree at the Hungarian Academy of Sciences, Budapest, in 1994.

He is a Senior Researcher with the Institute for Electric Power Research, Budapest. His research interests are applied control theory, computer simulation, neural-network-based signal processing, and malfunction recognition. He has more than 100

technical publications.



**Zoltán Benyó** received the M.Sc. degree in electrical engineering from the Technical University of Budapest, Budapest, Hungary, in 1961. He received the Ph.D. and the D.Sc. degree from the Hungarian Academy of Sciences, Budapest, in 1975 and 1993, respectively.

He has been teaching at the university since 1961. In 1994, he became a full-time Professor with the Department of Control Engineering and Information Technology. His research and education areas are process control theory, especially the modeling and identification of physiological systems. He has published about 150 papers and authored or co-authored some 15 textbooks.