Derivation of Respiratory Signals from Multilead ECGs

Article *in* Computers in Cardiology · January 1985

CITATIONS
386

READS
4,662

4 authors, including:

Sara Mantero
Politecnico di Milano
79 PUBLICATIONS **4,940** CITATIONS

SEE PROFILE

Derivation of Respiratory Signals from Multi-lead ECGs

George B. Moody(*), Roger G. Mark(*), Andrea Zoccola(+), and Sara Mantero(+)

Massachusetts Institute of Technology, Cambridge, Mass., and Beth Israel Hospital, Boston, Mass., USA (*); Politecnico di Milano, Milan, Italy (+)

This article originally appeared in *Computers in Cardiology* (Washington, DC: IEEE Computer Society Press). Please cite this publication when referencing this material. A sample implementation of the algorithm described in this paper is available in source form here.

Summary

Knowledge of respiratory patterns would be clinically useful in many situations in which the ECG, but not respiration, is routinely monitored. We describe a signal-processing technique which derives respiratory waveforms from ordinary ECGs, permitting reliable detection of respiratory efforts. Central and mixed apnea, hypopnea, and tachypnea may be identified with confidence. In many cases, obstructive apnea and changes in tidal volume are also clearly visible in the ECG-derived respiratory signal (EDR). We compare examples of the EDR with conventional respiration measurements. In the context of multiple-lead arrhythmia detectors, the additional computation required for recovery of the EDR is insignificant. The technique is applicable to both real-time monitors and tape systems, requires no supplementary transducers or hardware modifications, and yields significant information of clinical value.

Relationships of cardiac and respiratory rhythms

The clinical significance of certain cardiac arrhythmias can be understood only with reference to respiration. The normal respiratory cycle is accompanied by changes in autonomic tone which modulate heart rate, causing sinus arrhythmia. Apnea may be associated with tachycardia, increased ventricular ectopy, or asystole. Stress, congestive heart failure, and chronic lung disease may result in both tachypnea and tachyarrhythmia. Thus, simultaneous observation of the ECG and the respiratory cycle over long periods is often clinically useful.

Respiration monitoring techniques

Methods of respiration monitoring fall into two categories. Devices such as spirometers and nasal thermocouples measure air flow into and out of the lungs directly. Respiration can also be monitored indirectly, by measuring body volume changes; transthoracic inductance and impedance plethysmographs, strain gauge measurement of thoracic circumference, pneumatic respiration transducers, and whole-body plethysmographs are

examples of indirect techniques. Each method has unique advantages and disadvantages. Direct measurements are the most accurate, but interfere with normal respiration. The whole-body plethysmograph can be highly accurate and does not interfere with respiration, but requires immobilizing the patient. Other techniques require frequent recalibration but the transducers (belts or electrodes) do not significantly burden a stationary patient; these are the only methods suitable for ambulatory respiration monitoring.

Measurement of mean axis direction

ECGs recorded from the surface of the chest are influenced by motion of the electrodes with respect to the heart, and by changes in the electrical impedance of the thoracic cavity. The expansion and contraction of the chest which accompanies respiration results in motion of chest electrodes. Short-term changes in thoracic impedance reflect the filling and emptying of the lungs, a phenomenon which is the basis of impedance plethysmography.

These physical influences of respiration result in amplitude variations in the observed ECG (figure 1). In terms of the equivalent dipole model of cardiac electrical activity, respiration induces an apparent modulation in the direction of the mean cardiac electrical axis. This phenomenon is independent of what is generally known as electrode motion artifact, an intermittent and generally aperiodic signal component which results from mechanical deformation of the electrode/skin interface.

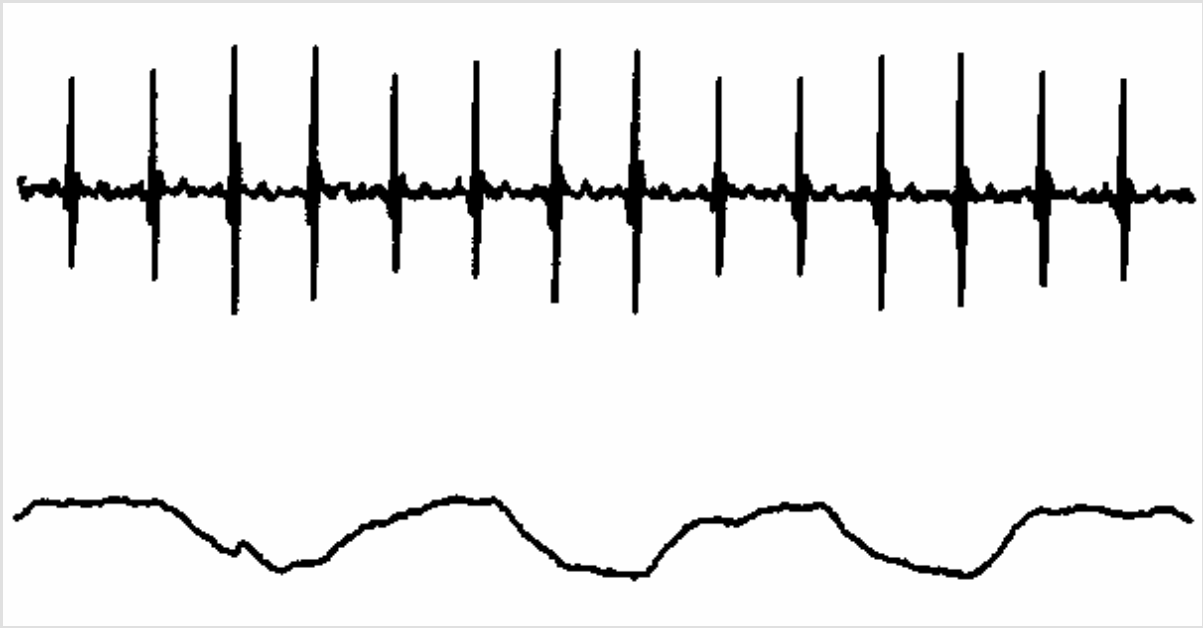

Figure 1. Respiration-induced modulation of QRS amplitude. Upper trace: ECG; lower trace: respiration measured by a pneumatic respiration transducer (PRT) placed around the chest. Duration: 10 seconds.

Several years ago, Pinciroli[1] began studying methods for determining the direction of the axis, in order to create ``virtual ECG leads'' which would represent what might be obtained from electrodes fixed in position relative to the heart. We expected that fluctuations in axis direction measurements would reflect the physical influences of respiration on the ECG, and began a study to determine if these measurements could be used to derive information about respiration.

ECG-derived respiratory signals

This relationship was confirmed by comparing axis direction measurements with simultaneously recorded measurements of chest circumference using a mercury strain gauge; later studies used pneumatic respiration transducer (PRT) measurements. Axis direction measurements based on chest electrodes correlate better with chest circumference and impedance than with abdomen measurements, or with direct measurements.

Although many techniques for measuring the direction of the axis work well, we found that shown in figure 2 both accurate and computationally simple. After subtracting the baseline, the area of each normal QRS complex in each of two leads is measured over a fixed window (the width of which is determined during the learning phase of the ECG analysis program to match the interval from the PQ junction to the J-point of a normal QRS). Area measurements such as these are made routinely by many arrhythmia detectors, either for direct use in feature-extraction approaches, or for normalization in template-matching methods. Since the window width is fixed, the area is proportional to the mean amplitude of the signal, hence to the projection of the mean cardiac electrical vector on the lead axis. Assuming that the leads are orthogonal, the arctangent of the ratio of the areas measured in the two leads gives the angle of the mean axis with respect to one of the lead axes. If the leads are not orthogonal, a systematic but harmless error in axis direction estimation results from this computationally convenient assumption.

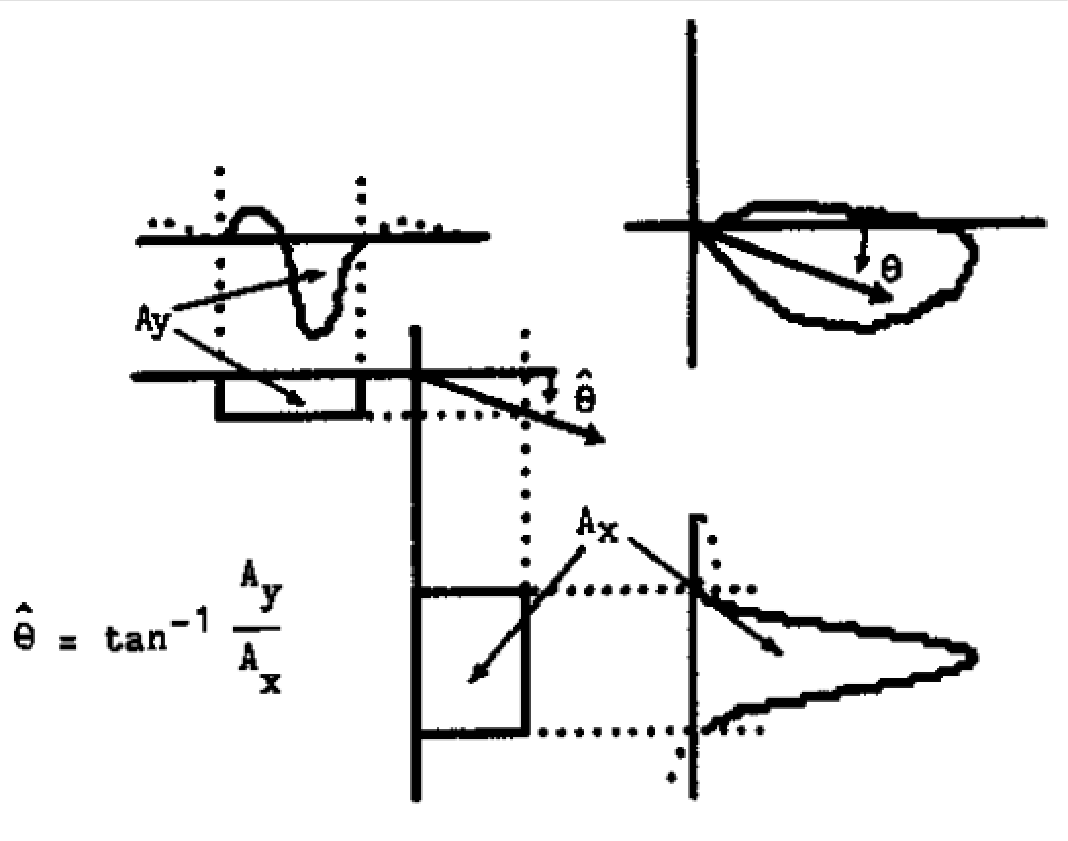

Figure 2. Estimation of the direction of the mean cardiac electrical axis from measurements of QRS area.

Axis direction measurement during the QRS complex provides one sample of the ECG-derived respiratory (EDR) signal per cardiac cycle. Given that the heart rate is almost always greater than twice the respiration rate, the frequency of respiratory effort can be measured well from this limited set of samples. Interpolation using cubic splines produces a continuous EDR signal (figure 3) which bears a remarkable resemblance to the signal obtained from a PRT chest measurement.

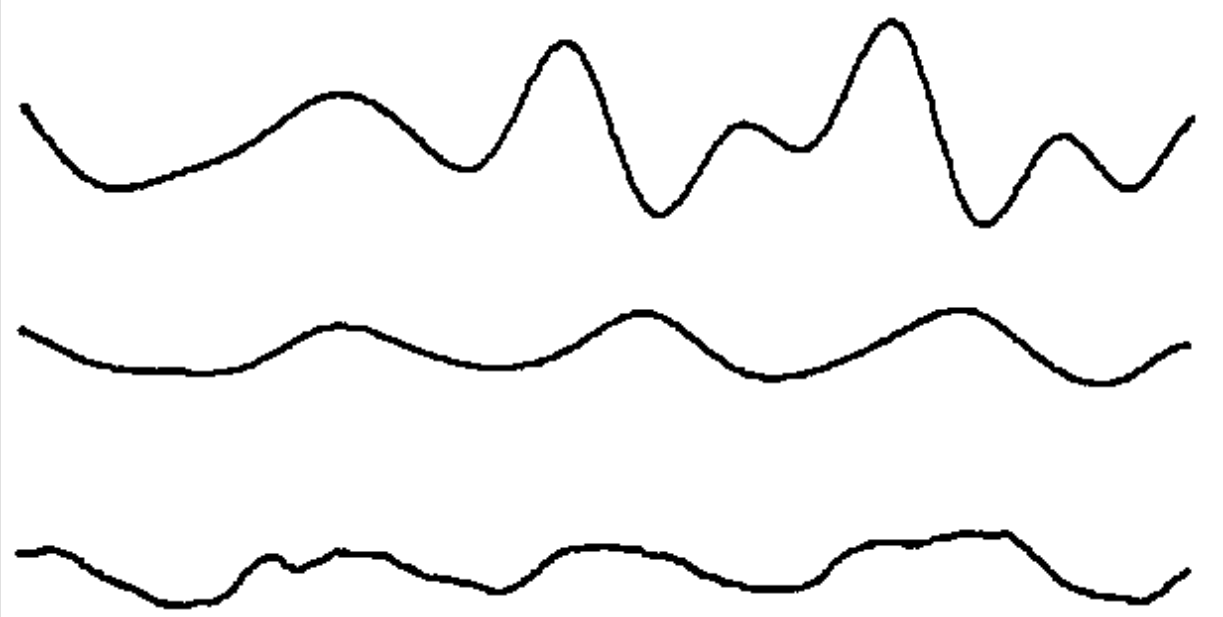

Figure 3. Normal respiration. Lower trace: chest PRT; center trace: mean cardiac electrical axis direction; upper trace: QRS area in V1. Cubic spline interpolation has been used between measured points in the center and upper traces. Duration: 10 seconds.

If small numbers of ectopic beats are present, they may be disregarded. When many similar ectopic beats occur, a better strategy is to make lead axis measurements for each morphology. Measurements for beats of different morphologies will generally differ by constant angles which can be determined from the differences of the means of the measurements for each morphology. Once these constant correction angles are known, measurements can be merged (figure 4). If ectopic beat morphologies are poorly differentiated (for example, if many fusion PVCs are present), the distributions of ectopic beat measurements will not match that for normal beats, and such measurements should be discarded rather than allowing them to corrupt the EDR.

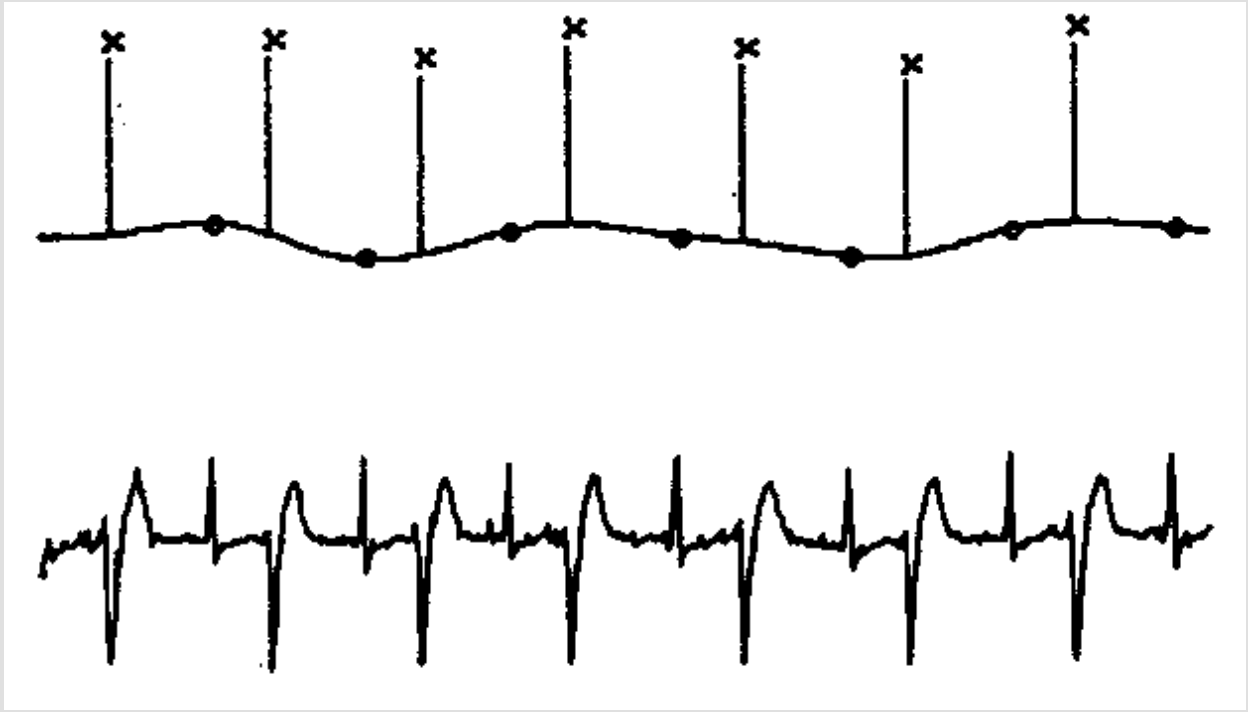

Figure 4. Figure 4. EDR (upper trace) derived from the ECG during ventricular bigeminy (lower trace). A constant correction angle is applied to the measurements obtained from the PVCs before merging them with data from normal beats. Duration: 10 seconds.

Additional samples of the EDR can be obtained by measuring the mean axis direction during the T-wave, which in general is not parallel to that observed during the QRS complex. T-wave axis measurements may be merged into a stream of QRS axis measurements by adding a correction angle as described above. Calculation of the correction angle should be made continuously in order to track ischemic changes, which can rotate the T-wave axis relative to the QRS axis.

If only one ECG lead is available, QRS area measurements from that lead can still be used in many cases as an approximation to the respiratory signal, The single-lead EDR works best if the lead axis is significantly different from the mean electrical axis, in order to obtain a relatively large signal. Since noise does not vary in proportion to the signal, the greatest signal-to-noise ratio is usually obtained when the lead axis is orthogonal to the mean electrical axis.

EDR signals may be analyzed to determine the frequency of respiratory effort by counting peaks which follow significant level changes. The normal range of respiration-induced axis shift is between 2 and 12 degrees, peak-to-peak. The amplitude of the EDR from any given set of electrodes is roughly proportional to respiratory tidal volume.

Evaluation

In order to evaluate the technique, and to explore its limitations, simultaneous recordings of EDR and respiration signals were made in the sleep laboratory at Beth Israel Hospital in Boston. Two roughly orthogonal ECG chest leads, and chest and abdomen PRT signals, were recorded for each of seven patients who were referred to the laboratory with diagnosed or suspected sleep apneas. Of this group, obstructive apneas were recorded in four patients, including one who also had mixed apneas; central apneas were observed in a fifth patient, hypopneas in a sixth, and the seventh patient had normal respiration. Twenty-nine hours of recordings were analyzed using a two-lead arrhythmia detector[2] which had been modified to calculate the EDR.

Figure 5 shows a central apnea, as seen from the chest PRT and from the EDR. The central apneas observed were easily identifiable in both signals.

Figure 5. Central apnea. Lower trace: chest PRT; upper trace: EDR. Duration: 30 seconds.

Figure 6 shows a rather remarkable event which begins with a 45-second obstructive apnea (the beginning is not shown). After nearly 35 seconds have passed, several sinus pauses occur, culminating in a 5.2 second asystole. Shortly after the heartbeat resumes, a central apnea begins, and persists for 15 seconds. The figure shows a tidal volume signal formed by combining the chest and abdomen PRT signals, and a single-lead EDR (the second ECG lead was not recorded during this event). The obstructive apnea cannot be unambiguously diagnosed from the single-lead EDR in this case, but the central apnea is quite clear.

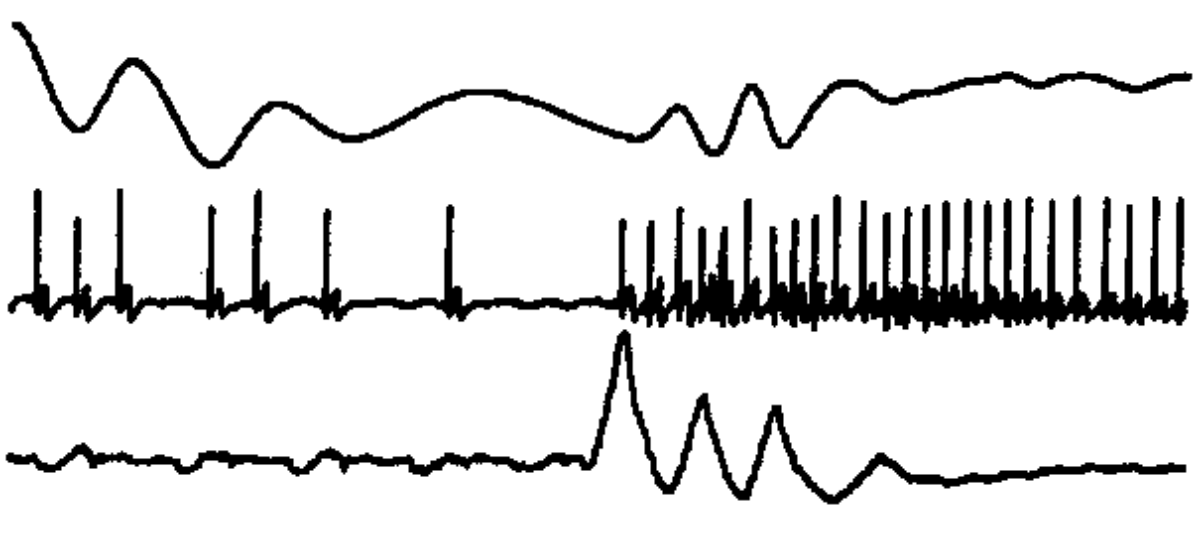

Figure 6. Mixed apnea. Lower trace: tidal volume (from chest and abdominal PRT signals); upper trace: single-lead EDR from ECG in center trace. Duration: 35 seconds.

The arrhythmia in this case is the result of a respiratory disturbance, and its clinical significance can be appreciated only if this is known. A cardiac pacemaker is not indicated on the basis of this asystole.

Reliable detection of obstructive apneas using indirect techniques requires both chest and abdominal measurements, since persistent respiratory efforts during obstructive apneas are often indistinguishable from normal respiration on the basis of a single signal. If both measurements are available, obstructive apneas are indicated by out-of-phase signals ("paradoxical respiration"), which cancel each other when added together if the instrument has been calibrated properly. Often the amplitude of each signal is substantially lowered during obstructive apnea, and this characteristic is shared by the EDR (figure 7). Although some episodes may not be detectable on the basis of the EDR, it appears unlikely that chronic obstructive apnea could go unnoticed using this technique.

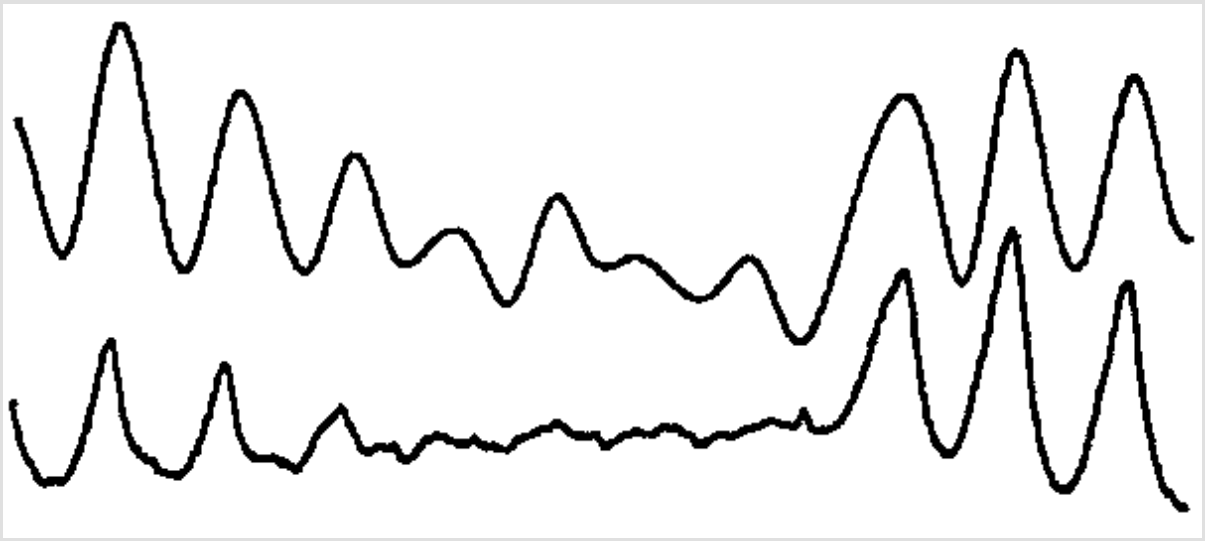

Figure 7. Obstructive apnea. Duration: 30 seconds.

Changes in respiration rate are easily detected (figure 8). Artifact can cause false peaks to appear in the EDR, however, and the best strategy for interpreting an apparent sudden increase in rate is to ignore it unless it persists for several cycles or reappears in the same context. Since the EDR is sparsely sampled, it night be thought to be quite noise-sensitive. In practice, although noise in the PRT signal is sometimes visible in the EDR (figure 9), in many cases the EDR is cleaner.

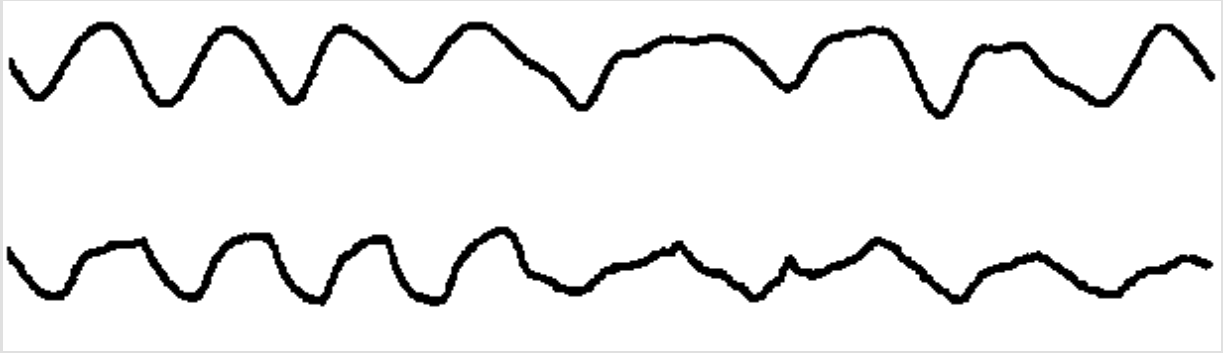

Figure 8. Hypopnea. Duration: 30 seconds.

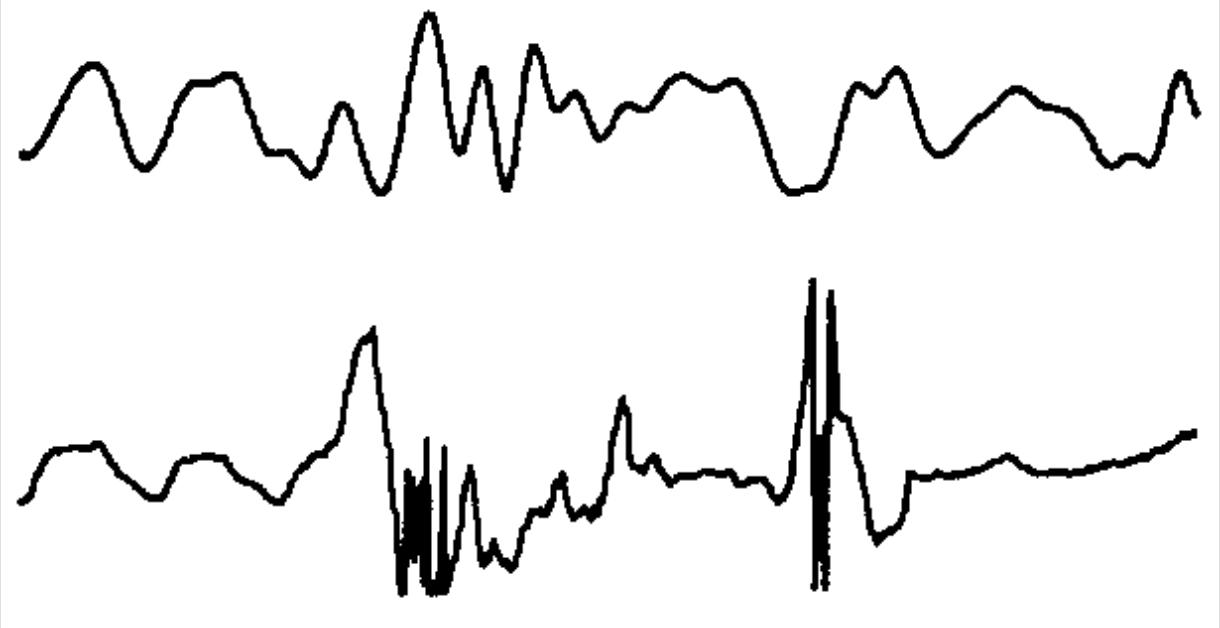

Figure 9. Noise in PRT (lower) and EDR (upper) signala. Duration: 30 seconds.

Examination of the MIT/BIH and AHA arrhythmia databases [3,4] showed that signals qualitatively similar to respiration waveforms could be recovered in all cases. Since no independent respiration measurements are available, the fidelity of the recovered signals cannot be evaluated.

The technique has also been applied successfully to recordings made on infants at risk of sudden infant death syndrome (SIDS). For these infants, long-term respiratory monitoring for detection of central apnea is of critical importance. Derivation of a respiratory signal from the ECG offers the possibility of reducing the number of transducers needed for monitoring SIDS infants.

Conclusions

Beat-by-beat measurements of mean cardiac electrical axis direction relative to lead axis direction have been shown to be strongly correlated with conventional measurements of respiration. The phenomenon has been observed in a wide variety of data. The frequency of respiratory efforts may be measured easily and accurately from the ECG-derived respiration (EDR) signal. Respiratory disturbances which are reflected in changes of respiration frequency are clearly observable in the EDR. Disturbances which are reflected only in tidal volume changes, such as obstructive apneas, may not always be detected using this method, although tidal volume changes are often visible in the EDR.

The technique is computationally simple, and applicable to any type of automated ECG analysis. It can be incorporated into real-time arrhythmia monitors as well as tape

scanning systems without the need for additional transducers or hardware redesign. Although the method works best when two orthogonal ECG leads are available, it is possible to generate an EDR from only one lead. With an insignificant amount of additional computation, any automated system for ECG analysis can use this technique to produce significant, previously unavailable information of clinical value.

Acknowledgments

The authors wish to thank M. Mograss and N. Logowitz of the Beth Israel Hospital Sleep Laboratory for their assistance, advice, and encouragement; and F. Pinciroli of the Politecnico di Milano, whose idea for improving ECG signal quality provided the impetus for the present study.

References

1. Pellegrini, A., Pinciroli, F., Rossi, R., and Vergani, L. Virtual leads for ambulatory ECG monitoring: Remarks on the persistency of their direction. *Computers in Biomedical Research*, 18:62-78. 1985.
2. Moody, G.B., and Mark, R.G. Development and evaluation of a two-lead EKG analysis program. *Computers in Cardiology*, 9:39-44. Long Beach, California: IEEE Computer Society. 1982.
3. Mark, R., Schluter, P., Moody, G., Devlin, P., and Chernoff, D. An annotated database for evaluating arrhythmia detectors. In *Frontiers of Engineering in Health Care* pp. 205-210. Proc. 4th Annual Conf. IEEE Engineering in Medicine and Biology Society. 1982.
4. Hermes, R. and Oliver, G. Use of the American Heart Association database. In *Ambulatory Electrocardiographic Recording*, pp. 165-181. Eds. Wenger, N., Mock, M., and Ringqvist, R. Yearbook Med. Pub. 1980.