Significance of Respiratory Artifact in the Electrocardiogram

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Electrocardiographic artifact is generally considered to be a nuisance. Respiratory artifact, however, is a physiologic signal that may carry useful diagnostic information. Our goal was to evaluate the characteristics, prevalence, and clinical significance of respiratory artifact observed in electrocardiograms (ECGs). ECGs exhibiting repetitive microoscillations were systematically collected. The morphologic characteristics of the microoscillations were analyzed and their association with the respiratory cycle was evaluated using simultaneous respiratory waveform tracings. The presence and rate of respiratory artifact were correlated with the patient’s clinical status, including medical diagnoses and the need for ventilatory support. During a 30-month period, respiratory artifact was detected in 320 12-lead ECGs. It was best seen in leads II, III, aVF, and V5. Respiratory artifact occurred during the inspiratory phase and its rate correlated precisely with objective measures of the respiratory rate. The majority of patients with respiratory artifact revealed diseases of the respiratory (26.6%) and circulatory systems (24.0%) and, of note, respiratory artifact was never detected in patients with normal cardiorespiratory function; 43.5% of patients with respiratory artifact required ventilatory support, including 28.2% requiring continuous mechanical ventilation. Successful treatment of the underlying condition resulted in a decrease in the rate or in complete disappearance of the respiratory artifact. In conclusion, respiratory artifact is commonly seen in routine 12-lead ECGs of hospitalized patients. It is associated with a high-risk state of increased work of breathing due to either compromised cardiac or pulmonary function. Additionally, the presence of respiratory artifact enables precise evaluation of the respiratory rate—a commonly miscalculated vital sign. © 2008 Elsevier Inc. All rights reserved. (Am J Cardiol 2008;102:1090–1096)

Respiratory rate (RR) is a sensitive and reasonably specific indicator of acute respiratory function.1,2 Several studies have demonstrated the inaccuracy of RR measurement by physicians and nurses.3–5 Throughout the course of routine interpretations of electrocardiograms (ECGs), the presence of distinct repetitive high-frequency, low-amplitude artifact was observed. Additionally it was noted that this artifact was present in cases when patients presented with respiratory distress.6 The purpose of this study was to analyze a large number of consecutive ECGs that exhibited the cyclic appearance of respiratory artifact and to correlate its presence and rate to patient diagnoses and clinical conditions.

Methods

During the routine interpretation of 12-lead ECGs over a 30-month period at a large tertiary care hospital, consecutive ECGs that displayed repetitive microoscillations were prospectively collected. The population was a compilation of adult medicine, surgery, neurosurgery, obstetrics, gynecology, and trauma patients from regular and intensive care units. Emergency department patients were not included. The number of cases exhibiting respiratory artifact was correlated with the total number of ECG interpretations during the same time period. The study was approved by Carolinas Medical Center’s Institutional Review Board.

All 12-lead ECGs were recorded by a GE-Marquette system (General Electric, Milwaukee, Wisconsin). The ECGs were electronically stored. Standard amplifications, filter settings, and paper speed were used. To study the relation of respiratory artifact to the respiratory cycle, telemetry recordings exhibiting respiratory artifact were obtained from 25 patients in the intensive care unit who also had simultaneous mechanical respiratory waveform tracings. In 10 of these patients, 12-lead ECGs recorded the same day also demonstrated the presence of respiratory artifact.

The ECGs and 4× enlarged tracings were analyzed for the frequency content, amplitude, and duration of the respiratory artifact. The rate of respiratory artifact was approximated by counting the number of microoscillations in a 10-second recording and multiplying it by 6. When the RR is regular, a more accurate rate assessment is achieved by measuring ≥1 respiratory artifact cycles in millimeters and dividing it into 1,500 or its multiples.

The following clinical data were collected from patients’ medical records and by query of our CareScience database: patient demographics, major clinical diagnoses, clinical assessment at the time of recording of the index ECG, chest x-ray, and chest computed tomography findings, arterial oxygen saturations or arterial blood gas assessments, percentage of patients who required mechanical ventilation, percentage of patients requiring bilevel positive airway pressure (BiPAP) or continuous positive airway pressure (CPAP), and clinical interventions associated with changes...
Figure 1. Respiratory artifact in a 44-year-old man with quadriplegia and respiratory distress. (A) Spontaneous breathing during an episode of asystole (continuous recording). ECG monitor lead II and a simultaneous respiratory waveform tracing are shown. When it could be measured, there was a 440 to 460 ms time delay from the onset of the respiratory artifact to the upslope of the mechanical wave. The insert is a 4× enlargement of a respiratory artifact. Note a 60 ms by 0.2 mV initial deflection followed by 580 ms of microoscillations with a frequency content of about 40 cycles per second. (B) Spontaneous breathing with progressive tachypnea. (C) After intubation and mechanical ventilation, the respiratory artifact was no longer present.

Figure 2. Respiratory artifact indicating increased work of breathing and tachypnea in a 72-year-old man with crush injury to the chest. The patient was mechanically ventilated but agitated and fighting the respirator. Repetitive microoscillations are best seen in V4 to V6. The heart rate (HR) was 78 per minute, the RR was 32 per minute. The ECG was otherwise normal.
in the presence and rate of respiratory artifact. Simple descriptive statistics were used.

**Results**

During a 30-month period respiratory artifact was detected in 320 12-lead ECGs obtained from 248 patients. The number of ECGs that displayed distinct respiratory artifact constituted about 1% of the approximately 32,000 ECGs analyzed during the same time period.

In ECGs that displayed respiratory artifact, it was best seen in leads II (100%), aVF (97%), III (95%), V5 (63%), and V4 (52%). Leads I, V1, and V2 displayed the respiratory artifact in ≤10% of the study ECGs. In 25 randomly selected cases the amplitude, duration, and frequency content of the microoscillations were measured from 4× enlarged ECG recordings. The peak amplitudes ranged from 0.1 to 0.35 mV (mean ± SD, 1.80 ± 0.68). The duration of microoscillations measured 430 to 1,620 ms (mean, 865 ± 276). The frequency content ranged from 26 to 53 per second (mean, 40.3 ± 6.6). In a few cases a single 40 to 60 ms sharp upward deflection immediately preceded the high-frequency microoscillations.

On 25 occasions where simultaneous respiratory waveform monitoring was performed, each respiratory artifact was associated with a mechanical respiratory wave. It corresponded to the inspiratory cycle but there was a 40 to 400 ms time delay between the onset of the respiratory artifact and the onset of the inspiratory waveform in the mechanical
tracing. Figure 1 is a representative illustration of simultaneous ECG and respiratory waveform tracings in a telemetry recording. This particular patient had periods of asystole, which exposed several of the listed morphologic characteristics of the respiratory artifact.

Patient ages ranged from 23 to 92 years; 58% were men. Major diagnostic categories of patients with respiratory artifact included diseases of the respiratory system (26.6%) and circulatory system (24.0%). Within these categories asthma exacerbation and acute decompensated heart failure, respectively, were the most frequent findings. Other common diagnoses included infection, sepsis, trauma, adult respiratory distress syndrome, acute leukemia, diabetic ketoacidosis, renal failure, delirium tremens, postoperative state, and bleeding.

The RR calculated from the respiratory artifact ranged from 9 to 54 per minute (mean, 27.3 ± 8.6). In 81% of cases the RR was >20 per minute, which is usually considered to be the upper limit of normal for the RR. Even in those patients with a slow or normal RR, however, the presence of respiratory artifact signified cardiopulmonary compromise such as hypercarbic hypoventilation, large pleural effusion, pericardial mass, botulism, narcotic overdose, and severe emphysema. In 15 acutely or critically ill patients the 12-lead ECG was completely normal except for the presence of respiratory artifact.

When evaluating all ECGs with respiratory artifact, every patient was noted to have an abnormality in ≥1 of the following objective measures: chest x-ray, chest computed tomography, arterial oxygen saturation (≥91%), or arterial blood gas (pCO2 >60 mm Hg or pO2 <59 mm Hg). More importantly, 43.5% of patients with respiratory artifact required a medical intervention such as mechanical ventilation (28.2%) or noninvasive ventilation support of CPAP or BiPAP. Figures 2 to 4 are representative examples of respiratory artifact in patients with increased work of breathing and tachypnea associated with a variety of clinical conditions.

On many occasions treatment of the underlying process resulted in a substantial decrease in the rate or in complete elimination of respiratory artifact. In 24 cases the respiratory artifact was immediately abolished when the patient was sedated, intubated, and when mechanical ventilation was started (Figures 1 and 5). Reemergence of spontaneous
respirations while on the ventilator was associated with reemergence of respiratory artifact (Figure 2). In 13 cases other successful therapeutic interventions resulted either in abolition of the respiratory artifact (Figure 6) or in a substantial decrease in the rate of the respiratory artifact (Figure 7).

Discussion

Our study demonstrates that repetitive microoscillations are commonly found in 12-lead ECGs of hospitalized patients and, when noted, they are often associated with significant cardiopulmonary disease. At our institution approximately 1 in every 100 ECG shows this phenomenon. These microoscillations represent inspiratory artifact. This finding indicates significant cardiopulmonary decline and a high-risk state; almost half of the patients with respiratory artifact needed some form of respiratory support, including 28% who required intubation and mechanical ventilation. Of striking consequence was that successful therapeutic interventions often resulted in a slower rate or complete elimination of the respiratory artifact.

There have been past attempts at deducing respiratory waveforms from the surface ECG using computerized techniques, but these did not lead to clinically useful results. Repetitive microoscillations similar to those described in this study have been previously noted. They were originally considered to represent atrial activity associated with intra-atrial block or atrial dissociation because solitary deflections preceding the microvibrations, similar to those shown in Figure 1 of the study, were believed to signify peaked P waves. Subsequent reports suggested that the apparent P waves actually represented artifact on the ECG produced by the accessory muscles of respiration. Respiratory artifact was considered to be an extremely rare phenomenon; in 1 study it was recognized in a single ECG of 18,000 recorded over a 3-year period.

Our study has proven that the repetitive microoscillations are respiratory artifacts associated with the inspiratory phase of respiration, but it did not provide a definitive clue to its source. The respiratory artifact is almost certainly related to increased muscle tension rather than the actual motion of the thoracic cage because its high-frequency components are inconsistent with passive motion artifacts,
the onset of respiratory artifact preceded the onset of the inspiratory waveform recorded in simultaneous mechanical tracings, and the respiratory artifact always disappeared when sedation and mechanical ventilation were started.

The respiratory artifact probably represents some form of surface electromyogram, which noninvasively measures muscle activity using surface electrodes placed on the skin overlying the muscle. The sharp upward deflections occasionally seen at the onset of the microoscillations may represent the tonic initiation of inspiratory effort. Normal respiratory muscle activity can only be recorded with amplification. Increased muscle tension and muscle force, however, may result in a marked increase in the electrical activity of the inspiratory muscles and thus in the amplitude of the surface electromyogram. It is conceivable that the amplitude of the respiratory artifact is affected by body habitus, subcutaneous emphysema, edema/anasarca, hemodialysis, or how tightly the leads are affixed, but these factors were not systematically analyzed.

There are limitations to this study. We did not review the ECGs of all patients with acute cardiorespiratory insufficiency and therefore do not have an indication about the sensitivity of this sign. The exact cause of the respiratory artifact and why it is best seen in the inferolateral leads remain uncertain. Future studies will have to establish if certain characteristics of respiratory artifact might help distinguish a primary cardiac versus pulmonary cause of labored breathing, and whether recognition of respiratory artifact on the ECG would change patient management and outcome.

14. Pullman SL, Goodin DS, Marquinez AI, Tabbal S, Rubin M. Clinical utility of surface EMG: report of the Therapeutic and Technology


