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Respiratory rate and pulmonary embolus

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Sir,

Dr Jolobe highlights the importance of the accurate measurement of respiratory rate in the detection of pulmonary embolus (PE) [1]. He cites the paper by Kline et al that reported normalization of respiratory rate after presentation did not reduce the probability of pulmonary emboli. Kline et al postulated that suspicion of PE is critical to its diagnosis: a clinician will not use a decision rule, a D-dimer, or any other diagnostic test for PE unless he or she believes a PE may have occurred, a suspicion which is often triggered by vital sign changes observed when the patient is first seen [2]. However, there are other signs that may raise the possibility of PE, such as diaphoresis, hypoxia, gasping, and accessory muscle use [3]. In my personal experience these signs of distress are often transient: within a few minutes patient can recover and seem perfectly well, only to died a short while later from a recurrent PE. Suffice it to say the clinical presentation of PE is notoriously non-specific [4] and it remains a frequent cause of death: 1% of all patients admitted to hospital die of acute PE and 10% of all in-hospital deaths are PE-related [5-7]. However, not all PE patients have adverse outcomes and clinical prediction rules, such as the Pulmonary Embolism Severity Index (PESI) and/or laboratory biomarkers, can identify low risk cases that can be safely treated as outpatients [8].

Increased respiratory rate has frequently been reported to occur in PE: in the Urokinase Pulmonary Embolism trial only 8% of patients with PE had a respiratory rate less than 16 breaths/min [9] and tachypnoea was the commonest sign observed in the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) [10]. Yet in this same study 30% of patients with PE had a respiratory rate less than 20 breaths per minute. Moreover in many studies the increase in respiratory rate noted is modest [2], PESI only includes an exceptionally high rate of 30 breaths per minute as a prognostic marker [11], while the abbreviated PESI does not include respiratory rate at all [12]. There are two possible explanations for these contradictory findings: one is that the increased respiratory associated with PE is either transient and/or remains within the normal range, the other is that increased respiratory rates are missed because they are not measured accurately.

Sudden pulmonary vascular obstruction should result in a sudden drop in the partial pressure of oxygen, which could be compensated for by either taking deeper breaths
and/or breathing more rapidly. For any level of alveolar ventilation there is an optimum respiratory rate at which the work of breathing is at a minimum, and any increase or decrease in rate will result a larger amounts of work. The work of breathing depends on lung compliance and airway resistance [13]. If there is high compliance then slower deeper breathing will generate less work, whereas if compliance is lowered faster breathing will be more efficient. Reduction of blood-flow through part of the lung causes its ventilation to become “dead-space”, which may be compensated for by changes in local airway calibre which reduces ventilation in areas of decreased blood-flow. These changes may result in a sudden reduction of lung compliance and increased airway resistance, which will force the patient to rapidly seek a new optimal rate of breathing [14]. This new rate may not necessarily be outside the normal range. The process may be somewhat similar to a cyclist trying to find the right gear to climb a hill: for a short while he or she might be peddling faster or slower than usual, but eventually the right gear will be found that will provide the optimal rate of peddling, which may be only slightly faster to the optimal rate for cycling on the flat. Although this is an attractive hypothesis that certainly fits in with my clinical experience, it can only be confirmed by large trials that continuously monitor respiratory rate before, during and after a pulmonary embolus. It may be sometime, if ever, before these trials can be performed.

The alternative explanation is that the tachypnoea associated with PE is caused by reflex mechanisms unrelated to hypoxaemia [15,16] and is not detected because respiratory rates are not measured accurately. This is highly likely as in many studies respiratory rate is counted by visual observation over 20 to 30 seconds [2]: if respiratory rate is counted over 30 seconds one third of patients with a respiratory rate over 20 breaths per minute will be missed, and two thirds missed if their rate was counted over 15 seconds [17]. In many cases this is the most likely explanation why patients with PE appear to have respiratory rates within the normal range.

Respiratory rate has become a well established valuable predictor of adverse outcomes based on studies that probably never measured it accurately [18]. How much more valuable might it prove to be if it the technology to measure it accurately and continuously were available? Continuous measurement might show that pulmonary emboli can be detected from sudden fluctuations in respiratory rate, or
other conditions may be suggested by changes in the depth of breathing as well as its rhythm and periodicity. In the mean time it must be recognise that measuring respiratory rate accurately over 60 seconds is of great clinical importance, and cannot be done without taking time and trouble.
References


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