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The last sigh and the death knell:

A view on lung hyperinflation from a millennium ago.

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Main letter text

Just before death, it is rather common that a moribund person takes a really deep breath that could scientifically be called a 'sigh', a tidal volume that is in fact close to two and a half times the normal. This very high tidal volume causes an important heart-lung interaction often culminating in a massive shift of the interventricular septum, leading to a reduction in the cardiac output thus heralding the death knell.

Here we would quote a couplet from a famous Persian poet and mathematician who lived about a millennium ago.

"And now by all the words the preacher saith,

I know that time, for me, is but a breath,

And all of living but a passing sigh,

A little wind that stirs the calm of death."

Hakim Omar Khayyam (1048-1131 CE)(1)

The above couplet elaborately and exquisitely sheds light on the underlying pathophysiological mechanism by which very large tidal volumes, when used in patients who have a low cardiac output and a hypovolemic functional status might cause severe hemodynamic impairment, and this is what the last sigh in fact may do. To translate it into our daily practice, it could be extrapolated that large tidal volumes (e.g. recruitment maneuvers at high airway pressure) should be employed with particular caution in patients with acute respiratory distress syndrome (ARDS) when their cardiac output is in a tenuous state and on its lower edge.

The physiological "*sigh*" (2-3 x tidal volume) normally occurs at variable frequencies (1-25 breaths /hour) during the course of spontaneous breathing (2). Physiologically, it could be said that the "*sigh*" breath is in fact initiated by the rapidly adapting stretch receptors employing an inspiration-augmenting reflex from the vagal afferents (3, 4). Again, in the same vein, Bell et al. (5) could

notice the return of augmented breath when an animal was subjected to hypoxic mixture in their experiments (6).

Hyperinflation of the lungs, as we all know, causes almost invariably a decrease in cardiac output by different combinations, between other mechanisms, of decreased right ventricular function, increased pulmonary resistance, ventricular interaction and increased left ventricular afterload, in variable proportions, depending on the patient's physiological status and on whether the ventilation is spontaneous or mechanically supported. Moreover, larger tidal volumes (15ml/kg), decrease the heart rate by a combination of both increased vagal tone (6), and abolition of sympathetic influence. The sympathetic withdrawal also potentiates arterial vasodilatation (7). To conclude, we may say that death is in fact made calmer and easier by the last "*sigh*" which brings in an overwhelming change in the heart-lung interaction, which the Persian Poet appreciated almost a millennium back and which we still consider when managing our patients.

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