Dynamic hyperinflation—the anaesthetist applying a tourniquet to the right heart

“The leprous distilment; whose effect holds such an enmity with blood of man that, steeti as quicksilver, it courses through the natural gates and alleys of the body,...”

Hamlet, Act 1 Scene 5

Even though the play antedates the publication of De Motu Cordis by nearly 30 years, the quotation was used as evidence that Shakespeare was familiar with William Harvey’s concept of circulation of blood.1

There has been a tendency for clinicians to underrate the importance of the right heart2 to flow “through the natural gates and alleys”. Transoesophageal echocardiography is opening a window under operative conditions and through it can be seen, in a way previously not possible, the changes of preload and afterload on atrial, ventricular, tricuspid valve and outflow tract function.2 Functionally, being in a Russian doll’s nest of anatomic and pressure layers, the right heart is weakened by being juxtaposed between the muscle-bound left ventricle and the lungs, and is sensitive and vulnerable to disease of both these organs and the pericardium. Anaesthetic insults primarily act on the myocardium and pulmonary vasculature but, by taking control of the airway with a tracheal tube, an anaesthetist breaches the integrity of, and imposes forces, that markedly influence the climate of the intrapleural layer. Additional, supranormal pressures of positive pressure ventilation (IPPV) may be enough to override the protective cushioning of the pericardium so that the right heart is compromised; sometimes, particularly in those with a predisposing pathophysiology, this effect is critical.

Ultimately, it is intra-alveolar pressure within the intrapleural layer that dictates forward flow of blood through the lungs. It is pathologically raised in several well known states encountered by anaesthetists. If expiratory airflow obstruction occurs then intra-alveolar pressure may remain positive throughout much of the respiration cycle. This auto or intrinsic PEEP (PEEPi) effect,3 is typically part of acute and chronic obstructive airways disease, notably asthma and emphysema, and of adult respiratory distress syndrome.4 Recently introduced treatment options for respiratory failure like lung transplantation,5 minimal access thoracic surgery5 and lung volume reduction have swelling the ranks of the predisposed and susceptible. Large lungs, too, can be dysfunctional such that PEEPi is generated: classically, for example, in the presence of inhaled foreign bodies, and central airways compression.8

To the list of primed can be added diverse causes of accidental expiratory retardation engendered by the processes of anaesthesia, such as obstructed ventilator tubing; faulty anaesthetic circuits; wet humidifying filters; narrow tracheal tubes, notably double lumen tubes9; and inappropriate ventilation techniques.1011 The latter can lead to barotrauma and the peculiar manifestation of pulmonary hypercompliance in a relatively non-compliant chest cavity, known as dynamic hyperinflation (DHI).

The imposition of IPPV may produce the increased pressure dynamic that results in a tamponading constrictive force being applied to the right heart; a “tourniquet effect” as gas is trapped by increments and successive breaths become, what is appropriately described as “stacked” with a net positive balance of gas. Cardiac sensitivity to the additive pressure effects of IPPV may result from the fluid dynamic changes of hypovolaemia, vasodilatation, superior vena cava obstruction and pleural or pericardial effusions.12

Of the three current defined variants of PEEPi—dynamic, occlusion and static—the latter are measurable easily in mechanically ventilated patients in intensive care units.1415 But from the operating table-side, the anaesthetist is most likely to be forewarned of the development of a compromising level by having a high index of suspicion and using right heart signs as surrogate barometry.217 A raised external jugular venous pressure and a fall in systolic arterial pressure are a common experience for most anaesthetists when anaesthesia is induced in chronic bronchitic patients and, though these usually resolve, such signs alert us to the risk of compounding PEEPi with deliberate or accidental PEEP. Arterial wave forms in the invasively monitored patient,18 by reflecting pulsus paradoxus, can also be a warning sign, as is a combination of oxygen desaturation and a fall in arterial pressure, as sensed by some forms of pulse oximeter.19 A deliberate manoeuvre of disconnecting the ventilator may be the ultimate test. A commensurate improvement in oxygen saturation and arterial pressure is virtually diagnostic of DHI being part of the pathophysiology; it should signal the use of a suitable ventilatory pattern.20

The best ways of reducing the constricting effects of DHI are by treating the various components logically. In so doing, the development of DHI should be preempted in some of the recognizably predisposed. Induction of anaesthesia with agents with venous constrictive activity, such as etomidate or ketamine (Feneck, personal communication), may be rational. Right heart support has received little coverage but the judicious use of colloid volume to optimize venous return, the use of inotropes to adjust venous tone and myocardial contractility, and agents that influence the pulmonary circulation all have a place theoretically.20 Influencing the airways should be considered in some cases. If there is evidence of reversibility or reactivity, small airways dysfunction may respond to bronchodilators or corticosteroids. In cases with central airways obstruction, which cannot physically be removed, consideration may need to be given to the use of tracheal or bronchial stents. The insertion of drainage systems will help in the event of emphysematous bullae expanding17 or the cases in which pleural or pericardial effusions are present.

But it is ventilation stratagems that are the mechanisms by which anaesthetists can counter PEEPi.
These should hinge on low-peak inflation pressures, prolonged expiratory intervals, and minimizing mean intrathoracic pressure. The deliberate addition of PEEP is a vexed question. As a general rule, PEEP is counter-productive, but in patients with collapsing small airways the internal splinting effect to maintain patency throughout the respiratory cycle may allow a reduction in the volume of trapped air. This process, part of optimization of PEEP, is usually not an option in the acute situation and more applicable to longer term management of acute lung injury in intensive care units. But optimal PEEP may counter some of the effects of the use of double lumen tubes in which a small unilateral lumen may contribute to the development of air trapping in patients undergoing surgery for respiratory failure.

Is DHI a problem that should concern anaesthetists working outside environments commonly dealing with the predisposed? There are scattered reports of unexpected cardiac arrests and fatalities in the literature. The common theme of suddenness and the suspected presence of conditions associated with a increased PEEPi are strong evidence that DHI is a part of the story, and that it is a phenomenon that should be more widely recognized by both those administering anaesthetics and those involved in resuscitation. Standard guidelines for resuscitation recommend manoeuvres that would be counter-productive in the event of gas trapping that is sufficient to impede right heart function.

Some personal knowledge of two unexpected deaths, in which embolic phenomena were suspected as causative because right heart dysfunction was an agonal event, but in which the working diagnoses could not be substantiated with angiography or at postmortem examination, would fit a hypothesis that DHI is the guise of a “leperous distilment” that can kill without leaving a pathological imprint. More chronic forms of air trapping lead to changes that are detectable histologically, but the cessation of ventilation as the preterminal event in an acute situation would allow lung deflation so that the crucial prima facie evidence, that of hyperinflation, would disappear. In the absence of any other explanation, it is appropriate to advise that if DHI is at all possible, then the disconnecting of the ventilator or stopping ventilation, albeit at an apparently critical moment during such a highly charged scenario as a cardiac arrest, may be life-saving. Such a manoeuvre would facilitate venous return and improve cardiac output. As an aide mémoire, adjust the old adage of presumed oesophageal intubation (“if in doubt, take it out”) to “if in doubt, let it (the trapped gas) out”; or, alternatively, “save a life, allow to expire!”

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References