Twenty-four hours later there was further deterioration in his condition with dynamic hyperinflation as a consequence of excessive end-expiratory ventilation. After consultation with the family, it was agreed that supportive measures only would be continued. These included biphasic positive airway pressure ventilation with \( P_{aw,1} \) via nasotracheal tube, giving blood gas analysis of \( pH \) 7.30, \( P_{aw,1} \), 57 mmHg, \( P_{aw,2} \), 68 mmHg, base excess \(-3.8 \) m. Twenty-four hours later there was further deterioration in his condition, and he died without active resuscitation being performed.

The sequence of events strongly suggested a respiratory cause for both arrests. Although it may be difficult to rule out tension pneumothorax clinically, there is considerable risk in arbitrary placement of an intercostal needle or drain in a patient with severe bullous chronic obstructive pulmonary disease. A subsequent radiograph showed no pneumothorax. A more plausible explanation is the development of dynamic hyperinflation as a consequence of excessive end-expiratory pressure in the respiratory system, or auto-positive end-expiratory pressure; this may lead to significant impedance to venous return and reduction in cardiac output. Although modern ventilators can be programmed to provide optimum cycle lengths, inspiratory-expiratory time ratios, variable inspiratory flow patterns, and external positive end-expiratory pressure to counter auto-positive end-expiratory pressure, such refinements are not available during manual ventilation in the heat of CPR. It is suggested that in this patient pressure in the respiratory system was so high that it shut down venous return. When CPR was discontinued and the endotracheal tube was removed, over the ensuing 5 min there was a gradual return of pressure in the respiratory system to atmospheric pressure, which allowed the heart to fill once more, leading to a return of spontaneous function.

References

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