well described by Pepe and Marin2 and is an important cause of hypotension in mechanically ventilated patients. The lungs are unable to exhale to FRC at the start of the next mechanically initiated inspiration because of airway obstruction. Positive end-expiratory intrathoracic pressure builds up, although this cannot be shown on the ventilator manometer. The patient described by Shennib et al could have had this cause for hypotension, rather than mediastinal emphysema. Restoration of blood pressure after sternotomy and recurrence of hypotension on closing the chest supports this view.

Auto-PEEP can be assessed by occluding the expiratory line at the end of the set exhalation period and delaying the next ventilator-delivered breath.4 Pressure in the tubing will equilibrate with intrathoracic pressure, and PEEP will be shown on the ventilator manometer. If this pressure is high, tidal volume or ventilatory rate needs to be decreased. Hypercapnia may result from this reduction of minute volume, but it is better to keep the patient hyperventilated and alive1 than risk the consequences of high Auto-PEEP.

This should always be considered before proceeding to surgical decompression of any pneumomediastinum or laying the chest cavity open.

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To the Editor:

Dr. Tse’s comment on the role of auto-PEEP in the development of hypotension in ventilated, airway-obstructed patients is well taken. Auto-PEEP can result in electromechanical dissociation by both increasing intrathoracic pressure and by disruption of the alveoli. The latter can further compound the situation by resulting in pneumothorax, pneumomediastinum and systemic air embolism (including coronary air embolism). When changing tidal volumes and ventilatory rates fail to reverse the deleterious status of these patients, as in our case, surgical decompression (including sternotomy) can buy time and may be life-saving.

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The Newest Quinolone Antibacterial Agents and Theophylline

To the Editor:

We have described the effect of new quinolone antibacterial agents on the serum concentration of theophylline. In this issue, we report the interaction of theophylline and quinolone antibacterial agents newly developed or under development in Japan.

Quinolone antibacterial agents tested were NY-198 (Hokuriku Seiyaku CO, Ltd), T-3262 (Toyama Chemical Co, Ltd) and AM-833 (Ro23-6240, Kyorin Pharmaceutical Co, Ltd). Chemical structures of these antibacterial agent, five healthy male volunteers received a sustained-release preparation of theophylline orally (200 mg bid for four days), followed by oral antibacterial agents for five days. The doses of NY-198, T-3262 and AM-833 were 200 mg tid, 150 mg tid and 200 mg bid, respectively. Serum theophylline levels were monitored at three and five days after the start of concomitant administration of antibacterial agents.

NY-198 and AM-833 showed no interaction effect. T-3262 showed a significant increase in serum theophylline level with a 1.23 times increase in Cmax and a 1.24 times increase in AUC, but no adverse reaction due to this increased theophylline level was noted. This effect is identical with that of ciprofloxacin and pefloxacin, and care should be taken when the theophylline is concomitantly administered with T-3262.

Furthermore, it is considered that NY-198 and AM-833, likeloxacin, may be used together with theophylline.

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FIGURE 1. Chemical structure of the newest quinolone antibacterial agents.