the potential false reassurance of a normal coagulation test and emphasizes the need for careful history taking. As outlined in our editorial, there are several congenital bleeding disorders, including mild VWD, which may present with a completely normal coagulation screen and therefore may be missed unless a very careful history and clinical assessment is undertaken. As there actually was a clear personal and family history of bleeding in this case that would have led to coagulation testing if the patient had been aware of it, one has to ask if routine testing to identify an extremely small number of cases whereby the patient is unaware of the history is warranted, given the large number of tests needed. It also highlights the importance of how questions are asked, as members of families unaware of the symptoms of mild bleeding disorders may consider pronged bleeding after minor trauma or procedures such as cuts or dental extraction as ‘normal’. Specific questioning about duration of bleeding after minor trauma or procedures can clarify this. By and large, both the BCSH guidelines2 and NICE guidance3 reflect the uncertainties about establishing a bleeding tendency but also agree that a perioperative coagulation screen in most instances should not be performed routinely. Although one could make a better case for routine testing in patients awaiting surgery with a high bleeding risk, the arguments regarding poor sensitivity and specificity of these tests remain. In conclusion, we do not claim that history taking will entirely solve the problem of preoperative coagulation status but that it is more rational than relying on routine coagulation testing in unselected patients.

Conflict of interest
None declared.

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New insights into the pathophysiology of aspiration pneumonia
Editor—We report a case of pulmonary aspiration of gastric contents during induction of anaesthesia in a monkey, which may contribute to a better understanding of the respective roles of acidity and particulate materials in the pathophysiology of this syndrome.

During an experimental study, a Cynomolgus monkey (4.2 yr old, 7.1 kg), treated in accordance with the Guide for the Care and Use of Laboratory Animals, was adequately fasted and anaesthetized with ketamine i.v. and inhalation of isoflurane. The animal suddenly vomited during laryngoscopy, before tracheal intubation. The vomit was removed from the oral cavity, and the trachea was then intubated and ventilated with a Servo™ 900D Siemens (tidal volume: 70 ml, ventilatory frequency: 30 bpm, end-expiratory pressure: 5 cm H₂O, oxygen inspiratory fraction: 100%). There was symmetric expiratory wheeze on auscultation which was not improved by isoflurane inhalation. The animal was cyanosed and oxygen saturation was 80%. Inspiratory airway pressure increased. The tidal volume was adjusted to keep the inspiratory plateau pressure between 30 and 35 cm H₂O. Mechanical ventilation was maintained for 2 h with no improvement and the animal was eventually euthanized. The lungs were removed and prepared for histological examination. Macroscopic examination revealed an inflammatory infiltrate of neutrophils, with an extensive recruitment of inflammatory cells around food particles (Fig. 1). Necropsy also revealed a large duodenal tumour, which was probably responsible for the increased gastric pressure and failure to empty gastric content, despite adequate starvation.

Aspiration of gastric contents is a major risk factor for acute lung injury, but to date this has been studied essentially in animal models. Data on the immediate lung pathology in humans are not available and most come from studies of the lungs after late open-lung biopsy. Most of the research on the effects of gastric content inhalation has focused on the effect of acidity rather than that of the particulate components of the aspirate, and only rodents have been used as models.1 Hydrochloric acid (HCl) has been widely used in both HCl-treated mice and rats to induce a diffuse inflammatory infiltrate.2 3 The observations in this report allowed us to study lung damage in accidental conditions mimicking Mendelson’s syndrome in humans. Indeed, it provided an opportunity to observe the effects of gastric content as a whole (acidity and particulate matter) shortly after the incident in a primate whose anatomy is more relevant to humans than that of rodents. In the lung tissue section, a marked accumulation of inflammatory cells around food particles was observed. This suggests that particulate food material is probably not only responsible for airway obstruction but may also contribute directly to inflammatory damage. A synergistic role of acid and food particles in the pathogenesis of acute lung injury induced by gastric aspiration has already been suggested in rodent models.4 5 Aspiration of a combination of acid and small gastric particles in mice led to increased albumin concentrations and inflammatory mediators (tumour necrosis factor-α, interleukin-6) in bronchoalveolar lavage, in contrast to the injuries caused by either dilute HCl or small non-acidified gastric particles alone.5 However, the exact mechanism involved remains
unclear. Inert glass particles of the same size and concentra-
tion as gastric particles do not produce similar pulmonary injury. One hypothesis is that bile salts or gastrointestinal secretions may be absorbed with food particles and result in slowing the neutralization of acid in lung fluids that are buffered by proteins and the bicarbonate system. Our observation of the early phase of aspiration pneumonia in a monkey sheds new light on the pathophysiology of lung lesions in this syndrome.

Conflict of interest

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Fig 1. Light microscopy of lung tissue section, 2 h after inhalation of gastric contents showing particulate food material with patchy inflammatory infiltrates and alveolar oedema (stained with haematoxylin and eosin, magnification ×40).