Difficulty of establishing preoperative coagulation status

Editor—We read with interest the editorial proposing that the routine measurement of a patient’s coagulation status should be abandoned in the absence of a family or personal history, an acute illness, or anticoagulation therapy. The authors are correct that questioning the patient about clotting abnormalities and bleeding disorders should form an essential part of the pre-anaesthetic medical assessment. However, it is possible that patients themselves may be unaware that there is an issue to report. Some years previously, a young man presented for major cancer surgery, having previously undergone a staging laparoscopy, followed by chemotherapy. He was then seen by a consultant at a preoperative anaesthetic clinic, after a surgical assessment. As normal practice at the time, a routine coagulation sample had been analysed along with the other routine investigations. The activated partial thromboplastin time (APTT) was found to be prolonged, at 1.29 (normal maximum 1.18), although platelet count and international normalized ratio were normal. Initially, this was thought to be a spurious finding, but examination of past records revealed that after the laparoscopy, and before the chemotherapy, a postoperative set of blood tests had included a clotting screen, which showed the APT to be 1.33. By chance, the patient’s parents had also attended the preoperative anaesthetic appointment to support him. Initially, full questioning revealed no evidence of any personal or family history of clotting abnormality. However, the evidence of the prolonged APPT prompted a detailed inter familial discussion. It was then revealed that as a child, the patient had suffered excessive bleeding after a minor procedure, and he then admitted to bleeding freely after minor trauma, such as shaving cuts. In addition, it was also revealed that the patient’s female siblings had received blood transfusions after surgery of moderate severity and that a maternal sibling had also experienced bleeding difficulties. Soon, the classic autosomal dominant pattern of a previously unrecognized von Willebrand’s clotting abnormality was revealed. Discussion with the haematologist led to the operation being postponed for a short time so that surgery could proceed with adequate haematological cover. The diagnosis of von Willebrand’s was confirmed, and the operation was successful, and uneventful. Although in many cases the performance of coagulation tests may be redundant due to the variation in normal findings, and the rarity of finding a meaningful abnormality, reliance on a carefully elicited history as a screening tool to prompt investigation of a possible disorder may be unreliable. The correct use of routine preoperative clotting tests situation is compounded by the non-specificity of NICE guidance in some situations. As the above story reveals, even with appropriate questioning, no history may be forthcoming in the first instance as the patient may be unaware that they have a problem, or unaware of disease patterns within the extended family. This is not just the case with von Willebrand’s, but also with other clotting disorders such as Factor XI deficiency, and Factor VII disorders. In this case, it was only the presence of the abnormal blood test and the fortuitous attendance of parents that prompted the family reflection that revealed the pattern of disease. Although von Willebrand’s disease is a common coagulation disorder in humans, the family were unaware until that point they had a genetically associated coagulation problem, despite a series of incidents. In this case, preoperative coagulation testing prevented a potential disaster. Although anaesthetists should ensure that they do their best to ask patients specifically about bleeding disorders, using this as a screening tool to determine whether preoperative clotting tests is unreliable. The promotion of greater awareness of blood disorders in the general public may be the most effective answer.

Conflict of interest

None declared.

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Reply from the authors

Editor—We thank Drs Watts and Joseph for their letter regarding preoperative coagulation status in response to our Editorial. They describe the case of a young man requiring major surgery who was unaware of a personal and family history of bleeding but which was discovered after a prolonged activated partial thromboplastin time (APTT) that triggered detailed history taking from family members, leading to a diagnosis of von Willebrand disease (VWD). The authors suggest that a ‘potential disaster’ was avoided by routine testing and that reliance on a bleeding history as a screening tool may be unreliable. We note that the patient had undergone staging laparoscopy uneventfully prior to any APTTs being performed, suggesting that the bleeding disorder here was mild. Although we agree that there is currently no method for fully establishing the bleeding risk in the perioperative period, their letter also highlights
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 prolonged 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 guidelines and NICE guidance 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.

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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. Microscopic 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