surface required for assembly of the platelet-bound coagulation enzymes, and the production of enough thrombin to cause substantial fibrin formation. Platelet-derived micro-particles such as P-selectin, which are formed during the process provide a mechanistic route for amplifying thrombus formation on a thrombogenic surface [4,7].

Leukocyte activation (CD11b upregulation) also occurs within minutes leading to adhesion while tissue factor expression occurs over hours. Complement activation occurs at all these time scales [3].

Researchers should be aware that the choice of the storage tube can influence TEG® variables. The advice is that reference intervals for TEG® variables be established, and this is likely to be specific for the nature of the tube used. The values for *r* and MA obtained during this study fell within the references ranges for our own analyser for citrated samples.

Our results suggest that glass is a more potent surface for activation of the coagulation cascade than plastic. This is reflected in lower *r* values and greater MA values for glass tubes when compared to plastic tubes.

In conclusion, both glass and plastic tubes show no significant effect between 45 min and 4 h. Glass tubes are associated with shorter *r* values and higher MA when compared with plastic tubes.

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Fatal aluminium phosphide poisoning

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EDITOR:
Aluminium phosphide is used to control rodents and pests in grain-storage facilities. When aluminium phosphide comes into contact with water, it releases large quantities of phosphine (PH_3), a very toxic gas and a mitochondrial poison. We present a case report of suspected inhalation exposure to phosphine gas in a manufacturing facility for aluminium phosphide fumigants.

Case report

A 45-yr-old male was admitted to the hospital with a history of severe diarrhoea, nausea and vomiting for 3 days. He was treated with intravenous saline without improvement and his clinical picture deteriorated with the appearance of dyspnoea. His symptoms had started soon after he had used an insecticide (Fosguard which contains phosphine) to clean a small and non-

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air-conditioned room. The patient was not on any medication, had no history of alcohol abuse and had an unremarkable medical record. On admission, the patient was comatose (Glasgow Coma Scale 6/15) with increased respiratory rate ($42\,min^{-1}$), increased heart rate ($80\,min^{-1}$) and decreased mean arterial pressure (30 mmHg). He was transferred to the intensive care unit (ICU) where a chest X-ray showed extensive bilateral alveolar-interstitial infiltrate compatible with acute respiratory distress syndrome. On blood gas analysis, the PaO_2/F_iO_2 ratio was 140, pH 6.98 and base excess −15. He had severe metabolic acidosis, liver dysfunction, renal insufficiency and disseminated intravascular coagulation with leucocytes $2500\,mm^{-3}$, platelets $43\,000\,mm^{-3}$, alanine aminotransferase $2934\,IU\,L^{-1}$, aspartate aminotransferase $2740\,IU\,L^{-1}$, urea $425\,mg\,dL^{-1}$, creatinine $6.5\,mg\,dL^{-1}$, potassium $6.8\,mmol\,L^{-1}$, amylase $487\,IU\,mL^{-1}$ and International normalized ratio 2.29. Respiratory support, veno-venous haemofiltration, inotropic support and bicarbonate infusion were immediately started, but the patient eventually died 10 h later.

Discussion

Phosphine is mainly used as a fumigant in pest control. Human exposure to phosphide fumigants includes both pesticide applicators and individuals in the vicinity of application to be at risk of accidental exposure and injury from phosphine inhalation. Studies on isolated rat liver showed that mitochondrial oxygen uptake is inhibited by phosphine owing to its reaction with cytochrome *c* and cytochrome *c* oxidase. Although the exact mechanism of action of phosphine in man is not known, non-competitive inhibition of mitochondrial cytochrome oxidase in mouse liver, housefly and granary weevil is mentioned by some authors [1].

The initial clinical manifestations of mild phosphide inhalation may mimic upper respiratory-tract infection including cough, nausea, vomiting, diarrhoea, headache, fatigue and dizziness. In severe exposure, lung irritation with persistent coughing, ataxia, paraesthesia, tremor, diplopia, hypotension, weak pulse and jaundice may also occur. Severe metabolic acidosis, cardiovascular collapse, oliguria, proteinuria and finally anuria may occur, which may require haemodialysis. Death, which may be sudden, usually occurs within 4 days but may be delayed for 1–2 weeks. Postmortem examinations have revealed focal myocardial infiltration and necrosis, pulmonary oedema and widespread small vessel injury. The diagnosis of phosphine poisoning is easy. A silver nitrate-impregnated paper test can be used to test the breath and gastric fluid of the patients exposed to phosphine/phosphide: silver nitrate and phosphine/phosphides react to form silver phosphide, which confirms the diagnosis. Other laboratory investigations including full blood count, haemoglobin, haematocrit, arterial blood gas analyses, renal and liver function tests and cardiopulmonary monitoring and investigations are essential for the assessment of organ effects [1–4]. Failure in diagnosis and delayed therapy have contributed to mortality. Singh and colleagues [5] demonstrated that non-survivors had more severe hypotension and metabolic acidosis than survivors who had more severe vomiting at aluminium phosphide poisoning.

As initial signs and symptoms of intoxication from phosphine gas may be non-specific and transient, there is a need for improved recognition of the potential hazards associated with phosphide fumigants and phosphine gas. The signs and symptoms appear rapidly and become established within hours. The initially severe clinical situation improves fast. Cases of this rare mode of intoxication should be carefully followed and treated in the ICU.

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