

# Effects of storing blood in citrated silicone-coated glass tubes vs. citrated plastic tubes on thromboelastograph variables

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**EDITOR:**

We have read with interest the article by Willschke and colleagues [1] and a similar article by Camenzind and colleagues [2]. This study was conducted to challenge the finding that Thromboelastography (TEG<sup>®</sup>) values deteriorate with storage in silicone-coated glass tubes but not in plastic polypropylene tubes [1]. Willschke and colleagues demonstrated that TEG<sup>®</sup> blood coagulation variables in recalcified citrated blood deteriorated in silicone tubes as opposed to plastic tubes. The basis for this claim was a two-sample analysis, which demonstrated a change. Before we began a larger study of platelet function we wished to know whether there was a need to standardize the time in glass storage tubes prior to analysis. We sought to overcome this by using three-sample analysis for both glass and plastic tubes.

A comparative *in vitro* observational study was undertaken using blood from ten healthy volunteers. All were ASA physical status I, and aged between 18 and 60 yr. Any with coagulation disorders, liver or renal disease, oral or systemic anticoagulation and the concurrent use of non-steroidal anti-inflammatory drugs were excluded.

A 22-G cannula was inserted into a forearm free-flowing vein and the first 3 mL of blood taken was discarded. A further 25 mL of blood was taken and divided into three glass tubes (Greiner, Germany), each holding 4.5 mL of blood, and three plastic tubes (Greiner, Germany), each holding 3 mL of blood.

All tubes contained 0.105 M sodium citrate in a ratio of volume to whole blood 1:9. The tubes were labelled 1, 2 and 4 h to correspond to when they were to be analysed and were left at room temperature for a minimum of 1 h after filling, to stabilize [2].

All processing was conducted using our Thromboelastograph 5000<sup>®</sup> analyser (Haemoscope, IL, USA), maintained on a regular service contract

and subject to regular quality-control procedure. Samples were managed in accordance with the Haemoscope<sup>®</sup> manual at 37°C.

Two variables were compared, reaction time (*r* value) and maximum amplitude (MA) at 1, 2 and 4 h. Results were analysed using analysis of variance.

The sample size of the study was based on a clinically important difference of 5 mm for MA using a standard deviation (SD) of 4.1 [2] to achieve a power of 0.85 with significance level 0.05. This estimated that 20–25 samples would be required. There were 30 samples for each group in our study.

No significant trend was observed over 1–4 hours in both glass and plastic tubes for both *r* and MA (Table 1). There was however a significant difference between glass and plastic tubes for all *r* values (*P* value = 0.0017) and for all MA values (*P* value = 0.017).

The marked differences in *r* value and MA in glass tubes compared to plastic tubes is likely to be associated with activation of the coagulation cascade. Blood–material interactions trigger a complex series of events including protein adsorption, platelet and leukocyte activation/adhesion, and the activation of complement and coagulation; they are highly interlinked [3]. Each component is associated with a different time scale [3]. Protein adsorption and Factor XII activation occurs within seconds of blood–material contact, producing low levels of thrombin.

Platelet adhesion, activation and aggregation [4–6] occur within minutes [3], creating the phospholipid

**Table 1.** MA and *r* values (mm) over 1, 2 and 4 h expressed as mean (SD).

Time (h)	Glass ( <i>n</i> =10)		Plastic ( <i>n</i> =10)	
	MA	<i>r</i> value	MA	<i>r</i> value
1	51.4 (7.1)	17.9 (5.9)	45.7 (9.48)	66.6 (25.6)
2	51.4 (7.69)	19.2 (7.16)	44.6 (7.27)	62.1 (21.2)
4	51.9 (7.29)	19.8 (7.3)	42.9 (15.8)	68.5 (22.3)

No significant trend was observed over 1–4 h in both glass and plastic tubes for both *r* and MA. There was however a significant difference between glass and plastic tubes for all *r* values (*P* value = 0.0017) and for all MA values (*P* value = 0.017). MA is a measure of dynamic strength of the fibrin clot. *r* value is the time latency from the time the blood was placed in the TEG<sup>®</sup> analyser until initial fibrin.

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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<sup>®</sup> variables. The advice is that reference intervals for TEG<sup>®</sup> 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 reference 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<sub>3</sub>), 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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