APPARENT VISCOSITY OF ARTIFICIAL RED-WHITE AND WHITE THROMBOM DURING
ANESTHESIA AND SURGERY. L. R. Mcintyre, and B. Asafraci. Medical Research,
Kemansau Memorial Institute, Sydney Hospital, Sydney 2000, and Department
of Medicine, University of Sydney, Sydney 2006, Australia, and Department
of Medicine, Hadassah University Medical School and Hospital, Mount
Snezak, Jerusalem, Israel.

This is a pilot study intended to explore some aspects of thrombus
formation which might be of value in understanding the dynamics of anesthe-
sia, on the one hand, and the effects of anesthesia and surgery on tissue
perfusion, on the other. Patients studied included six subjects undergoing
operations for retinal detachment (1), upper abdominal surgery (1), and a
major orthopedic procedure (1). Artificial thrombi of morphology of red/
white and white arterial thrombi, were formed in vitro by means of VFPV,
variable frequency thrombo viscometer, at temperature 37°C, on freshly shed
blood, at mean shear rates of 36.8 and 80 sec⁻¹. Blood samples were drawn
immediately prior to commencement of anesthesia, and then at half-hourly
intervals. Anesthesia was induced with chloropentone sodium, and halothane
or meperidine and droperidol. In general, the apparent viscosity of artifi-
cial thrombi increased during surgery.

CLOTTING STUDIES PERFORMED ON BLOOD STORED IN HALF-STRENGTH ACD-A. J. H.
Mishler and J.R. Barley, Department of Haematology, Radcliffe Infirmary, Oxford,
England.

Six hundred ml of whole blood from each of five healthy male donors was equally divided and
stored at 4°C in either standard ACD-A (2.4g trisodium citrate, 0.8g citric acid, 2.45g dex-
trose/4l anticoagulant solution, pH 4.7) or half-strength ACD-A (1.1g trisodium citrate, 0.8g
citric acid, 2.45g dextrose, pH 4.3) to determine if low citrate concentrations adversely effec-
ted the following: prothrombin time (PT), thrombin time (TT), Kaolin- cephalin clotting time
(KCCT), ethanol gel (E) and fibrinogen levels. Low citrate concentrations had no significant
effect (Student's t-test for paired scores) on any clotting indices tested (see table below).

<table>
<thead>
<tr>
<th>ACD-A Strength</th>
<th>PT (sec)</th>
<th>TT (sec)</th>
<th>KCCT (sec)</th>
<th>E (g%)</th>
<th>Fibrinogen (mg/dl)</th>
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<tr>
<td>Standard</td>
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<td></td>
<td></td>
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<tr>
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<td>7d</td>
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<td>9.6</td>
<td>53.8</td>
<td>negative</td>
<td>224.6</td>
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<td>10.8</td>
<td>52.4</td>
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<td>21.4</td>
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<td>50.6</td>
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<td>204.8</td>
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<tr>
<td>Half</td>
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<td></td>
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<tr>
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<td>202.4</td>
</tr>
</tbody>
</table>

ABNORMAL PLATELET ULTRASTRUCTURE IN FULMINANT HEPATIC FAILURE. Bullock, G. *,
Weston, R. F., Rubin, R. H. *, Roberts, J. *, Langley, P. G. *, White, V. * and
Williams, R. *.

Platelets obtained from eight patients with varying degrees of liver damage
have been studied with respect to their ultrastructure. These platelets were
isolated from platelet-rich plasma which had been utilised in the pharma-
cological studies described by Dr. Weston (previous abstract) and were
compared with control platelets isolated from five normal subjects. The latter
were chosen for normal bleeding time and response of their platelets to
aggregation with ADP and collagen.

Marked differences were seen between control platelets and those from the
test group in that there was an alteration in the microtubular content and
disposition. In addition, these changes were partially reversed during the
recovery period suggesting that production of new normal platelets was taking
place. This is one of the few conditions where platelet structure has been
related with a clinical disorder.