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The Relation between the Platelet-activated Clotting Test (HemoSTATUS) and Blood Loss after Cardiopulmonary Bypass

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Background: **Platelet dysfunction is one of several major causes of bleeding after cardiopulmonary bypass. A timely, simple, point-of-care determinant of platelet function recently became available for clinical use. Adding platelet-activating factor to conventional activated clotting time methods (platelet-activated clotting test [PACT]) produces rapid results (<15 min) and may yield a measure of platelet responsiveness and whole-blood procoagulant activity.**

Methods: **Blood samples were drawn from 100 patients after cardiac surgery on their arrival in the intensive care unit for PACT, platelet count, prothrombin time (PT), and activated partial thromboplastin time (aPTT). Cumulative blood loss at 4, 8, and 12 h after arrival in the intensive care unit and perioperative transfusion requirements were quantitated. Coagulation tests and mediastinal blood loss were compared using the Spearman rank test and Pearson correlation. The sensitivity and specificity of the laboratory tests for predicting blood loss were analyzed using the receiver operating characteristic method.**

Results: **The PT was the only test that correlated with blood loss at 4, 8, and 12 h. The PACT did not correlate with blood loss at 4, 8, or 12 h, nor did the PACT correlate with the PT or the aPTT. The sensitivity and specificity of the PACT were less than those of the PT in predicting blood loss. Only the PT correlated with platelet and fresh frozen plasma transfusion.**

Conclusions: **The PT correlated with blood loss and transfusion requirements and was superior to PACT, aPTT, and platelet count for predicting excessive blood loss after cardiopulmonary bypass. (Key words: Cardiac surgery; coagulation; laboratory analysis; monitoring; platelet function.)**

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PATIENTS undergoing cardiac surgery that uses cardiopulmonary bypass (CPB) have some degree of acquired platelet dysfunction as a result of dilutional, activating, consumptive, and destructive actions.[1–4] Severe platelet dysfunction may impair hemostasis and require transfusion of blood products.[5] The absence of a clinical test of platelet function limits rational transfusion practices.

No specific, reliable, technically easy, or rapid point-of-care test of platelet function exists.[6,7] The Thromboelastogram Maximal Amplitude (TEG-MA, Haemoscope, Inc., Skokie, IL) is commonly used to assess platelet function, but it requires >30 min for results to be produced.[8–10] Although it is not useful in all cases, nor has it been correlated with benchmark tests of platelet function, a TEG-MA result <50 mm has been shown to identify patients at risk for excessive blood loss who may respond to desmopressin therapy.[11]

Traditionally, activated clotting time (ACT) has been used to assess anticoagulation during CPB. The ability to predict platelet dysfunction during or after CPB would greatly benefit clinicians in the decision to transfuse allogeneic platelets. A newly developed assay, the platelet-activated clotting test (PACT; HemoSTATUS, Medtronic, Parker, CO), may be a useful test of platelet responsiveness or whole-blood procoagulant activity in patients undergoing cardiac surgery. The PACT is performed by using varying concentrations of platelet-activating factor (PAF) added to the reagent mixture of the ACT. Platelet-activating factor is a potent, endogenous platelet activator that stimulates *in vitro* thrombosis, indicating platelet responsiveness. The response to such an endogenous stimulant may provide a measure of platelet activity. Despotis *et al.*[12] recently described their experience with the PACT. They reported strong correlations ($r = -0.82$) between the PACT performed when patients arrive in the intensive care unit (ICU) and postoperative blood loss. They also found that recovery of PAF accelerated coagulation after administra-

Table 1. Demographic, Surgical, and Blood Loss Data

Age (yr)	65 ± 12
Gender (% female/% male)	31/69
Height (cm)	170 ± 10
Weight (kg)	82.7 ± 16.3
Body surface area (m^2)	1.9 ± 0.2
Type of surgery	
Primary CABG, valve, or myectomy (%)	62
Repeat or combined procedures (%)	38
Heparin dose (units)	21,000 ± 6,200
Protamine dose (mg)	250 ± 37
CPB duration (min)	87 ± 45

Cumulative mediastinal blood loss	Range	25th–75th Percentile	Median	Mean ± SD
4 h*	40–1,500	150–410	234	336 ± 279
8 h*	100–2,190	290–700	395	534 ± 397
12 h*	190–3,520	400–940	585	726 ± 533

Data are percent or mean ± SD; N = 100.

CABG = coronary artery bypass graft surgery.

* Shapiro–Wilcoxon normality test (indicating nonnormal distribution).

tion of 1-deamino-8-D-arginine vasopressin (DDAVP) or platelet therapy. They concluded that the PACT would be useful to identify patients at risk for excessive blood loss who would benefit from the administration of DDAVP, platelet transfusion, or both.

Our group recently reported a weak correlation ($r = -0.30$) between the PACT performed immediately after protamine administration and blood loss in the first 4 h after cardiac surgery.[13] We also found that the sensitivity and specificity of the TEG-MA were superior to those of PACT in predicting postoperative blood loss. The study by Despotis *et al.*[12] performed PACT when patients arrived in the ICU. Further, the increased duration of CPB and hypothermia in the Despotis *et al.* study likely resulted in a greater degree of platelet dysfunction. It is possible that the PACT may be most sensitive in the setting of severe platelet dysfunction. The mixed results by these two groups warrant further investigation.

In a recent preliminary report, Shore-Lesserson *et al.*[14] could not correlate the PACT with blood loss after CPB. They did, however, find that the PACT accurately trends defects in platelet function that occur during CPB by comparing it with flow cytometric methods, yet this was not true in the postoperative period.

We hypothesized that this new test of platelet function (PACT) performed when patients arrive in the ICU would be predictive of excessive blood loss after CPB.

Materials and Methods

After we received institutional review board approval and patient consent, 100 nonconsecutive adult patients undergoing elective cardiac surgery requiring CPB were recruited. No alterations in surgical, anesthetic, or postoperative care occurred as a result of participation in this study. Patients received an intravenous fentanyl, midazolam, and pancuronium anesthetic supplemented with inhaled isoflurane. Heparin (9,000 units/m^2 of body surface area) was administered intravenously before establishing CPB.[15] An additional 10,000 units of heparin was added to the CPB circuit prime volume of 1.6–1.8 l. The ACT was measured 10 min after heparin administration and every 30 min during CPB. Additional heparin was administered if the ACT was <450 s. Cardiopulmonary bypass was performed under normothermic or mildly hypothermic conditions (34–37°C), at flows of 2.4 l/min/m^2, and without the use of ultrafiltration. Membrane oxygenators were used in all patients. Intraoperative erythrocyte salvage was used for all patients. The anesthetic and surgical teams were unaware

Table 2. Summary Laboratory Results for the PACT, Platelet Count, PT, and aPTT

Characteristic	N	Mean ± SD	Median	25th/75th Percentile
PACT clotting time				
Baseline	100	725 ± 125	706	639/835
Channel 1	100	659 ± 217	592	519/723
Channel 2	100	630 ± 204	583	512/687
Channel 3	100	548 ± 123	522	464/622
Channel 4	100	452 ± 102	426	385/516
Channel 5	100	396 ± 105	370	328/452
Channel 6	100	340 ± 135	316	282/367
PACT clot ratio				
Channel 3	99	0.12 ± .09	0.12	0.08/0.17
Channel 4	99	0.27 ± 0.11	0.27	0.21/0.34
Channel 5	98	0.36 ± 0.10	0.37	0.31/0.42
Channel 6	99	0.46 ± 0.07	0.47	0.43/0.51
PACT% maximal				
Channel 3	99	23.7 ± 17.4	23	15/33
Channel 4	99	53.2 ± 21.4	52	41/66
Channel 5	98	71.0 ± 21.4	72	60/82
Channel 6	98	91.5 ± 15.0	92	84/99
Platelet count ($\times 10^9$/L)	98	128 ± 43.7	117	98/153
PT (s)	82	12.4 ± 2	11.9	11.0/13.0
aPTT (s)	89	41 ± 14	38	34/45
INR	82	1.24 ± 0.2	1.2	1.1/1.3

PACT = platelet activated clotting time; PT = prothrombin time; aPTT = activated partial thromboplastin time; INR = international normalization ratio.

Table 3. Relationship between Cumulative Mediastinal Blood Loss and PACT, Platelet Count, PT, and aPTT

Characteristic	N	4 h		8 h		12 h	
		r	*P* Value	r	*P* Value	r	*P* Value
PACT clotting time							
Baseline	100	0.277	0.005	0.247	0.013	0.241	0.015
Channel 1	100	0.309	0.0018	0.267	0.007	0.242	0.015
Channel 2	100	0.31	0.0017	0.261	0.008	0.242	0.016
Channel 3	100	0.24	0.017	0.191	0.028	0.187	0.074
Channel 4	100	0.185	0.012	0.141	0.042	0.129	0.067
Channel 5	100	0.315	0.0014	0.238	0.017	0.192	0.051
Channel 6	100	0.401	<0.0001	0.312	0.0016	0.235	0.018
PACT clot ratio							
Channel 3	99	0.15	0.86	0.18	0.99	0.186	0.97
Channel 4	99	0.186	0.42	0.20	0.36	0.20	0.37
Channel 5	98	0.03	0.67	0.08	0.59	0.11	0.58
Channel 6	99	0.12	0.77	0.13	0.75	0.16	0.72
PACT% maximal							
Channel 3	99	0.156	0.87	0.188	0.99	0.187	0.97
Channel 4	99	0.178	0.42	0.191	0.36	0.193	0.36
Channel 5	98	−0.08	0.67	−0.002	0.59	0.03	0.58
Channel 6	98	0.11	0.77	0.12	0.59	0.148	0.72
Platelet count ($\times 10^9$/L)	98	−0.12	0.077	−0.08	0.25	−0.09	0.33
PT (s)	82	0.61	<0.0001	0.53	<0.0001	0.52	<0.0001
aPTT (s)	89	−0.02	0.54	−0.02	0.04	−0.03	0.62

PACT = platelet activated clotting time; PT = prothrombin time; aPTT = activated partial thromboplastin time; r = Spearman's rank correlation.

of PACT results, and no clinical decisions were made based on PACT data.

Perioperative medications and surgical events were recorded. Whole-blood samples were obtained from a radial or femoral arterial catheter after operation within 1 h of patients' arrival in the ICU for a platelet count, prothrombin time (PT), activated partial thromboplastin time (aPTT), and PACT. Cumulative mediastinal blood loss (MBL) was recorded 4, 8, and 12 h after patients arrived in the ICU.

Laboratory PT and aPTT tests were performed on plasma from a 2.7-ml blood sample drawn into a vacu-

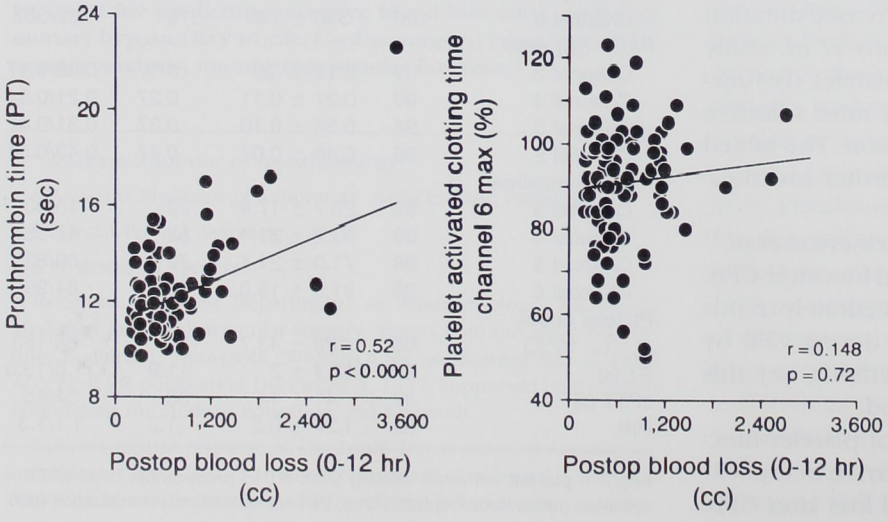

Fig. 1. Correlation of prothrombin time and platelet activated clotting test with postoperative mediastinal blood loss (at 0–12 h).

tainer containing 0.3 ml of 3.8% sodium citrate. The results, expressed in seconds, were determined as a function of time to clot formation determined by an MLA Electra-700 coagulation timer (Medical Laboratory Automation, Pleasantville, NY). The PT was performed using tissue thromboplastin and calcium (Dade-Innovin; Baxter Diagnostics, Miami, FL). The aPTT was determined using platelin-L and platelin-L calcium chloride (Organon Teknika Corp., Durham, NC).

The platelet count was determined electronically by an automated hemocytometer, Coulter STKS (Coulter Diagnostics, Hilaeh, FL), from a 3-ml blood sample placed into a vacutainer containing ethylene diamine tetracetate.

The PACT test was performed on a modified Hepcon HMS device (HemoSTATUS, Medtronic HemoTec, Parker, CO). Platelet procoagulant activity is determined by measuring the PAF-induced shortening of the kaolin-activated ACT. A syringe containing 3 ml whole blood was secured in the HMS machine. Blood (0.35 ml) was automatically distributed to each of six channels in the self-contained PACT cartridge. The cartridge contained all reagents necessary to perform the PACT test. Platelet-activated factor was present in channels 3, 4, 5, and 6 in concentrations of 1.25, 6.25, 12.5, and 150 nM, respectively. Channels 1 and 2 (with no PAF) served as controls. To increase the sensitivity of this method, ACTs were prolonged by adding 3 units/ml of heparin to each of the six channels during manufacturing. The PACT values were expressed as absolute clotting time, as a clot ratio, and as a percentage of the maximal response. The clot ratio value is calculated with the following formula for each PAF concentration: clot ratio = 1 − (ACT/control ACT). Clot ratio values were expressed as a percentage of maximal normal response in a normal patient reference population (Medtronic HemoTec) and were calculated using the mean clot ratio values obtained from channel 6 (150 nM PAF). The mean ± SD clot ratio values for the series of healthy volunteers in channel 6 was 0.51 ± 0.06. The percentage of maximal response = clot ratio/0.51 × 100. The clotting time, clot ratio, and percentage of maximal response are reported. In channels 5 and 6 (12.5 and 150 nM PAF), platelet stimulation was submaximal and maximal, respectively.

Sensitivity, specificity, and negative and positive predictive values were analyzed using the receiver operating characteristic method curves.[16] When there is a definition of disease state and abnormal test results, the receiver operating characteristic statistical method evaluates diagnostic accuracy. The two disease states of bleeding after CPB were determined by > an average of 100 ml/h and 200 ml/h chest tube blood loss in the first 4 h in the ICU. These values were chosen because the 100 ml/h rate has limited clinical significance, whereas the 200 ml/h rate would be considered clinically important. The laboratory cut point is the test value at which any observed value above it is considered a positive indication of disease (an abnormal test result) and any value below is considered a negative indication of the disease. With the receiver operating characteristic analysis, the cut point is changed throughout the potential range of the test being studied to examine the sensitivity and specificity of the test. The test result in which sensitivity and specificity together are at a maximum is the value frequently chosen to differentiate normal from the disease state. We defined sensitivity as the percentage of patients with excessive bleeding who also had an abnormal test result. Specificity was defined as the percentage of patients without excessive bleeding who also had a normal test result. Negative predictive value was defined as the percentage of patients with a normal test result who did not have excessive bleeding. Positive predictive value was defined as the percentage of patients with an abnormal test result who had excessive bleeding. A perfect test would have 100% sensitivity and specificity.

The Spearman rank correlation test (nonparametric analysis) was used to assess the degree of association between laboratory values (PACT, platelet count, PT, and aPTT) and MBL. Spearman rank correlation was also used to compare the relation between PACT values and platelet count, PT, and aPTT. After log transformation of blood loss data and univariate adjusting for the PT, the relation between cumulative blood loss and PACT, platelet count, and aPTT was completed using the Pearson correlation. A probability value ≤ 0.05 was considered significant.

Results

Table 1 summarizes demographic, surgical, and blood loss data. Table 2 shows all laboratory values. None of the percentage-maximal values of the PACT (channels 3, 4, 5, and 6) correlated with MBL or platelet count at 4, 8, or 12 h (table 3, fig. 1). There were no significant correlations between the PACT, clot ratio, and percentage-maximal values and blood loss. The PACT clotting time value did correlate with blood loss, but the platelet

Table 4. Relationship between PACT and Standard Coagulation Tests

Characteristic	N	Platelet Count		PT		aPTT	
		r	*P* Value	r	*P* Value	r	*P* Value
PACT clotting time							
Baseline	100	−0.42	<0.0001	0.58	<0.0001	0.44	<0.0001
Channel 1	100	−0.36	0.0003	0.62	<0.0001	0.36	0.0005
Channel 2	100	−0.46	<0.0001	0.55	<0.0001	0.39	0.0001
Channel 3	100	−0.44	<0.0001	0.52	<0.0001	0.24	0.043
Channel 4	100	−0.44	<0.0001	0.44	<0.0001	0.17	0.087
Channel 5	100	−0.44	<0.0001	0.44	<0.0001	0.09	0.19
Channel 6	100	−0.37	<0.0001	0.39	0.0003	0.06	0.19
PACT clot ratio							
Channel 3	99	−0.18	0.64	0.20	0.30	0.30	0.26
Channel 4	99	−0.11	0.87	0.26	0.01	0.27	0.29
Channel 5	98	−0.02	0.84	0.20	0.07	0.33	0.22
Channel 6	99	0.02	0.83	0.28	0.11	0.35	0.15
PACT% maximal							
Channel 3	99	−0.18	0.64	0.21	0.30	0.30	0.26
Channel 4	99	−0.11	0.87	0.26	0.02	0.27	0.29
Channel 5	98	−0.003	0.84	0.11	0.07	0.35	0.22
Channel 6	98	0.02	0.83	0.28	0.11	0.35	0.15

PACT = platelet activated clotting time; PT = prothrombin time; aPTT = activated partial thromboplastin time; r = Spearman's rank correlation.

count and aPTT did not. There was a strong and significant correlation between the PT and blood loss at 4 h ($r = 0.61$; $P < 0.0001$), 8 h ($r = 0.53$; $P < 0.0001$), and 12 h ($r = 0.52$; $P < 0.0001$). There were only a few small but significant correlations between the PACT percentage-maximal values and PT and aPTT (table 4). The PACT clotting time value did correlate with the PT, aPTT, and platelet count. After log transformation of blood loss data and univariate adjusting for the PT, none of the PACT values or platelet counts correlated with blood loss at 4, 8, or 12 h (table 5). After adjusting for PT, only the aPTT significantly correlated with blood loss at 4 h. Ten patients received antifibrinolytic therapy (aprotinin, tranexamic acid, or ϵ-amino caproic acid). Complete separate analyses of the antifibrinolytic group and the parent group did not reveal any significant difference, and thus complete data are presented here.

The PACT had sensitivity and specificity rates less than PT, aPTT, and platelet count in predicting excessive blood loss after CPB (table 6, fig. 2). The cut points that produce maximal sensitivity and specificity for each particular test were outside the normal range for these laboratory values and are shown in the second column of table 6. Only channel 6 of the PACT weakly correlated with transfusion of platelets (table 7). The PT, however, strongly correlated with transfusion of platelets and fresh frozen plasma.

Discussion

The sensitivity and specificity of the PT was greater than the PACT for predicting excessive blood loss, and only the PT, not the PACT, aPTT, or platelet count, correlated with MBL at 4, 8, and 12 h. In addition, only the PT strongly correlated with transfusion of allogeneic platelets and fresh frozen plasma. However, the PACT clotting time value, not thought to be a primary measure of platelet function, did correlate with blood loss and the PT, aPTT, and platelet count.

Predicting excessive microvascular bleeding after CPB caused by platelet dysfunction, dilutional or consumptive coagulopathy, fibrinolysis, or heparin rebound remains an elusive goal. The receiver operating curve analysis of the data presented here suggests that the PT is superior to the PACT, platelet count, or aPTT in predicting blood loss. The lack of correlation between the PACT and 4-h blood loss and CPB is important because interventions to improve hemostasis are usually initiated in the immediate postoperative period. It is during this immediate post-CPB period that platelet function usually begins to recover.[5]

Despotis *et al.*[12] reported strong correlations ($r = -0.82$) between the PACT performed on arrival to the intensive care unit and MBL at 4 h. They also noted

Table 5. Univariate Relationship between Cumulative Blood Loss and PACT, Platelet Count, and aPTT

	LN Blood Loss					
	0–4 h		0–8 h		0–12 h	
	P Value	*P* Value*	*P* Value	*P* Value*	*P* Value	*P* Value*
PACT clotting time						
Baseline	0.013	0.599	0.031	0.674	0.061	0.966
Channel 3	0.028	0.626	0.078	0.818	0.130	0.896
Channel 4	0.041	0.401	0.118	0.593	0.203	0.894
Channel 5	0.007	0.167	0.030	0.285	0.066	0.575
Channel 6	0.003	0.100	0.009	0.175	0.024	0.379
PACT% maximal						
Channel 3	0.719	0.563	0.474	0.800	0.415	0.907
Channel 4	0.606	0.440	0.405	0.635	0.354	0.728
Channel 5	0.804	0.249	0.890	0.389	0.818	0.527
Channel 6	0.962	0.443	0.795	0.639	0.727	0.745
Platelet count ($\times 10^9$/L)	0.249	0.287	0.546	0.431	0.531	0.449
aPTT (s)	0.593	0.034	0.883	0.112	0.866	0.147

LN = log transformation; PT = prothrombin time; aPTT = activated partial thromboplastin time.

* Pearson correlation, univariate *P* value after adjusting for PT.

superior sensitivity and specificity of the PACT for predicting excessive blood loss compared with routine coagulation tests. They found that recovery of PAF accelerated coagulation after administration of DDAVP or platelet therapy, and they concluded indirectly that the PACT would be useful in identifying patients at risk for excessive blood loss who would benefit from administration of DDAVP or platelet transfusion.

In another study we previously reported a weak correlation ($r = -0.30$) between the PACT drawn after protamine administration and blood loss at 4 h.[13] The difference between PACT measurements obtained immedi-

Table 6. Sensitivity, Specificity, and Predictive Value of Laboratory Tests for Blood Loss after Cardiopulmonary Bypass

Test	Value	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)	Negative Predictive Value (%)
Greater than an average of 200 ml/h chest tube loss for 4 h					
PACT clot ratio (% max)					
Channel 5	70%	50	67	8	94
Channel 6	95%	57	54	7	92
Platelet count	110×10^9/L	71	57	9	95
PT	12.5 s	71	79	17	95
aPTT	40 s	75	60	15	94
Greater than an average of 100 ml/h chest tube for 4 h					
PACT clot ratio (% max)					
Channel 5	75%	50	48	20	70
Channel 6	80%	58	56	30	72
Platelet count	110×10^9/L	64	61	30	82
PT	14.0 s	68	68	37	78
aPTT	40 s	50	55	23	76

PACT = platelet activated clotting test; PT = prothrombin time; aPTT = activated partial thromboplastin time; Value = laboratory cut point that produces maximal sensitivity and specificity.

ately after termination of CPB and those obtained within the first hour of admission to the ICU may be important because platelet function spontaneously begins to recover within a few hours after CPB. It is possible that the PACT may be most sensitive in the setting of severe platelet dysfunction. Shore-Lesserson *et al.*[14] could not correlate the PACT with blood loss, yet they did report that the PACT accurately trends defects in platelet function associated with CPB.

The mixed results found by these groups warrant further investigation. The present study was conducted to duplicate the Despotis study methods and determine the relation between the PACT drawn within the first hour in the ICU and blood loss. We could not confirm the previously reported strong correlations. This may reflect, in part, even greater decreases in platelet function due to increased duration of CPB or systemic hypothermia in the study by Despotis *et al.* or the use of a different heparin management protocol.

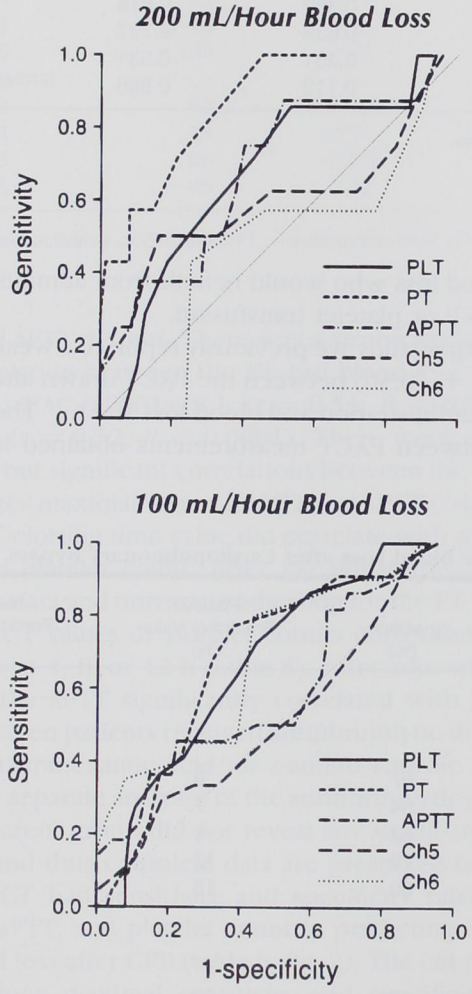

Fig. 2. Receiver operating characteristic curves for the ability of tests of coagulation function to identify patients with blood loss of more than an average of 200 ml/h and 100 ml/h for each of the first 4 h after operation. PLT = platelet count, PT = prothrombin time, aPTT = activated partial thromboplastin time, Ch5 = platelet activated clotting test channel 5, Ch6 = platelet activated clotting test channel 6. A test that lies on the line of identity has no predictive value.

Table 7. Relationship between Coagulation Tests and Transfusion of Hemostatic Factors

		Platelets		FFP	
Characteristic	N	r	*P* Value	r	*P* Value
PACT clot ratio					
Channel 3	99	0.05	0.63	0.10	0.36
Channel 4	99	0.05	0.61	0.15	0.15
Channel 5	99	0.03	0.76	0.07	0.52
Channel 6	98	0.19	0.05	0.20	0.06
PACT% maximal					
Channel 3	99	0.05	0.59	0.11	0.30
Channel 4	99	0.05	0.60	0.15	0.14
Channel 5	98	0.01	0.90	−0.02	0.86
Channel 6	98	0.19	0.05	0.20	0.06
Platelet count ($\times 10^9$/L)	98	−0.13	0.23	−.014	0.18
PT (s)	82	0.59	<0.0001	0.42	0.0001
aPTT (s)	89	0.12	0.25	−0.06	0.57

FFP = fresh frozen plasma; PT = prothrombin time; aPTT = activated partial thromboplastin time.

The PACT also did not correlate with MBL at 8 or 12 h in this study. This, however, is not unexpected because in many patients coagulopathy resolves within 6–8 h of surgery, and, as such, additional MBL is usually limited or unrelated to a coagulopathy. Our findings regarding the routine coagulation test cut points at which maximal sensitivity and specificity occur are similar to those previously reported by our group, suggesting excellent reproducibility of data and methods.[17,18] Channels 5 and 6 contain the highest concentration of PAF and thus provide for submaximal and maximal platelet stimulation of procoagulant activity. Although hypothermia can induce platelet dysfunction, all patients in this study underwent CPB at normothermic or mildly hypothermic conditions (34–37°C). The exclusion of the patients undergoing moderate or deep hypothermic CPB limited hypothermic-induced platelet dysfunction and provided a relatively homogenous population.

We found no correlation between the PACT and platelet count, and only channel 4 of the PACT weakly

correlated with the PT and aPTT. Platelet count and platelet function do not correlate with each other and platelet count correlates poorly with post-CPB bleeding and transfusion requirements.[19] However, platelet function as measured by the TEG-MA does correlate with post-CPB hemorrhage.[13,20]

Platelet dysfunction is one of the major causes of hemostatic abnormalities after CPB.[2-4,20] There is no reliable, rapid, or accurate test of platelet function available for use in the operating room or ICU. The ability to assess functional platelet reserve (platelet procoagulant activity) during and immediately after CPB would allow physicians to administer platelets and other blood products with more confidence. This could reduce the amount of blood products administered and also their attendant risks and expense.[21]

The PACT may quantify platelet procoagulant function after CPB, but in this study it did not correlate with blood loss, other tests of coagulation function, or transfusion requirements. The strong correlation between the PT and both blood loss and hemostatic transfusion requirements may reflect the importance of nonplatelet factors in postoperative blood loss after CPB or indicate that the PACT is measuring some aspect of platelet function that is not affected by CPB. Based on the results of this study, the sensitivity and specificity of the PACT in predicting blood loss are inadequate and routine clinical use of the PACT cannot be supported at this time. The use of the PACT clotting time value may be of benefit and needs to be evaluated further.

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