Anticoagulation: how to deal, how to monitor

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IRCCS Policlinico San Donato - Milano

ECLS Registry Report

International Summary

January, 2016

Extracorporeal Life Support Organization
2800 Plymouth Road
Building 300, Room 303
Ann Arbor, MI 48109

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Cardiac Complications (0-30 days)

	No. Reported	*% Reported*	*No. Survived*	*% Survived*
Mechanical: Oxygenator failure	428	6.3%	105	25%
Mechanical: Raceway rupture	18	0.3%	6	33%
Mechanical: Other tubing rupture	34	0.5%	11	32%
Mechanical: Pump malfunction	104	1.5%	31	30%
Mechanical: Heat exchanger malfunction	32	0.5%	18	56%
Mechanical: Clots: oxygenator	780	11.5%	243	31%
Mechanical: Clots: bridge	248	3.7%	76	31%
Mechanical: Clots: bladder	378	5.6%	101	27%
Mechanical: Clots: hemofilter	284	4.2%	70	25%
Mechanical: Clots: other	926	13.7%	320	35%
Mechanical: Air in circuit	212	3.1%	63	30%
Mechanical: Cracks in pigtail connectors	39	0.6%	19	49%
Mechanical: Cannula problems	404	6.0%	141	35%
Hemorrhagic: GI hemorrhage	73	1.1%	10	14%
Hemorrhagic: Cannulation site bleeding	720	10.7%	230	32%
Hemorrhagic: Surgical site bleeding	1,995	29.5%	618	31%
Hemorrhagic: Hemolysis (hgb > 50 mg/dl)	756	11.2%	209	28%
Hemorrhagic: Disseminated intravascular coagulation (DIC)	267	4.0%	47	18%
Neurologic: Brain death clinically determined	71	1.1%	0	0%
Neurologic: Seizures: clinically determined	437	6.5%	141	32%
Neurologic: Seizures: EEG determined	205	3.0%	65	32%
Neurologic: CNS infarction by US/CT	229	3.4%	63	28%
Neurologic: CNS hemorrhage by US/CT	760	11.2%	183	24%

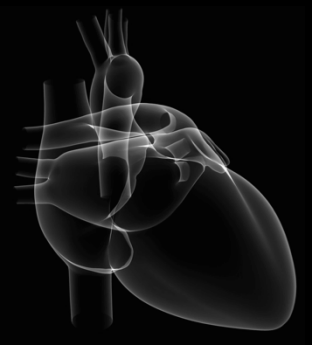
Cardiac Complications (31 days and < 1 year)	*No. Reported*	*% Reported*	*No. Survived*	*% Survived*
Mechanical: Oxygenator failure	318	7.1%	93	29%
Mechanical: Raceway rupture	21	0.5%	10	48%
Mechanical: Other tubing rupture	25	0.6%	6	24%
Mechanical: Pump malfunction	81	1.8%	26	32%
Mechanical: Heat exchanger malfunction	12	0.3%	5	42%
Mechanical: Clots: oxygenator	379	8.5%	156	41%
Mechanical: Clots: bridge	128	2.9%	49	38%
Mechanical: Clots: bladder	174	3.9%	66	38%
Mechanical: Clots: hemofilter	157	3.5%	52	33%
Mechanical: Clots: other	471	10.6%	197	42%
Mechanical: Air in circuit	108	2.4%	49	45%
Mechanical: Cracks in pigtail connectors	35	0.8%	13	37%
Mechanical: Cannula problems	237	5.3%	85	36%
Hemorrhagic: GI hemorrhage	88	2.0%	12	14%
Hemorrhagic: Cannulation site bleeding	556	12.5%	218	39%
Hemorrhagic: Surgical site bleeding	1,375	30.8%	540	39%
Hemorrhagic: Hemolysis (hgb > 50 mg/dl)	429	9.6%	140	33%
Hemorrhagic: Disseminated intravascular coagulation (DIC)	154	3.5%	35	23%
Neurologic: Brain death clinically determined	133	3.0%	0	0%
Neurologic: Seizures: clinically determined	368	8.3%	104	28%
Neurologic: Seizures: EEG determined	151	3.4%	49	32%
Neurologic: CNS infarction by US/CT	214	4.8%	73	34%
Neurologic: CNS hemorrhage by US/CT	276	6.2%	79	29%

Cardiac Complications (1 year and < 16 years)

	No. Reported	*% Reported*	*No. Survived*	*% Survived*
Mechanical: Oxygenator failure	285	7.7%	126	44%
Mechanical: Raceway rupture	22	0.6%	10	45%
Mechanical: Other tubing rupture	36	1.0%	13	36%
Mechanical: Pump malfunction	64	1.7%	32	50%
Mechanical: Heat exchanger malfunction	13	0.3%	7	54%
Mechanical: Clots: oxygenator	299	8.0%	152	51%
Mechanical: Clots: bridge	81	2.2%	42	52%
Mechanical: Clots: bladder	72	1.9%	37	51%
Mechanical: Clots: hemofilter	88	2.4%	29	33%
Mechanical: Clots: other	405	10.9%	226	56%
Mechanical: Air in circuit	100	2.7%	44	44%
Mechanical: Cracks in pigtail connectors	33	0.9%	14	42%
Mechanical: Cannula problems	224	6.0%	107	48%
Hemorrhagic: GI hemorrhage	107	2.9%	29	27%
Hemorrhagic: Cannulation site bleeding	685	18.4%	373	54%
Hemorrhagic: Surgical site bleeding	1,012	27.2%	507	50%
Hemorrhagic: Hemolysis (hgb > 50 mg/dl)	308	8.3%	139	45%
Hemorrhagic: Disseminated intravascular coagulation (DIC)	152	4.1%	52	34%
Neurologic: Brain death clinically determined	227	6.1%	0	0%
Neurologic: Seizures: clinically determined	155	4.2%	38	25%
Neurologic: Seizures: EEG determined	44	1.2%	15	34%
Neurologic: CNS infarction by US/CT	180	4.8%	72	40%
Neurologic: CNS hemorrhage by US/CT	154	4.1%	32	21%

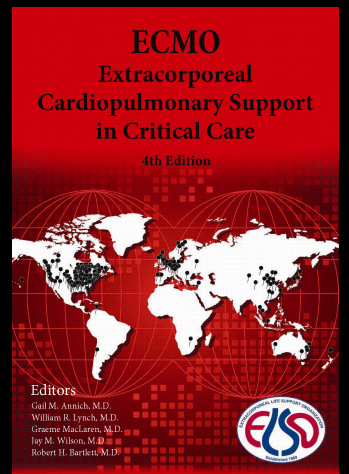
Cardiac Complications (16 years and over)	*No. Reported*	*% Reported*	*No. Survived*	*% Survived*
Mechanical: Oxygenator failure	626	7.4%	228	36%
Mechanical: Raceway rupture	6	0.1%	2	33%
Mechanical: Other tubing rupture	14	0.2%	4	29%
Mechanical: Pump malfunction	64	0.8%	21	33%
Mechanical: Heat exchanger malfunction	8	0.1%	5	63%
Mechanical: Clots: oxygenator	776	9.2%	329	42%
Mechanical: Clots: bridge	54	0.6%	30	56%
Mechanical: Clots: bladder	17	0.2%	8	47%
Mechanical: Clots: hemofilter	113	1.3%	28	25%
Mechanical: Clots: other	521	6.2%	212	41%
Mechanical: Air in circuit	100	1.2%	30	30%
Mechanical: Cracks in pigtail connectors	26	0.3%	3	12%
Mechanical: Cannula problems	361	4.3%	132	37%
Hemorrhagic: GI hemorrhage	360	4.3%	91	25%
Hemorrhagic: Cannulation site bleeding	1,574	18.6%	618	39%
Hemorrhagic: Surgical site bleeding	1,730	20.5%	581	34%
Hemorrhagic: Hemolysis (hgb > 50 mg/dl)	481	5.7%	152	32%
Hemorrhagic: Disseminated intravascular coagulation (DIC)	306	3.6%	67	22%
Neurologic: Brain death clinically determined	354	4.2%	0	0%
Neurologic: Seizures: clinically determined	135	1.6%	33	24%
Neurologic: Seizures: EEG determined	50	0.6%	10	20%
Neurologic: CNS infarction by US/CT	322	3.8%	75	23%
Neurologic: CNS hemorrhage by US/CT	184	2.2%	17	9%

Which is the ideal anticoagulant for ECMO?



Which is the ideal anticoagulant for ECMO?

An ideal anticoagulation for ECMO should inhibit platelet and coagulation system activation within the extracorporeal circuit, be easily titrated to clinical effect, yet still allow enough endogenous coagulation activity to prevent bleeding in the patient.

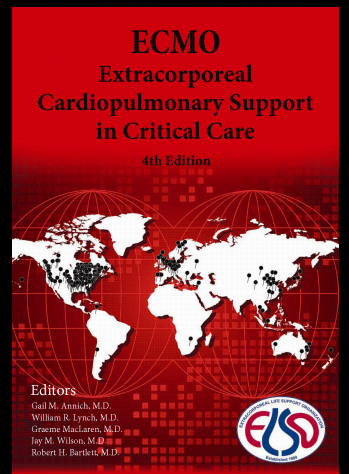


Lequier L et al, ECMO: Extracorporeal Cardiopulmonary Support in Critical Care 4th Edition

Which is the ideal anticoagulant for ECMO?

An ideal anticoagulation for ECMO should inhibit platelet and coagulation system activation within the extracorporeal circuit, be easily titrated to clinical effect, yet still allow enough endogenous coagulation activity to prevent bleeding in the patient.

Such an ideal anticoagulant is not currently available and therefore unfractionated heparin remains default anticoagulant.



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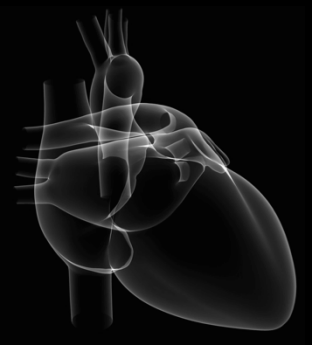
WHY DOES AN ECMO PATIENT MAY BLEED?

- Perioperative coagulopathy in post-cardiotomy ECMO
- Need for anticoagulation during ECMO
- Peri-procedural coagulopathy
- Acquired von Willebrand disease
- Heparin-like effect



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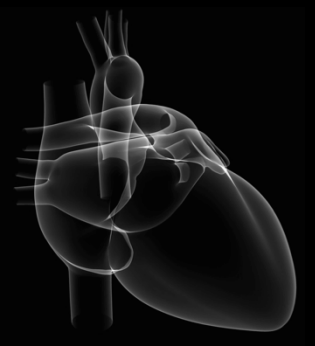
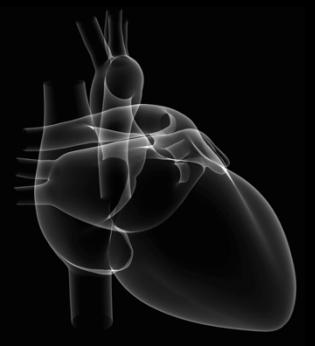


Perioperative coagulopathy in post-cardiotomy ECMO

Patients who need ECMO as a rescue approach after cardiac surgery and difficult weaning from CPB have usually experienced a prolonged surgery and CPB



Perioperative coagulopathy in post-cardiotomy ECMO

As a result, they invariably suffer from a complex coagulopathy which includes:

1. Thrombocytopenia
2. Platelet dysfunction
3. Low fibrinogen levels
4. Reduced levels of AT III
5. Reduced levels of soluble coagulation factors
6. Acidosis and hypothermia are common…

Perioperative coagulopathy in post-cardiotomy ECMO

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6. Acidosis and hypothermia are common…

Moderate-degree acidosis is an independent determinant of postoperative bleeding in cardiac surgery.

Ranucci M[1], Baryshnikova E, Simeone F, Ranucci M, Scolletta S.

Abstract

BACKGROUND:

Acidosis is a well-known factor leading to coagulopathy. It has been widely explored as a risk factor for severe bleeding in trauma patients. However, no information with respect to acidosis as a determinant of postoperative bleeding in cardiac surgery patients exists. The objective of this study is to investigate the role of acidosis and hyperlactatemia in determining postoperative bleeding and need for surgical revision in cardiac surgery patients.

METHODS:

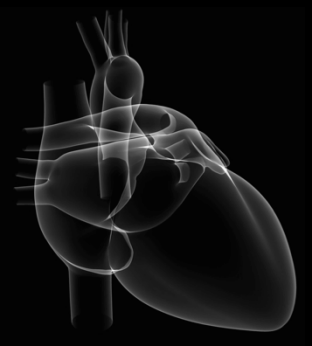
Retrospective analysis on 4,521 patients receiving cardiac operations in two institutions. For each patient the preoperative data and operative profile was available. Arterial blood gas analysis data at the arrival in the intensive care unit were analyzed to investigate the association between acidosis (pH < 7.35), hyperlactatemia (> 4.0 mMol/L) and postoperative bleeding and surgical revision rate.

RESULTS:

After correction for the potential confounders, both acidosis (P=0.001) and hyperlactatemia (P=0.001) were significantly associated with the amount of postoperative bleeding. Hyperlactatemia was an independent risk factor for postoperative bleeding even in absence of acidosis. Overall, surgical revision rate was 5.6% in patients with hyperlactatemia and no acidosis; 7.7% in patients with acidosis and hyperlactatemia, and 7.2% in patients with acidosis and no hyperlactatemia. All these values are significantly (P=0.001) higher than the ones in patients without acidosis/hyperlactatemia (2%).

CONCLUSIONS:

Even a moderate degree of postoperative acidosis is associated with a greater postoperative bleeding and surgical revision rate in cardiac surgery patients. Correction of acidosis with bicarbonate does not lead to an improvement of the postoperative bleeding asset.



Ranucci M; Minerva Anestesiol. 2015 Aug;81(8):885-93

Acidosis and Coagulopathy

The Differential Effects on Fibrinogen Synthesis and Breakdown in Pigs

Wenjun Z. Martini, PhD, and John B. Holcomb, MD

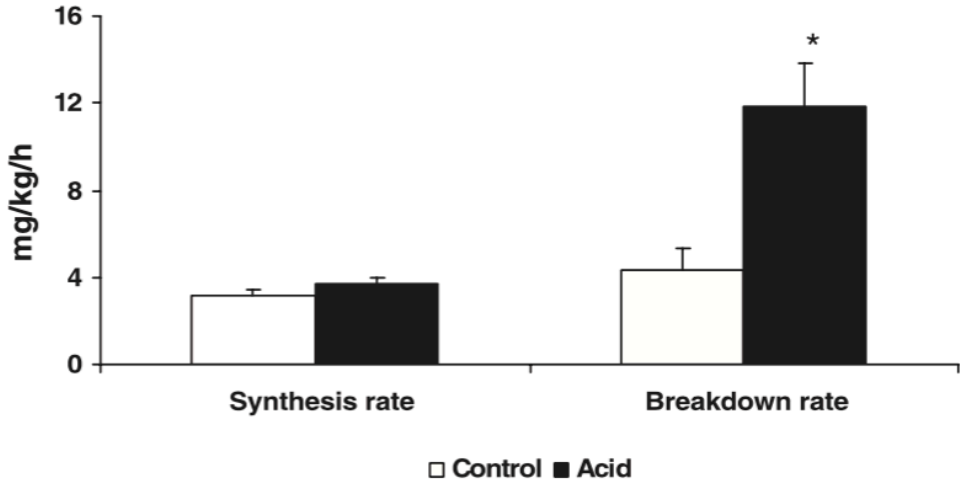
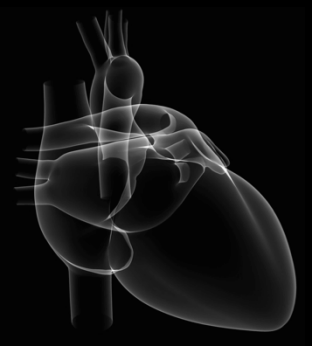


FIGURE 1. Effects of acidosis on fibrinogen synthesis and breakdown rates. *$P < 0.05$ compared with values in the control group.



Martini W and Holcomb J; Ann Surg 2007;246: 831–835

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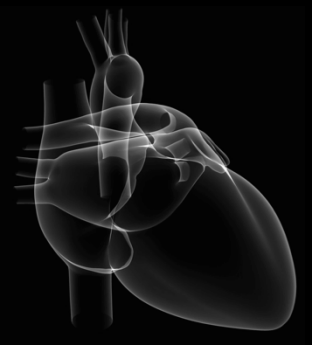
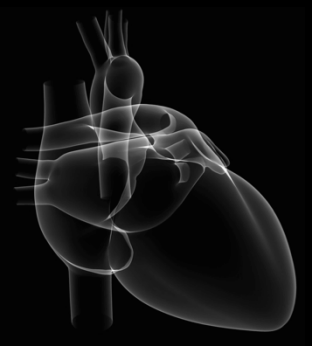


ANTICOAGULATION ON ECMO: WHY?

1. TO AVOID CLOT FORMATION IN THE CIRCUIT
2. TO AVOID CLOT FORMATION IN THE PATIENT



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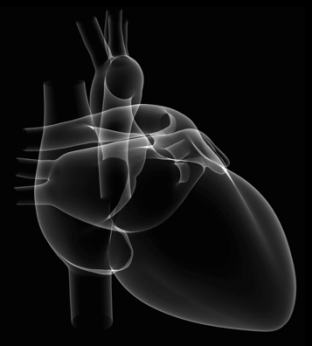
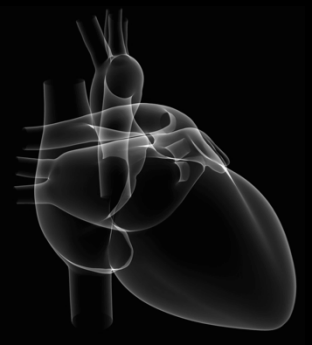
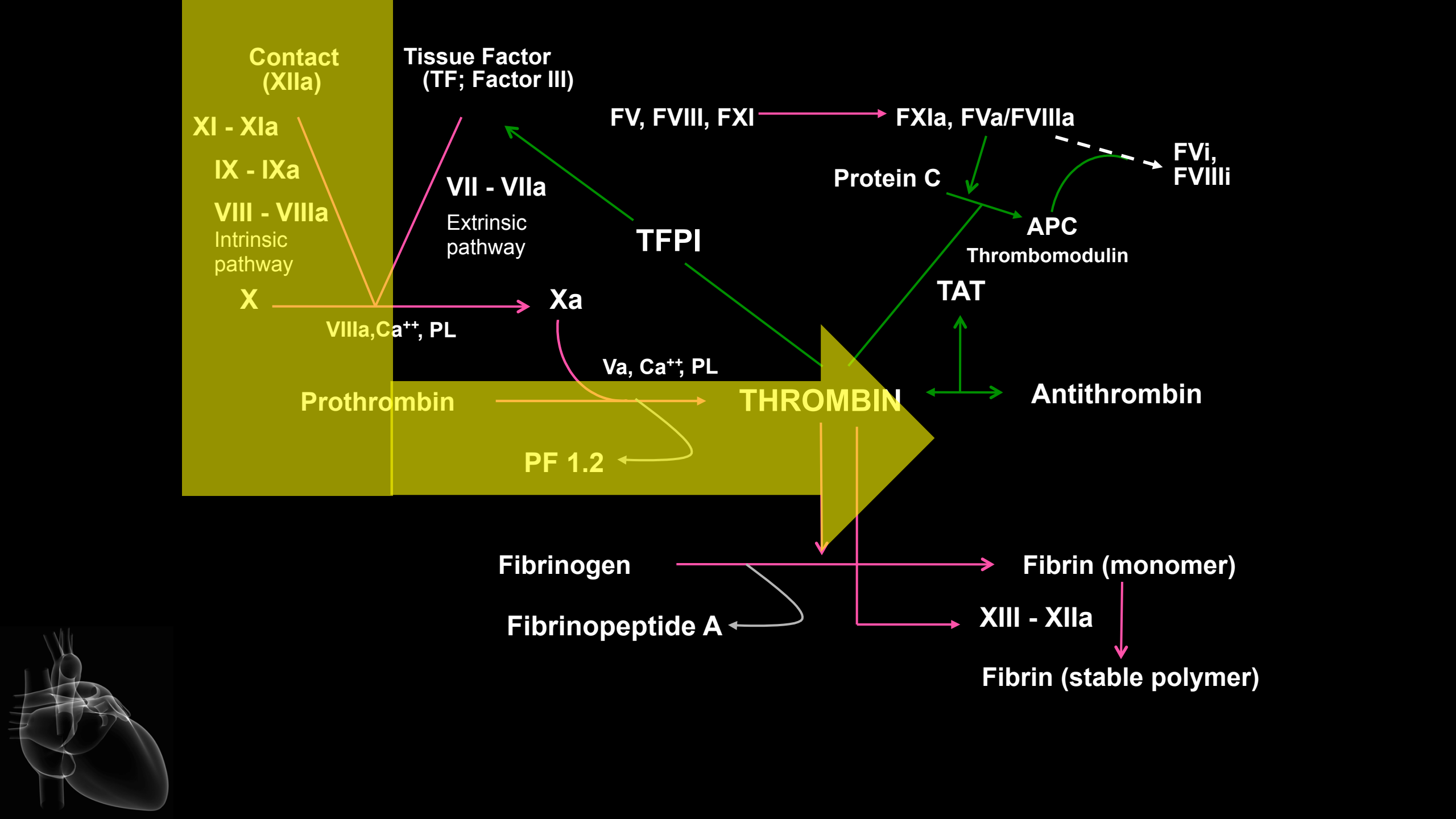


WE WANT TO AVOID…..

Material-dependent blood activation

Mechanical: Clots: oxygenator	568	9.4%	236	42%
Mechanical: Clots: bridge	44	0.7%	24	55%
Mechanical: Clots: bladder	12	0.2%	5	42%
Mechanical: Clots: hemofilter	93	1.5%	25	27%
Mechanical: Clots: other	376	6.2%	148	39%




Contact
(XIIa)
Tissue Factor
(TF; Factor III)
XI - XIa
IX - IXa
VIII - VIIIa
Intrinsic
pathway
VII - VIIa
Extrinsic
pathway
FV, FVIII, FXI
FXIa, FVa/FVIIIa
FVi,
FVIIIi
Protein C
APC
Thrombomodulin
TFPI
TAT
X
Xa
VIIIa,Ca++, PL
Va, Ca++; PL
Prothrombin
THROMBIN
Antithrombin
PF 1.2
Fibrinogen
Fibrin (monomer)
Fibrinopeptide A
XIII - XIIa
Fibrin (stable polymer)

ANTICOAGULATION ON ECMO: WHY?

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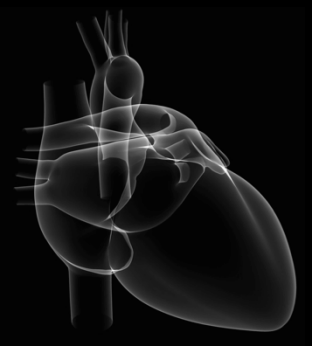
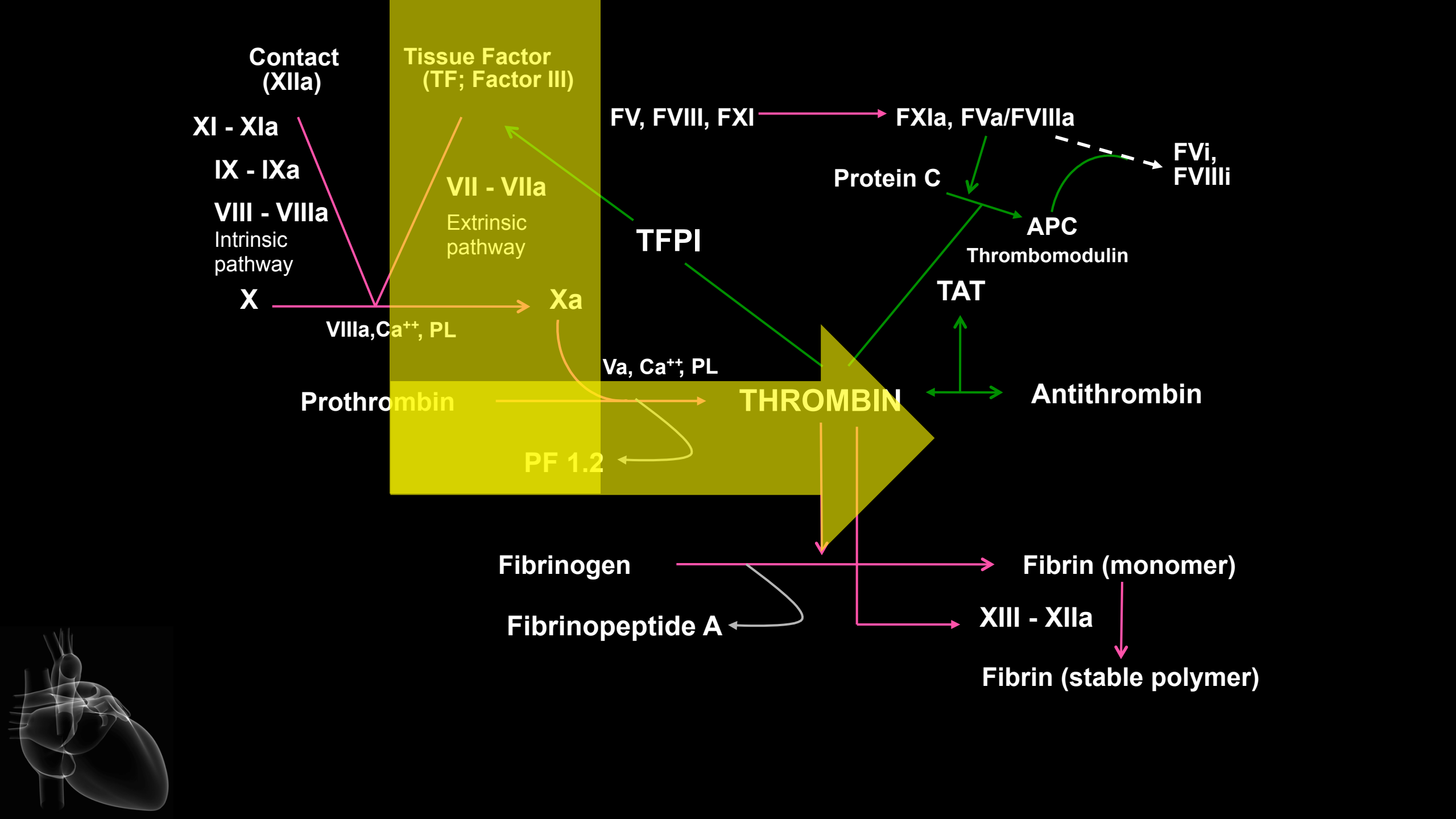
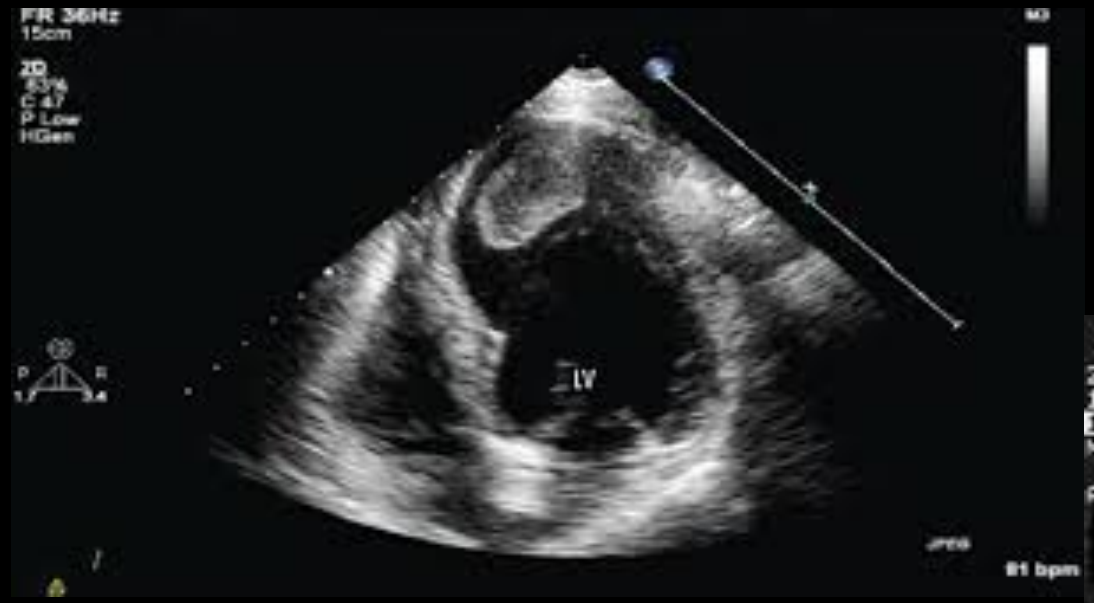
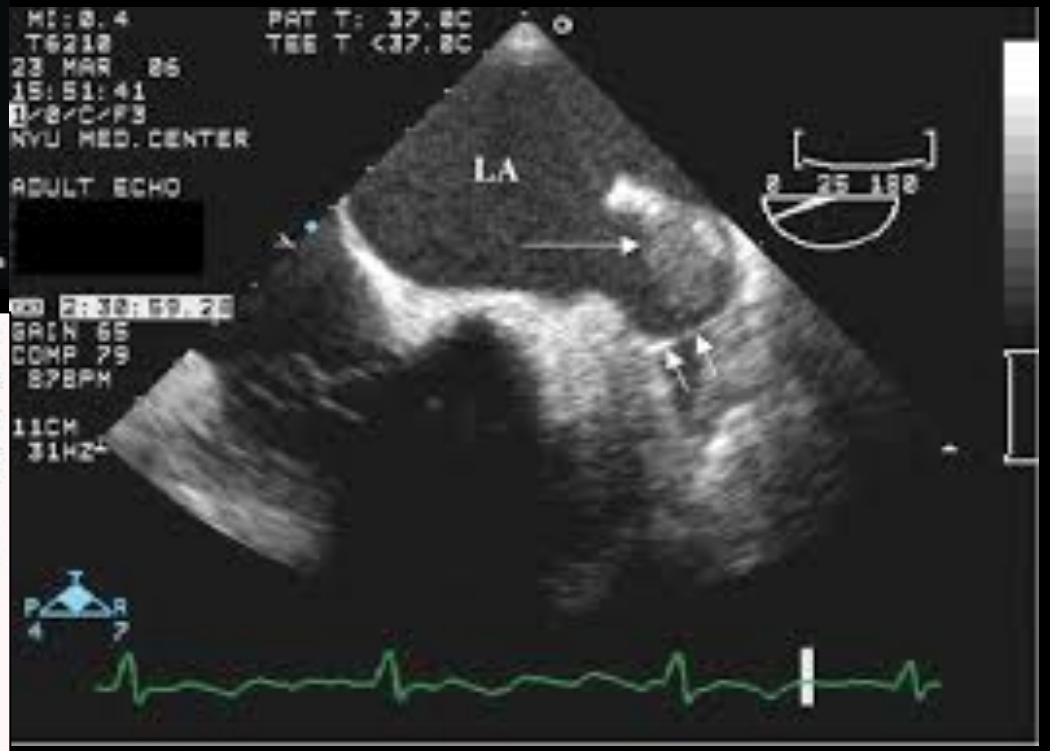
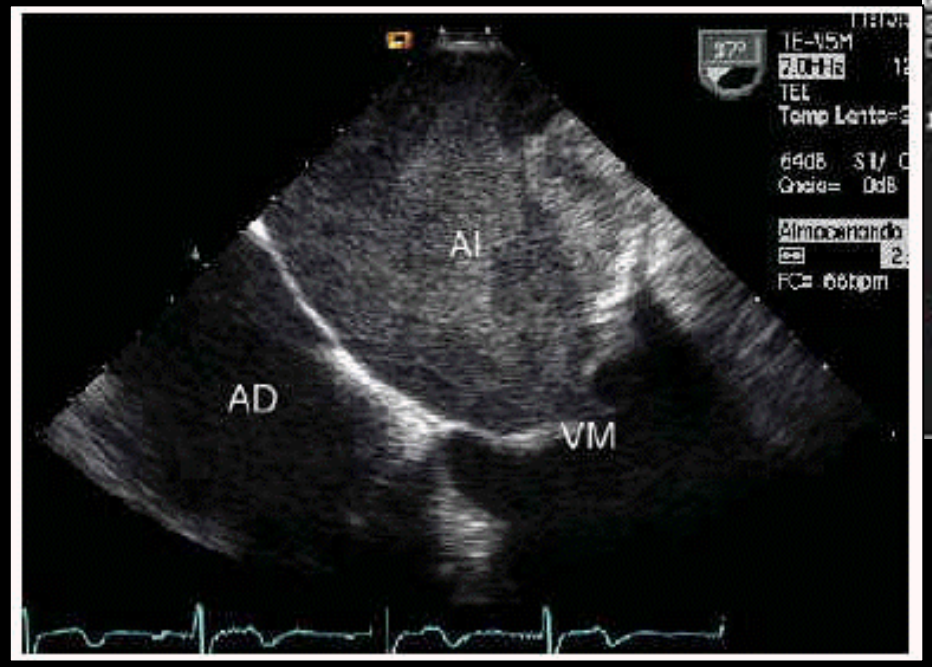



Contact
(XIIa)
Tissue Factor
(TF; Factor III)
XI - XIa
IX - IXa
VIII - VIIIa
Intrinsic
pathway
VII - VIIa
Extrinsic
pathway
X
Xa
VIIIa,Ca++, PL
TFPI
FV, FVIII, FXI
FXIa, FVa/FVIIIa
FVi,
FVIIIi
Protein C
APC
Thrombomodulin
TAT
Va, Ca++; PL
Prothrombin
THROMBIN
Antithrombin
PF 1.2
Fibrinogen
Fibrin (monomer)
Fibrinopeptide A
XIII - XIIa
Fibrin (stable polymer)


FR 36Hz
15cm
2D
LV
81 bpm


MI:0.4
PAT T: 37.8C
T6210
TEE T <37.8C
23 MAR 06
15:51:41
NYU MED. CENTER
ADULT ECHO
LA
GAIN 65
COMP 79
11CH
31HZ


AI
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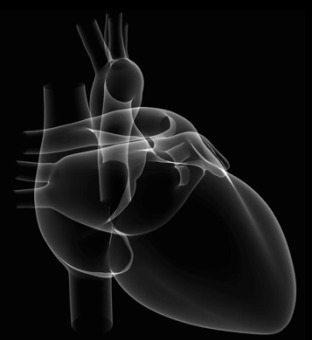
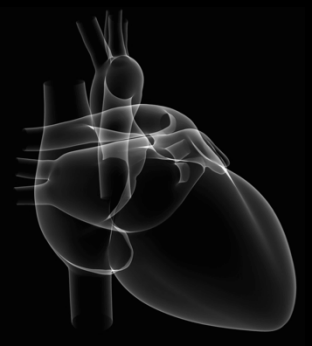
FC= 66bpm



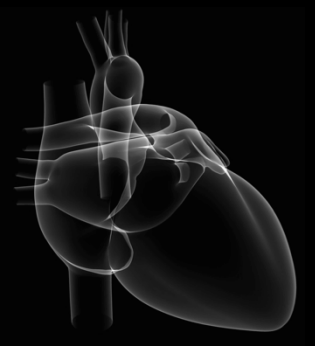
THE TEN COMMANDMENTS OF ANTICOAGULATION ON ECMO



1. AN ECMO IS NOT A CPB
2. LOW HEPARIN DOSE MAY ONLY CONTROL LOW THROMBIN GENERATION
3. THE ECMO CIRCUIT IS NOT THE ONLY SOURCE OF CLOTS
4. BEWARE OF BLEEDING
5. BEWARE OF THROMBOSIS
6. THE HEPARIN DOSE IS NOT A CONSTANT
7. CONSIDER THE COFACTORS (PLATELET COUNT AND AT III LEVELS)
8. FIBRINOGEN IS IMPORTANT BUT ONLY AT THE ECMO ONSET (24 HOURS)
9. THERE ARE ALTERNATIVES TO HEPARIN
10. WE DON’T KNOW ANYTHING ON ANTICOAGULATION IN ECMO

ALTERNATIVES TO HEPARIN

1. Bivalirudin is a direct thrombin inhibitor
2. It works independently from AT levels
3. Continuous infusion, rapid onset/offset
4. Successfully used when HIT on ECMO or even in non-HIT ECMO patients



RESEARCH Open Access

Bivalirudin-based versus conventional heparin anticoagulation for postcardiotomy extracorporeal membrane oxygenation

Marco Ranucci[1*], Andrea Ballotta[1], Hassan Kandil[1], Giuseppe Isgrò[1], Concetta Carlucci[1], Ekaterina Baryshnikova[1] and Valeria Pistuddi[1], for the Surgical and Clinical Outcome Research Group

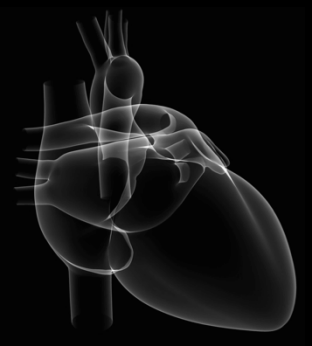
Abstract

Introduction: Extracorporeal membrane oxygenation (ECMO) after cardiac operations (postcardiotomy) is commonly used for the treatment of acute heart failure refractory to drug treatment. Bleeding and thromboembolic events are the most common complications of postcardiotomy ECMO. The present study is a retrospective comparison of the conventional heparin-based anticoagulation protocol with a bivalirudin-based, heparin-free protocol. Endpoints of this study are blood loss, allogeneic blood product use, and costs during the ECMO procedure.

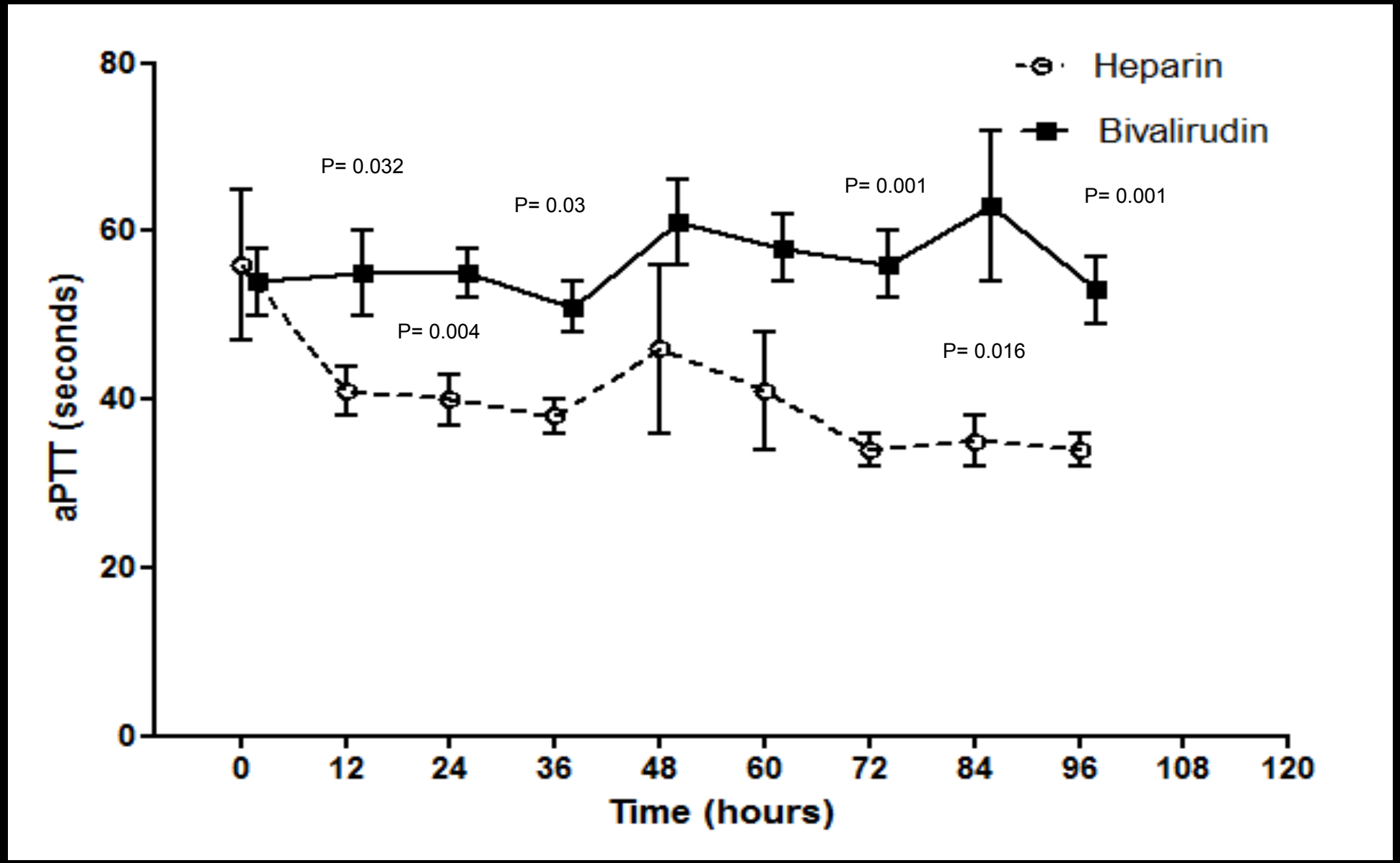
Methods: A retrospective study was undertaken in the setting of cardiac surgery, anesthesia, and intensive care departments of a university research hospital. Twenty-one patients (12 adults and nine children) who underwent postcardiotomy ECMO from 2008 through 2011 were retrospectively analyzed. The first consecutive eight patients were treated with heparin-based anticoagulation (H-group) and the next 13 consecutive patients with bivalirudin-based anticoagulation (B-group). The following parameters were analyzed: standard coagulation profile, thromboelastographic parameters, blood loss, allogeneic blood products use, thromboembolic complications, and costs during the ECMO treatment.

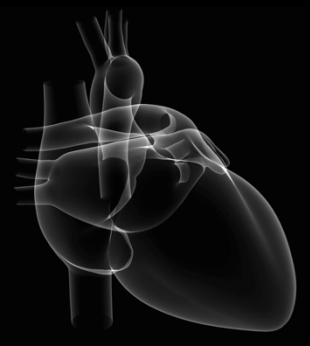
Results: Patients in the B-group had significantly longer activated clotting times, activated partial thromboplastin times, and reaction times at thromboelastography. The platelet count and antithrombin activity were not significantly different, but in the H-group a significantly higher amount of platelet concentrates, fresh frozen plasma, and purified antithrombin were administered. Blood loss was significantly lower in the B-group, and the daily cost of ECMO was significantly lower in pediatric patients treated with bivalirudin. Thromboembolic complications did not differ between groups.

Conclusions: Bivalirudin as the sole anticoagulant can be safely used for postcardiotomy ECMO, with a better coagulation profile, less bleeding, and allogeneic transfusions. No safety issues were raised by this study, and costs are reduced in bivalirudin-treated patients.



Ranucci M et al, Crit Care. 2011;15(6):R275





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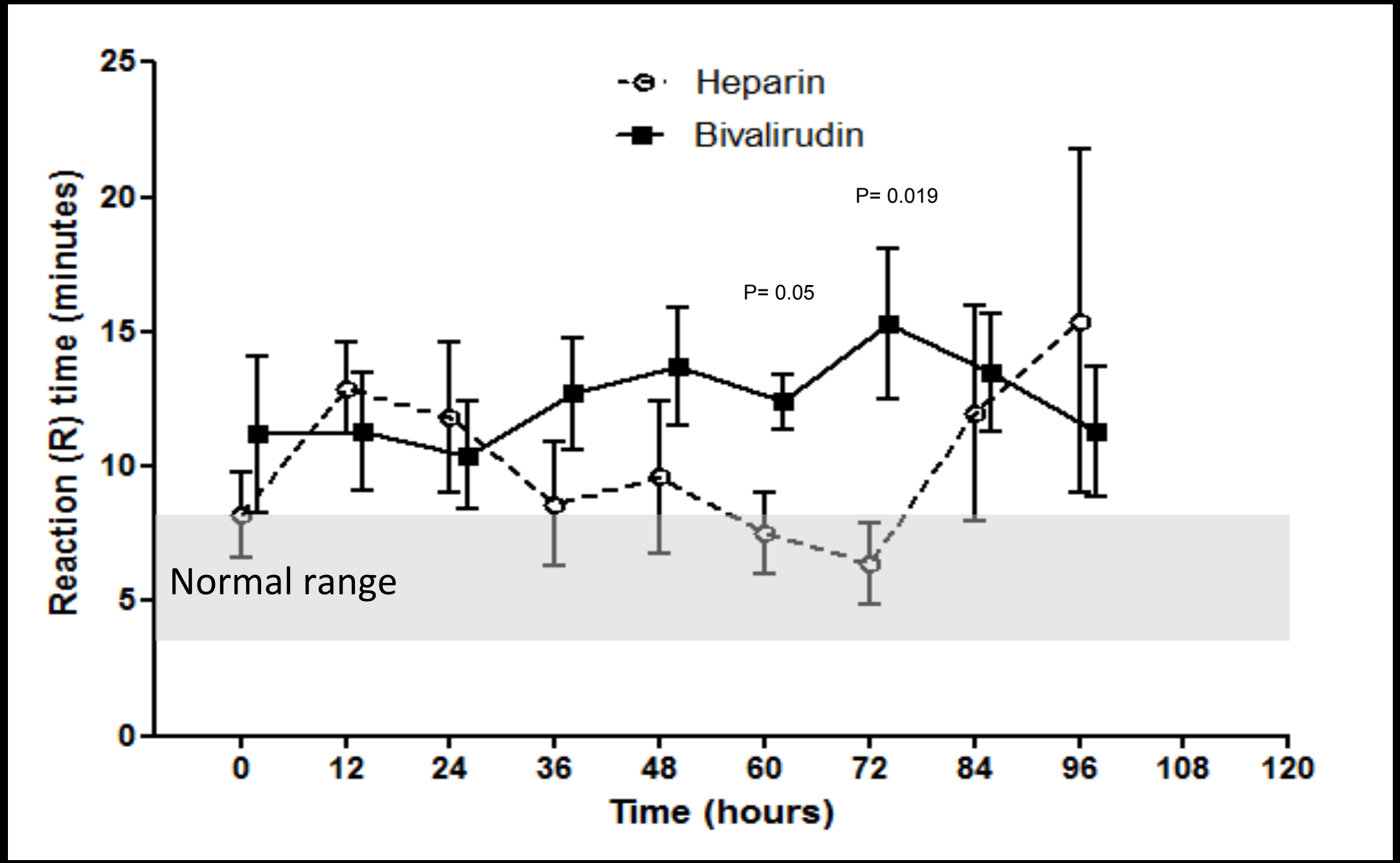

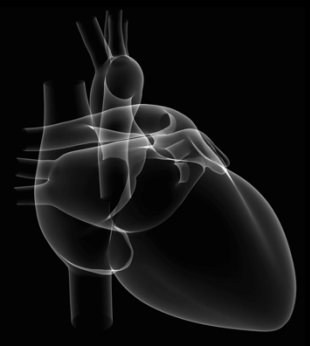

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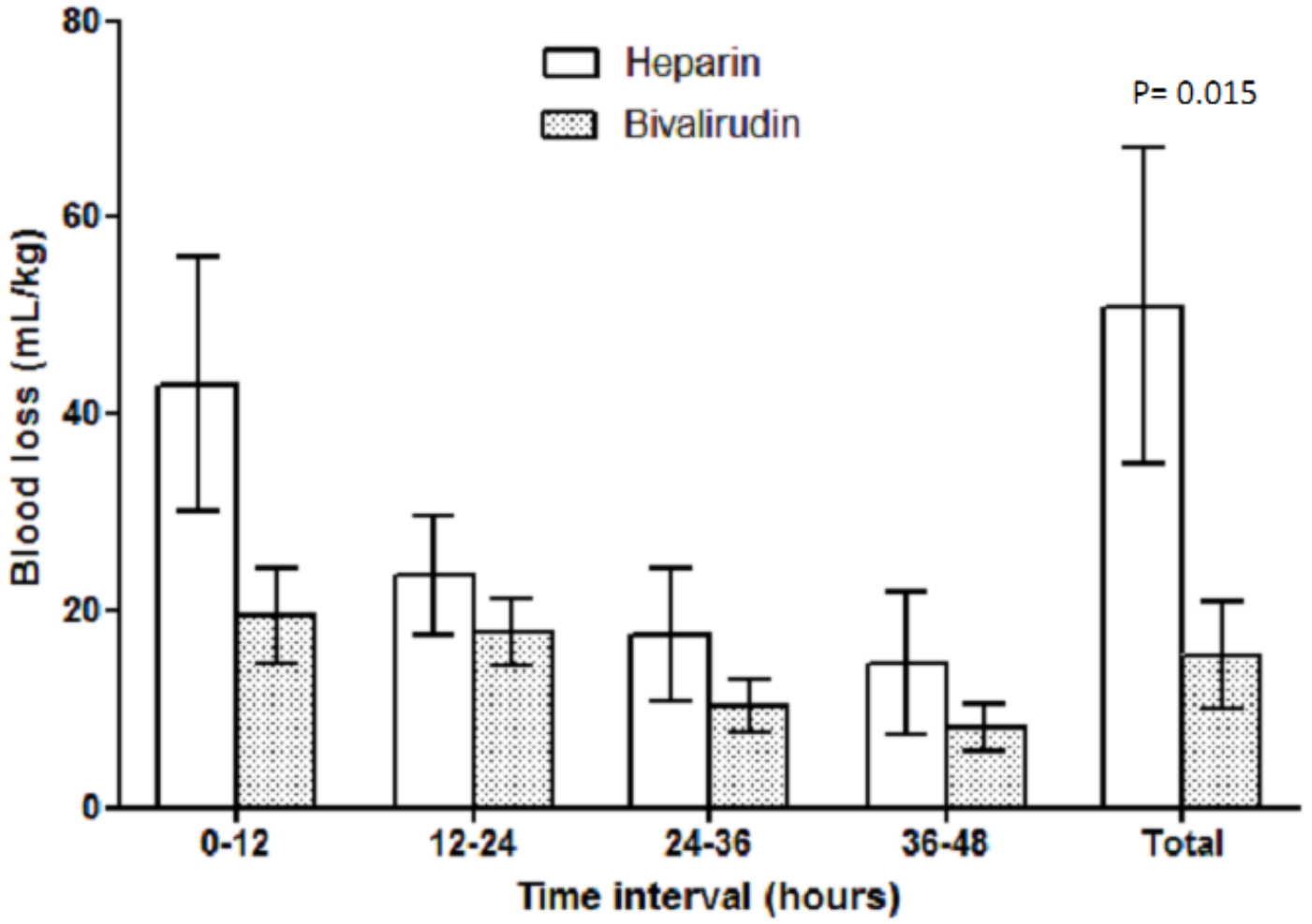
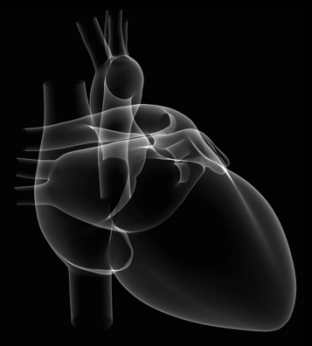


Figure 1



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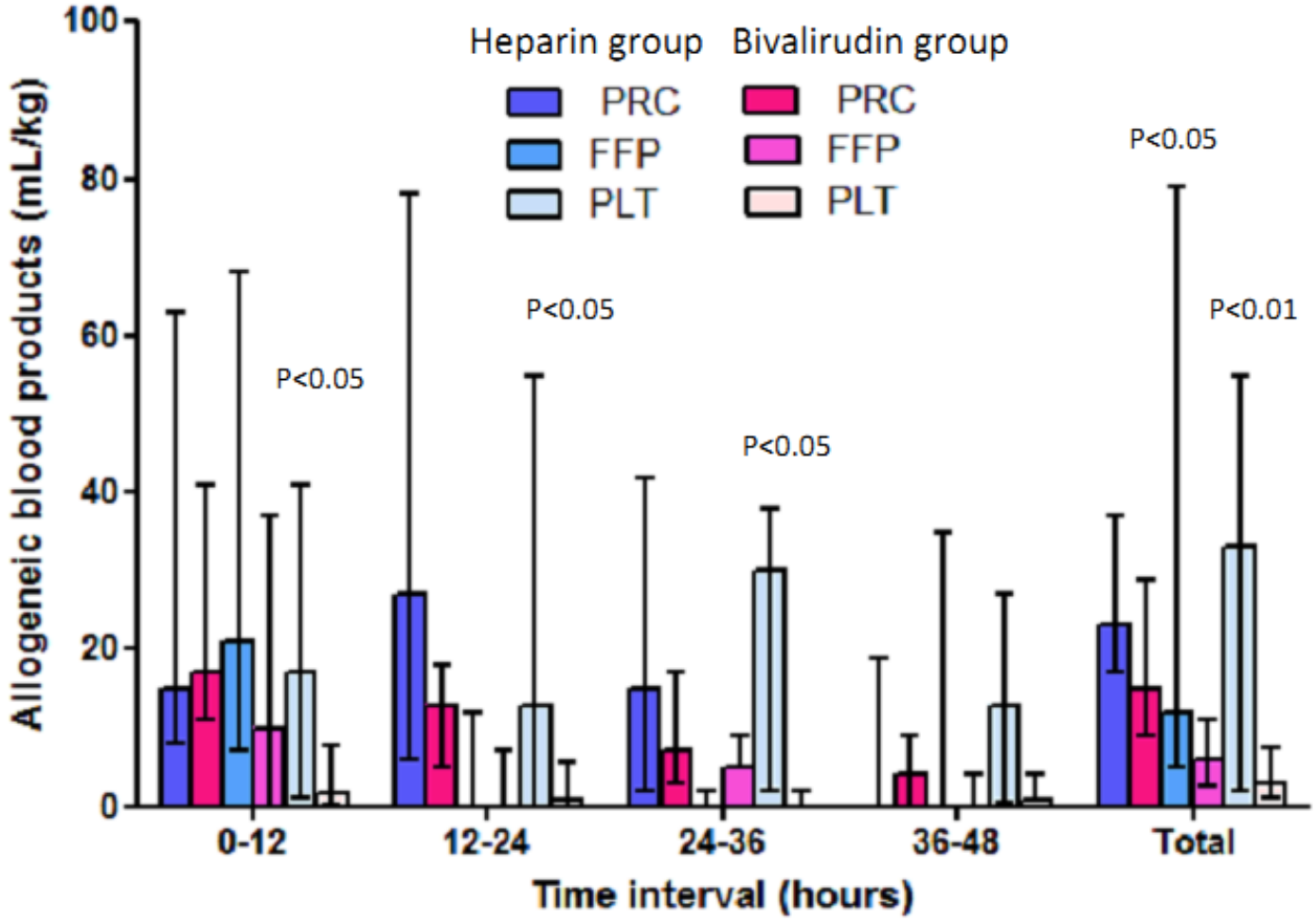
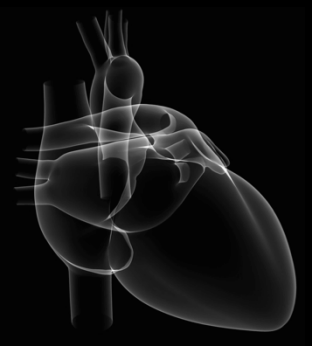


Figure 2



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WHY DOES AN ECMO PATIENT MAY BLEED?

- Perioperative coagulopathy in post-cardiotomy ECMO
- Need for anticoagulation during ECMO
- Peri-procedural coagulopathy
- Acquired von Willebrand disease
- Heparin-like effect

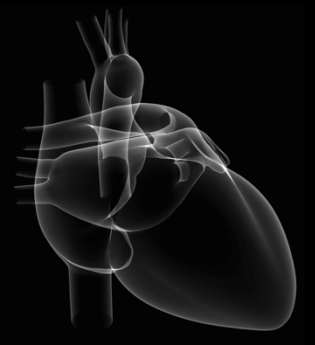
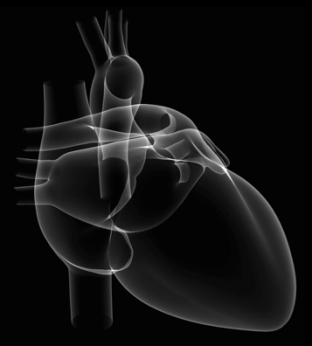


Table 1. Main factors contributing to hemorrhagic and thrombotic complications in ECMO

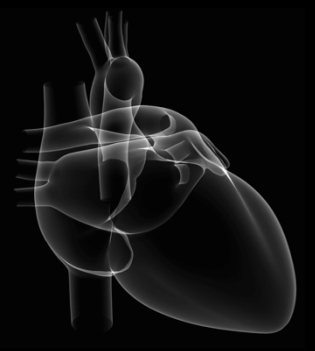
Pro-hemorrhagic factors	Pro-thrombotic factors
Excessive heparin anticoagulation	Inadequate heparin anticoagulation
Consumption of coagulation factors	Acquired antithrombin deficiency
Low fibrinogen levels	Protein C-S complex consumption
Thrombocytopenia	Tissue factor pathway inhibitor consumption
Platelet dysfunction	Endothelial dysfunction
Hyperfibrinolysis	Heparin-induced thrombocytopenia
Acquired von Willebrand disease	Blood stagnation in the cardiac chambers
Surgical site bleeding	Endotoxins



PERI-PROCEDURAL COAGULOPATHY

1. Coagulation factors consumption (rarely a problem, thrombin generation is low, may be locally high…)

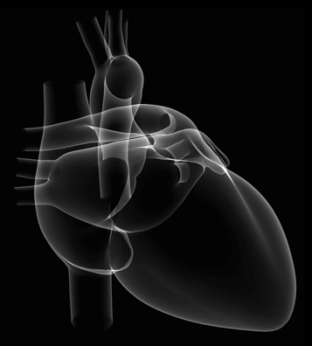
Therefore, very rarely there is the need for PCCs or rFVIIa..
FFP may be useful if volume replacement is needed.



PERI-PROCEDURAL COAGULOPATHY

2. Low levels of fibrinogen. Usually at the onset in post-cardiotomy ECMO; subsequently, fibrinogen levels rise, unless in case of continuous bleeding

Fibrinogen concentrate is the best source. Target levels unknown, possible 1.0-1.5 g/dL or 10 mm at FIBTEM

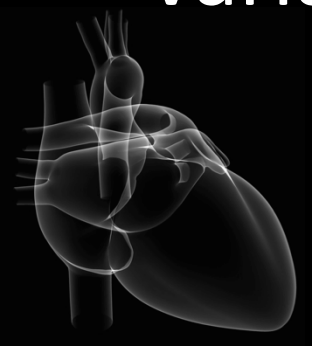


PERI-PROCEDURAL COAGULOPATHY

3. Low platelet count/function

Platelets are continuosly consumed and activated on ECMO. Heparin is bound by platelets. Therefore, the lower is the platelet count, the more UFH will work…

Platelet concentrates are the only option. Target values variable.

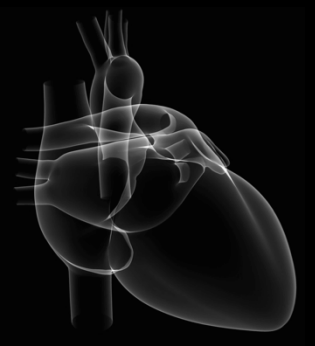


PERI-PROCEDURAL COAGULOPATHY

4. Low AT levels

AT is continuosly consumed during UFH anticoagulation on ECMO. The lower is the AT activity, the less UFH will work

Purified AT is the best source. Target value 70%?



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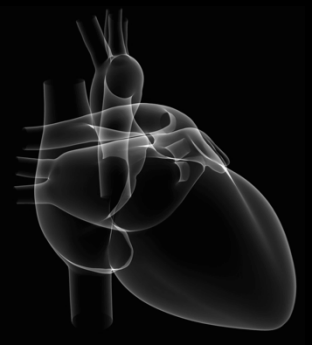


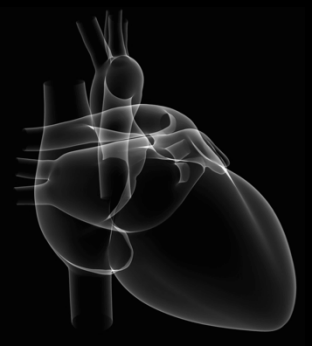
Shear Stress

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

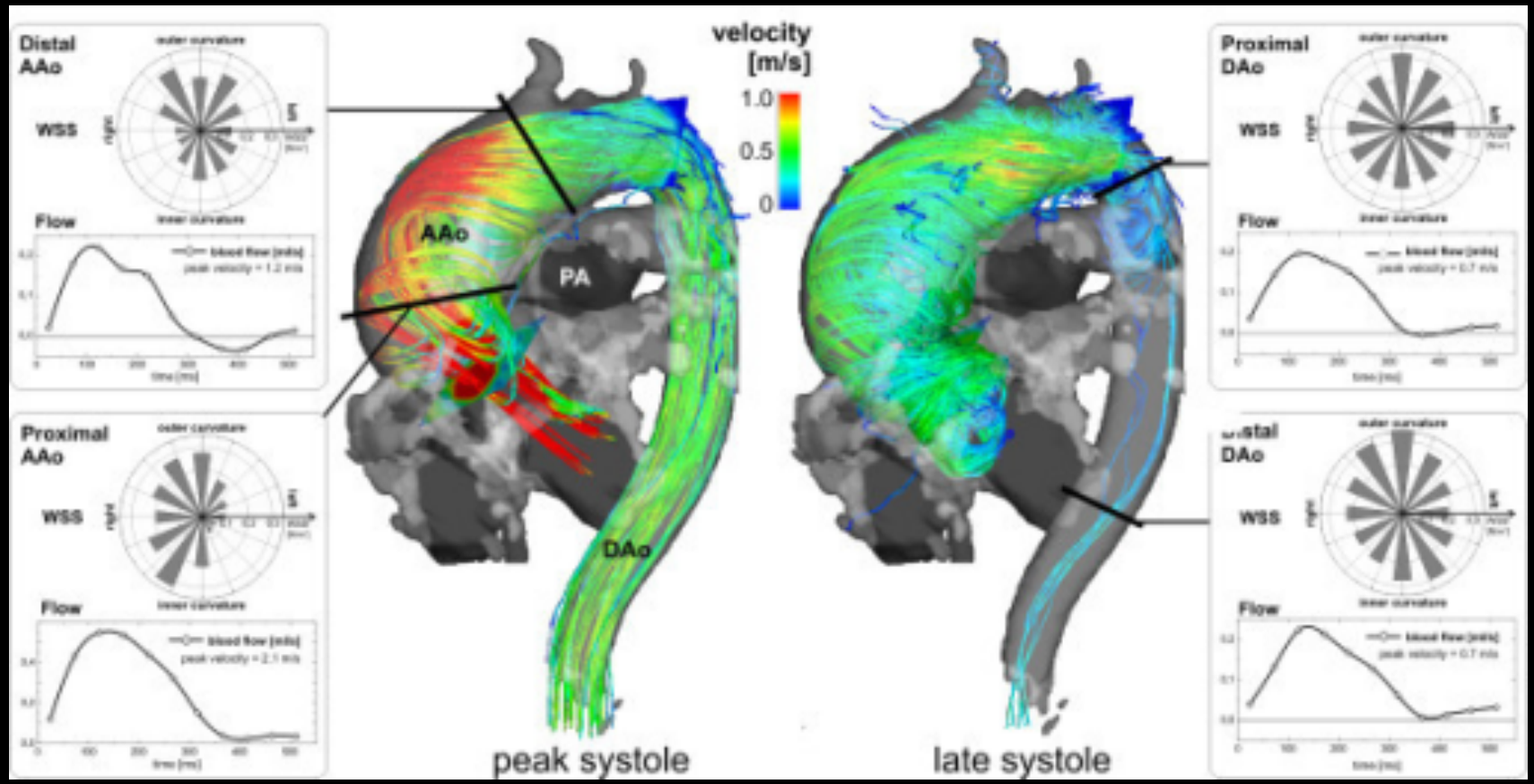
Acquired von Willebrand Syndrome in Aortic Stenosis

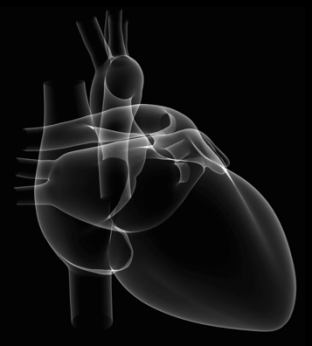
André Vincentelli, M.D., Sophie Susen, M.D., Thierry Le Tourneau, M.D., Ph.D., Isabelle Six, Ph.D., Olivier Fabre, M.D., Francis Juthier, Anne Bauters, Christophe Decoene, M.D., Jenny Goudemand, M.D., Ph.D., Alain Prat, M.D., and Brigitte Jude, M.D., Ph.D.



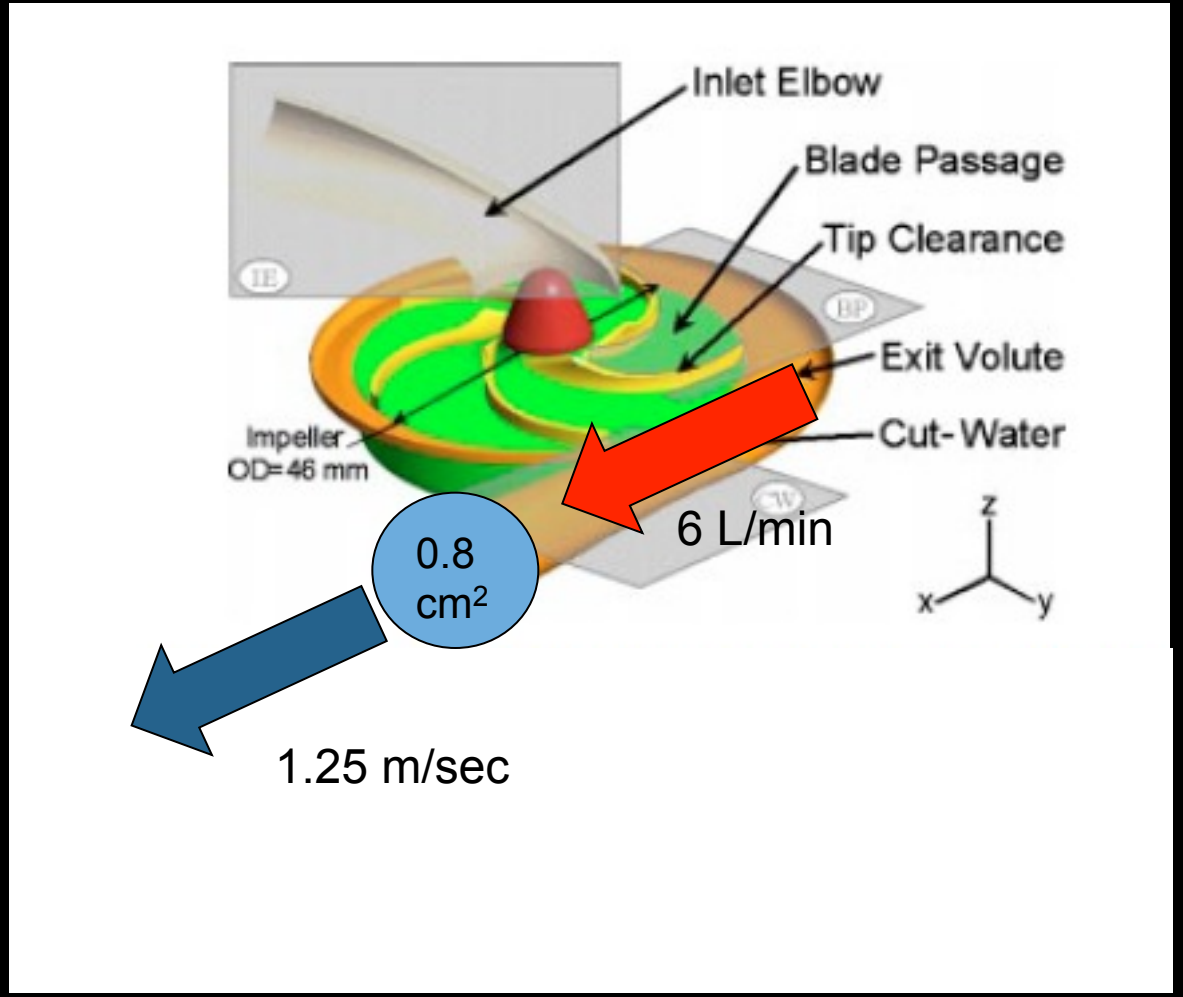
Vincentelli A et al; N Engl J Med. 2003 Jul 24;349(4):343-9

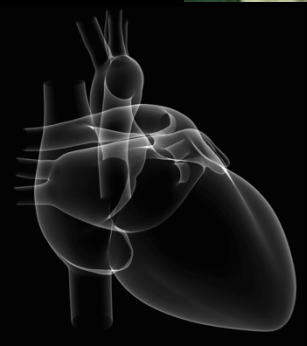
Shear Stress





ECMO, centrifugal pumps, and avWD



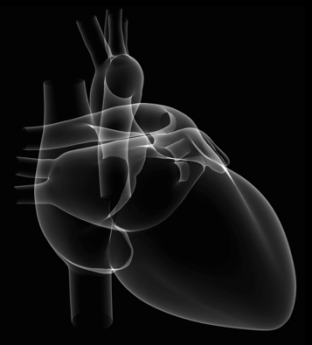


Intensive Care Med (2012) 38:62–68
DOI 10.1007/s00134-011-2370-6

ORIGINAL

Claudia Heilmann
Ulrich Geisen
Friedhelm Beyersdorf
Lea Nakamura
Christoph Benk
Georg Trummer
Michael Berchtold-Herz
Christian Schlensak
Barbara Zieger

Acquired von Willebrand syndrome in patients with extracorporeal life support (ECLS)



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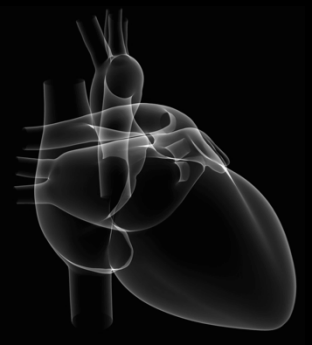


RESEARCH Open Access

Heparin-like effect in postcardiotomy extracorporeal membrane oxygenation patients

Marco Ranucci*, Ekaterina Baryshnikova, Giuseppe Isgrò, Concetta Carlucci, Mauro Cotza, Giovanni Carboni and Andrea Ballotta

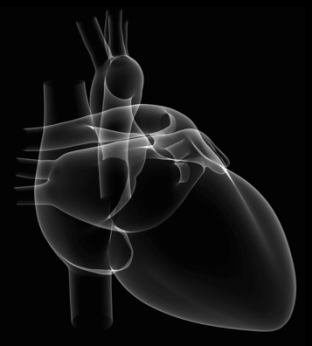
Abstract

Introduction: Unfractionated heparin (UFH) is the anticoagulant of choice for extracorporeal membrane oxygenation (ECMO), but bivalirudin can be used as an alternative. The purpose of the present study is to investigate the existence of a heparin-like effect (HLE) during heparin-free ECMO.

Methods: This is a retrospective study on patients treated with ECMO and receiving bivalirudin as the sole anticoagulant. Thromboelastography (TEG) tests with and without heparinase were recorded during the ECMO duration. A total of 41 patients (22 pediatrics and 19 adults) treated with ECMO after cardiac surgery procedures and receiving only bivalirudin-based anticoagulation were studied. Based on the presence of a different reaction time (R-time) between the TEG test with heparinase or without heparinase we defined the presence of a HLE. Survival to hospital discharge, liver failure, sepsis, bleeding and transfusion rate were analyzed for association with HLE with univariate tests.

Results: HLE was detected in 56.1% of the patients. R-times were significantly shorter in tests done with heparinase versus without heparinase during the first seven days on ECMO. Patients with HLE had a significantly ($P = 0.046$) higher rate of sepsis (30%) than patients without HLE (5.6%) at a Pearson's chi-square test.

Conclusions: A heparin-like effect is common during ECMO, and most likely due to a release of heparinoids from the glycocalyx and the mast cells, as a consequence of sepsis or of the systemic inflammatory reaction triggered by the contact of blood with foreign surfaces.



Ranucci et al. Critical Care 2014, 18:504

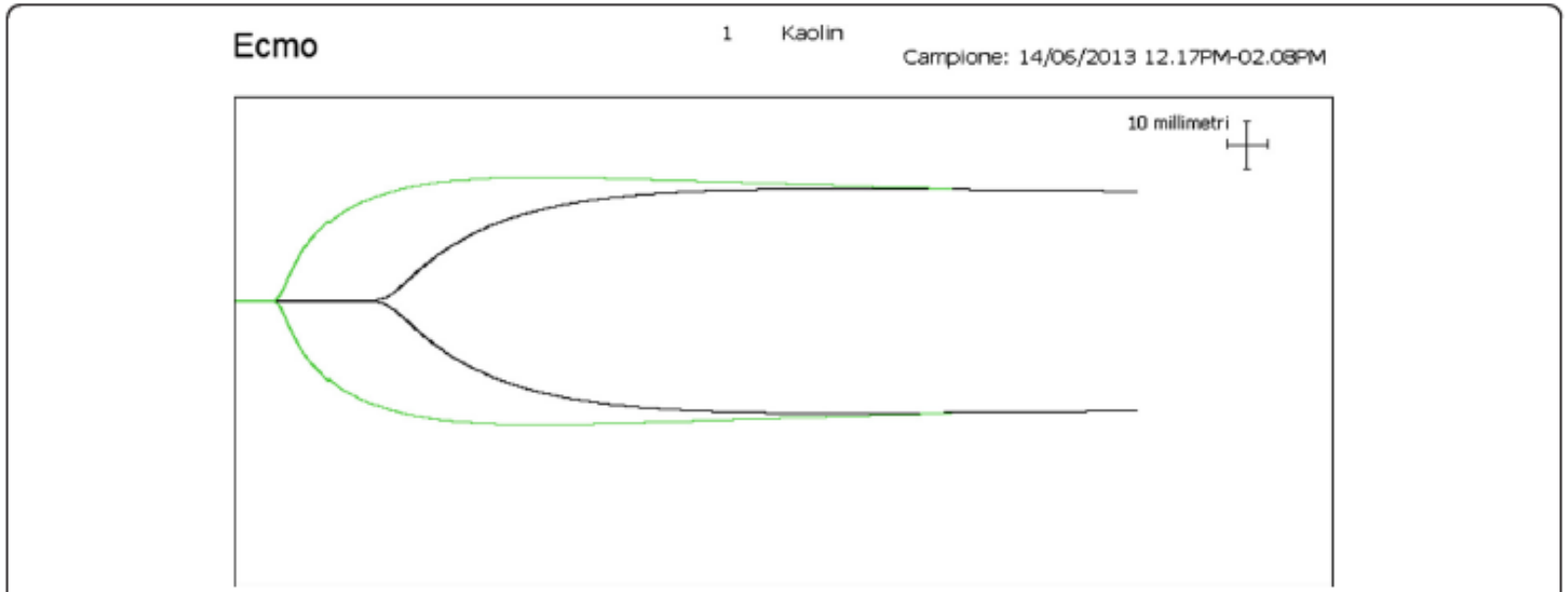
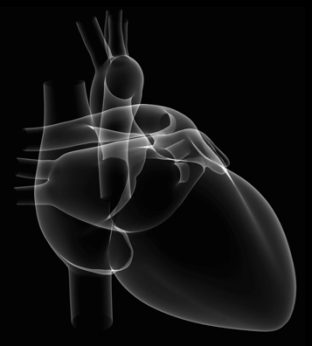


Figure 1 An example of heparin-like effect on thromboelastography (kaolin activation). Black tracing, test without heparinase; green tracing, test with heparinase. ECMO, extracorporeal membrane oxygenation.



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Key messages

- During ECMO without heparinization, the patients experience an HLE in about 50% of cases
- The HLE is evident during the first week on ECMO, and decreases with time after the seventh day on ECMO
- The HLE is not due to liver failure or heparin coating of the ECMO system
- There is an association between the HLE and the onset of sepsis, with a sepsis rate during ECMO of 30% in patients with an HLE, and of 6% in those without this phenomenon.

Ranucci et al. Critical Care 2014, 18:504

How to monitor?

HEPARIN-BASED ANTICOAGULATION
Dose range 20-70 IU/kg/min

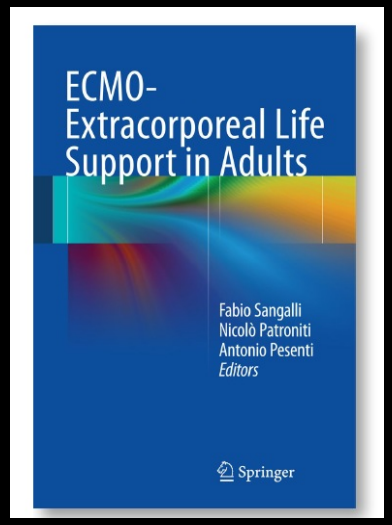
aPTT (50/70 sec) plus…

HEPARIN-BASED ANTICOAGULATION
Dose range 20-70 IU/kg/min

aPTT (50/70 sec) plus…

Table 2. The optimal hemostatic pattern for the ECMO patient

Parameter	Suggested value
Activated clotting time (seconds)	180 – 220
Anti Factor Xa activity	0.3 - 0.7 IU/ml
International normalized ratio	1.3 – 1.5
R – time at thromboelastography (seconds)	16 – 25
Fibrinogen (mg/dL)	> 100
Maximum clot firmness at FibTEM (mm)	> 10
Antithrombin activity (%)	70 – 80
Platelet count (cells/mmc)	> 80,000 (bleeding patients/high risk)
	> 45,000 (no bleeding / low risk)
D-dimers (µg/L)	< 300



Sangalli F et al; ECMO-Extracorporeal Life Support in Adults; Springer; 2014

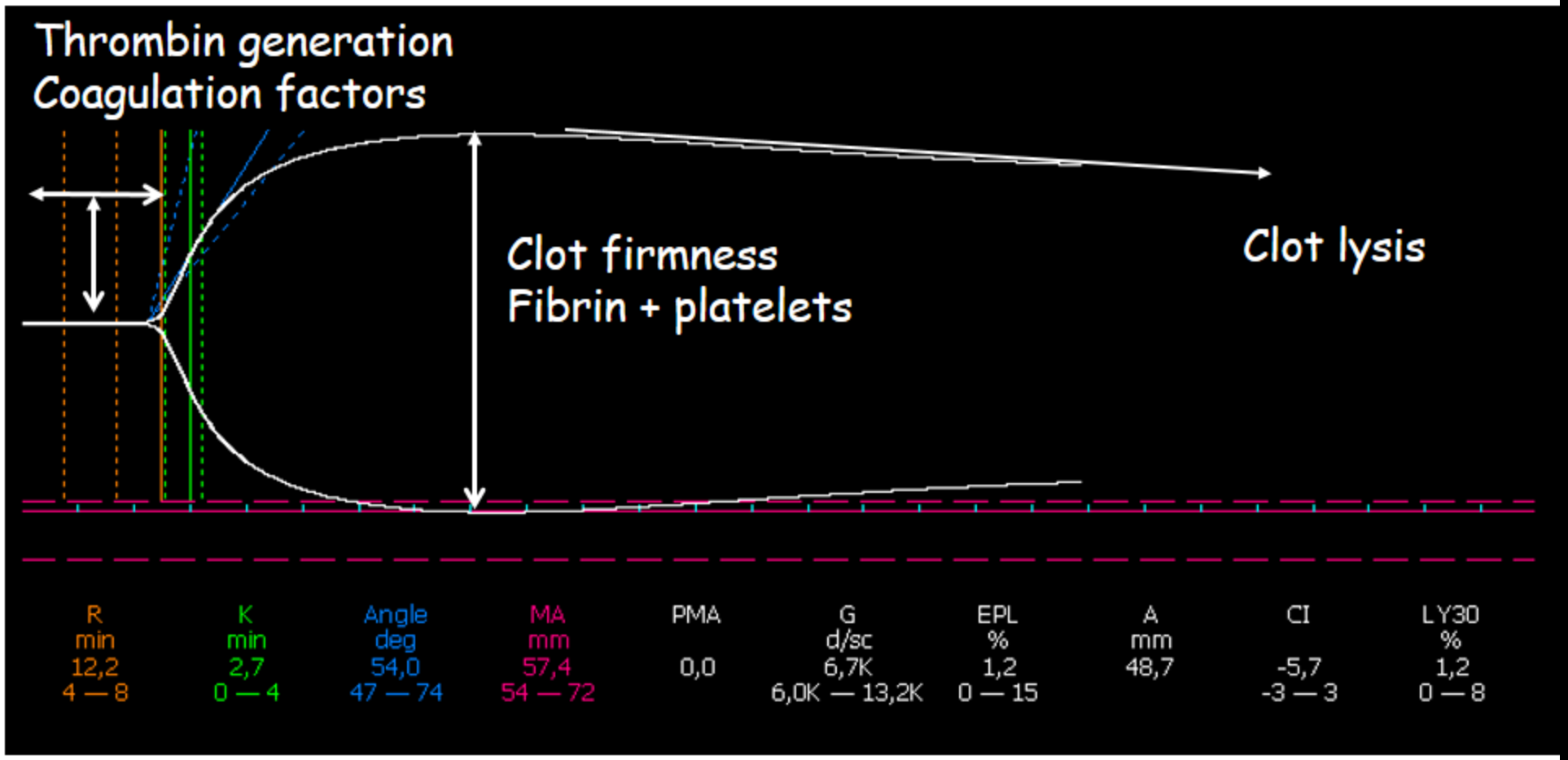


Figure 1. Thromboelastographic tracing

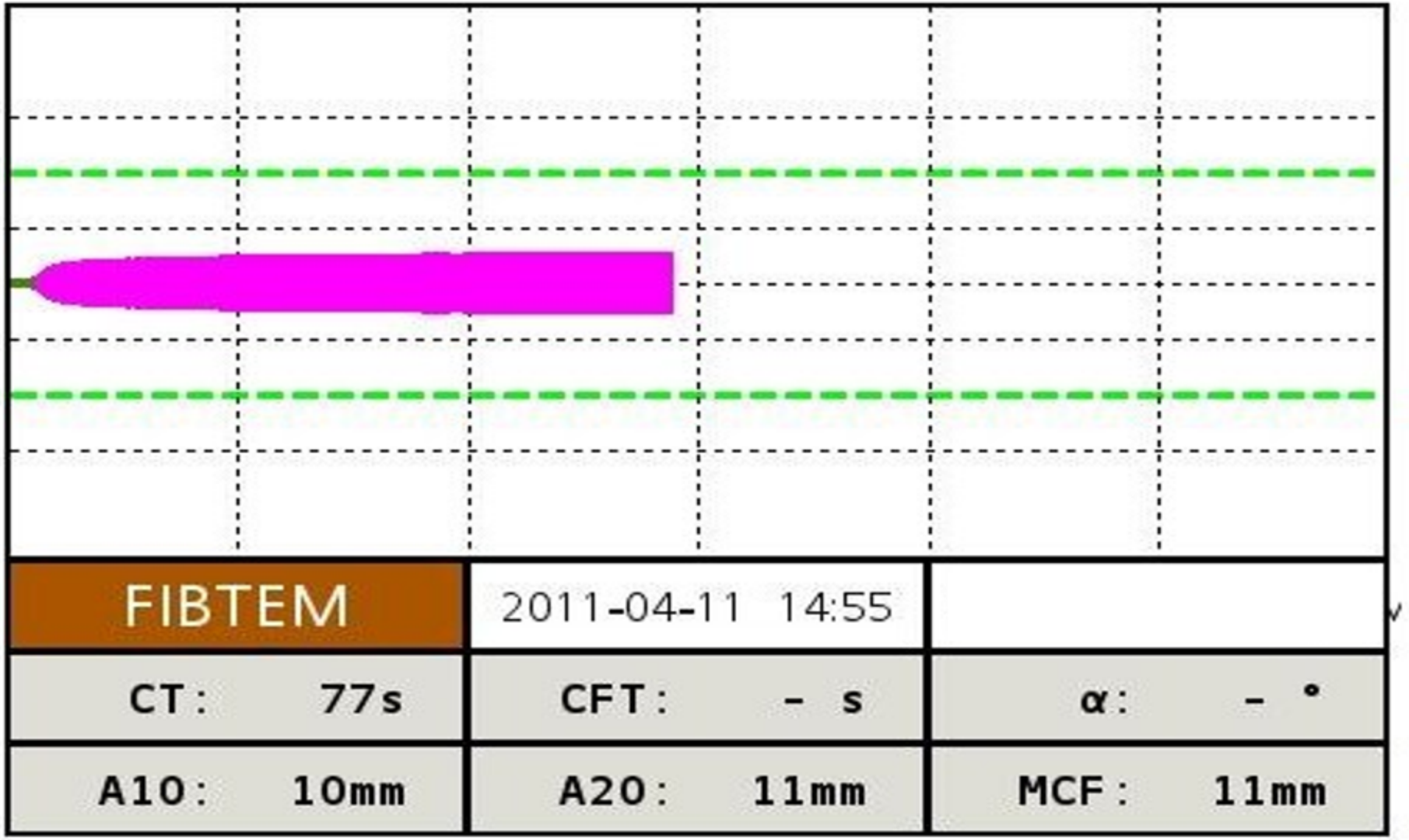
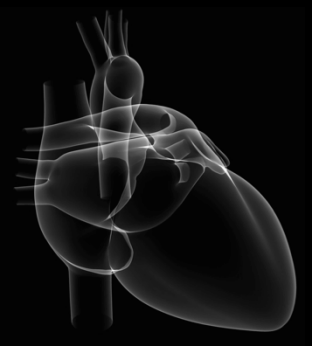


Figure 3. ROTEM analysis for fibrinogen concentration (FIBTEM)

Variability in anticoagulation management of patients on extracorporeal membrane oxygenation: an international survey

ATIII measurements (n=117 respondents)	Routinely	60 (51%)
	Occasionally	36 (31%)
	Never	21 (18%)
ATIII monitoring frequency (n=89 respondents)	Every 1–8 h	7 (8%)
	Every 9–12 h	17 (19%)
	Every 13–24 h	45 (51%)
	Only as needed	20 (22%)
Anti-factor Xa measurements (n=115 respondents)	Routinely	46 (40%)
	Occasionally	29 (25%)
	Never	40 (35%)
Anti-factor Xa monitoring frequency (n=66 respondents)	Every 1–8 h	15 (23%)
	Every 9–12 h	12 (18%)
	Every 13–24 h	27 (41%)
	Only as needed	12 (18%)
TEG measurements (n=116 respondents)	Routinely	21 (18%)
	Occasionally	29 (25%)
	Never	66 (57%)

* All parameters refer to a typical ECMO patient at the respondent's ECMO center; ACT: activated clotting time; SD: standard deviation; CBC: complete blood count; ECMO: extracorporeal membrane oxygenation; LDH: lactate dehydrogenase; AT: antithrombin; TEG: thromboelastogram

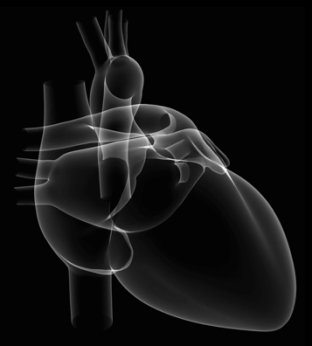


Bembea m ET AL; Pediatr Crit Care Med. 2013 February ; 14(2): e77

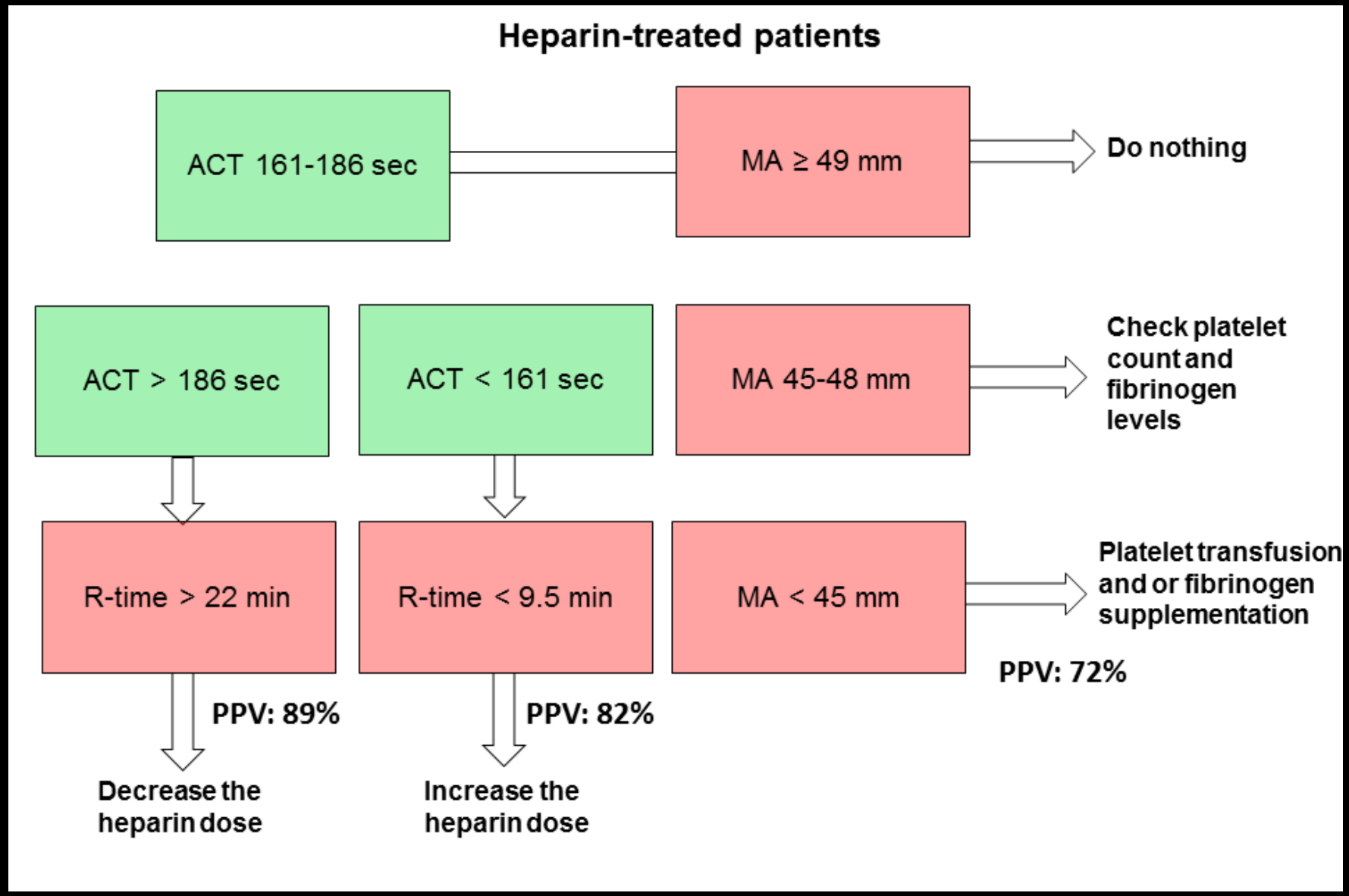
Table 2. Properties of the ACT for predicting an aPTT on target

Heparin-treated patients (N=29)					
ACT range (seconds)	Sensitivity	Specificity	PPV	NPV	P
161 – 186	51.5%	75.2%	61%	67.4%	0.001
R-time range (minutes)	Sensitivity	Specificity	PPV	NPV	P
9.5 – 22	54.3%	57.4%	46.3%	65%	0.254
Bivalirudin-treated patients (N=38)					
ACT range (seconds)	Sensitivity	Specificity	PPV	NPV	P
174 – 209	26.7%	77.6%	58.6%	47.2%	0.380
R-time range (minutes)	Sensitivity	Specificity	PPV	NPV	P
9.1 – 15.8	51.5%	65.8%	64.5%	52.9%	0.010

ACT: activated clotting time; aPTT: activated partial thromboplastin time; R: reaction



Ranucci et al, data under submission, 2015



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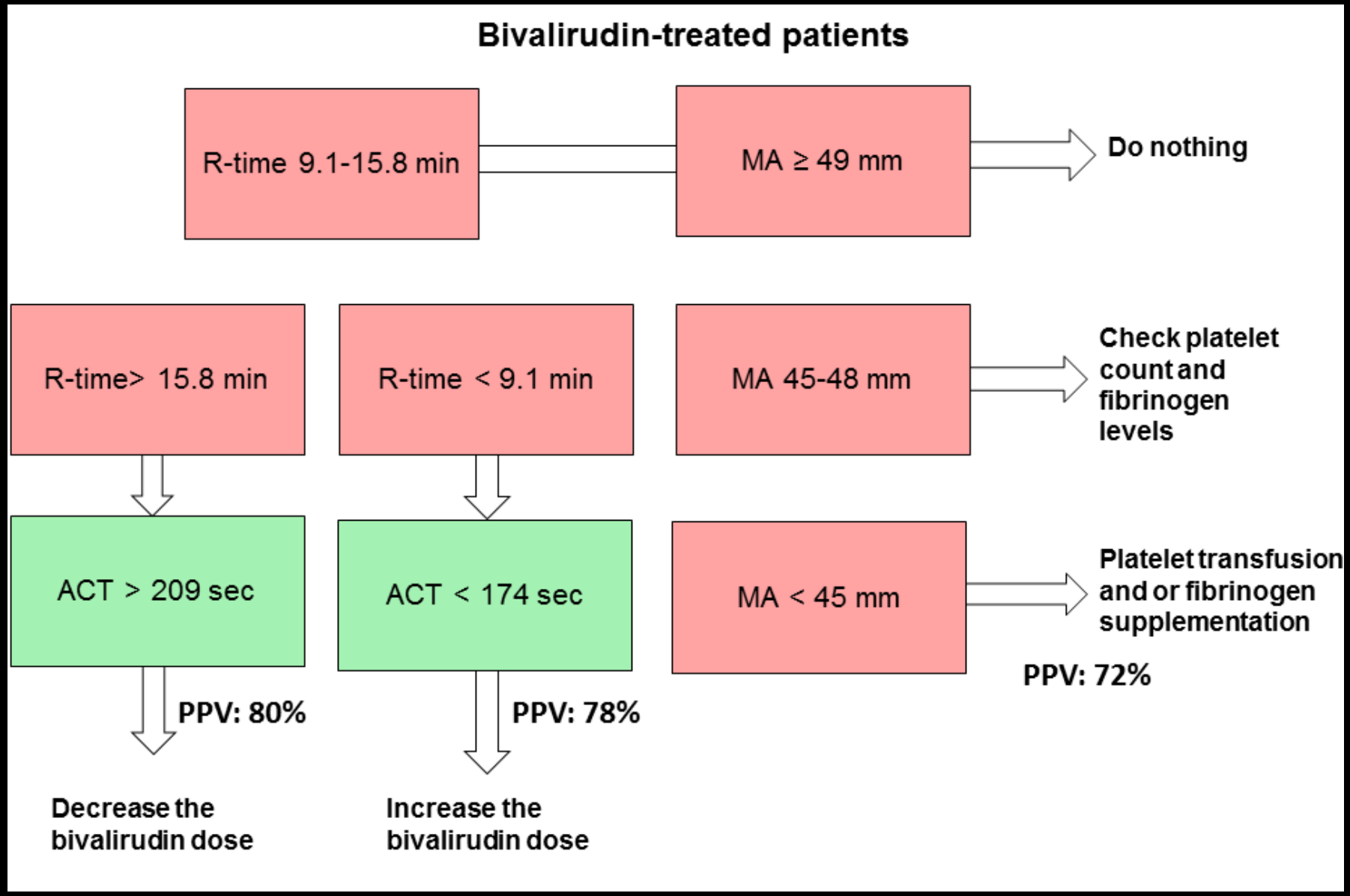

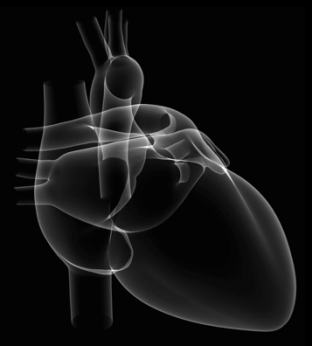

Ranucci et al, data under submission, 2015

CONCLUSIONS

1. There are very little topics that deserve more studies and knowledge than coagulation management on ECMO
2. Point-of-care monitoring tools are required
3. Alternatives to UFH should be tested in large series