Guidelines of platelets transfusion in anesthesia

Essay
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List Of Abbreviations

ACEI:	Angiotensin Converting Enzyme Inhibitors.
ADP:	Adenosine diphosphate.
AIDS:	Acquired Immun Deficiency syndrome.
ATP:	Adenosine triphosphate.
BC:	Buffy Coat.
CMV:	CytoMegalo Virus.
DDAVP:	1-Desamino-8-D-Arginine VasoPressin.
DIC:	Disseminated Intravascular Coagulopathy.
DVT:	Deep Venous Thrombosis.
FFP:	Fresh Frozen Plasma.
FNHTR:	Febrile Non-Hemolytic Transfusion Reactions.
GP:	Glycoprotein.
GVHD:	Graft Versus Host Disease.
HIT:	Heparin Induced Thrombocytopenia.
HITT:	Heparin Induced Thrombocytopenia and Thrombosis.
HIV:	Human Immunodeficiency Virus.
HLA:	Human Leucocytic Antigen.
HNA:	Human Neutrophil Antibodies.
HPA:	Human Platelet Antigen.
HUS:	Hemolytic Uremic Syndrome.
Ig:	Immunoglbulin .
IL:	Interlukins.
ITP:	Idiopathic Thrombocytopenic Purpura.

List Of Abbreviations (Cont...)

IVIG:	IntraVenous ImmunoGlobulin.
LDH:	Lactate DeHydrogenase.
LTA:	optical Light Transmission Aggregometry.
NSAIDs:	Non Steroidal Anti-Inflammatory Drugs.
P_2:	Purinergic receptors.
PAS:	Platelet Additive Solution.
PC:	Platelet Concentrates.
PE:	Pulmonary Embolism.
PG:	Prostaglandin.
PPP:	Platelets Poor Plasma.
PRP:	Platelets Rich Plasma.
PTP:	Post Transfusion Purpura.
ROTEM:	Rotational Thromboelastography.
TA-GVHD:	Transfusion Associated Graft Versus Host Disease.
TEG:	Thromboelastography.
TNF:	Tumor Necrosis Factor.
TRALI:	Transfusion related Acute Lung Injury.
TTP:	Thrombotic Thrombocytopenic Purpura.
$TXA_{2:}$	Thromboxane A_2.
vWD:	von Willebrand Disease.
vWF:	von Willebrand Factor.
WHO:	World Health Organization.

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Objectives:

The aim of this essay is to discuss the perioperative indications and complications of platelets transfusion. Also, to review different preparations of platelets .

Introduction

Human blood consists of three components which are red blood cells (erythrocytes), white blood cells (leukocytes) and platelets (thrombocytes). Every cell has its own importance. Platelets play a very important role in healing process and the formation of blood clots at the time of injury. They are microscopic irregular shaped cells without a nucleus. These are derived from the bone marrow cells known as megakaryocytes. A normal platelet count range varies from 150,000 to 400,000 per µl (microlitre) in a mammal. Even a slight increase or decrease in this range can result in dangerous consequences ***(Sánchez et al., 2007).***

The haemostatic system consists of platelets, coagulation factors, and the endothelial cells lining the blood vessels. Under normal circumstances, the resistance of the endothelial cell lining to interactions with platelets and coagulation factors prevents thrombosis. When endothelial continuity is disrupted and the underlying matrix is exposed, a coordinated series of events are set in motion to seal the defect (primary hemostasis). Platelets play a primary role in this process, interacting with subendothelium-bound von Willebrand factor (vWf) via the membrane glycoprotein (GP) Ib complex. This initial interaction (platelet adhesion) sets the stage for other adhesive reactions that allow the platelets to interact with each other to

form an aggregate. When platelets are activated, negatively charged phospholipids move from the inner to the outer leaflet of the membrane bilayer. This negative surface provides binding sites for enzymes and cofactors of the coagulation system, resulting in the formation of a clot (secondary hemostasis) ***(Lassila et al., 2012).***

Hence, primary hemostatic disorders are characterized by prolonged bleeding time, and the characteristic physical examination findings are petechiae and purpura. In comparison, defects in secondary hemostasis result in delayed deep bleeding (eg, into muscles and joints) and the characteristic physical examination finding is hemarthrosis. Hemarthrosis and muscle hematomas are not present in primary hemostatic disorders. Platelets disorders may be due to disorders in platelets number or disorders in platelets function ***(Van Veen et al., 2010).***

Aspirin and clopidogrel therapy have become standard therapies for preventing recurrent atherothrombotic events and for preventing primary events in many high-risk patients. The two drugs affect platelet function via different mechanisms. Aspirin reduces thromboxane-mediated platelet aggregation, while clopidogrel partially prevents ADP-induced platelet activation. Because patients exhibit substantial variability in their response to these therapeutics, laboratory measurement of platelet function may some day play a role in successful patient

management. These laboratory tests include Optical Light Transmission Aggregometry**,** Cartridge-based Platelet Aggregation**,** in vitro Bleeding Time **or** Thromboelastography ***(Madsen et al., 2010).***

Platelet transfusions play an important role in the prevention of bleeding in severely thrombocytopenic patients. It may be used to stop bleeding due to thrombocytopenia and in patients with severely impaired platelet function ***(Boekhorst et al., 2005).***

Like other blood components, platelet transfusions have achieved a high degree of safety as far as transmission of viral diseases is concerned. However, transfusion of platelet concentrates is accompanied by a high frequency of febrile and anaphylactoid reactions. In rare cases, recipients of platelet concentrates are threatened by severe reactions as septic complications due to bacterial contamination of platelet concentrates, transfusion-related acute lung injury and severe anaphylactic episodes ***(Ahrens et al., 2008).***

Platelets are either isolated from collected units of whole blood and pooled to make a therapeutic dose or collected by apheresis, sometimes concurrently with plasma or red blood cells. The industry standard is for platelets to be tested for bacteria before transfusion to avoid septic reactions, which can be fatal ***(Stroncek and Rebulla, 2007).***

Pooled random donor platelet concentrates are prepared from platelets that have been harvested by centrifuging units of whole blood. Up to 8 units of platelets, each from a separate donor, can be pooled into a single bag for transfusion. All units are from the same ABO type. If ABO compatible platelets are unavailable, ABO incompatible platelets can be substituted with very little risk ***(Stroncek and Rebulla, 2007)***.

Apheresis platelets are collected from a single donor and are equivalent to ~4-6 pooled units. An apheresis platelet concentrate contains 200-400mL of plasma. They may be collected as a random unit (random apheresis platelets) or be obtained for a specific recipient from a family member or a volunteer HLA compatible "directed" donor ***(Vamvakas, 2009).***

Functions of platelets

I. ***Formation of Platelets*:**

The fragmentation of bone marrow cells known as megakaryocytes produces platelets. Every single megakaryocyte cell produces 5,000 to 10,000 platelets. Platelets have a life span of 5 to 9 days and a normal human adult produces $1x10^{11}$ platelets everyday. The extra or reserve platelets are stored in the spleen and are released at the time of need. Phagocytes and Kupffer cells destroy the old platelets hence, giving chance to the new ones to take their place (Figure 1) ***(O'Connell et al., 2008).***

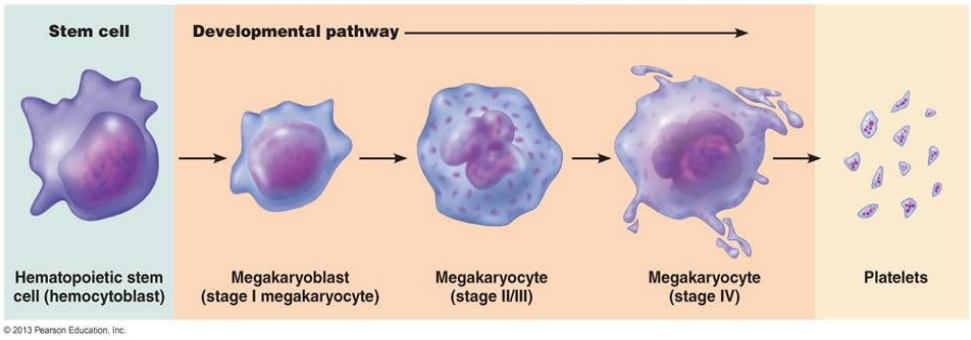

Figure (2): Steps of platelets formation **(O'Connell *et al.*, 2008).**

II. Normal Platelet Count:

Normally, a person's platelet count is 150,000 to 400,000 per µl (microlitre). The normal platelet count range in children is between 150,000-450,000 per µl ***(Van Veen et al., 2010).***

There is a slight decrease in the platelet count in pregnancy as there is an increase in the blood produced while the number of platelets remain the same. It is quite normal till 100,000 platelets per µl but if the number goes below this, then it can be a medical emergency. Some women may experience a slight downfall in the platelet count before menstruation ***(Van Veen et al., 2010).***

III. Functions of Platelets :

1) Platelets interact with the coagulation factors in a complex way to arrest bleeding or generate thrombi (Hemostasis).

2) Platelets cause leukocytes to accumulate around the platelet plug; that is, they may release chemotactic substances.

3) Platelets release vasoactive amines to regulate vascular tone.

4) Platelets may release hydrolytic and proteolytic enzymes directly into the intimal and subintimal structures provoking changes that may eventually lead to atheroma.

5) Platelets act to transport serotonin from sites of synthesis (the gut) to other sites of function (Figure 2).

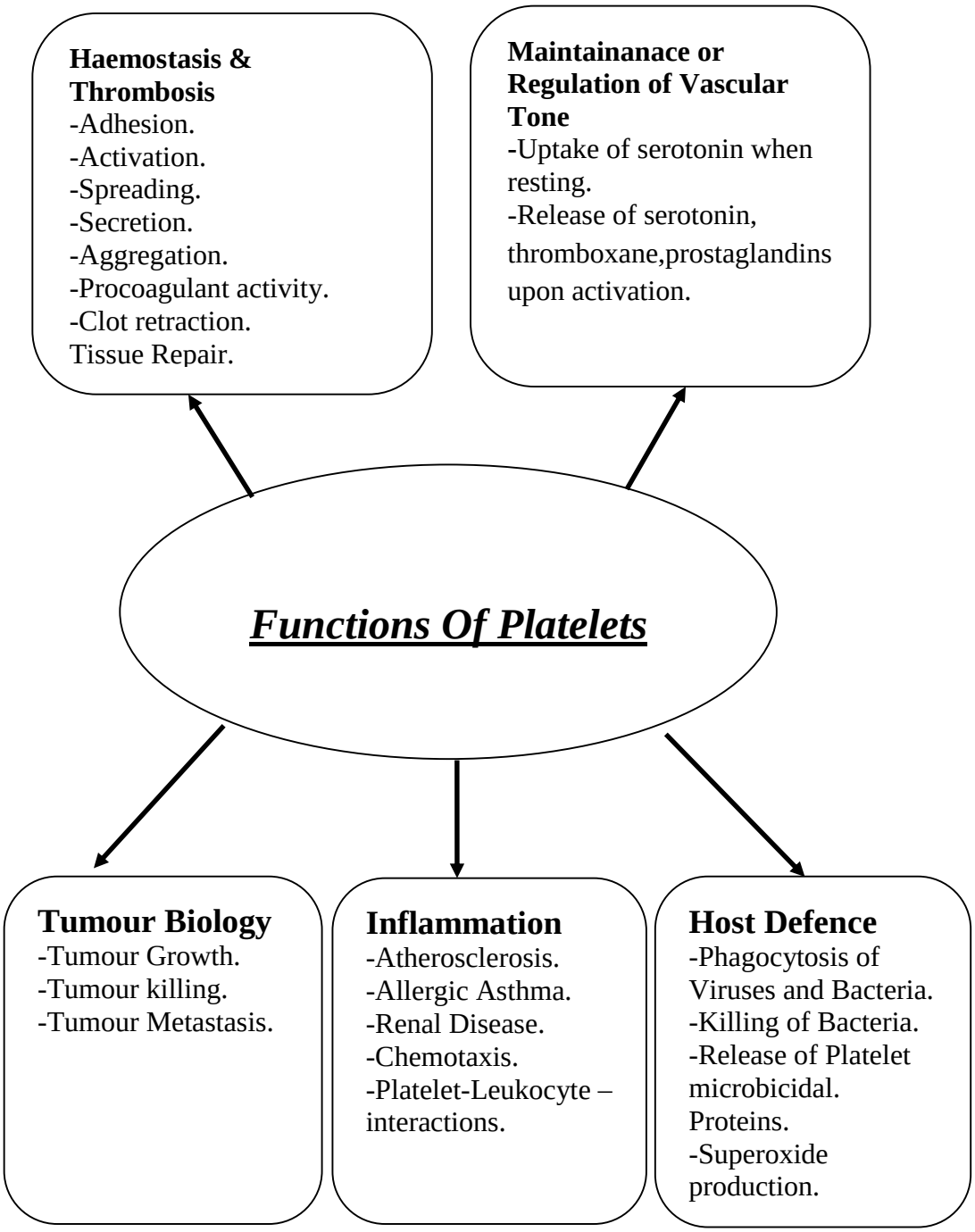

Figure (2): Functions of platelets ***(McAleer et al., 2006).***