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plasmapheresis in pediatrics

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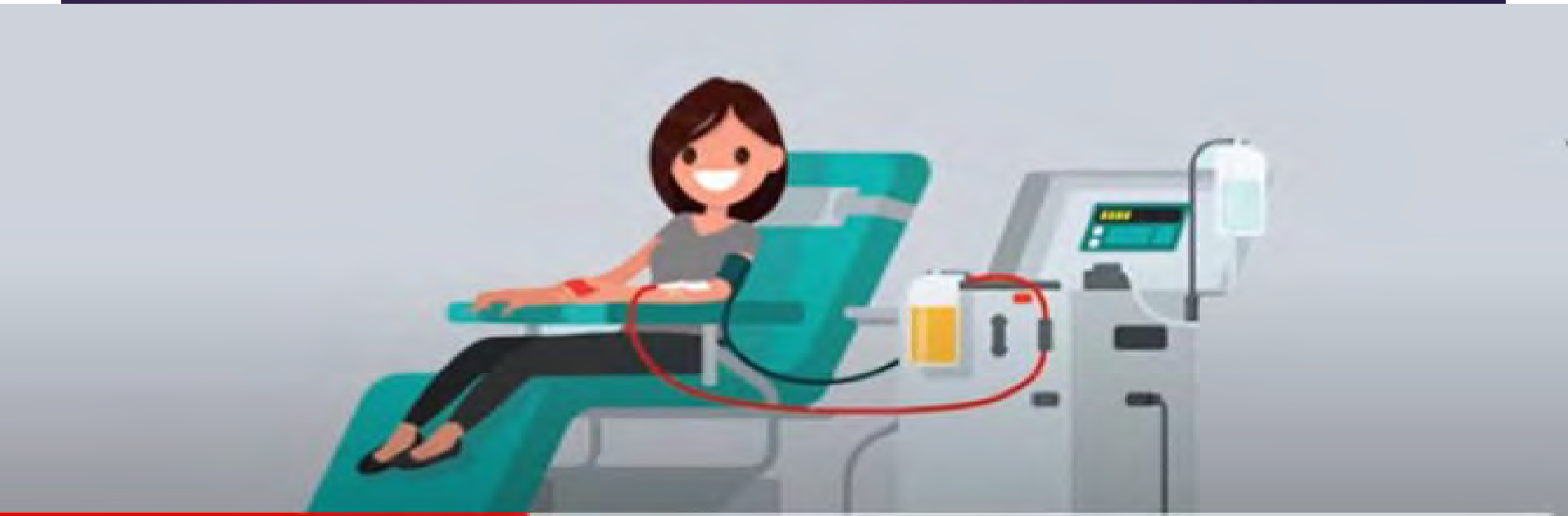
Assuit University



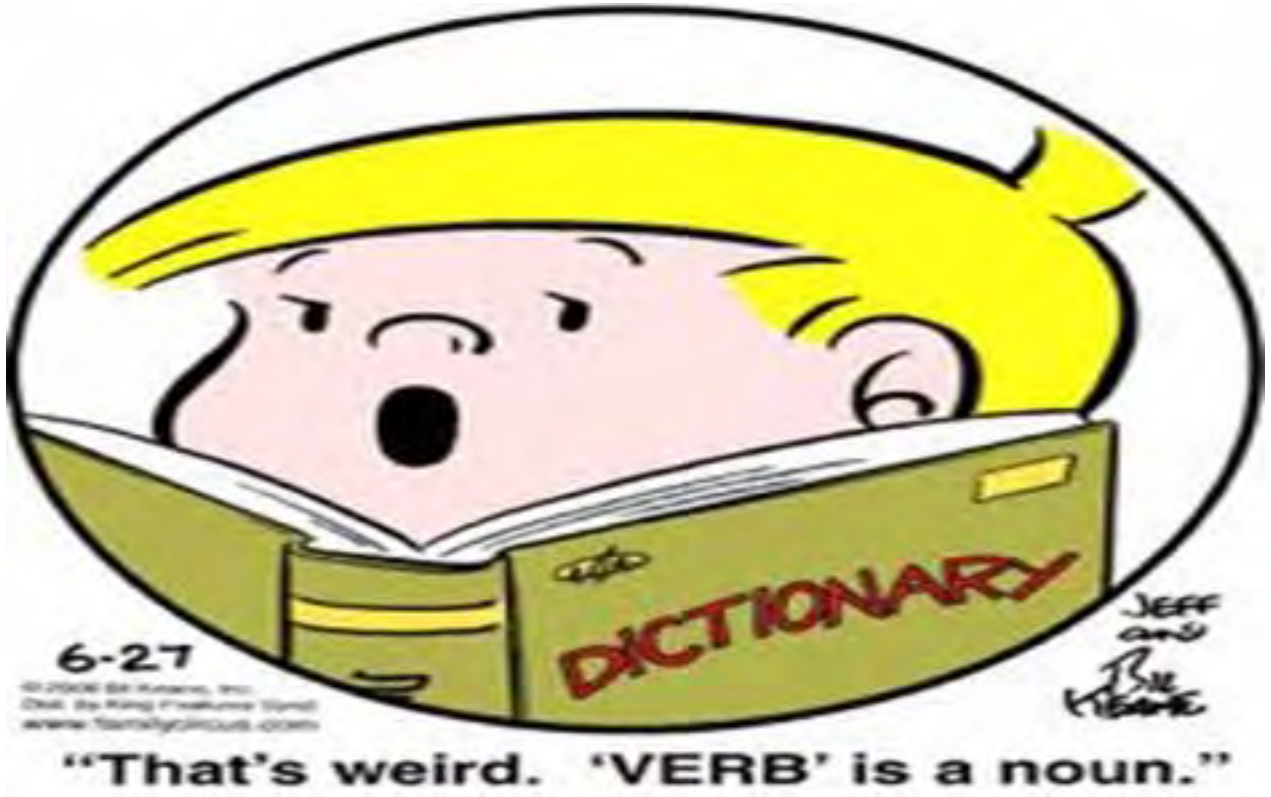
Agenda

1. Introduction
2. Indication of therapeutic plasma exchange in children
3. When? , how ? and how long?
4. Technical aspects of pediatric plasma exchange
5. complication

Introduction

Apheresis is derived from a Greek word meaning to take away by force.

- Donation
- Therapeutic



Historical background

- It is unclear when the notion of therapeutic removal of blood components first origined , but it was flourishing even before Hippocrates in the fifth century BC
- Bloodletting to remove evil “humors” was common place medical practice

Edwin papyrus

Therapeutic apheresis

- Plasmapheresis
- Cytaphersis
  - Leucopheresis (lymphocytapheresis)
  - Plateletpheresis
  - RBCs(Exchange transfusion)

Apheresis in Clinical Practice

- **Removal Of Antibodies**
  - -TTP
  - -Guillain Barre Syn.
  - -Myasthenia Gravis
  - -Goodpasture's Syn.
  - -Antibody mediated graft rejection
  - -Cryoglobulinemia
  - -Immune complexes; SLE
- **Removal Of Excessive Or Abnormal Substances**
  - -Paraproteinemia (Waldenstrom's M.)
  - -Cholesterol in Hypercholesterolemia
- **Removal Of Toxins**

Padmanabhan A, Connelly-Smith L, Aqui N, Balogun RA, Klingel R, Meyer E, Pham HP, Schneiderman J, Witt V, Wu Y, Zantek ND, Dunbar NM, Schwartz GEJ. Guidelines on the Use of Therapeutic

PLASMA REMOVAL WITH RETURN OF CORPUSCLES (PLASMAPHAERESIS)

FIRST PAPER

JOHN J. ABEL, L. G. ROWNTREE, and B. B. TURNER

Journal of Pharmacology and Experimental Therapeutics July 1914, 5 (6) 625-641;

- John Jacob Abel in 1914, who showed, together with his team, that large amounts of plasma could be extracted periodically from dogs as long as the red blood cells were re infused

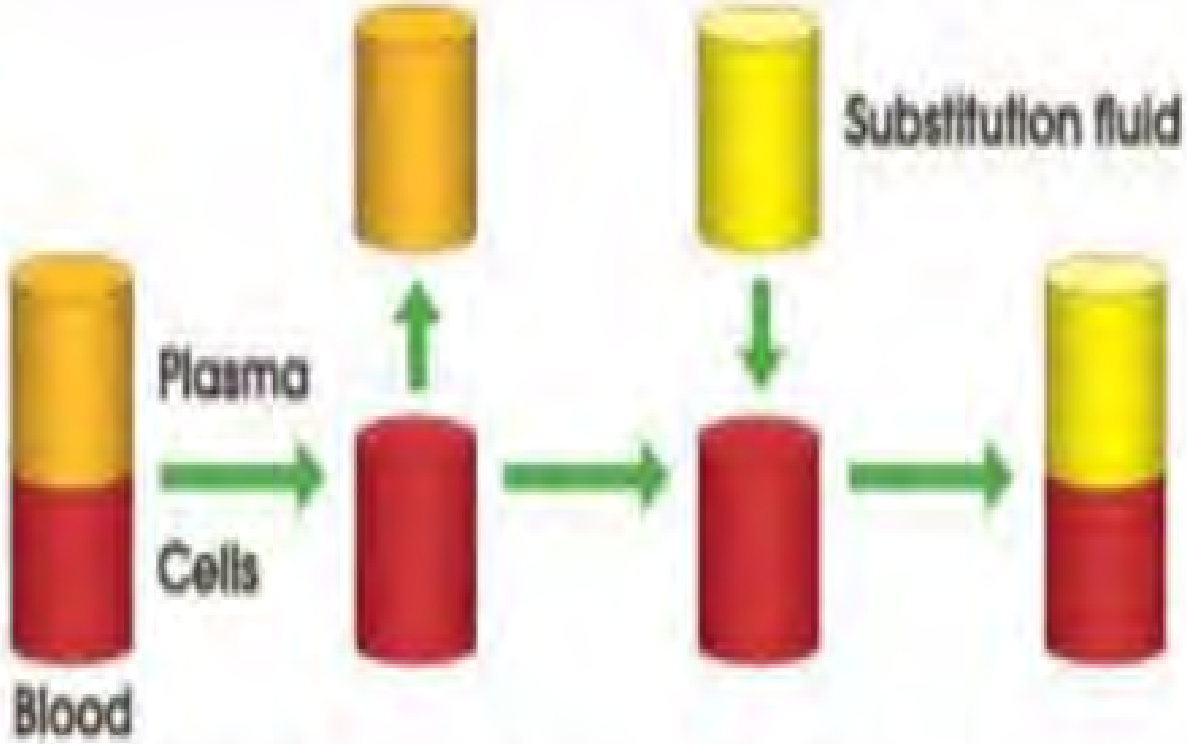

Therapeutic plasma exchange (TPE)
Plasma pharesis

plasma pharesis

- is a procedure in which plasma of patient or donner is removed without the use of colloid replacement solution.
- less than 15% of total plasma volume

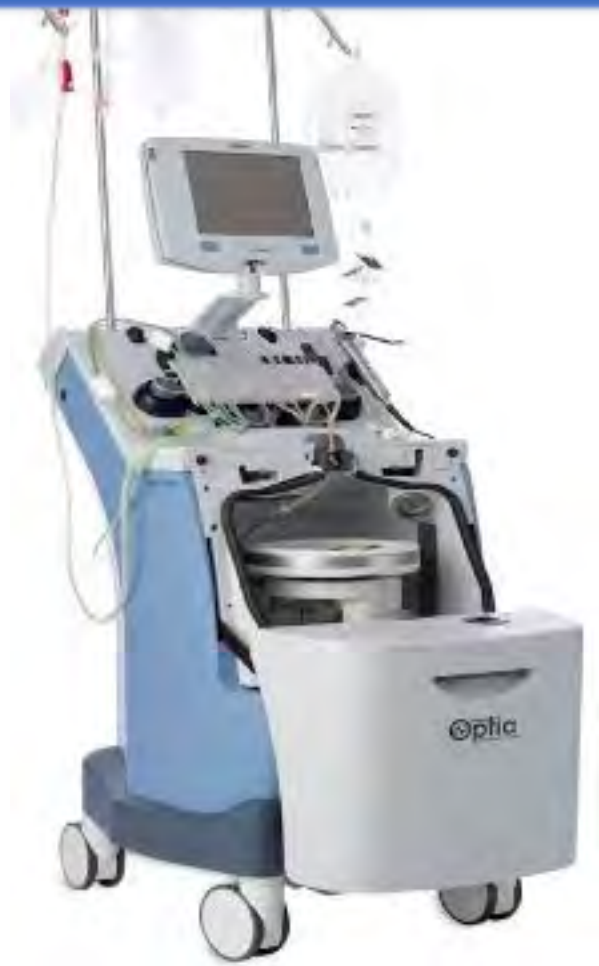
Therapeutic plasma exchange (TPE)

- A therapeutic procedure in which plasma is removed and replaced with a replacement solution such as colloid solution (e.g., albumin and/or plasma) or a combination of crystalloid/colloid solution.



Plasmapheresis is a process in which plasma is separated from blood cells and then plasma is replaced with another solution like albumin or FFP

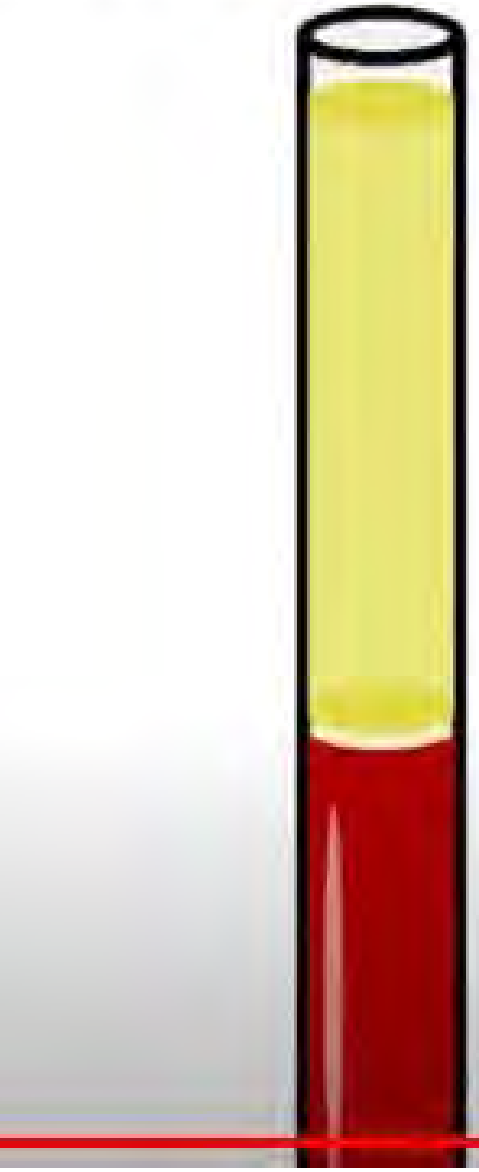
Methods of therapeutic plasma exchange



2 types of device:

- One that separates the plasma from the cellular components based on size (Filtration-based apheresis), and
- One that separates components based on density (Centrifugation-based apheresis)

www.iplasma.life



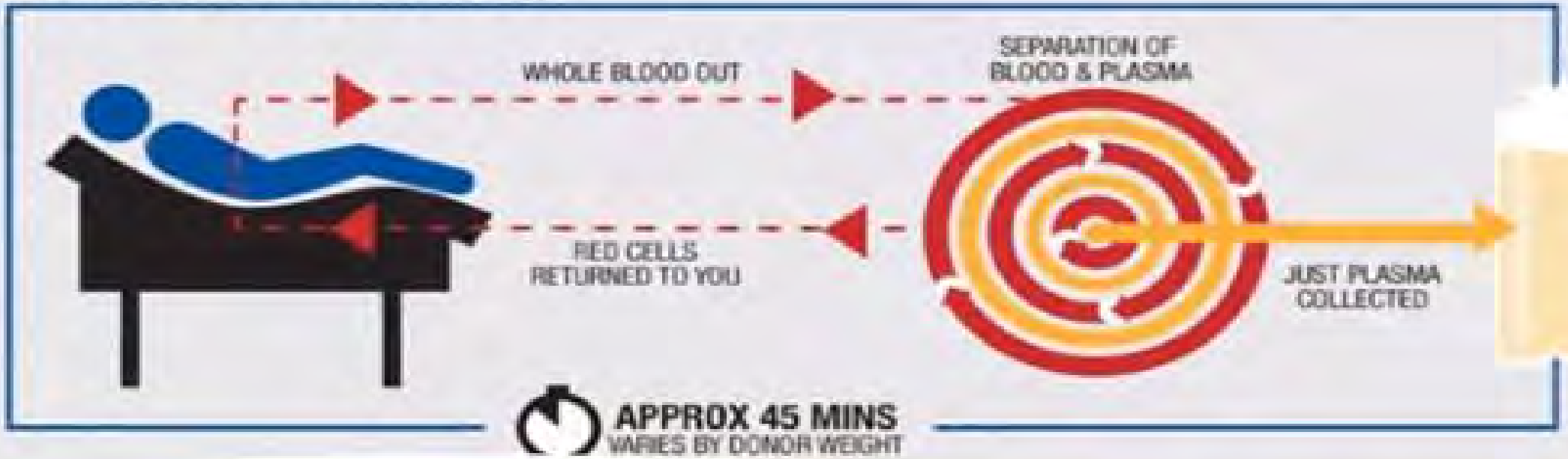
55%

PLA

45%

RED BLOOD CELL,
WHITE BLOOD CELL
AND PLATELETS

PLASMAPHERESIS



BUN	Creatinine	VitB12	β2-microglobulin	κ Light Chain	λ Light Chain	Albumin	IgG	IgM
0.06	0.113	1.355	11.8	25	50	66	160	950

Small Molecules

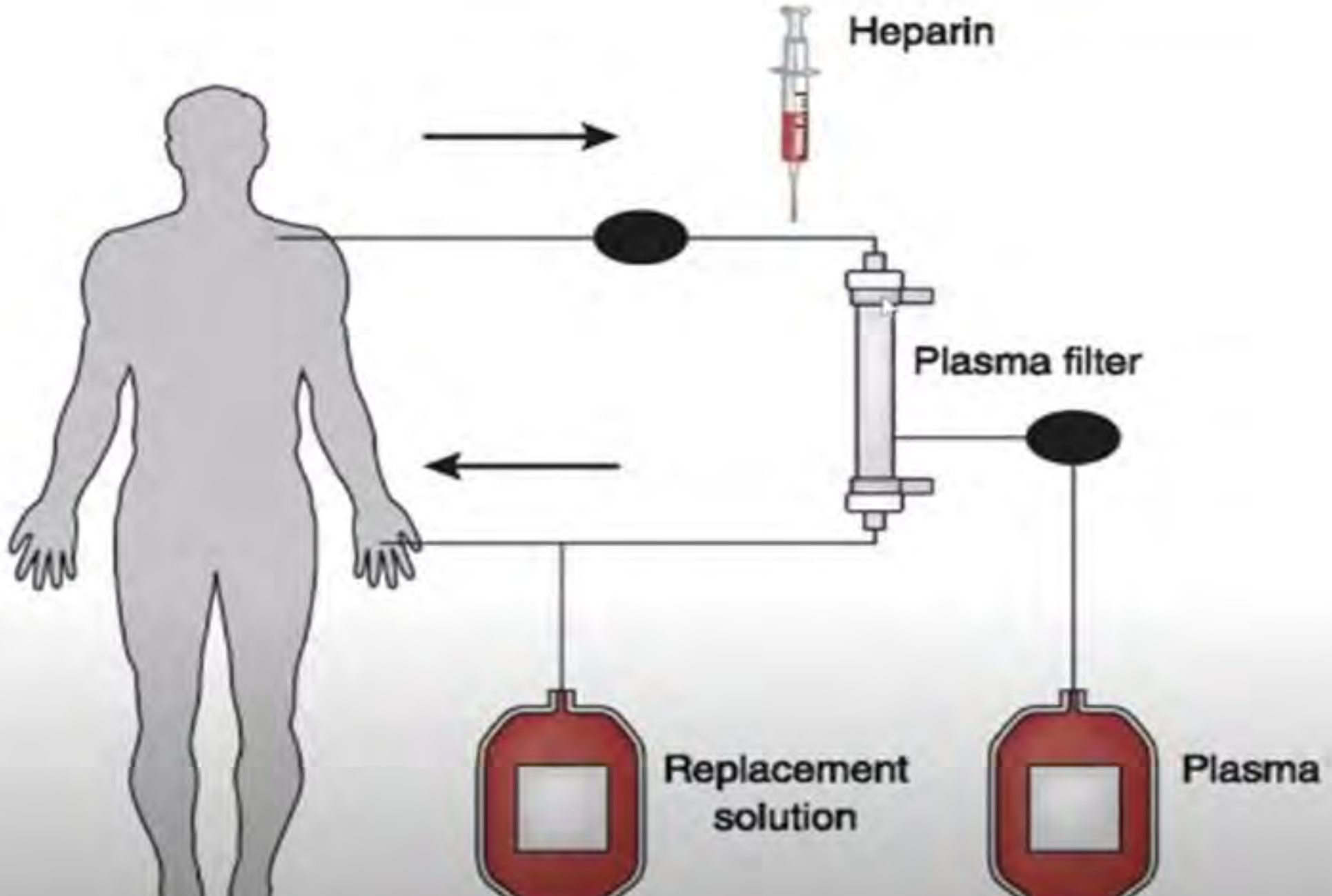
Middle Molecules

Large Molecules

Hemodialysis: Diffusion Clearance

Hemofiltration: Convective Clearance

Therapeutic Plasma Exchange




System parameters
Dialysis representation
Cum. Blood Vol.
3500
128
25.0

Technical considerations

Membrane apheresis:

Advantage:

- Fast and efficient plasmapheresis
- No citrate requirement
- Can be adapted for cascade filtration

Disadvantage:

- Removal of substance limited by sieving coefficient of membrane
- Unable to perform cytapheresis
- Requires high blood flows, central venous access
- Requires heparin anticoagulation limiting use in bleeding disorders


when
How
For
How
long

Therapeutic plasma exchange (TPE)

- In clinical practice, there are two rationales for using TPE:

1) to remove an inciting circulating pathogenic molecule (antibody, immune complex, toxin, etc.)
2) to replace a deficient factor, as in systemic thrombotic microangiopathy.

Indication of therapeutic plasma exchange in children

- **Renal diseases**
  - **Renal transplant conditions**
    - ABO-incompatible kidney transplant
    - Pretransplant desensitization
    - Antibody-mediated rejection
    - Recurrent focal segmental glomerulosclerosis

- **Immune-mediated glomerular disease**
  - Anti-glomerular basement membrane disease
  - ANCA-associated RPGN
  - IgA nephropathy
  - Henoch–Schonlein purpura nephritis
  - Other immune-mediated glomerulonephritis
  - Cryoglobulinemia

- **Others**
  - Atypical hemolytic uremic syndrome
  - Thrombotic thrombocytopenic purpura
  - Sepsis with multiorgan failure
  - Myeloma cast nephropathy

- **Hematologic**
  - ABO-incompatible stem cell transplant
  - Autoimmune hemolytic anemia (Cold agglutinin)
  - Catastrophic antiphospholipid antibody syndrome
  - Aplastic anemia (pure red cell aplasia)
  - Hyperviscosity in monoclonal gammopathies
  - Post-transfusion purpura

Neurologic

- Acute disseminated encephalomyelitis
- Guillain–Barre syndrome
- Myasthenia gravis
- PANDAS, Sydenham's chorea
- Chronic focal encephalitis
- Multiple sclerosis
- Lamber–Eaton myasthenic syndrome
- Lupus cerebritis
- Neuromyelitis optica

Metabolic

- Familial hypercholesterolemia (homozygotes, small blood volume)
- Mushroom poisoning
- Refsum's disease
- Wilson's disease, fulminant

Table 2.

Indications for therapeutic apheresis in diseases involved kidney and their pathogenic factors.

Medical disciplines	Diseases	Pathogenic factors
Primary kidney diseases	FSGS	Circulatory permeability factors
	MN	PLA2R Ab and THSD7A Ab
	Anti-GBM glomerulonephritis (Goodpasture's syndrome)	Anti-GBM Ab
Secondary kidney diseases	ANCA-associated vessel vasculitis	Anti-MPO or anti-PR3 Ab
	TTP	ADAMTS-13 Ab, ICs
	aHUS	Complement regulatory components or autoantibodies
	SLE	Anti-dsDNA Ab, anti-nuclear Ab, ICs
KT	ABO-incompatible KT	Blood group isoagglutinins
	HLA-incompatible KT	HLA and non-HLA alloantibodies
	Ab-mediated allograft rejection	HLA and non-HLA alloantibodies

Ask A Question

DEALING WITH DIFFICULT PEOPLE

J Clin Apher. 2023 Apr;38(2):77-278. doi: 10.1002/jca.22043.

Guidelines on the Use of Therapeutic Apheresis in Clinical Practice - Evidence-Based Approach from the Writing Committee of the American Society for Apheresis: The Ninth Special Issue

Laura Connelly-Smith [1], Caroline R Alquist [2], Nicole A Aqui [3], Jan C Hofmann [4], Reinhard Klingel [5 6], Oluwatoyosi A Onwuemene [7], Christopher J Patriquin [8], Huy P Pham [9], Amber P Sanchez [10], Jennifer Schneiderman [11], Volker Witt [12], Nicole D Zantek [13], Nancy M Dunbar [14]

Affiliations + expand

PMID: 37017433 DOI: 10.1002/jca.22043

The American Society for Apheresis (ASFA) Categories

Category	Description
I	Disorders for which apheresis is accepted as **first-line therapy**, either as a primary standalone treatment or in conjunction with other modes of treatment.
II	Disorders for which apheresis is accepted as **second-line therapy**, either as a standalone treatment or in conjunction with other modes of treatment.
III	Optimum role of apheresis therapy is **not established**. Decision making should be individualized.
IV	Disorders in which published evidence demonstrates or suggests apheresis to be **ineffective** or **harmful**.

Table 3. Therapeutic apheresis for the treatment of kidney diseases: recommendation grades and indication categories in 2019 American Society for Apheresis guidelines [1].ABO incompatibleII

Disease	Indication	Apheresis	Category	Recommendation grade	Technical notes
FSGS	Recurrent in KT	PE/IAS	I	Grade 1B	Volume treated: TPE, LA, or IA with single use adsorbers: 1.0–1.5 TPV; IA with regenerative adsorbers: 2–3 TPV.Frequency: Daily or every other day at initiation of treatment. Subsequent frequency and duration based on patient response.
	Recurrent in KT/Steroid resistant in native kidney	LDL-A	II	Grade 2C	
	Steroid resistant in native kidney	PE	III	Grade 2C	
Anti-GBM glomerulonephritis	DAH	PE	I	Grade 1C	Volume treated: 1–1.5 TPVFrequency: daily or every other day for 14 days or until anti-GBM undetectable
	Dialysis-independence	PE	I	Grade 1B	
	Dialysis-dependence (Cr > 5.7mg/dl)	PE	III	Grade 2B	
ANCA-associated disease	MPA/GPA/RLV				Volume treated: 1–1.5 TPVFrequency: daily in DAH, typically every other day in absence of DAH
	RPGN, Cr ≥ 5.7mg/dl	PE	II	Grade 1B	
	RPGN, Cr < 5.7 mg/dl	PE	IIII	Grade 2C	
	DAH	PE	I	Grade 1C	
	EGPA	PE	III	Grade 2C	
SLE	Severe complications	PE	II	Grade 2C	Volume treated: 1–1.5 TPVFrequency: LN or DAH: daily or every other day; Other severe complications: 1–3 times per week. Typically course of 3–6 PE is enough to see response
TMA	TTP	PE	I	Grade 1A	Volume treated: 1–1.5 TPVFrequency: daily until platelets >150K and LDH near normal for 2–3 consecutive days, taper vs abrupt discontinuation practices vary
	STEC-HUS	PE/IAS	III	Grade 2C	Volume treated: 1–1.5 TPVFrequency: daily until improvement, no standardized approach exists
	aHUS				Volume treated: 1–1.5 TPVFrequency: daily until clinical response (complement mediated), daily or every other day for coagulation mediated TMA
	Factor H autoantibody	PE	I	Grade 2C	
	CF gene mutations	PE	III	Grade 2C	
KT					
ABO incompatible	Desensitization	PE/IAS	I	Grade 1B	Volume treated: 1 - 1.5 TPV Frequency: daily or every other day. antibody titer is less than critical threshold prior to before KT
	AMR	PE/IAS	II	Grade 1B	
ABO compatible	Desensitization	PE/IAS	I	Grade 1B	Volume treated: 1–1.5 TPVFrequency: usually 5 or 6, daily or every other day
	AMR	PE/IAS	I	Grade 1B	

Table 2 Summary of renal indications and recommended treatment for plasmapheresis in children

Diagnosis	ASFA category	Pathogenic molecule	Prescription			Notes	Selected reports on pediatric patient population
			Treatment volume and replacement fluid	Frequency	Duration/endpoint		
ABO-incompatible renal transplantation	II	Anti-A or Anti-B endothelial oligosaccharide antibodies	1–1.5 plasma volume 5 % Albumin, plasma (compatible with recipient and donor)	Daily or every other day for 2–5 days prior to transplant	Reduce IgM or IgG antibody titers ≤4 Usually 2–5 treatments	After transplant: consider daily TPE followed by IVIG for 2-3 days Follow daily ABO antibody levels after transplant ×2 weeks	[20, 21]
Desensitization before renal transplantation Living donor positive cross-match	II	Donor-specific HLA antibodies	1–1.5 plasma volume 5 % Albumin Plasma if coagulopathy, preoperative	Preop: Daily or every other day until negative cross-match	Follow donor-specific antibodies to determine further treatments Minimum 3 treatments after surgery		[10]

Table 2 (continued)

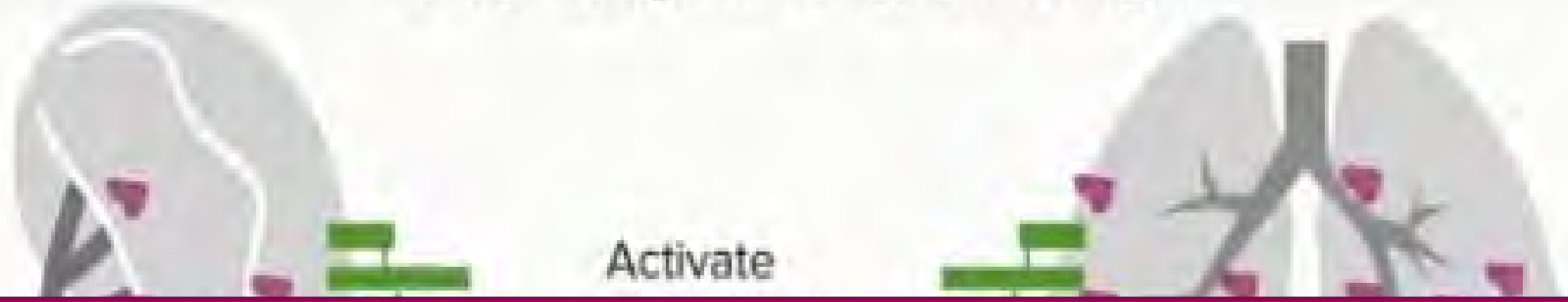
Diagnosis	ASFA category	Pathogenic molecule	Prescription			Notes	Selected reports on pediatric patient population
			Treatment volume and replacement fluid	Frequency	Duration/endpoint		
Atypical hemolytic uremic syndrome			1–1.5 plasma volume	Daily	Daily ×5 days, then 5 days/week ×2 weeks, then 3x/week ×2 weeks, taper as tolerated		[84, 90, 94]
Factor H antibody	I	Factor H antibody	5 % Albumin Plasma				
Complement mutations	II	Replace deficient factors, remove defective factors					
Thrombotic thrombocytopenic purpura	I	Replace deficient ADAMTS 13, ADAMTS 13 Antibody	1–1.5 plasma volume Plasma or cryo poor plasma	Daily	Daily until platelet count >150,000/L ×2 days, then taper		

TPE, Therapeutic plasma exchange; IVIG, Intravenous immunoglobulin; HLA, human leukocyte antigen; ASFA, American society for apheresis; IgM, Immunoglobulin M; IgG, Immunoglobulin G; ANCA, Anti-neutrophil cytoplasmic antibodies

plasmapheresis

Autoantibodies bind to glomerular/alveolar basement membrane

Recognize α_3 chain of type IV collagen



Given this situation, the 2021 KDIGO guidelines recommend that "glucocorticoids and plasmapheresis should be initiated as soon as possible (within 24 h)" even before the return of anti-GBM antibody data" when the disease is suspected

- it should be continued daily until the anti-GBM antibody disappear from the serum.
- It takes about 1 month for anti-GBM antibodies to disappear from the serum, and during this period, even if PEX is performed, the anti-GBM antibody increase the next day.
- Anti-GBM disease is usually" a one-hit phenomenon", and once the anti-GBM antibody has disappeared from the serum, it does not rise again.

Category description and recommendation grade of systemic vasculitidesin ASFA guideline [2, 3]

Disease	Indication	Modality	Category	Grade
Medium vessel vasculitis, MVV				
Polyarteritis nodosa, PAN		TPE	IV	1B
Small vessel vasculitis, SVV				
ANCA-associated vasculitis, AAV[a]				
Microscopic polyangiitis, MPA AAV	MPA/GPA/RLV: RPGN, Cr ≥ 5.7 mg/dl[b]	TPE	II	1B
Granulomatosis with polyangiitis (Wegener's), GPA	MPA/GPA/RLV: RPGN, Cr < 5.7 mg/dl[b]		III	2C
	MPA/GPA/RLV: DAH		I	1C

Ther Apher Dial. 2022 Jun; 26(3): 493–506.
Published online 2022 Mar 16. doi: 10.1111/1744-9987.13829
PMCID: PMC9311821
PMID: 35247230

Plasmapheresis for systemic vasculitis

Kazuhito Fukuoka,[✉1] Mitsumasa Kishimoto,[1] Takahisa Kawakami,[1] Yosinori Komagata,[1] and Shinya Kaname[1]

Associated Data

▸ **Data Availability Statement**

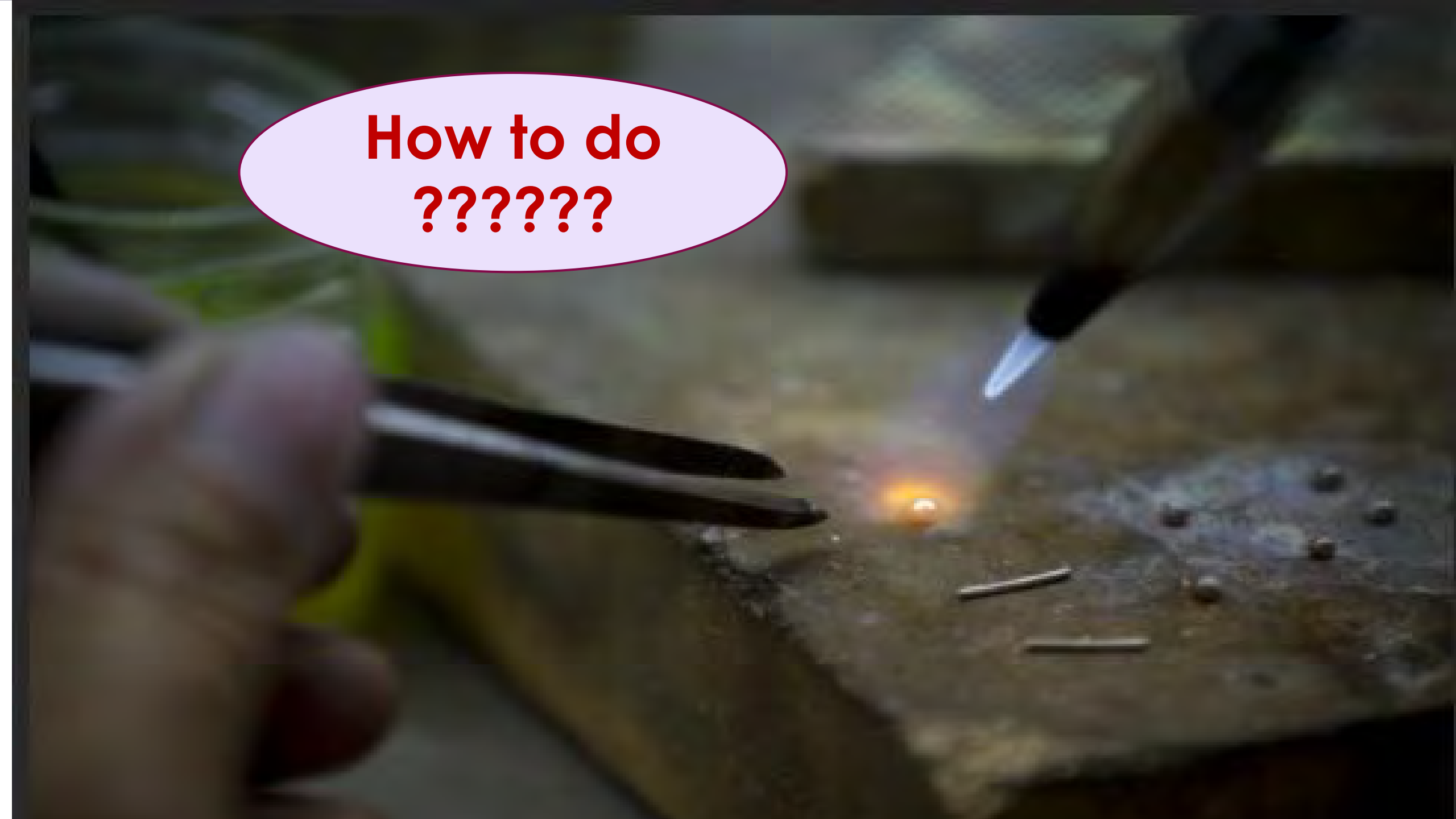
plasma exchange therapy (PEX) has been frequently used and is expected to be effective in some diseases, most of which are included in small vessel vasculitides. In particular, data showing efficacy have been accumulated for immune complex vasculitis, and the recommendation seems to be high. For instance, anti-GBM nephritis, concomitant use of PEX is essential and strongly recommended. On the other hand, for ANCA-related vasculitis among small vessel vasculitis, RCTs have recently shown negative results. In particular, the PEXIVAS trial statistically showed that PEX has no potential to reduce the mortality and renal death in AAV, but the ASFA, ACR, and KDIGO guidelines following this trial all regard PEX as salvage therapy or selective treatment for severe cases. As plasmapheresis is often performed in combination with other therapies, it is difficult to evaluate to clarify its efficacy on its own, and this predisposition may be pronounced in vasculitis, a rare disease. Although statistically significant differences are not apparent, the diseases that show a trend toward efficacy may possibly include treatment-sensitive subgroups. Further analysis is expected in the future.

TABLE 1 Category and Grade Recommendations for Therapeutic Apheresis

Disease	TA modality	Indication	Category	Grade	Page
Acute disseminated encephalomyelitis (ADEM)	TPE	Steroid Refractory	II	2C	187
Acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barré syndrome)	TPE	Primary Treatment	I	1A	189
	IA	Primary Treatment	I	1B	

Indications for Therapeutic Plasma Exchange and Its Related Outcomes

ASFA Categorization	Number of Patients	Survivors, n (%)	Diagnosis	Number of Patients, n (%)	Survival Rate, n (%)	*P*
I	13	10 (76)	Autoimmune encephalitis, other encephalitis (Hashimoto), refractory epilepsy	8 (9.5)	5 (62.5)	.020[b]
			HUS	3 (3.6)	3 (100)	
GBS	2 (2.4)	2 (100)				
II	5	4 (80)	Mushroom poisoning	3 (3.6)	3 (100)	
			AIHA	1 (1.2)	1 (100)	
SLE	1 (1.2)	0 (0)				
III	65	27 (41.4)	MOF + TAMOF (including sepsis or sepsis + MOF)	34 (40.4)	12 (35.2)	
			Liver Failure + hepatic encephalopathy	24 (28.5)	13 (54.1)	
			HLH-MAS	2 (2.4)	0 (0)	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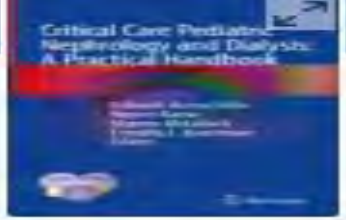


Plasmapheresis in Pediatric Renal Disease

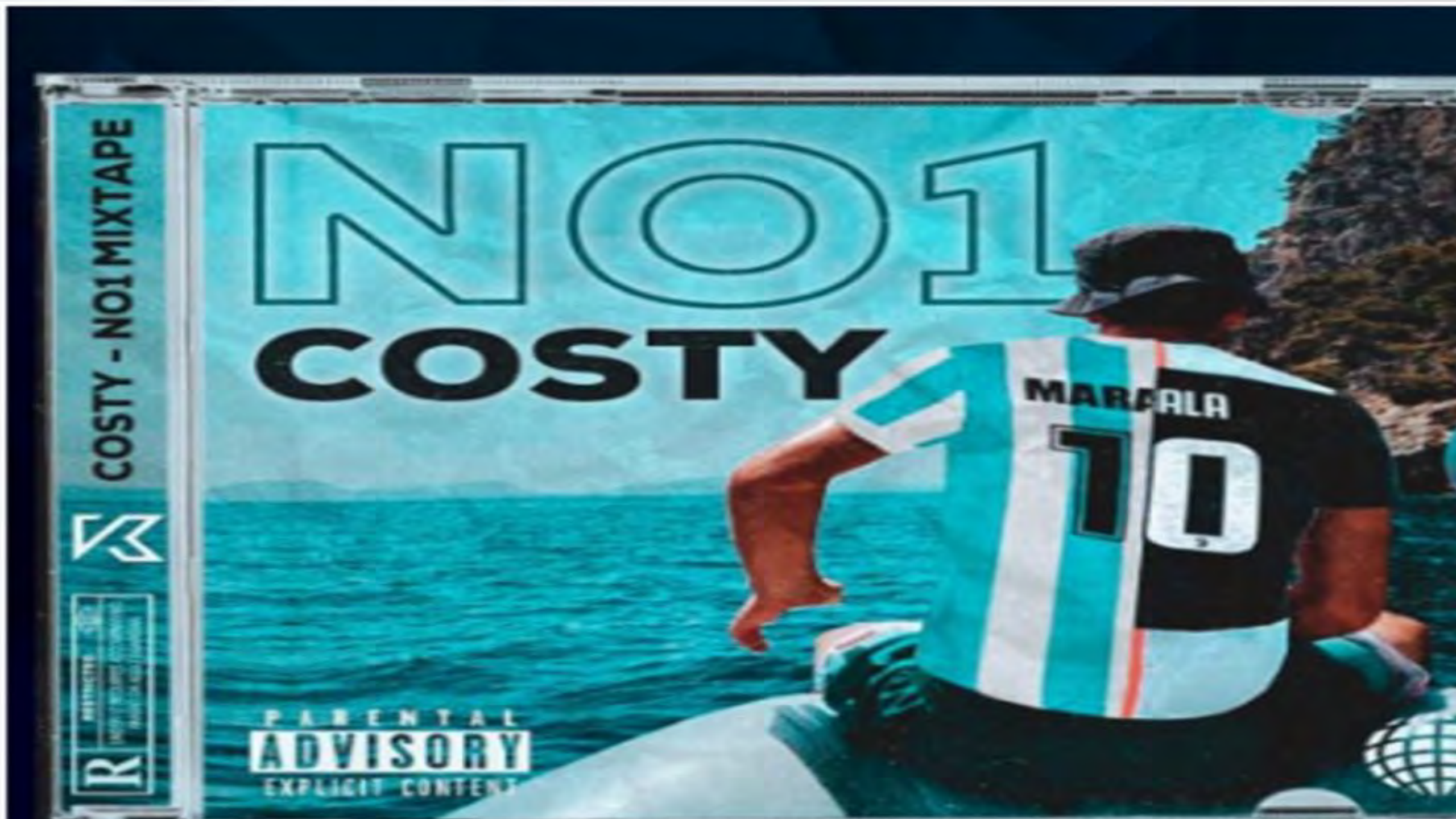
Daniella Levy-Erez & Haewon C. Kim

Chapter | First Online: 02 February 2019

TPE is not a benign procedure, and complications in children are higher compared with adults.
However, continuous improvement in techniques and instrument allow apheresis to be performed safely in children.

Table 13.1 Pediatric guidelines for central venous access devices for acute or short-term (<14 days) and chronic (>14 days) apheresis[a] [6]

Patient weight (kg)	Catheter/port size (French)[b]	
	Acute or short term (<14 days) (non-cuffed)	Chronic (>14 days) (cuffed)
<10	Single-lumen, 5 Fr Turbo-flo PICC[c]	Double-lumen, 6 Fr[d]
	Double-lumen, 7 Fr	
11–19	Single-lumen, 5 Fr Turbo-flo PICC[c]	Double-lumen, 6 Fr[d]
	Double-lumen, 8 Fr	Double-lumen, 8 Fr
20–29	Double-lumen, 8 Fr	Double-lumen, 8 Fr
		Single-lumen, 7.5[e], 6.6[f], 8[f] Fr port
30–39	Double-lumen, 9 Fr	Double-lumen, 8 Fr
		Single-lumen, 8 Fr port[f]
41–50	Double-lumen, 9 Fr	Double-lumen, 10 Fr
	Double-lumen, 11.5 Fr	Double-lumen, 11.4 Fr port[e]
		Single- lumen, 9.6 Fr IV Port[g]
>50	Double-lumen, 11.5 Fr, 12 Fr, or 13.5 Fr	Double-lumen, 10 Fr
		Single- lumen, 9.6 Fr IV Port[g] Double-lumen, 11.4 Fr port[e]


NO1
COSTY
COSTY - NO1 MIXTAPE
MARSALA
10
PARENTAL
ADVISORY
EXPLICIT CONTENT

Type	PF 1000 N	PF 2000 N
Effective membrane area (m^2)	0.15	0.35
Fiber dimensions		
Inner diameter (µm)	330	330
Wall thickness (µm)	150	150
Max. pore size (µm)	0.5	0.5
Sterilization agent	Ethylene oxide	

Components	Materials	
Membrane	Polypropylene	(PP)
Potting material	Polyurethane	(PUR)
Housing and caps	Polycarbonate	(PC)
Protective caps blood side	Polypropylene	(PP)

- The pressure gradient between arterial inlet and filtrate outle
should be strictly controlled. **Do not exceed** a transmembrane
pressure of:
  - PF 1000 N = 200 mmHg
  - PF 2000 N = 120 mmHg

- **PF 1000N:**
The bloodflow rate should not exceed 200 ml/min or fall below
50 ml/min. The optimal range is between 80 - 130 ml/min.

PF 2000N:
The bloodflow rate should not exceed 250 ml/min or fall below
100 ml/min. The optimal range is between 150 - 200 ml/min.

Table 3 Obligatory extracorporeal volume of equipment for continuous pediatric plasmapheresis

Plasmapheresis equipment	Obligatory extracorporeal volume of equipment (mL)
Baxter BM25 plus filter	Neonatal: 85–100 Pediatric: 101–116
Cobe spectra	170
Cobe Optia	185
Fenwal Amicus	160

Portion of Plasma Volume Exchanged *(Ve/EPV)*	Volume Exchanged *Ve (ml)*	Immunoglobulin Removed *MRR%*
0.5	1400	39
1	2800	63
1.5	4200	78
2	5600	86
2.5	7000	92
3	8400	95

For this reason, usually one, and at most 2 plasma volume equivalents (Ve/EPV) are exchanged during one plasmapheresis session

44

Table 4 Equations to estimate total blood volume in children

Equations	Blood volume
Russel 1949 [114]	
Weight (kg)	Blood volume (mL)
3–10	58.2 × weight (kg) + 42
10–20	82.7 × weight (kg) + 17
20–30	95.7 × weight (kg) − 274
Nadler 1962 [115]	
Sex	Blood volume (mL)
Male	$0.3669 \times \text{height (m)}^3 + 0.03219 \times \text{weight (kg)} + 0.6041$
Female	$0.3561 \times \text{height (m)}^3 + 0.03308 \times \text{weight (kg)} + 0.1833$
Geigy scientific tables 1970 [116]	
Age	Mean blood volume per weight (mL/kg)
24 h	83
3 months	87
6 months	86
1 year	80
6 years	80
10 years	75
15 years	71
Adult men	71
Adult women	70

Estimated plasma volume in ml is {0.0 65 × weight (kg)} × {1-hematocrit} × 1,000.

Replacement fluid

Choice of Replacement solution

- **Albumin**
- **Advantage:**
- No risk of hepatitis
- Stored at room temperature
- Allergic reaction are rare
- No concern about ABO blood group
- Depletes inflammation mediators

- **Disadvantage:**
- Expensive
- No coagulation factors
- No immunoglobulin's

- **Fresh Frozen Plasma**
- **Advantage:**
- Coagulation factors
- Immunoglobulin's " beneficial" factors complement

- **Disadvantage:**
- Risk of hepatitis, HIV transmission
- Allergic reaction
- Hemolytic reaction
- Must be thawed
- Must be ABO compatible
- Citrate load

FFP:

Specific indications:-

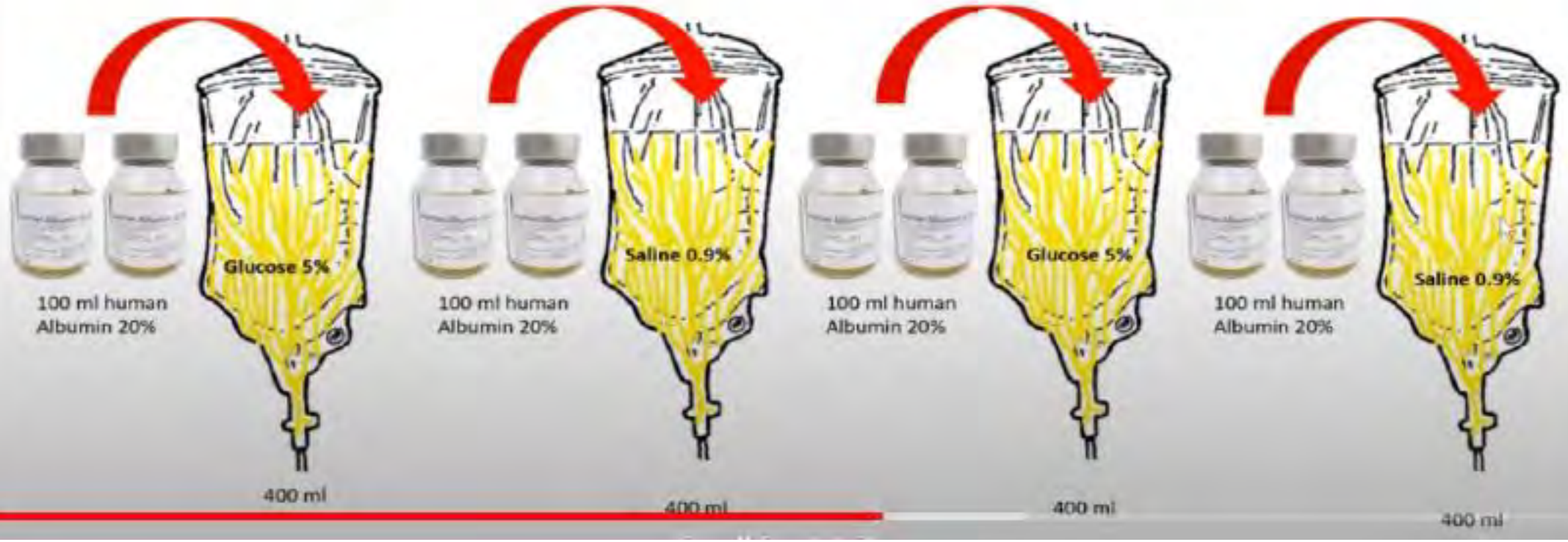
- TTP/HUS.
- preexisting defect in hemostasis.
- risk of cholinesterase depletion.
- when the fibrinogen level is low (< 125 mg/dL).

- minimum 2–5 ds pre- and post-surgery
- • For daily, multiple closely spaced (e.g., every other day) or TPEs with albumin replacement which result in a dilutional coagulopathy

esis

SUBSTITUTION OF TWO LITERS PLASMA BY FRESH FROZEN PLASMA

PREPARATION OF TWO LITERS PLASMA SUBSTITUTION BY HUMAN ALBUMIN



Anticoagulation

Comparison of anticoagulants

- SUMMARY

Citrate:

1. Familiar in blood-banking.
2. Short-acting: prescribe ratio to blood flow
3. No systemic anticoagulant effect; risk of "citrate toxicity"
4. Suitable for low-flow circuits

Heparin:

1. Familiar in dialysis.
2. Long-acting: prescribe units/ Kg body wt/ hour
3. Systemic anticoagulant; risk of bleeding; rare HIT
4. Suitable for high-flow circuits

Complication of plasmapeheresis

Related to vascular access:

- Hematoma
- Pneumothorax
- Retroperitoneal bleed

Related to the procedure

- Hypotension form externalization of blood in the extracorporeal circuit.
- Hypotension due to decrease intravascular oncotic pressure.
- Bleeding from reduction in plasma levels of coagulation factors
- Edema formation due to decrease intravascular oncotic pressure
- Loss of cellular elements (platelets)
- Hypersensitivity reactions

Complication of plasmapeheresis

Related to anticoagulation:

- Bleeding, especially with heparin
- Hypocalcemic symptoms (with citrate)
- Arrhythmias
- Hypotension
- Numbness and tingling of extremities
- Metabolic alkalosis from citrate


CHALLENGE
CHALLENGE
CHALLENGE

Editorial > Indian J Pediatr. 2021 Aug;88(8):745-746. doi: 10.1007/s12098-021-03821-6.

Epub 2021 May 26.

Pediatric Therapeutic Plasma Exchange: Coming of Age?

Saptarshi Mandal [1], Aditi Sinha [2]

Affiliations + expand

PMID: 34041697 DOI: 10.1007/s12098-021-03821-6

FULL TEXT LINKS

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ACTIONS

Cite

Collections

- Pediatric TPE constitutes less than one-twentieth of all apheresis procedures
- TPE in children is challenging because of

Currently available machines and disposables are designed for adult patients

To be able to perform the procedure safely in children using currently available device, careful consideration should be given primarily to three areas :

- intravascular volume red cell mass
- anticoagulation
- vascular access

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- TPE … llenging because of

lack evidence - based guidance.

Currently available machines and disposables are designed for adult patients


All for One
One for All

Thank you

Which of the following is correct regarding ASFA guidelines:

- A) Anti GBM nephritis is considered category II
- B) category III means PEX may be harmful
- C) It is adapted mainly for children
- D) TTP is considered category I

Which of the following is correct regarding PEX in pediatrics:

- A)catheter guidelines are the same for acute and chronic purpose.
- B) you can use salt free albumin in all indications of PEX
- C) review literature are abundant
- D) Heparin anticoagulation is more suitable for long circuits

Which of the following is correct regarding indications of PEX in pediatrics:

- A) PEX has very imp role in all types of ANCA associated vasculitis
- B) patients with GBS could have great benefits from PEX
- C) PEX has no role in TTP
- D) Role of PEX in Multiorgan failure is well established