Dr DAVY IP
CIC NEPHROLOGY VH
MUG 21 AUGUST 24

BLOOD PURIFICATION: THERAPEUTIC PLASMAPHARESIS

EXTRACORPOREAL THERAPY

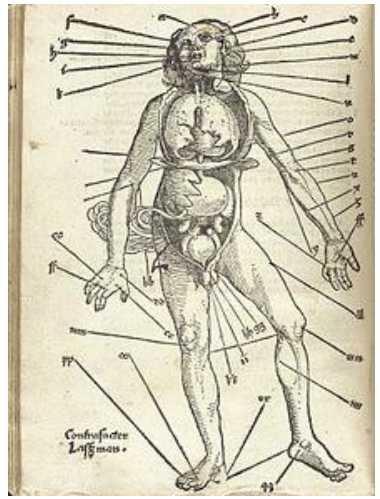
BLOOD PURIFICATION
- DIALYSIS/HAEMOFILTRATION
- APHARESIS
- ALBUMIN BASED DIALYSIS (‘MARS’)
- C02 DIALYSIS

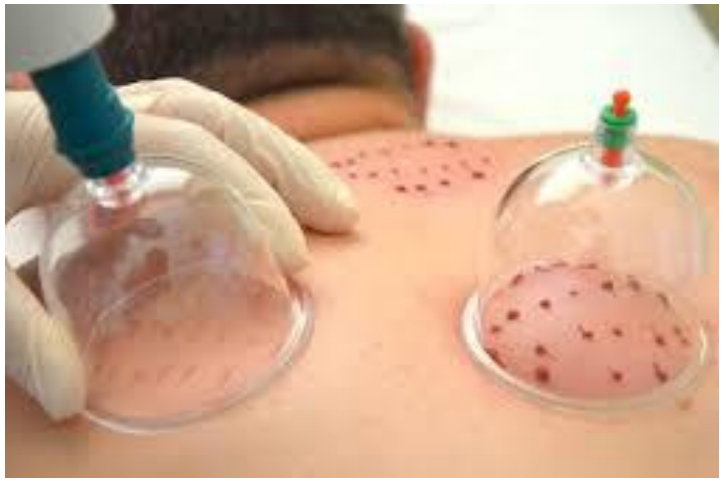
EXTRACORPOREAL MEMBRANE OXYGENATION

APHARESIS IS NOT NEW

Since ancient times…
Removal of pathogenic substances from the blood had been postulated to be therapeutic or even curative for some diseases.

Phlebotomy was known as bloodletting when it was first used and dates back to the ancient Egyptians, around 1000 BC.

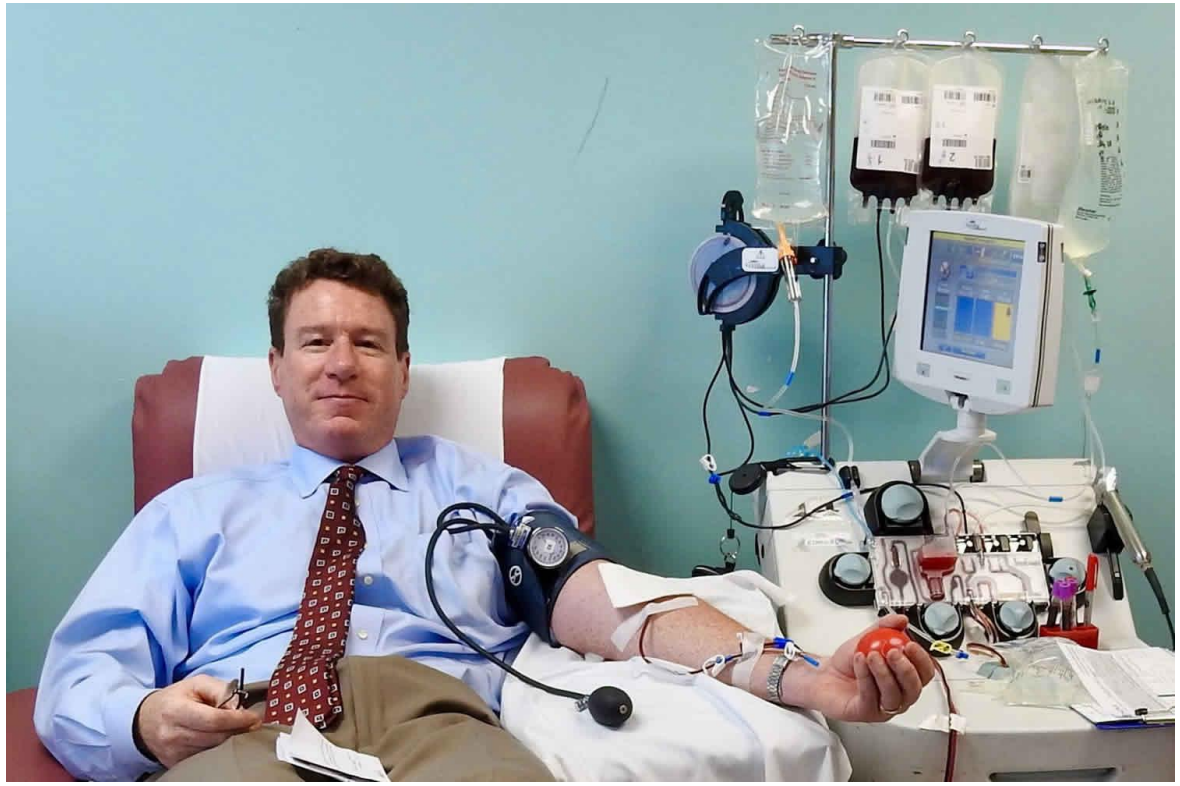




WHAT ABOUT APHARESIS NOW?

In the modern era…

The term apheresis refers to the general technique of extracorporeal removal of a blood constituent.

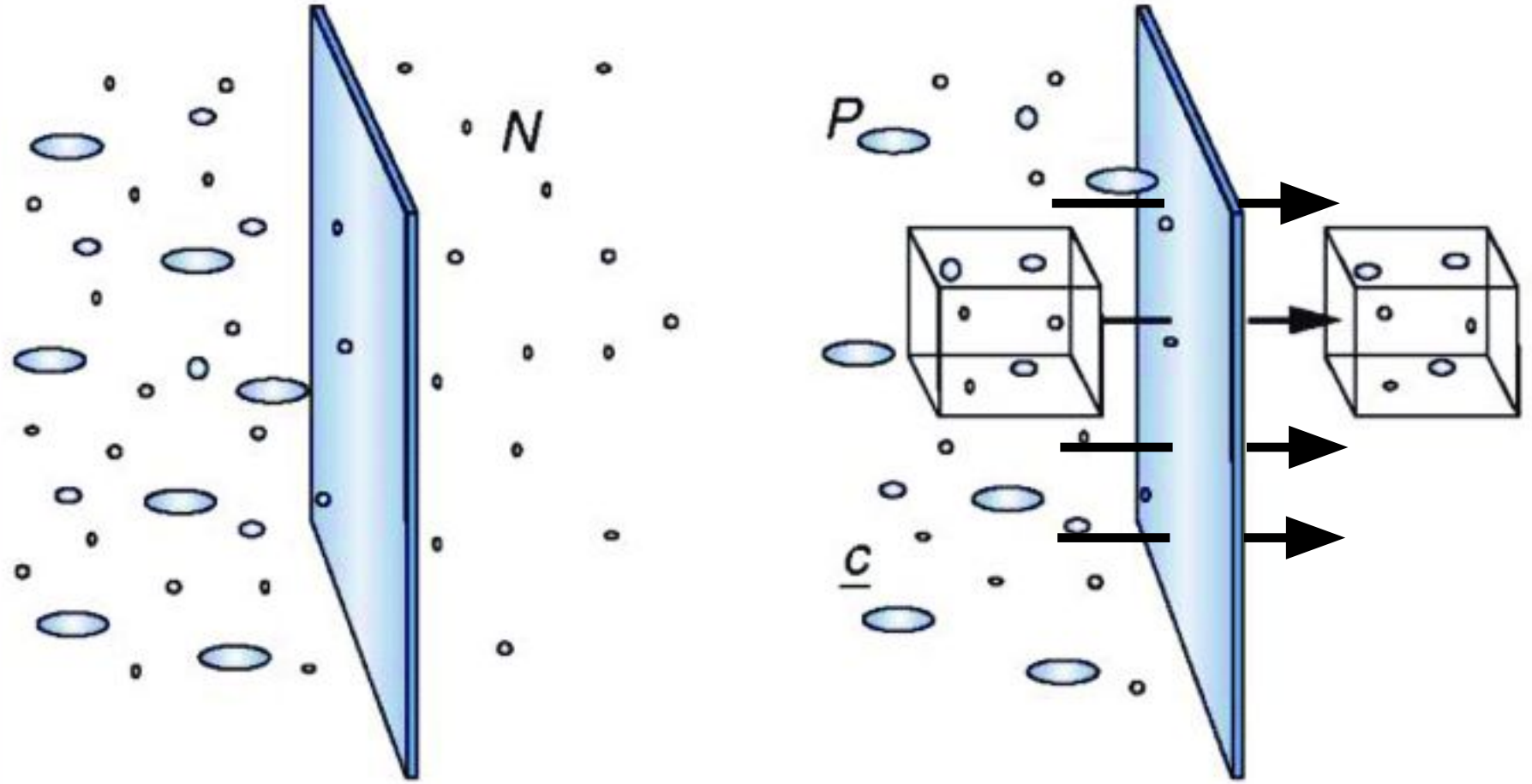


APHARESIS MODALITIES

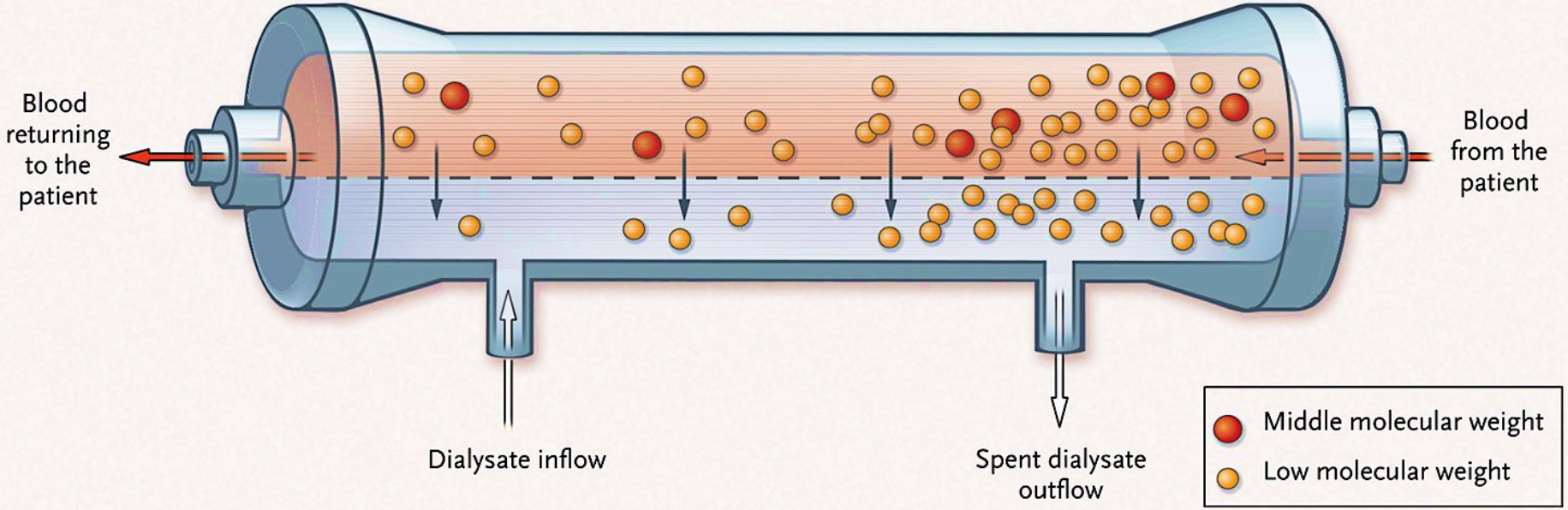
Procedure	Target Molecule
Adsorptive cytapheresis	Monocytes, granulocytes
ß$_2$-microglobulin column	ß$_2$-microglobulin
Double filtration plasmapheresis	Autoantibodies, immune complexes, lipoproteins
Erythrocytapheresis	Red blood cells
Extracorporeal photopheresis	Buffy coat (white blood cells and platelets)
Immunoadsorption	Immunoglobulins
Leukocytapheresis	White blood cells
Lipoprotein apheresis	Lipoprotein particles
Red blood cell exchange	Red blood cells (exchanged for replacement fluid)
Rheopheresis/ Double filtration plasmapharesis	High-molecular-weight plasma components (fibrinogen, α$_2$-macro-globulin, low-density lipoprotein cholesterol, and IgM)
Therapeutic plasma exchange	Plasma (exchanged for replacement fluid)
Thrombocytapheresis	Platelets

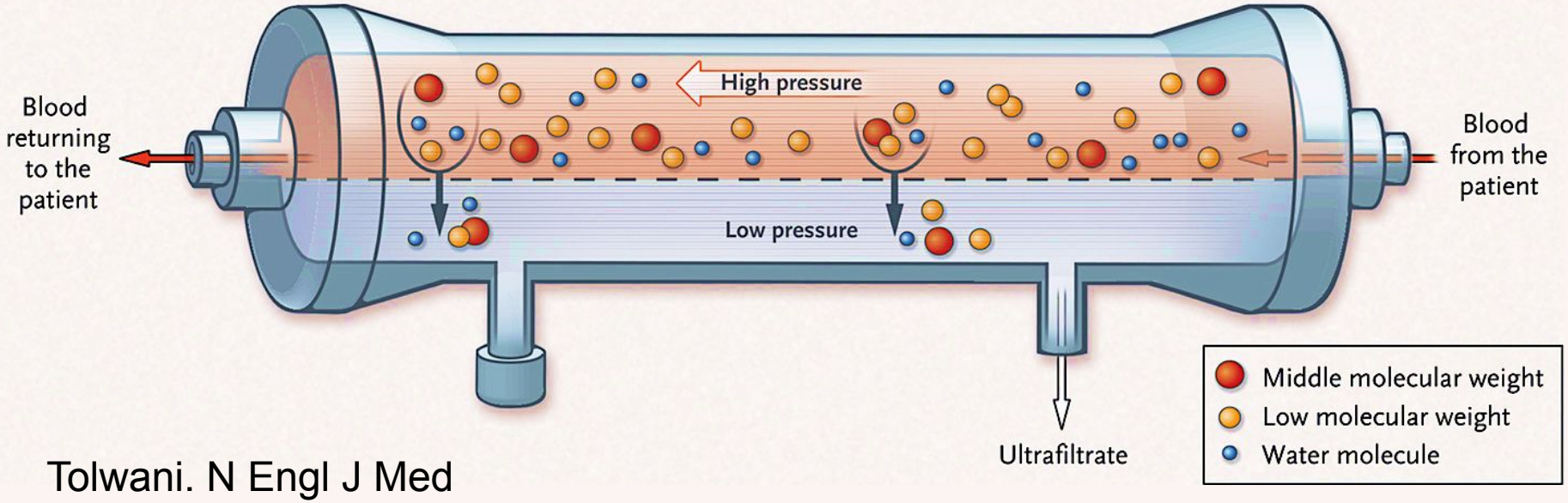
Adapted from Cervantes et al Am J Kidney Dis. 81(4):475-492 2023

DIFFUSION VS CONVECTION



DIFFUSION VS



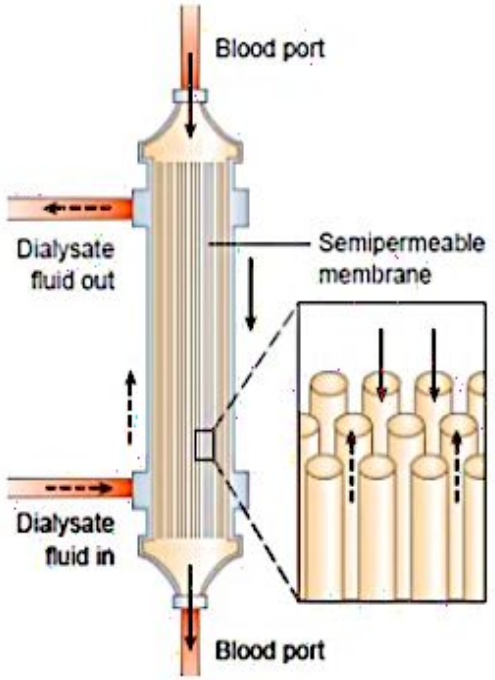


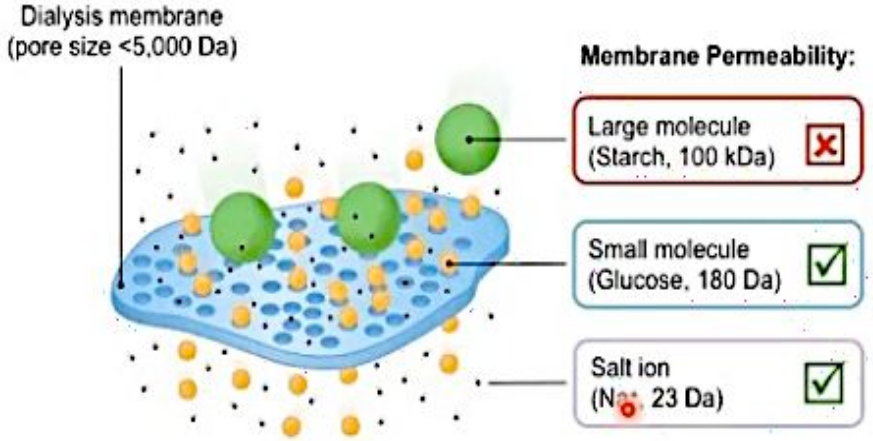
CONVECTION

Tolwani. N Engl J Med 2012;367:2505-2514

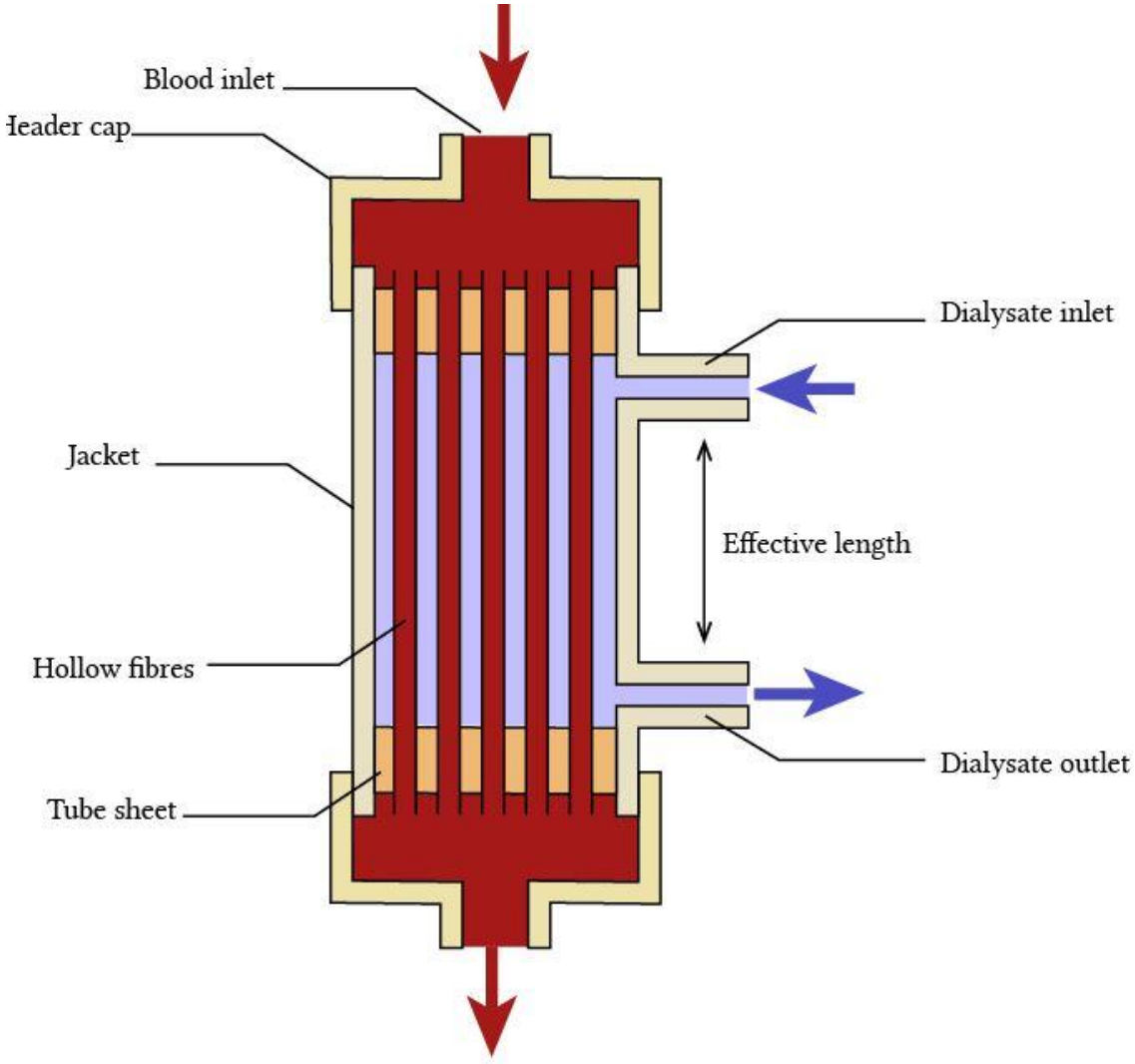
HOLLOW FIBRE TECHNOLOGY

- Many fine fiber tubes to allow contact surface area to be large
- Disposable dialyzers: single patient use to prevent cross-infection

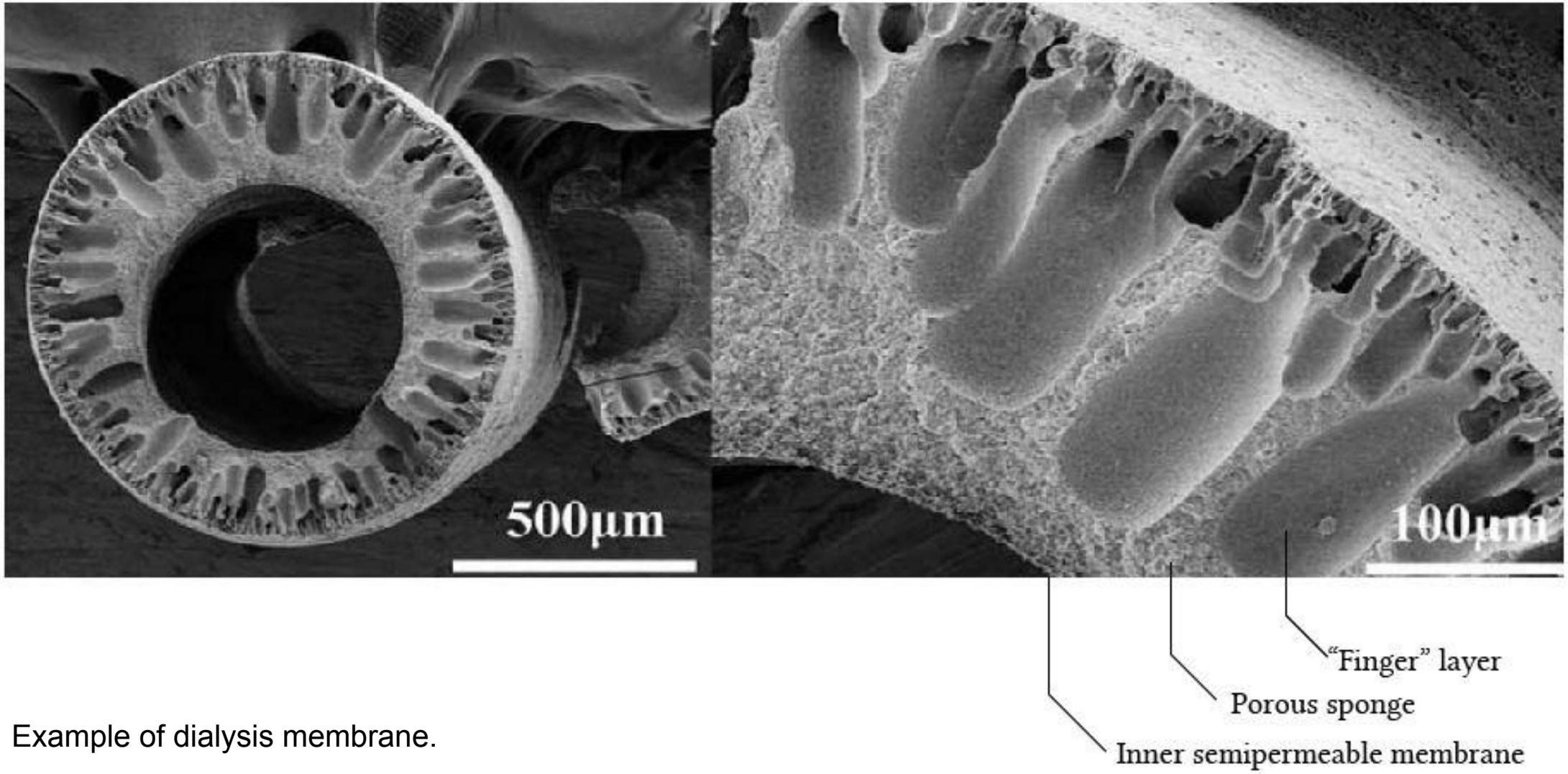




MEMBRANE SEPARATION



HOLLOW FIBRE TECHNOLOGY

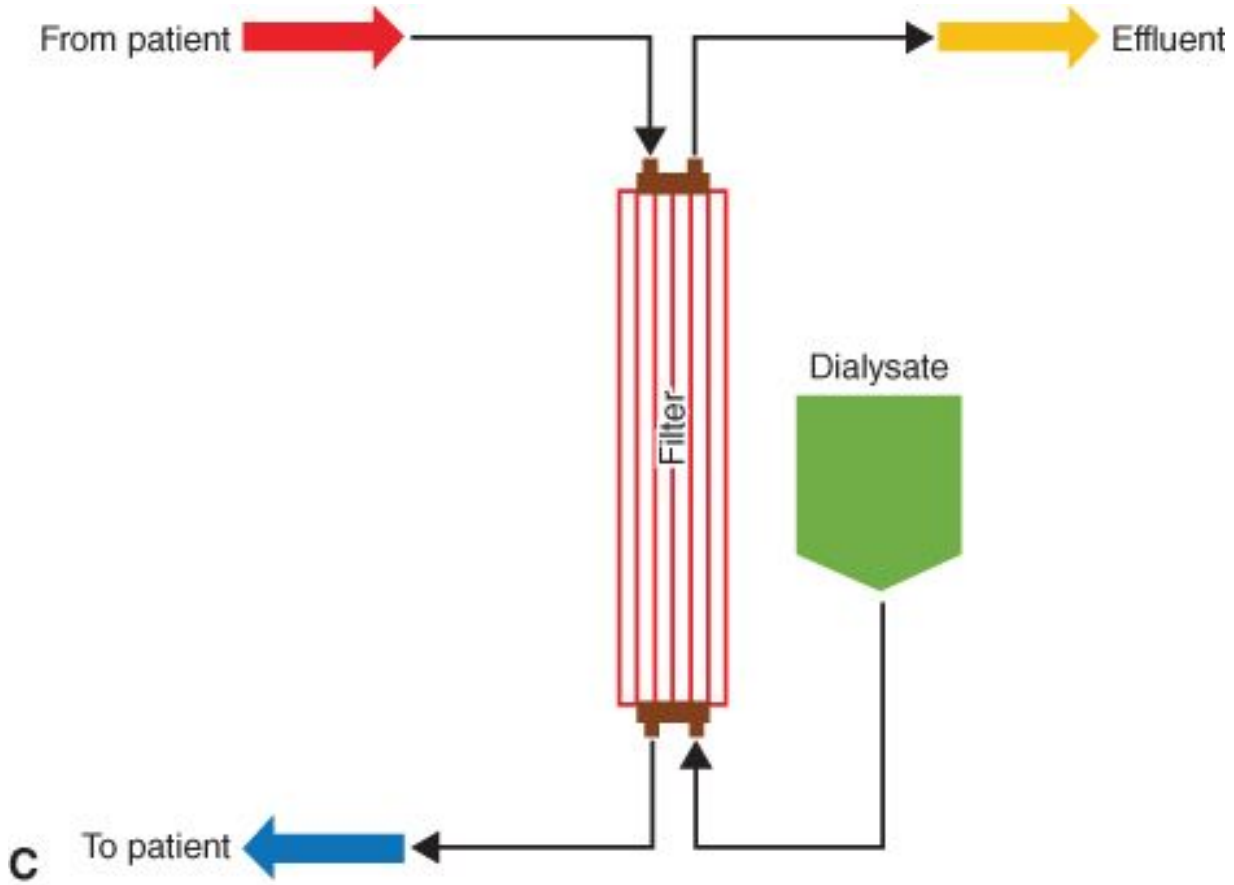


Example of dialysis membrane.

Zhang et al. RSC Adv., 2015,5, 21532-21543

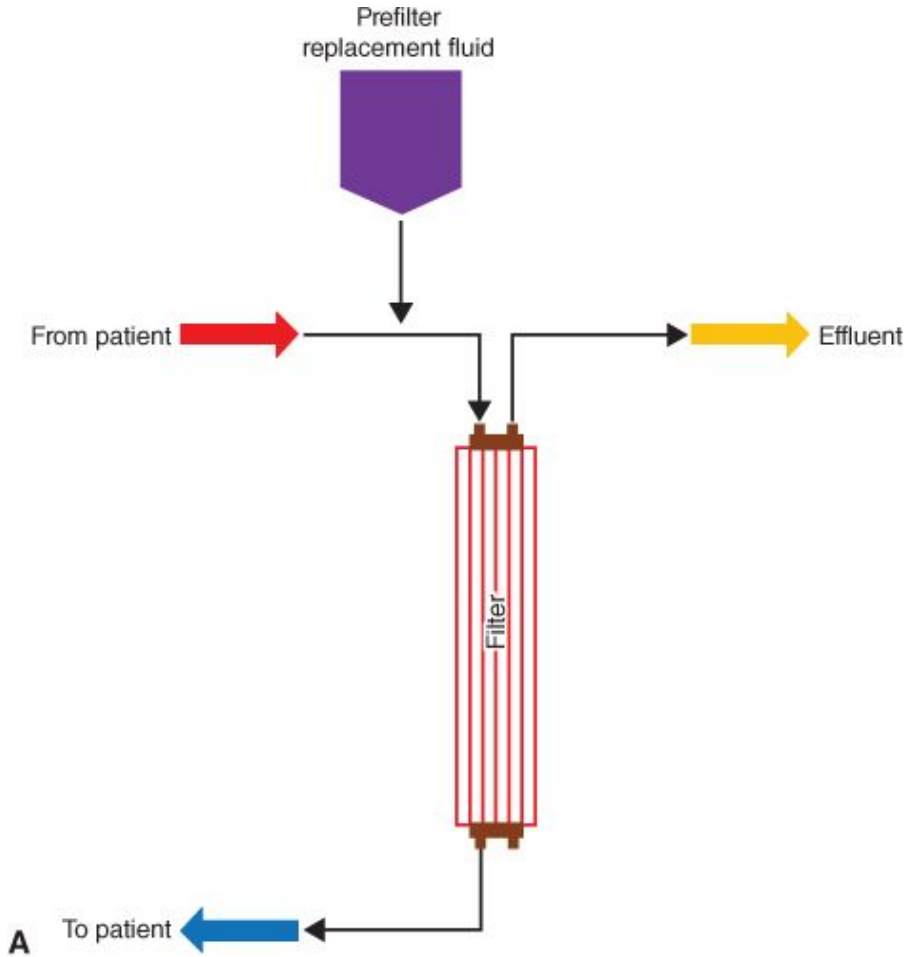
HAEMODIALYSIS = MAINLY DIFFUSION

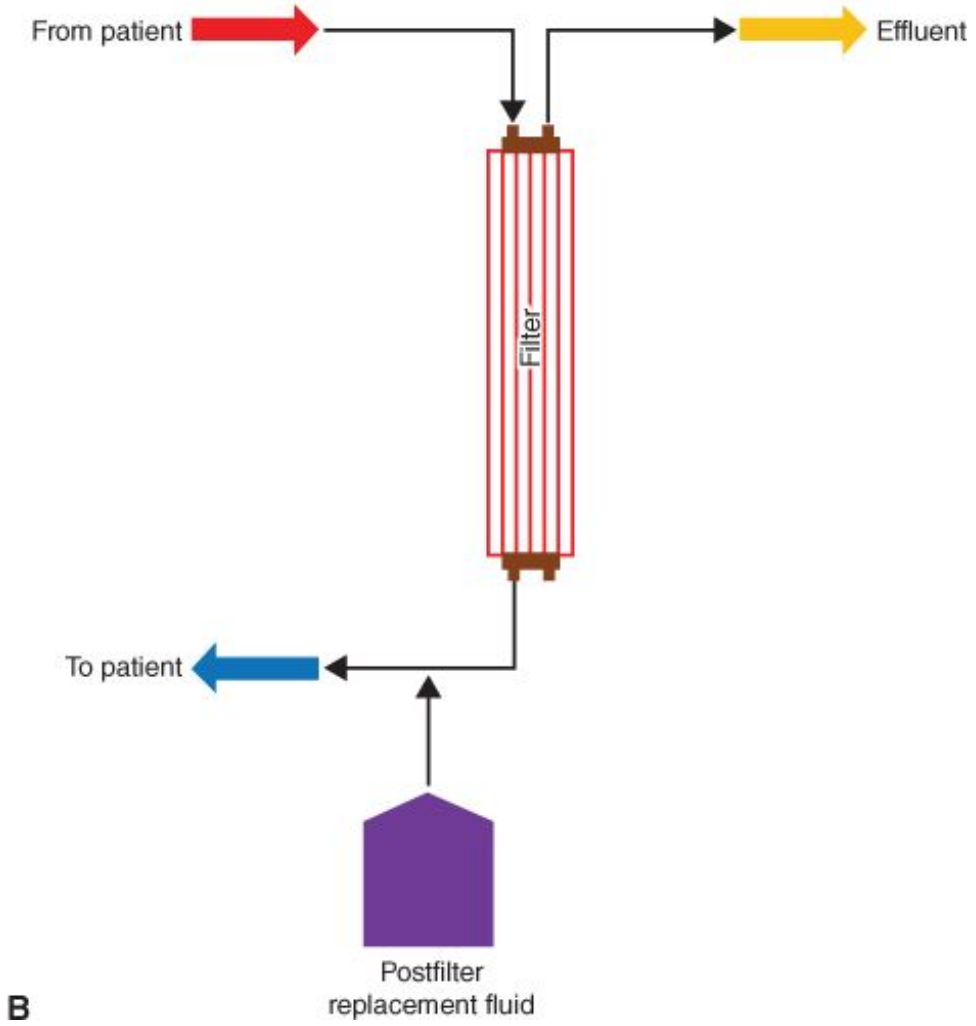
Intermittent or continuous therapy



HAEMOFILTRATION = CONVECTION

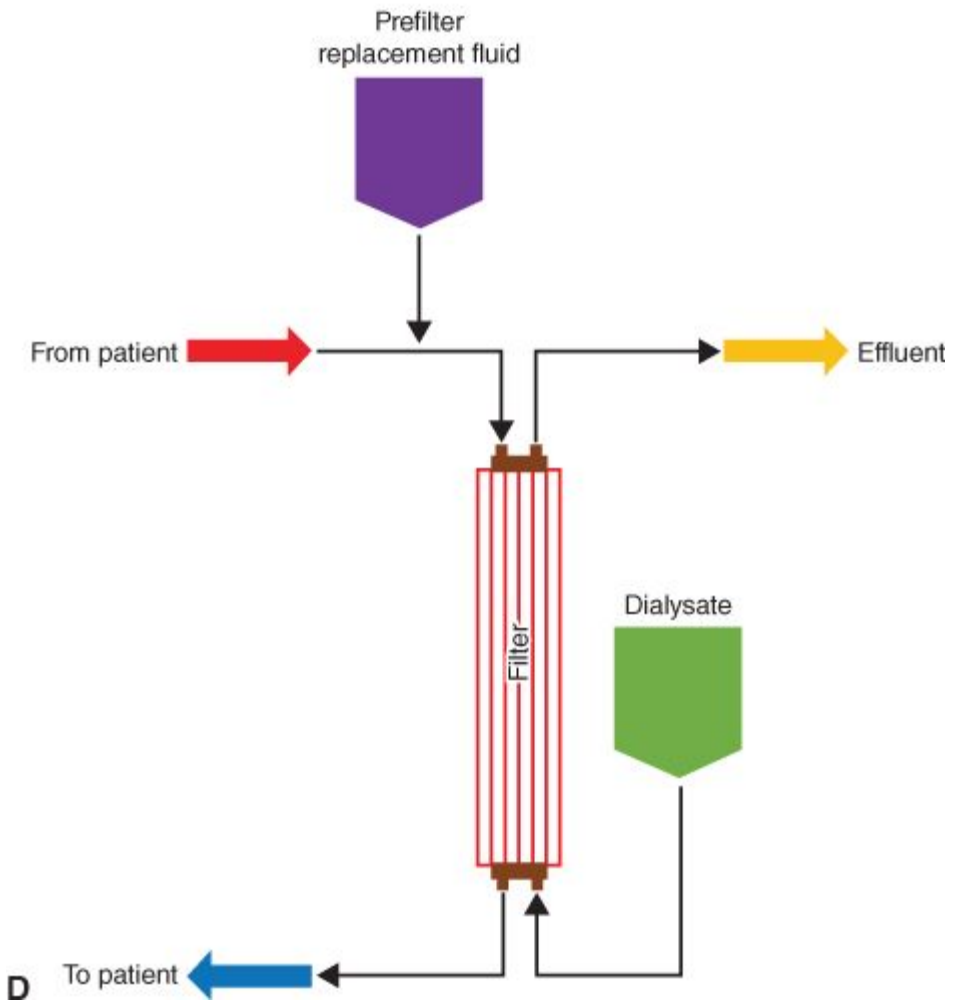
Usually, a continuous therapy

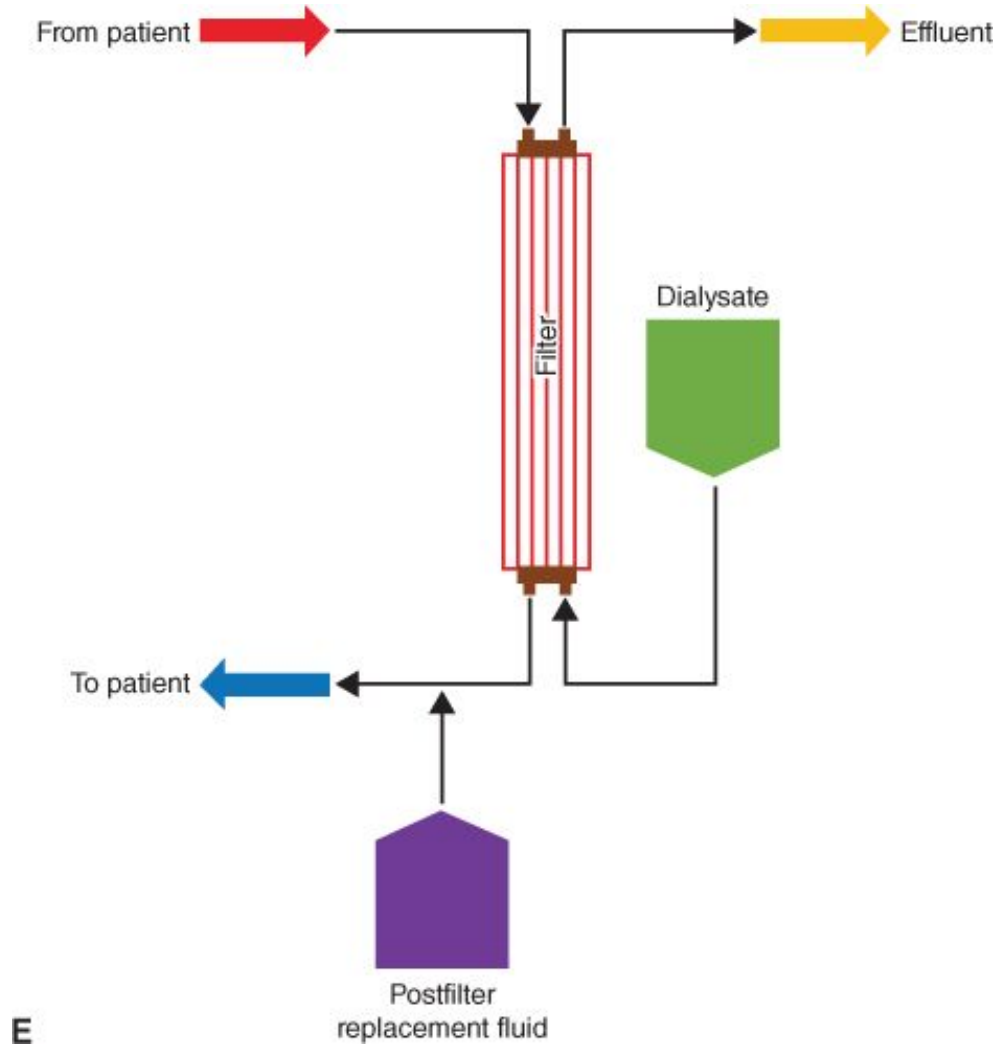




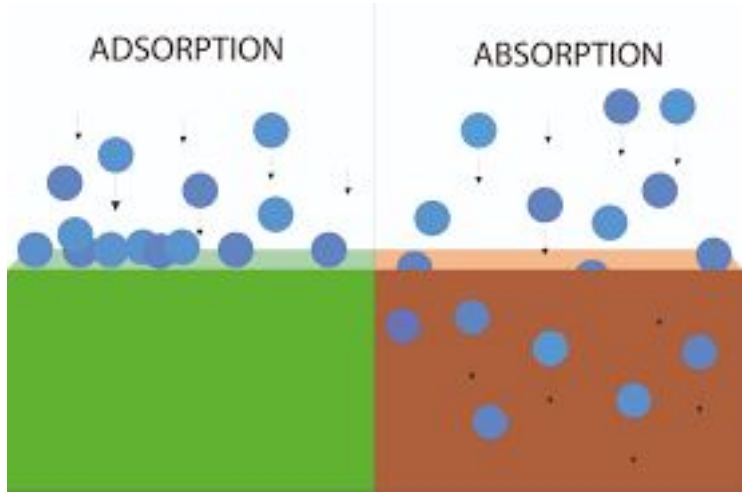
HAEMODIAFILTRATION = DIFFUSION + CONVECTION

Intermittent or continuous therapy

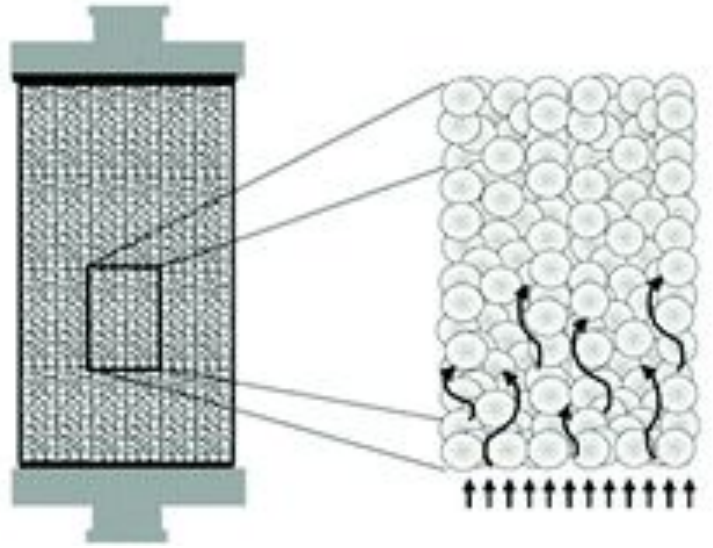




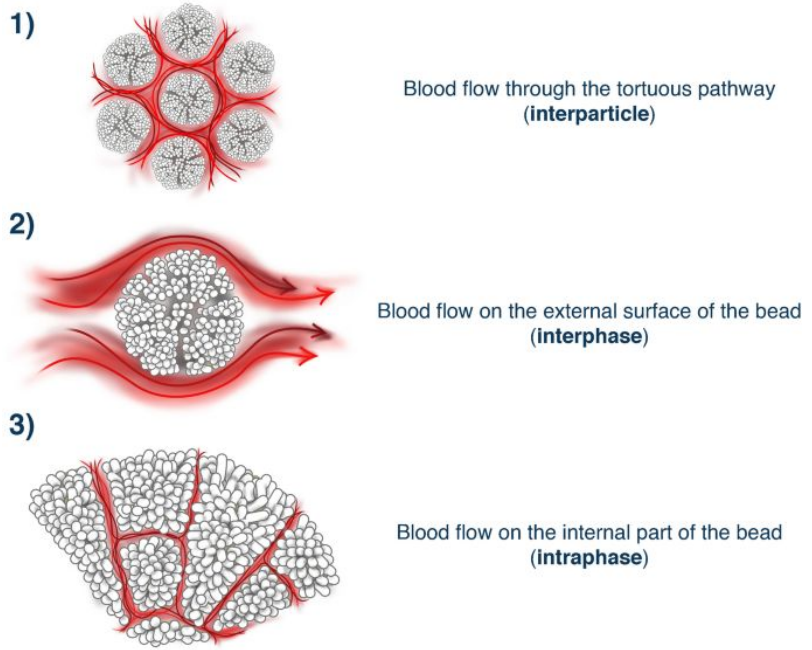
ADSORPTION



Adsorption is the adhesion of atoms, ions or molecules from a gas, liquid or dissolved solid to a surface. This process creates a film of the *adsorbate* on the surface of the *adsorbent*. In blood purification, hollow fibres or microspheres can be used as the adsorbent surface.

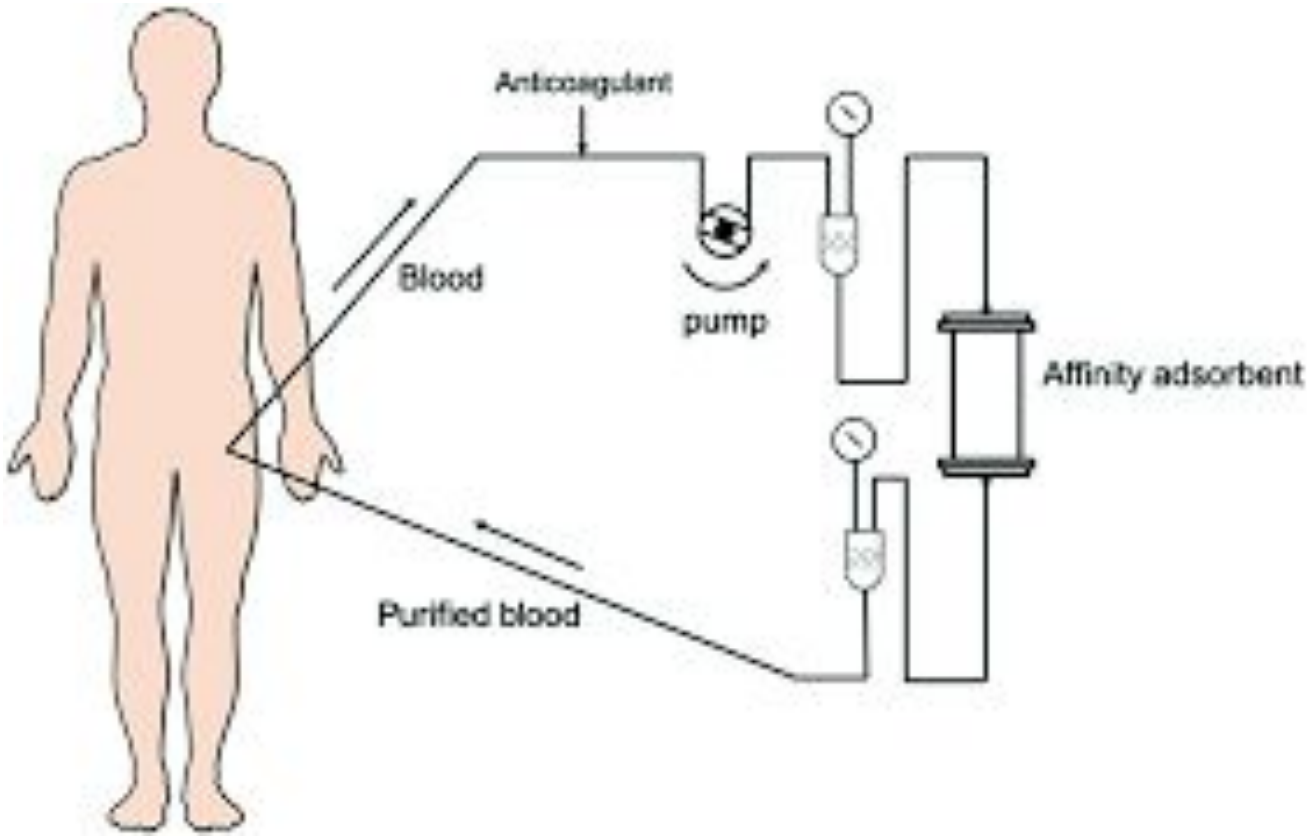


Cartridge filled with microspheres



Intensive Care Med **48**, 1397–1408 (2022)

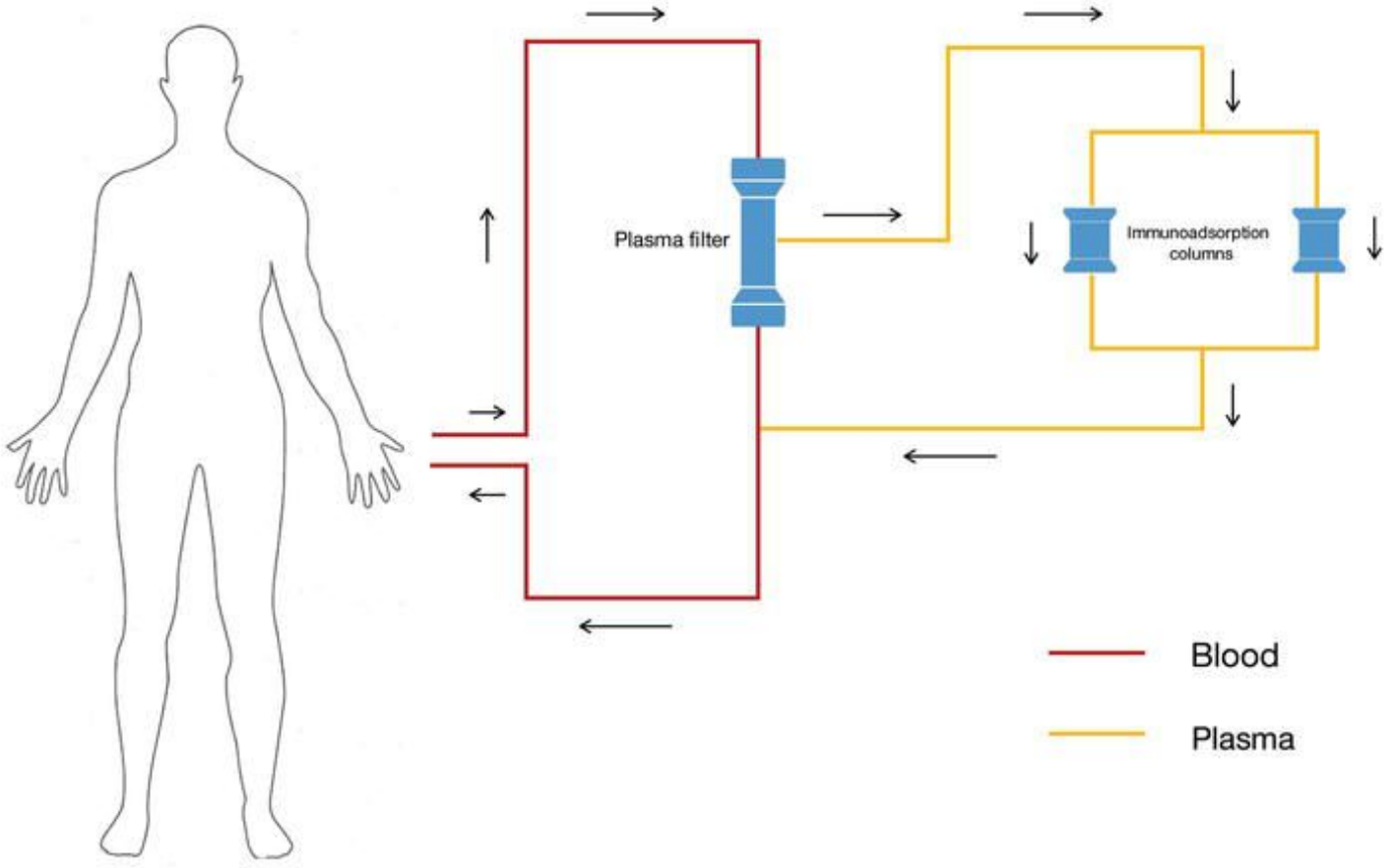
HAEMOPERFUSION



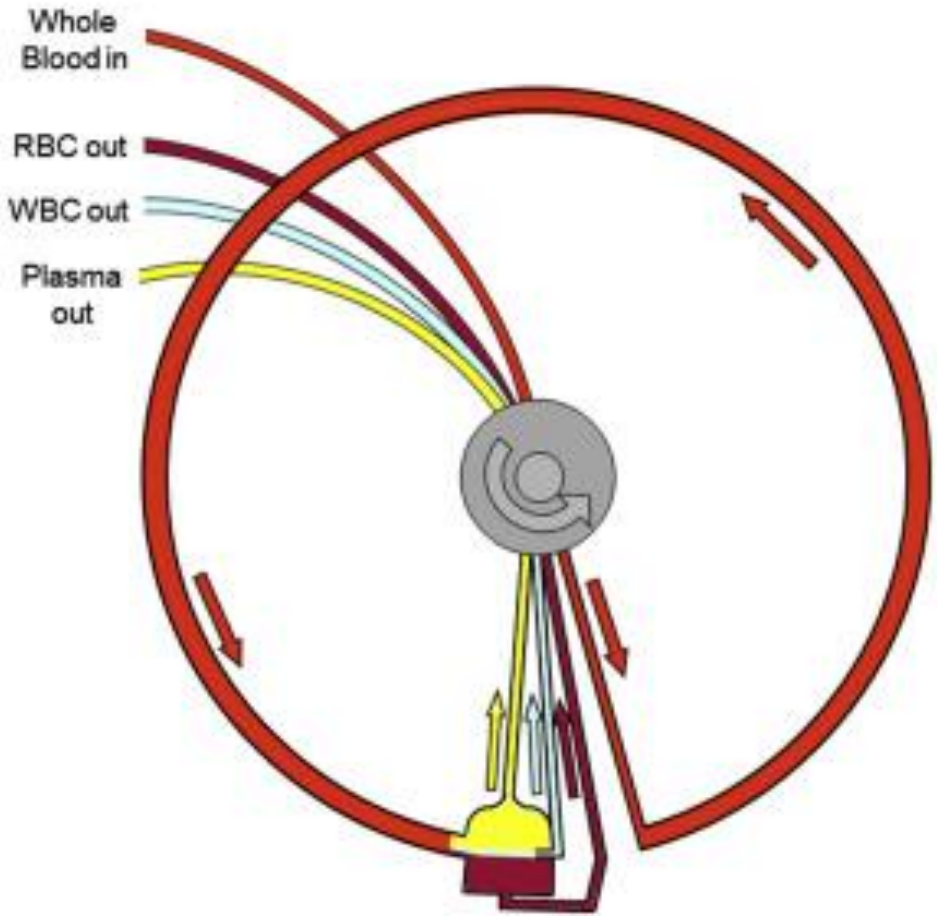
An extracorporeal procedure in which the anticoagulated patient's blood passes through a volume of adsorbent material (usually activated charcoal, microporous inorganic material, polymeric resins arranged in **microspheres** or **hollow fibre**)

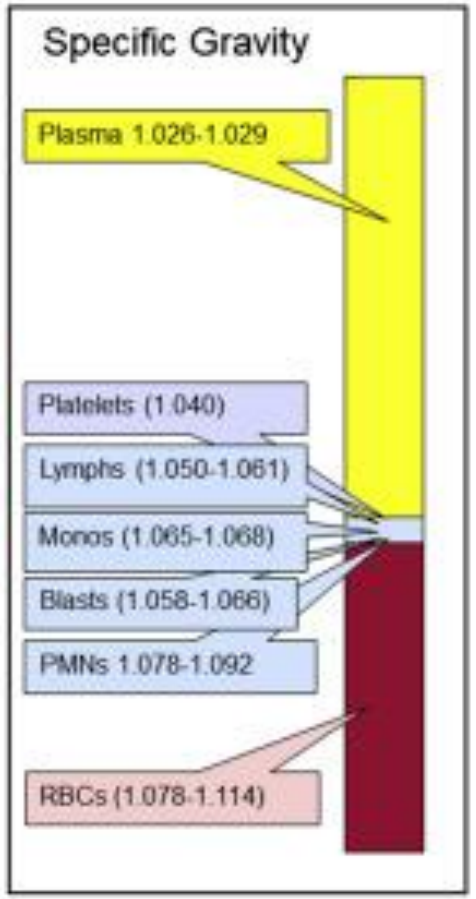
IMMUNOADSORPTION

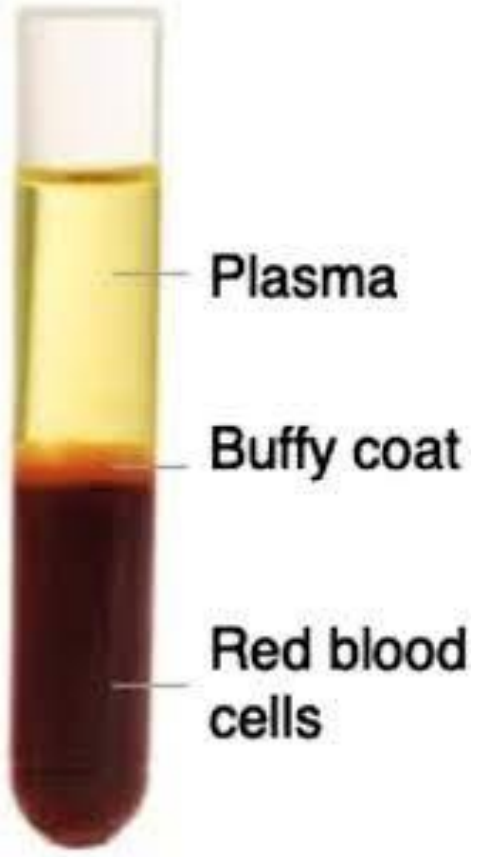
Immunoadsorption is a selective apheresis method for the removal of specific antibodies and immune complexes, leaving other plasma components and obviating the need for plasma replacement.



SEPARATION BY CENTRIFUGATION

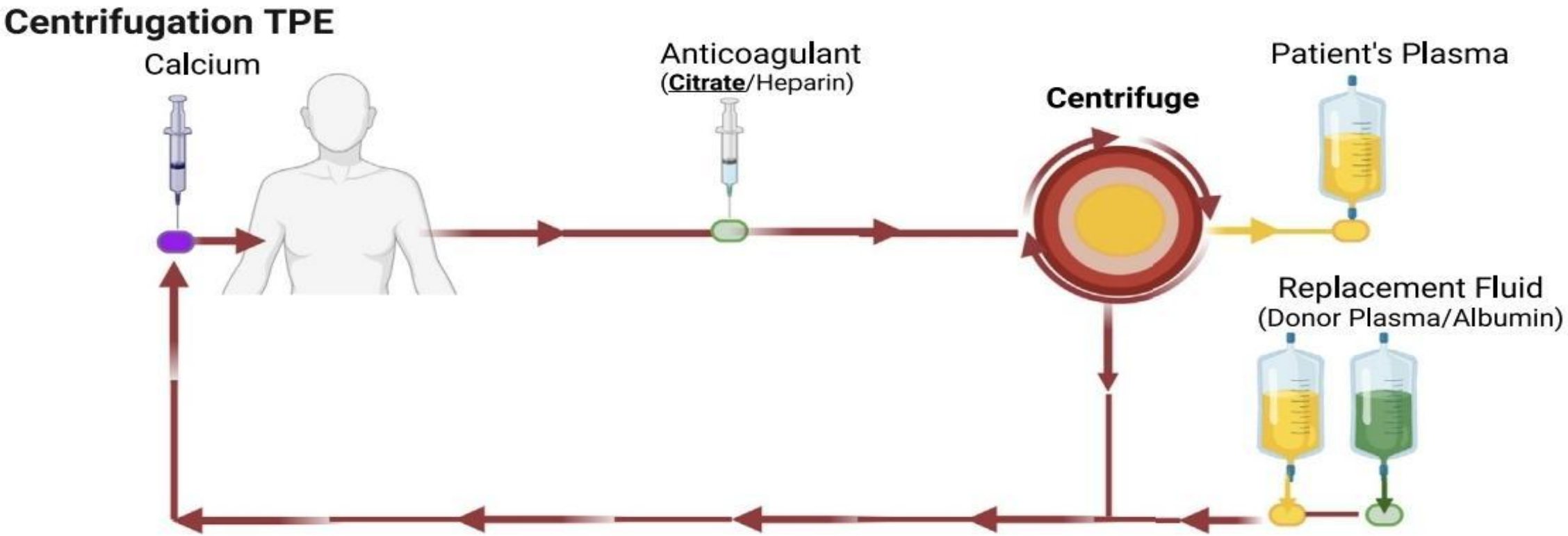






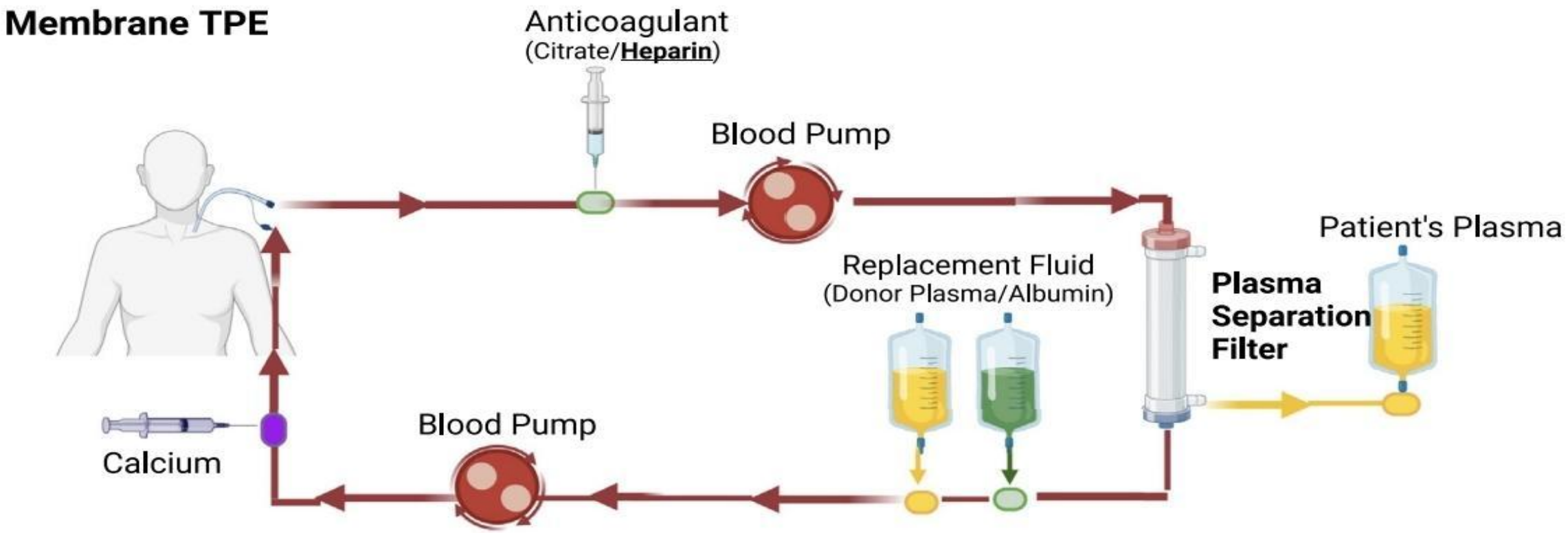
Molloy, E. et al. *Optimizing the Apheresis Product. Chimeric Antigen Receptor T-Cell Therapies for Cancer, 7–16* (2020).

THERAPEUTIC PLASMAPHARESIS



Cervantes et al Am J Kidney Dis. 81(4):475-492 2023

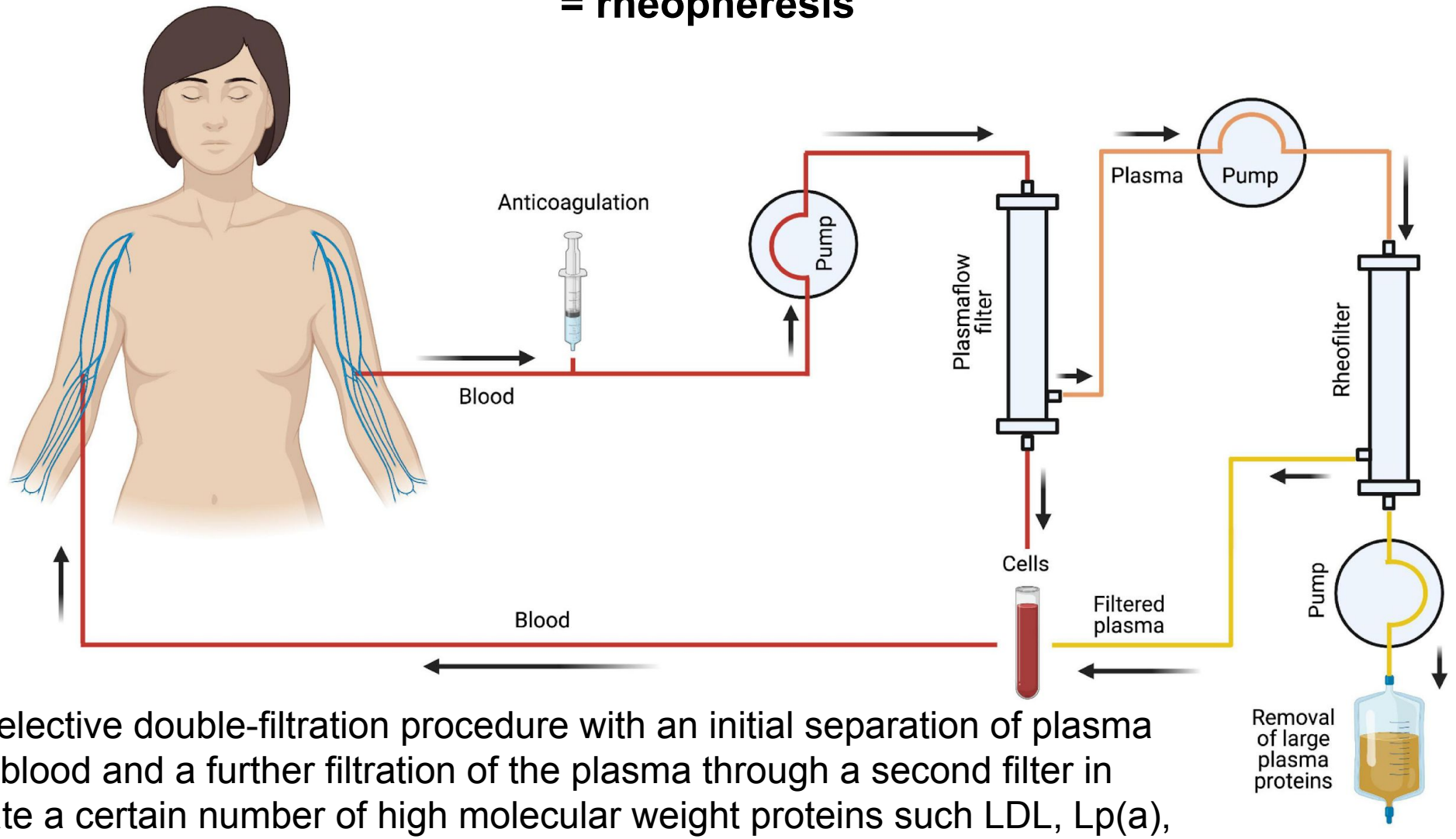
THERAPEUTIC PLASMAPHARESIS



Cervantes et al Am J Kidney Dis. 81(4):475-492 2023

DOUBLE FILTRATION PLASMAPHARESIS

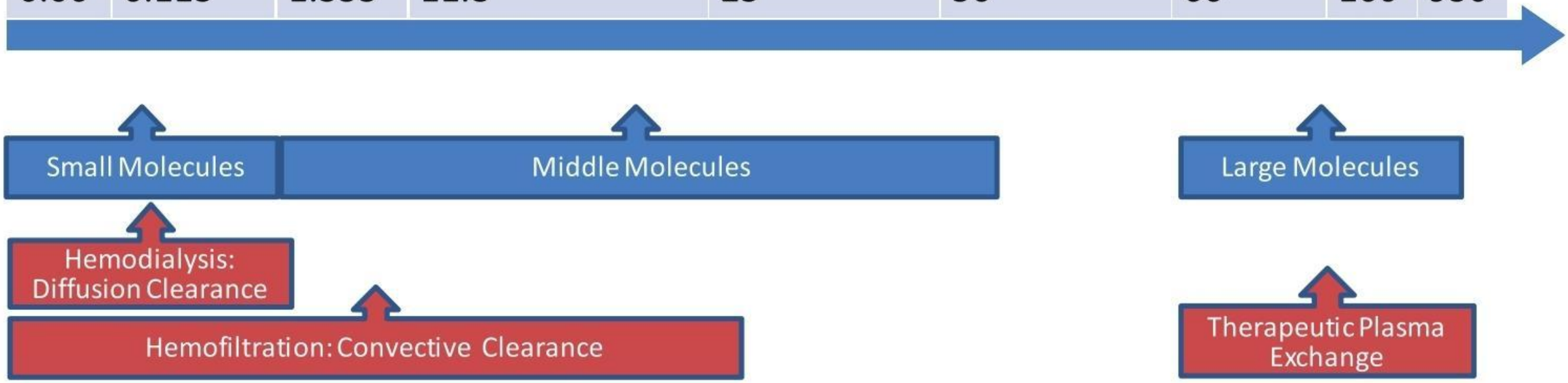
= rheopheresis



Extracorporal selective double-filtration procedure with an initial separation of plasma from the whole blood and a further filtration of the plasma through a second filter in order to eliminate a certain number of high molecular weight proteins such LDL, Lp(a), fibrinogen, α2-macroglobulin, von Willebrand Factor, and IgM.

EXTRACORPOREAL THERAPIES EFFECTIVENESS VS TARGET MOLECULES SIZES

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



Clin J Am Soc Nephrol 9: 181–190, 2014.

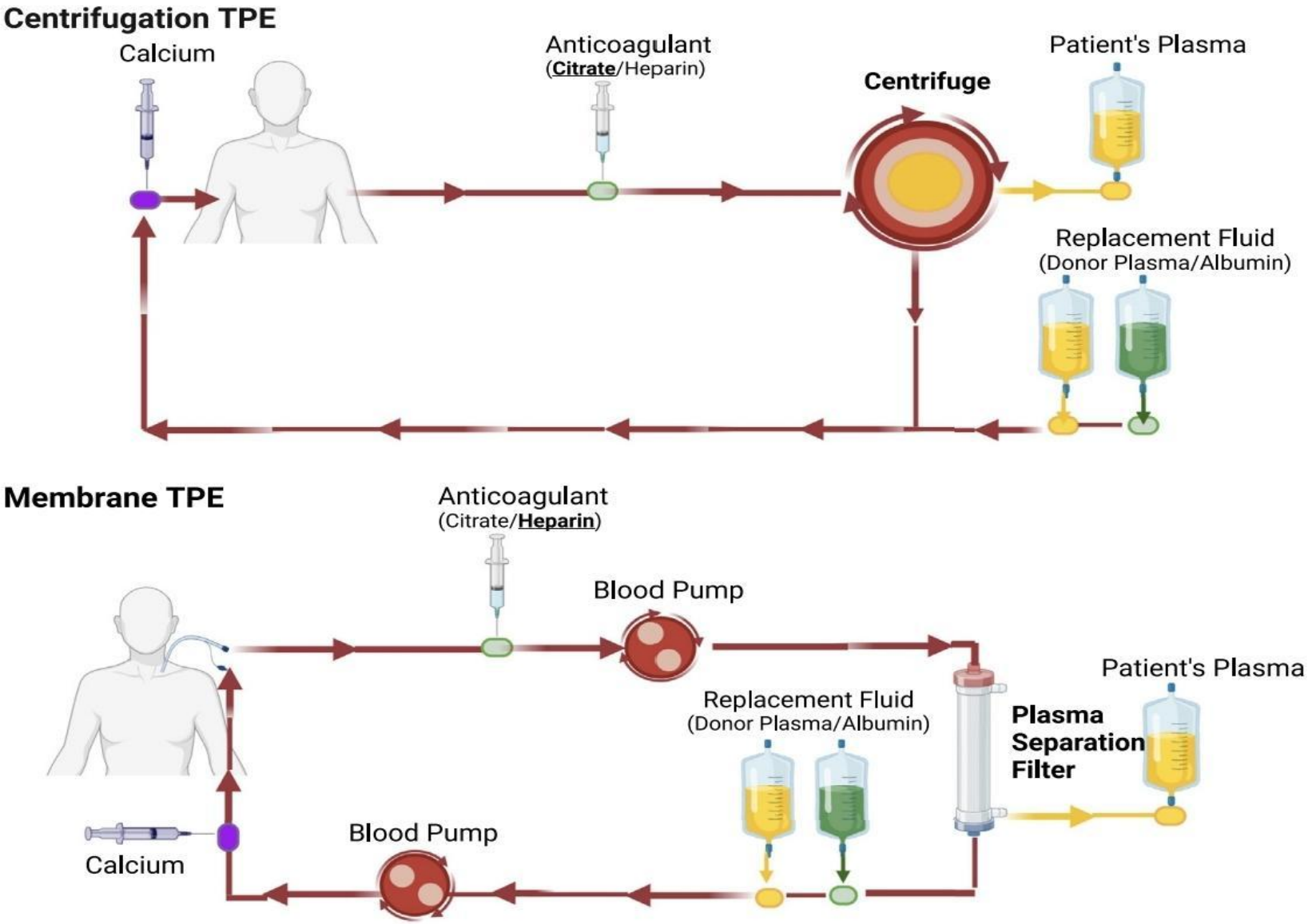
(THERAPEUTIC) PLASMAPHARESIS

Plasmapheresis removes plasma from a patient

Therapeutic plasma exchange (TPE) involves the removal of plasma from blood and replacement of the plasma with another fluid (crystalloid, colloid, allogenic plasma)

Can be membrane-based or centrifuged-based

THERAPEUTIC PLASMAPHARESIS



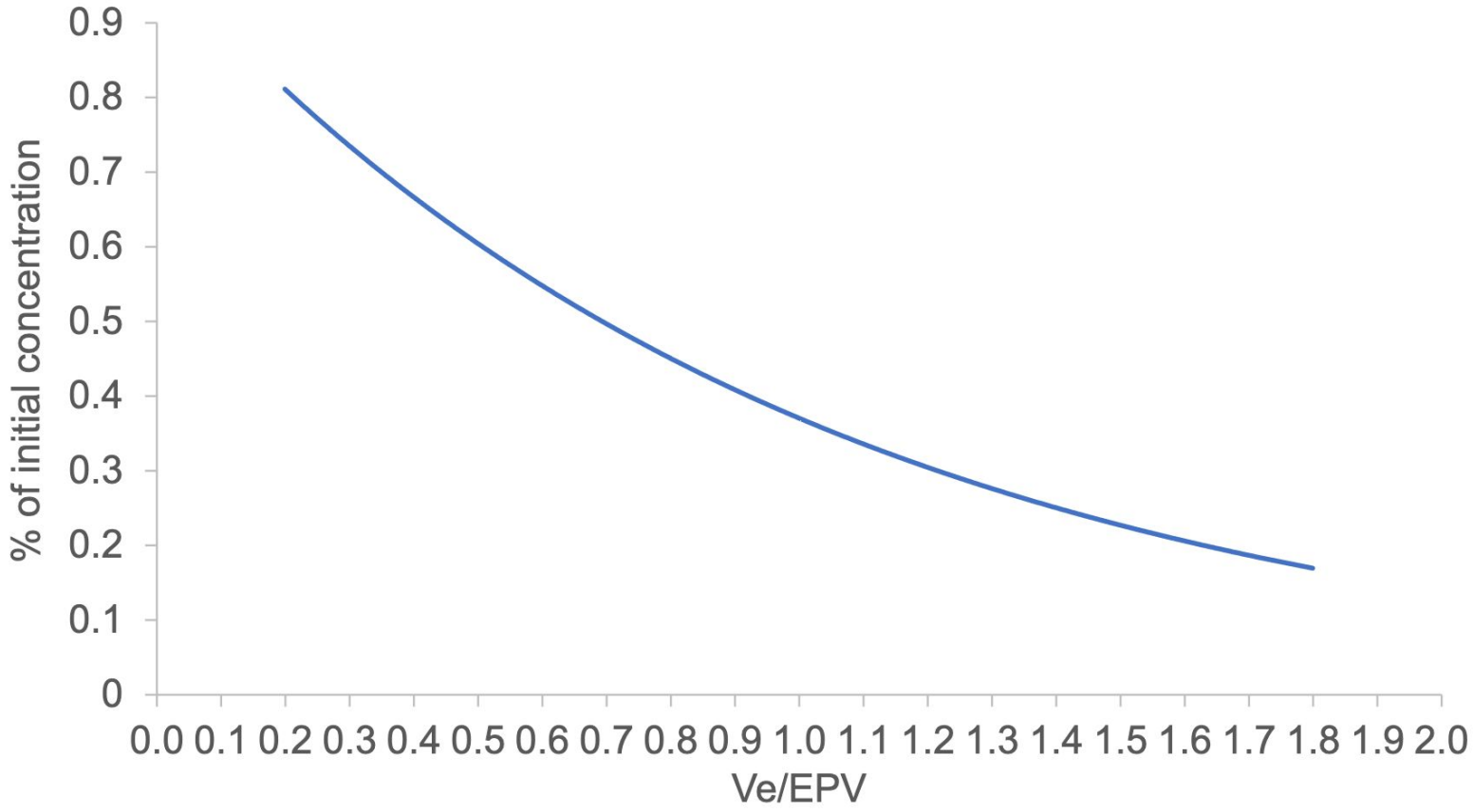
Cervantes et al Am J Kidney Dis. 81(4):475-492 2023

CENTRIFUGATION VS MEMBRANE FILTRATION

Characteristic	Therapeutic Plasma Exchange		Haemodialysis
	Centrifugation	Membrane Filtration	
Mechanism	Centrifugal force	Convection	Diffusion and/or convection
Blood flow, mL/min	10-150	150-200	Continuous: 100-300; intermittent: 200->400
Vascular access	Peripheral vein	Dual lumen catheter	AVF/AVG/Catheter
Blood volume in circuit, mL	180	125	160-280
Plasma extraction, %	80	30	NA
Molecular weight cutoff, Da	>15,000	>15,000	<15,000
Vd, L/kg	Low (<0.3)	Low (<0.3)	Moderate (≤1.5-2)
Protein binding, %	>80	>80	<80
Anticoagulation	Citrate	Heparin	Heparin

Adapted from Cervantes et al Am J Kidney Dis. 81(4):475-492 2023

TPE CLEARANCE



The relationship of percentage decrease in initial concentration for a given substance as a function of volume exchanged (Ve) relative to estimated plasma volume (EPV)

Kaplan, 1999 (A Practical Guide to Therapeutic Plasma Exchange; Blackwell Science)

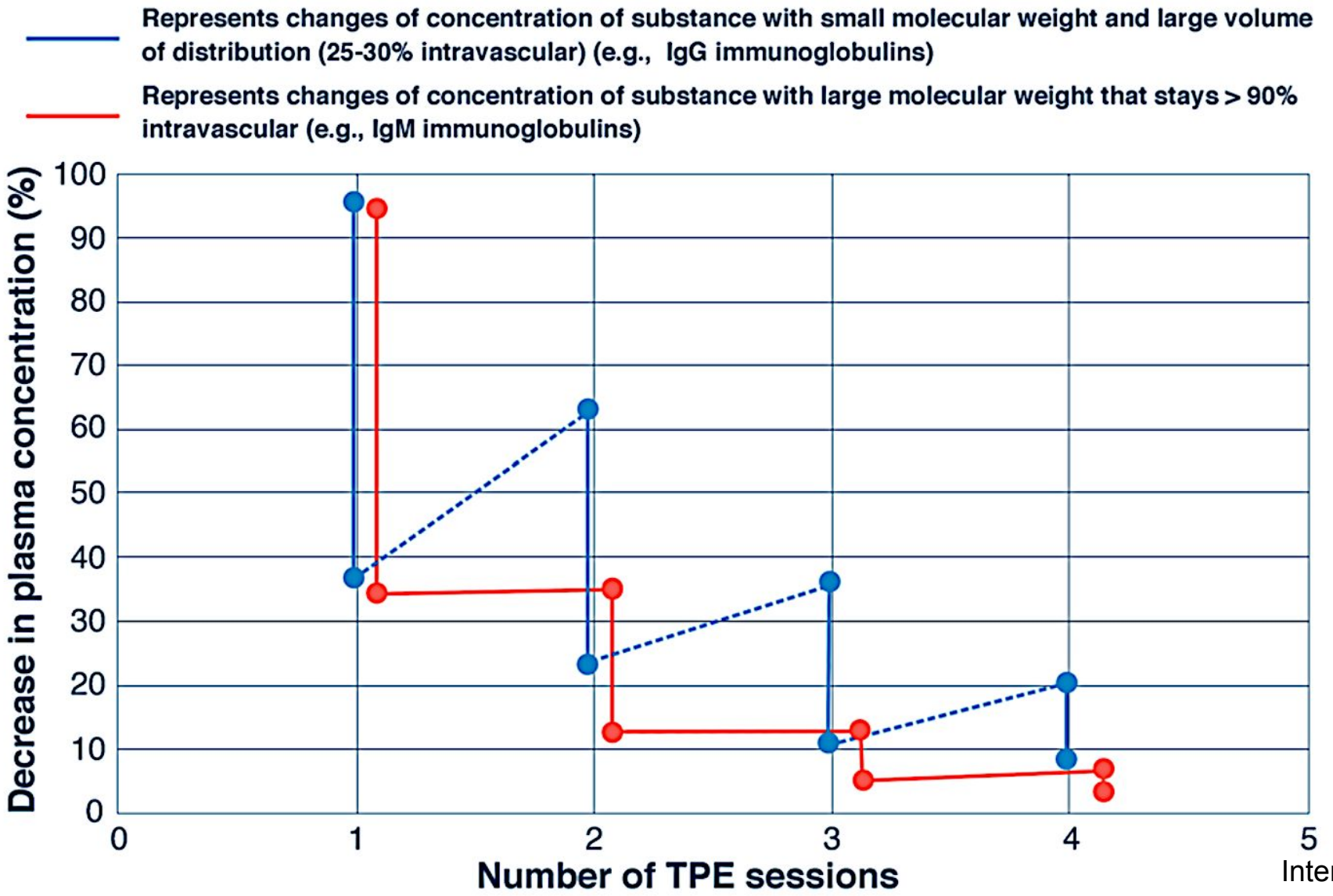
IDEAL TARGET MOLECULE ATTRIBUTES FOR TPE

Table 1. Ideal target molecule characteristics for therapeutic plasma exchange

Ideal Target Molecule Characteristic
Identified etiologic agent or toxic substance
High molecular mass (≥15,000 D)
Slow rate of formation
Low turnover
Low volume of distribution

Clin J Am Soc Nephrol 9: 181–190, 2014.

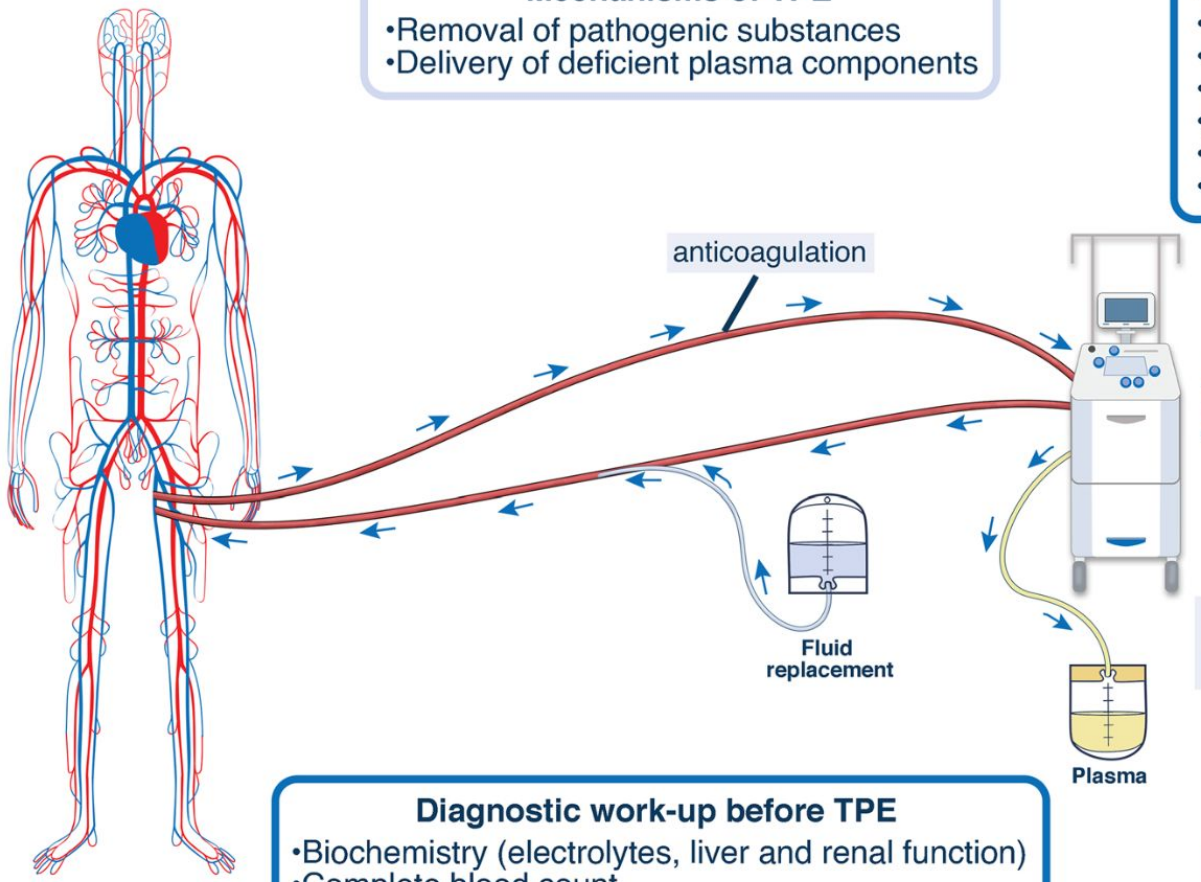
PLASMA CLEARANCE IN CONSECUTIVE TPE SESSIONS



1.2 plasma volume treated per session

Intensive Care Med (2022) 48:1382–1396

PRACTICAL OVERVIEW OF TPE



Mechanisms of TPE
- Removal of pathogenic substances
- Delivery of deficient plasma components

Prescription of TPE
- Vascular access
- Total plasma volume
- Number of sessions
- Interval between sessions
- Anticoagulation
- Type of replacement fluid
- Drug dosing

Diagnostic work-up before TPE
- Biochemistry (electrolytes, liver and renal function)
- Complete blood count
- Coagulation
- Disease-specific tests
- Infection status (HIV, hepatitis..)
- Blood type (ABO, Rhesus)

Monitoring during TPE
- Biochemistry
- Complete blood count
- Coagulation
- Disease-specific tests

Intensive Care Med (2022) 48:1382–1396

Journal of Clinical Apheresis

RESEARCH ARTICLE

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, Caroline R. Alquist, Nicole A. Aqui, Jan C. Hofmann, Reinhard Klingel, Oluwatoyosi A. Onwuemene, Christopher J. Patriquin, Huy P. Pham **... See all authors**

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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	
Acute liver failure	TPE-HV		I	1A	191
	TPE		III	2B	
Age related macular degeneration, dry	Rheopheresis	High-risk	II	2B	193
Amyloidosis, systemic	ß2-microglobulin column	Dialysis-related amyloidosis	II	2B	195
	TPE	Other causes	IV	2C	
Anti-glomerular basement membrane disease (Goodpasture syndrome)	TPE	Diffuse alveolar hemorrhage (DAH)	I	1C	197
	TPE	Dialysis- independence	I	1B	
	TPE	Dialysis-dependence, no DAH	III	2B	
Atopic (neuro-) dermatitis (atopic eczema), recalcitrant	ECP		III	2A	199
	IA		III	2C	

ACUTE INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY (GUILLAIN-BARRÉ SYNDROME)

Incidence: 1-2/100,000/yr	**Indication**	**Procedure**	**Recommendation**	**Category**
	Primary Treatment	TPE	Grade 1A	I
		IA	Grade 1B	I
# reported patients: >300	**RCT**	**CT**	**CS**	**CR**
TPE	21(1874)	0	NA	NA
IA	0	1(39)	6(105)	NA

Description of the disease

Guillain-Barré syndrome (GBS) is an acute, usually symmetrical, and typically ascending, paralyzing disorder caused by inflammation of the peripheral nerves. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP), which comprises up to 90% of GBS cases, is an acute progressive paralyzing illness affecting both motor and sensory peripheral nerves. The remainder of GBS cases are defined by presenting pathogenic and clinical features and classified as acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN), Miller Fisher syndrome, and acute autonomic neuropathy. Weakness or sensory impairment progresses over a period of 12 hours to 28 days before nadir is reached and may involve respiratory and oropharyngeal muscles in severe cases. Thus, mechanical ventilation is required for ~25% of patients. Autonomic dysfunction can cause variability in blood pressure and heart rate resulting in life threatening complications. Spontaneous recovery may occur; however, neurologic complications persist in up to 20% of patients, with half severely disabled at 1 year. Mortality is estimated at 3-5%. Guillain-Barré syndrome is usually preceded by infection or other immune stimulation that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots. Molecular mimicry between microbial and nerve antigens is a major mechanism behind the development of the disorder, as suggested by the association with *Campylobacter jejuni* infection, and the increase of GBS incidence in regions with Zika virus outbreaks. However, how the immune response is shifted towards unwanted autoreactivity is still not well understood. Autoantibodies against various gangliosides, notably GM1 and GD1a, play a role, particularly in AMAN and Miller Fisher syndrome subtypes.

Current management/treatment

Since spontaneous recovery is anticipated in most patients, supportive care is the mainstay of treatment in ambulatory patients. Severely affected patients may require intensive care, mechanical ventilation, and assistance through paralysis and necessary rehabilitation over several months to a year or more. Corticosteroids are not beneficial in the disorder. In trials using TPE and/or IVIG in GBS, AIDP patients represented the majority compared to other variants. TPE was the first therapeutic modality to impact the disease favorably and several major RCTs have confirmed its efficacy. An international RCT compared TPE, IVIG and TPE followed by IVIG in 383 adult patients with severe AIDP and found all three modalities to be equivalent (Plasma Exchange/Sandoglobulin Guillain-Barre Syndrome Trial Group, 1997). There were no differences in the three treatment groups in mean disability improvement at 4 weeks nor the time to be able to walk without assistance (TPE group 49 days, IVIG group 51 days and TPE/IVIG group 40 days). IA avoids the need of replacing human plasma products and was used in one CT and several CS with similar efficacy as TPE. Other therapeutic modalities studied include cerebrospinal fluid filtration, double filtration plasmapheresis, and drug targeting of complement activation. Since IVIG is readily available and a more convenient form of immunomodulatory treatment, it is frequently used as initial therapy; the typical dose is 0.4 g/kg for 5 consecutive days.

Rationale for therapeutic apheresis

The favored pathogenesis of GBS is autoimmune antibody-mediated damage to peripheral nerve myelin. The results of several CTs comparing TPE to supportive care alone indicate that TPE can accelerate motor recovery, decrease time on the ventilator, and decrease time to attainment of other clinical milestones. While recovery with TPE is improved, the duration of disability from AIDP remains significant. The Cochrane Neuromuscular Disease Group review of TPE in AIDP performed in 2012 and updated in 2017 concluded that TPE is an effective treatment of GBS and should be initiated within 7 days of disease onset (Chevret, 2017). It was further concluded that TPE has beneficial effect in severely and mildly affected individuals; with significantly increased proportion of patients able to walk after 4 weeks. Furthermore, TPE was reported to be more cost effective in India than IVIG (Maheshwari, 2018). A priori combining of TPE and IVIG in sequential order was not advantageous and is not recommended. There are insufficient data to conclude on the efficacy of TPE after IVIG failure. Treatment decisions must be made on a case-by-case basis.

Technical notes

Since autonomic dysfunction may be present, affected patients may be more susceptible to intravascular volume shifts during apheresis treatments and should be monitored carefully. Relapses may occur in up to 5-10% of patients 2-3 weeks following either treatment with TPE or IVIG. When relapses occur, additional TPE is typically helpful.

Volume treated: TPE: 1-1.5 TPV; IA: up to 3 TPV **Frequency:** Every other day or daily

Replacement fluid: TPE: Albumin or Plasma; IA: NA

Duration and discontinuation/number of procedures

The typical TPE strategy is to exchange 1-1.5 plasma volumes 5-6 times over 10-14 days, some patients may need additional treatments. Considerations for IA are essentially identical.

Keywords: Acute inflammatory demyelinating polyradiculoneuropathy, GBS, Guillain-Barré syndrome

ASA GUIDELINES FOR AIDP/GBS

The results of several CTs show :
TPE can accelerate motor recovery, decrease time on the ventilator, and decrease time to attainment of other clinical milestones.

The review of TPE in AIDP updated in 2017 concluded that TPE is an effective treatment of GBS and should be initiated within 7 days of disease onset

Furthermore, TPE was reported to be more cost effective in India than IVIG (Maheshwari, 2018).

Combining of TPE and IVIG in sequential order was not advantageous

There are insufficient data to conclude on the efficacy of TPE after IVIG failure.

Treatment decisions must be made on a case-by-case basis.

The typical TPE strategy is to exchange 1-1.5 plasma volumes 5-6 times over 10-14 days, some patients may need additional treatments. Replacement fluid: Albumin or Plasma

NEUROLOGICAL INDICATIONS FOR TPE

Disease	Indication	Category	**Evidence**
Acute disseminated encephalomyelitis	Steroid-refractory	II	2C
AIDP (including GBS)	**Primary treatment**	**I**	**1A**
CIDP (Chronic inflammatory demyelinating polyradiculoneuropathy)		**I**	**1B**
Lambert-Eaton myasthenic syndrome		II	2C
Multiple sclerosis	Acute attack/relapse	II	1A
Myasthenia Gravis	**Short-term treatment** Long-term treatment	**I** II	**1B** 2B
Neuromyelitis optica spectrum disorders	Acute attack/relapse	II	1B
N-methyl-D-aspartate receptor antibody NMDA encephalitis		**I**	**1C**
Paraproteinemic demyelinating neuropathies Chronic acquired demyelinating polyneuropathies	**IgG/IgA/IgM**	**I**	**1B**
PANDAS (pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections)	Exacerbation	II	1B
Phytanic acid storage disease (Refsum disease)		II	2C
VGKC (voltage-gated potassium channel) antibody–related diseases		II	1B

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TRANSPLANT INDICATIONS FOR TPE

Disease	Indication	Category	Evidence
Cardiac	Desensitization	II	1C
HSCT	Major ABOi HPC(M) Major ABOi HPC(A)	II II	1B 2B
Liver	**Desensitization, ABOi living donor**	**I**	**1C**
Kidney, ABO compatible	**Antibody mediated rejection** **Desensitization, living donor**	**I** **I**	**1B** **1B**
Kidney, ABO incompatible	**Antibody mediated rejection** **Desensitization, living donor**	**I** **I**	**1B** **2B**

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PATIENT SL

49 year old male. Past history of SLE and HD for KF.

Living donor kidney transplant from his wife in 2017 (in India)

Baseline creatinine last year 100. Admitted with creatinine of 250 in December 2023,

Given IV methyl prednisolone 0.5/day for 3 days

Kidney biopsy done one week after

Not responded, anti-thymocyte globulin (ATG-Thymoglobuline) given

Biopsied on 20/2/24, reported locally as 'AMR or infection'. Second opinion sought abroad.

CRP normal and no clinical signs of infection

High dose IV IgG given on 24/2/24 and PEX x5 started in 25/2/24

Creatinine is still stuck at 320

Patient went to India. Second biopsy. Creatinine went up to 700.

Started on HD, antibacterials, antifungals and valganciclovir

Back to Mauritius – all immununosuppression stopped

HD stopped after 3 months

RENAL/MULTISYSTEM INDICATIONS FOR TPE

Disease	Indication	Category	Evidence
Anti-GBM disease (Goodpasture syndrome)	**Not receiving dialysis at presentation**	**I**	**1B**
CAPS (catastrophic antiphospholipid syndrome)		**I**	**2C**
FSGS	**Recurrent in kidney transplant**	**I**	**1B**
Myeloma cast nephropathy		II	2B
SLE	**Severe complications**	**I**	**2C**
AAV (ANCA-associated vasculitis)	TPE MPA/GPA/RLV: RPGN, SCr ≥500µmol/l MPA/GPA/RLV: Diffuse alveolar haemorrhage	II I	1A 1C
Vasculitis, other	Hepatitis B polyarteritis nodosa	II	2C

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HAEMOTOLOGICAL INDICATIONS FOR TPE

Disease	Indication	Category	Evidence
Autoimmune hemolytic anemia, severe	Severe cold agglutinin disease	II	2C
Cryoglobulinemia	Severe/symptomatic	II	2A
Hyperviscosity in hypergammaglobulinemia	**Symptomatic** **Prophylaxis for rituximab**	**I** **I**	**1B** **1C**
TMA, complement mediated	**CFH autoantibody**	**I**	**2C**
TMA, drug associated	**Ticlopidine**	**I**	**2B**
TMA, TTP (Thrombotic thrombocytopenic purpura)		**I**	**1A**

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OTHER INDICATIONS FOR TPE

Disease	Indication	Category	Evidence
Acute liver failure	**(needs high volume TPE)**	**I**	**1A**
Wilson disease, fulminant		**I**	**1C**
Familial hypercholesterolemia	Homozygotes/heterozygotes	II	1B
Steroid-responsive encephalopathy associated with autoimmune thyroiditis (Hashimotoencephalopathy)		II	2C
Thyroid storm		II	2C
Overdose, envenomation, and poisoning		iI - III	2C

Evidence based on case reports and series.

TPE most used in amanita mushroom poisoning. Other uses: castor bean ingestion, pesticide/organophosphate poisoning, snake or spider bites, scorpion or Africanized bee stings.

Various drug overdoses/ poisonings – best used for the removal of drugs with a low volume of distribution (<0.2 L/ kg) and/or high-plasma protein binding (>80%). Important factors include the timing of poisoning.

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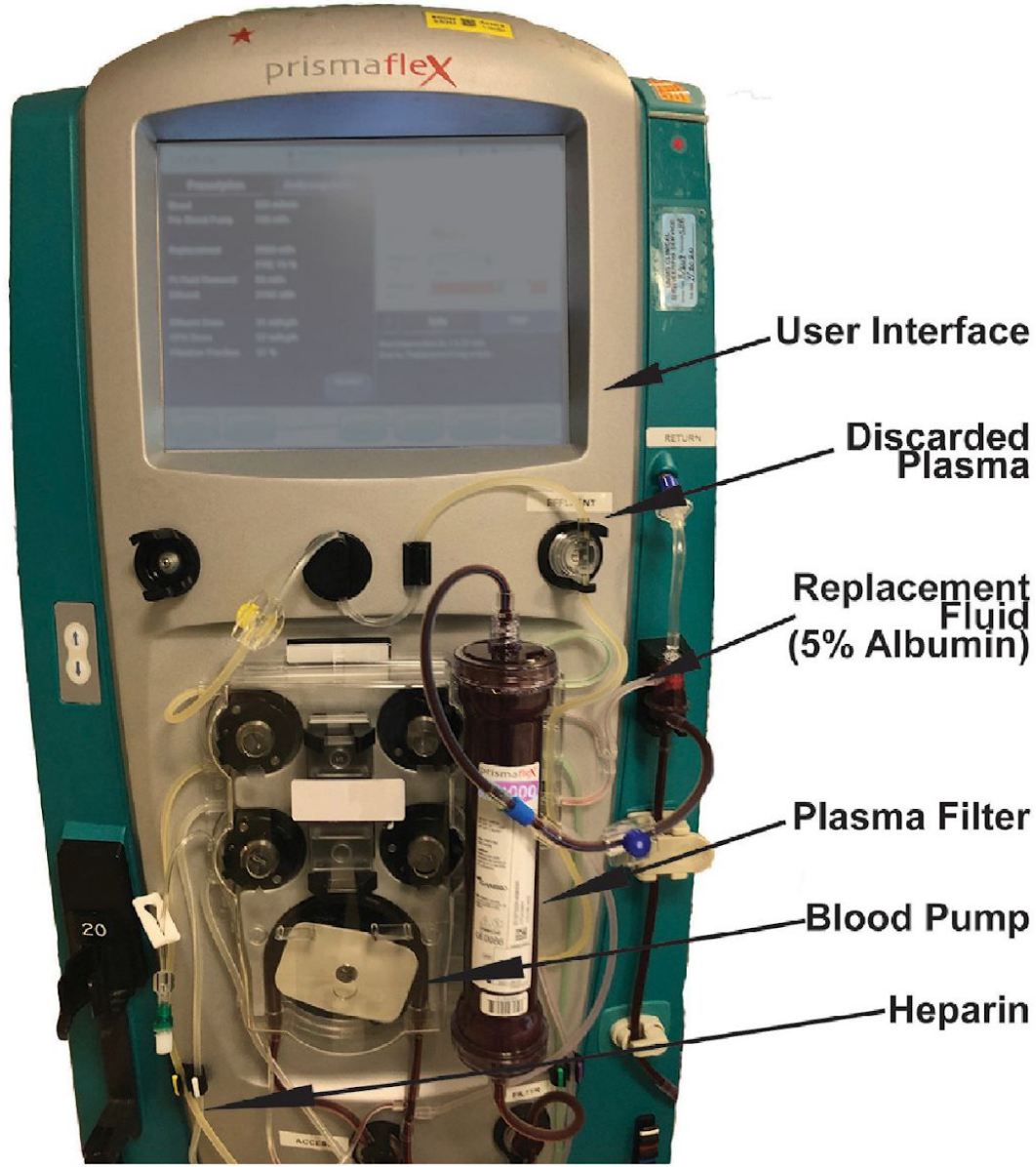
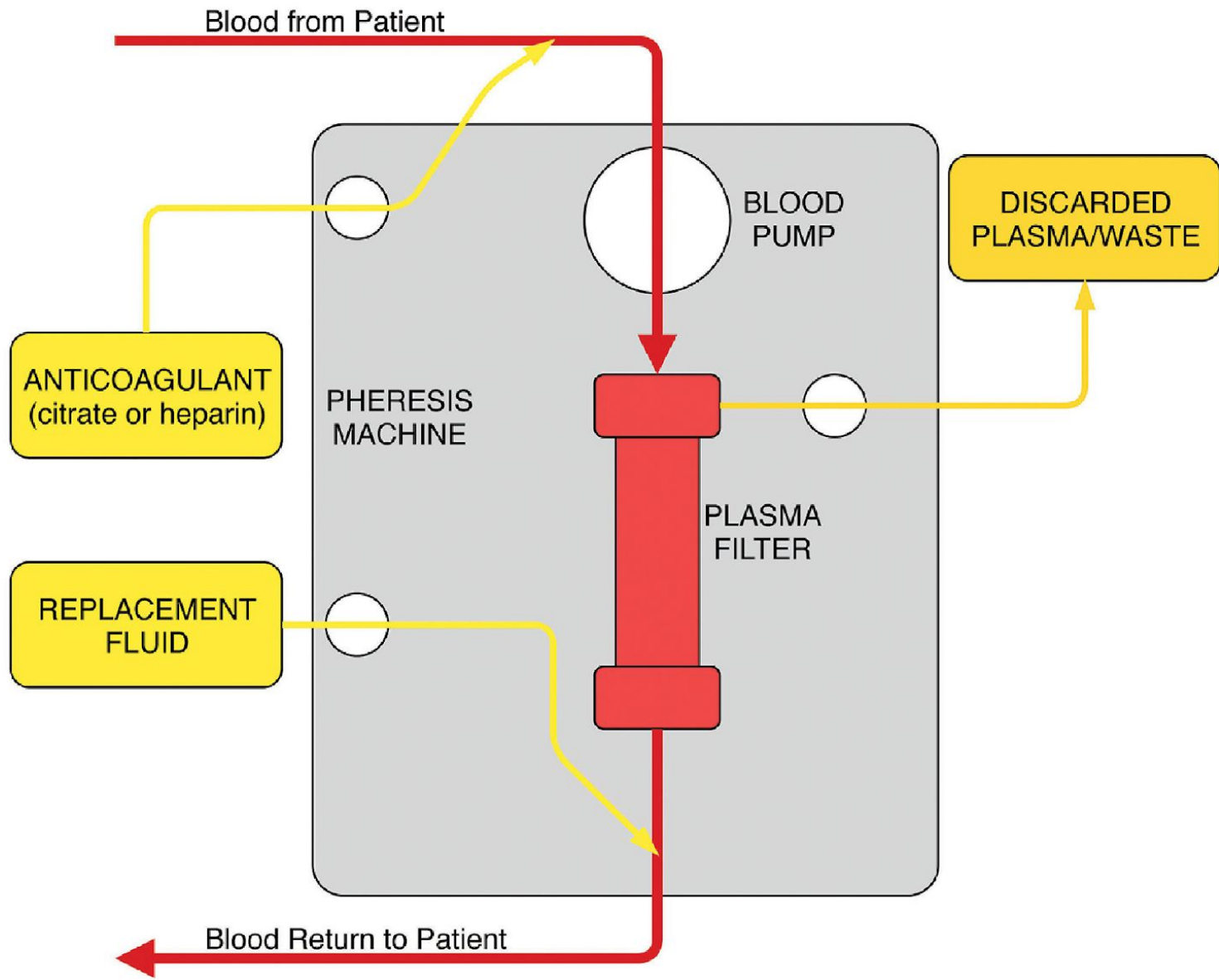

prismaflex
User Interface
Discarded Plasma
Replacement Fluid (5% Albumin)
Plasma Filter
Blood Pump
Heparin


Blood from Patient
BLOOD PUMP
DISCARDED PLASMA/WASTE
ANTICOAGULANT (citrate or heparin)
PHERESIS MACHINE
PLASMA FILTER
REPLACEMENT FLUID
Blood Return to Patient


--:--
Chat
People
Raise
React
View
More
Camera
Mic
Share
Leave
Nadine from Chemtech (External)
Neressa Moodley
DI

NEPHROLOGY UNIT

THERAPEUTIC PLASMA EXCHANGE PRESCRIPTION AND SESSION SHEET

(please file in casenotes)

NID														
Surname														
Forenames														
Unit no.									DOB					

Date of TPE session:	**No of sessions planned:**	**Session number:**
Indication for TPE:		**Patient weight:** **kg**
Ca: **mmol/l** **Alb:** **g/l** **Corr. Ca:** **mmol/l** If alb < 40g/l, use Corrected Ca = measured Ca x 0.02(40-Alb) to decide on IV calcium gluconate supplementation		**INR:** **Fibrinogen:** **APTT:** **Haematocrit:**

Estimated patient plasma volume EPPV: **litres**

To calculate EPPV, you may use the following approximation for *adult* patients with average body habitus:
EPPV (litres) = approx. 40ml/kg (weight of patient). A more precise equation for all *adult* patients especially for those with extremes of body habitus is **EPPV (litres) = 0.065 x weight (kg) x (1 – haematocrit)**

Litres to exchange: **litres (to the nearest 0.2l)**

Recommended volume of plasma to be exchanged (in most cases 3 to 4.5l) =

- 1 to 1.5 x EPPV in almost all indications of TPE
  (closer to 1 in most indications of TPE, closer to 1.5 for TTP/aHUS/ other thrombotic microangiopathies)
- 2-3 x EPPV in fulminant acute liver failure

Exchange fluid type

20% Human Albumin solution **ml**
N. Saline/ Ringer's lactate* **ml**
Fresh frozen plasma **ml**

Guidance on choice on fluid:

Most cases will require only a mix of 20% Human Albumin Solution and either N. Saline NS or Ringer's Lactate (RL) in the proportion of 1 part HAS to 4-5 part NS/RL. RL is preferred in patients with impaired kidney function (and its associated metabolic acidosis) but should not be used in liver failure or lactic acidosis.

Use fresh frozen plasma (FFP) for 25% (up to 50% in severe cases) of the exchange fluid with the rest made up of a mixture of 20% HAS and NS/RL in any of the following situations

□ Pulmonary or other active haemorrhage
□ Within 5 days of a kidney biopsy or surgery
□ INR/ APTT>1.4 or decreased fibrinogen
□ Daily regime of TPE sessions

Use FFP for the totality of the exchange fluid if the patient is receiving TPE for

□ TTP, aHUS or thrombotic microangiopathies
□ acute liver failure
□ severe coagulopathy

Volume of 10% calcium gluconate: **ml**

The volume of 10% calcium gluconate to be given during the TPE can be calculated according to the table on the right. Additional boluses of 10ml may be needed if FFP is used.

(Corr.) Calcium (mmol/l)	Volume of Ca gluconate (ml)
< 2.2	30
2.2-2.4	20
2.4-2.6	10
>2.6	0

Heparin loading dose: **iu** **Heparin maintenance dose:** **iu/hour**

The usual loading dose is 1000iu and the maintenance dose is 500iu/hr but should be adjusted according the individual clotting/bleeding risk.

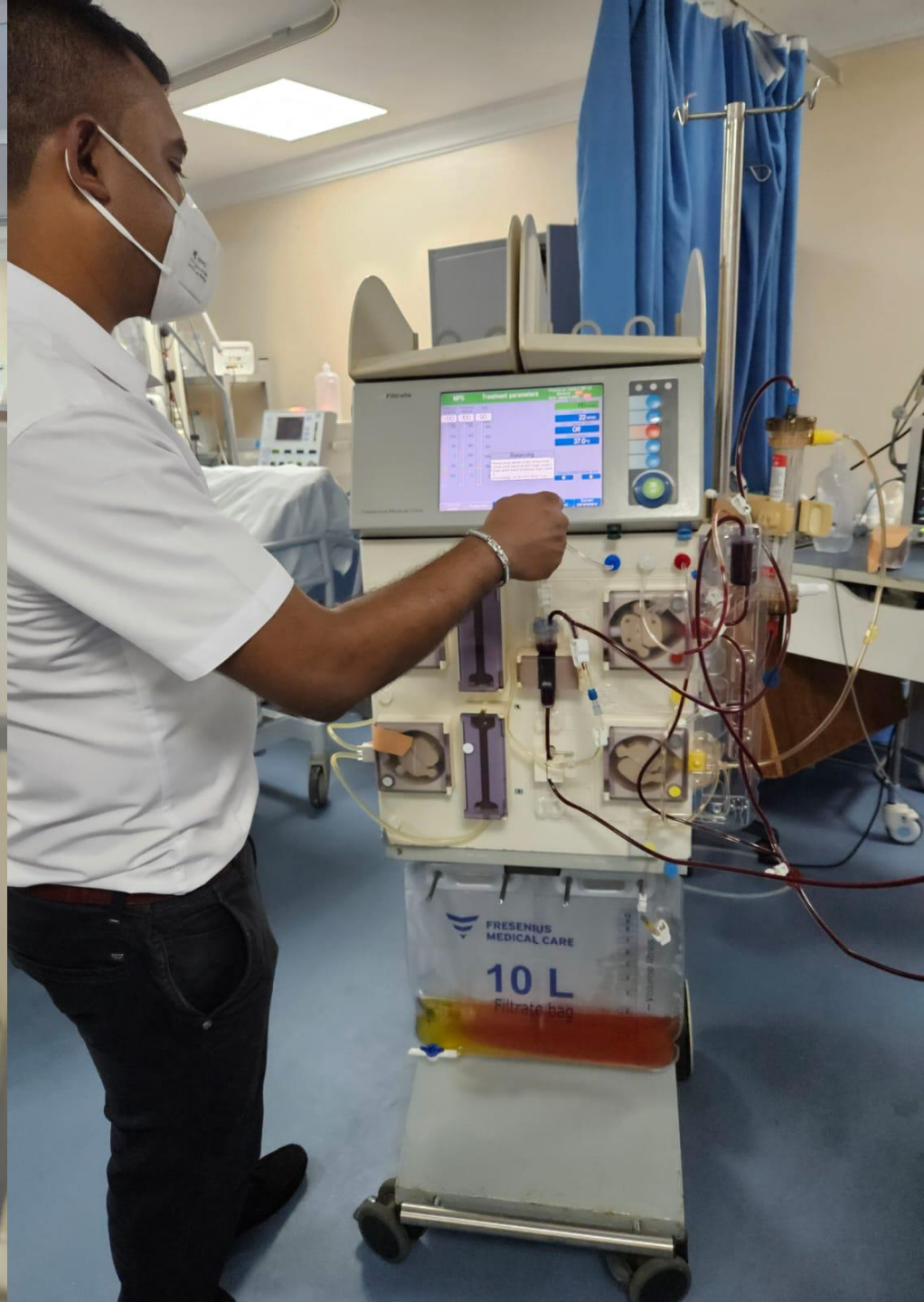
IV Medications as required

Chorpheniramine 10mg IV hydrocortisone 100/200*mg 10% calcium gluconate: 10-20ml

Prescriber (Sp or CIC) **Date:**

Name: **Signature:**


KN95


FRESENIUS
MEDICAL CARE
10 L
Filtrate bag

COMPLICATIONS OF TPE

Complication	Mechanism	Frequency
Access-related		
Peripheral access	Hematomas, nerve damage, sclerosis of veins/ arteries	1.48%
CVC	Thrombosis, infections, pneumothorax, arterial puncture, air embolism	0.11%-0.36% (more complications in subclavian [60%] vs jugular [20%] CVCs)
Ports	Early: pneumothorax, hematomas, arrhythmia, arterial puncture; late: thrombosis, port-pocket infection, pinch-off syndrome	18%
AVF/AVG	Thrombosis	12%-20%
	Inadequate maturation	60%
Anticoagulation-related		
Hypomagnesemia	Citrate chelation	NA
Thrombocytopenia	Heparin-induced thrombocytopenia	1%-5% (not specific to TPE)

COMPLICATIONS OF TPE

Complication	Mechanism	Frequency
Procedure-related		
Anemia	Hematocrit may decrease 10% due to intravascular expansion with hyperoncotic fluids; hemolysis if hypo-oncotic priming solutions used	NA
Hypotension, dyspnea, chest pain	Complement-mediated membrane bioincompatibility; ethylene oxide hypersensitivity	0.4%-15%
Thrombocytopenia	Loss of platelets in the discarded plasma, circuit clotting, or dilutional effect by replacement fluid	NA
Vitamin deficiencies	Depletion of protein-bound vitamins (A, B_6, B_{12}, C, and E and β-carotene) of 24%-48% with rebound to pretreatment levels within 24 h	NA

COMPLICATIONS OF TPE

Complication	Mechanism	Frequency
Replacement fluid–related		
Anaphylactoid reactions	Transfusion of IgA in donor plasma to patients with selective IgA deficiency; contamination with bacteria, endotoxins, pyrogens; presence of prekallikrein activator and bradykinin (ACEI); antibodies to polymerized albumin (rare)	0.02%-0.07%
Coagulopathy	Depletion of coagulation factors and its inhibitors related to albumin replacement alone (Table 4)	0.06%-0.14% for thrombosis, 0.06% for bleeding
Electrolyte/acid base abnormalities	Hypokalemia (albumin), hypocalcemia (frozen plasma), hypomagnesemia (frozen plasma), metabolic alkalosis (frozen plasma)	9%-19.6% for hypocalcemia, 0.03% for alkalosis
Infection	Hypogammaglobulinemia (albumin), viral transmission (frozen plasma)	NA
Transfusion-related lung injury	Transfusion of donor antibodies (frozen plasma)	NA
Hypervolemia	Administration of replacement fluid	NA

FIRST YEAR OF TPE IN MOHW

Hospital	Sex	Age	Indication	No of sessions
AGJH	M	46	Guillain-Barre Syndrome	4
BCH	F	48	SLE - complications	4
BCH	M	43	Guillain-Barre Syndrome	4
BCH	M	50	SLE - complications	4
VH	M	76	Guillain-Barre Syndrome	4
VH	M	38	Acute antibody mediated rejection	5
SSRNH	M	72	Myasthenia Gravis	4
VH	F	13	Guillain-Barre Syndrome	5

THANK YOU!

https://karger.com/bpu/article/53/5/358/867584/Nomenclature-of-Extracorporeal-Blood-Purification