



Εκλεκτική αφαίρεση σε ειδικά μόρια

Έκτωρ Άννινος, ΓΝΑ Αλεξάνδρα

Οκτώβριος 2019

Μέθοδοι αφαίρεσης

- ανοσοπροσρόφηση (immunoadsorption)
- προσρόφηση σε φίλτρο κυτταρίνης-θειικής δεξτράνης (dextran sulphate-cellulose adsorption)
- συστήματα καθίζησης της LDL σε ηπαρινισμένο εξωσωματικό κύκλωμα (heparin extracorporeal LDL precipitation system)
- πλασμαφαίρεση διπλής διήθησης και την πλασμαφαίρεση με θερμοδιήθηση (double filtration plasmapheresis and thermofiltration plasmapheresis)
- άμεση προσρόφηση λιποπρωτεϊνών με την χρήση αιμοδιήθησης (direct adsorption of lipoprotein (DALI) using haemoperfusion)

Table 1. ASFA 2010 indication categories, recommendation grade and TA modality in CVDs

Disease	Category	Grade	TA modality	
Cardiac allograft rejection				
Prophylaxis	I	1A	ECP	extracorporeal photopheresis
Treatment of rejection	II	1B	ECP	
Treatment of antibody-mediated rejection	III	2C	TPE	therapeutic plasma exchange
DCM				
NYHA II-IV	III	2B	IA	immunoadsorption
NYHA II-IV	III	2C	TPE	
FH				
Homozygotes	I	1A	Selective removal	
Heterozygotes	II	1A	Selective removal	
Homozygotes with small blood volume	II	1C	TPE	
Hyperviscosity in monoclonal gammopathies				
Treatment of symptoms	I	1B	TPE	
Prophylaxis for rituximab	I	1C	TPE	
TTP	I	1A	TPE	

Table 2. Patients' treatment criteria

Treatment criteria published by international organisations

The Food and Drug Administration (FDA) – USA

- (1) Functional homozygotes with LDL >500 mg dL⁻¹
- (2) Functional heterozygotes with no known CVD but LDL >300 mg dL⁻¹
- (3) Functional heterozygotes with known CVD and LDL >200 mg dL⁻¹ (2)

The International Panel on Management of FH – Spain

- (1) FH homozygotes
- (2) Heterozygotes with symptomatic coronary artery disease in whom LDL is >4.2 mmol L⁻¹ (162 mg dL⁻¹) or decreases by $<40\%$ despite maximal medical management (2)

The German Federal Committee of Physicians and Health Insurance Funds – Germany

- (1) FH homozygotes
- (2) Patients with severe hypercholesterolaemia in whom maximal dietary and drug therapy for >1 year has failed to lower cholesterol sufficiently (2)

The Hyperlipidaemia Education & Atherosclerosis Research Trust UK (HEART-UK) – UK

- (1) FH homozygotes in whom LDL is reduced by $<50\%$ and/or >9 mmol L⁻¹ (348 mg dL⁻¹) with drug therapy
 - (2) FH heterozygotes or a 'bad family history' with objective evidence of coronary disease progression and LDL >5.0 mmol L⁻¹ (193 mg dL⁻¹) or decreases by $<40\%$ despite drug therapy
 - (3) Progressive coronary artery disease, severe hypercholesterolaemia and Lp(a) >60 mg dL⁻¹ in whom LDL remains elevated despite drug therapy (2)
-

Table 1 Guidelines for using lipoprotein apheresis

Country	Recommendation
USA	<ul style="list-style-type: none">• Homozygous FH: LDL-c \geq 500 mg/dl (12.9 mmol/L) on maximal possible drug therapy• Heterozygous FH: LDL-c \geq 300 mg/dl (7.8 mmol/L) (0–1 additional risk factor), LDL-c \geq 200 mg/dl (5.2 mmol/L) (\geq 2 additional risk factors or additional high lipoprotein(a)), LDL \geq 160 mg/dl (4.1 mmol/L) (if at very high risk)
Germany	<ul style="list-style-type: none">• Homozygous FH• Severe hypercholesterolaemia (including but not restricted to heterozygous FH): LDL-c elevated on maximal possible drug therapy (taking the overall risk of the patient into account)• Lipoprotein(a): progressive CVD (clinically and on imaging) despite optimal control of all other risk factors and lipoprotein(a) \geq 60 mg/dl
Japan	<ul style="list-style-type: none">• Homozygous FH• Heterozygous FH: total cholesterol \geq 250 mg/dl (6.5 mmol/L) on maximal possible drug therapy
UK	<ul style="list-style-type: none">• Homozygous FH: LDL-c reduction $<$ 50% on max. drug therapy or LDL-c \geq 350 mg/dl (9.1 mmol/L)• Other hypercholesterolaemia (including heterozygous FH): CVD progression and LDL-c \geq 190 mg/dl (4.9 mmol/L) or lower if lipoprotein(a) elevated or LDL-c reduction $<$ 40%
Australia	<ul style="list-style-type: none">• Homozygous FH: LDL-c \geq 270 mg/dl (7.0 mmol/L) on maximal possible drug therapy• Heterozygous FH: CVD and LDL-c \geq 193 mg/dl (5.0 mmol/L) on maximal possible drug therapy• Alternative criteria (homozygous FH and heterozygous FH): $<$ 50% reduction on maximal possible drug therapy
Spain	<ul style="list-style-type: none">• Homozygous FH• Heterozygous FH: LDL-c \geq 200 mg/dl (5.2 mmol/L) with CVD or \geq 300 mg/dl (7.8 mmol/L) without CVD

Lipoprotein Apheresis Systems

HELP: Heparin-induced extracorporal LDL precipitation	Based on the precipitation of apolipoprotein B containing lipoproteins in acidic conditions by forming complexes with other proteins
DALI: Direct adsorption of lipoproteins	Positively charged apolipoprotein B binds to negatively charged polyacrylate anions
Liposorber: Dextran sulfate	Positively charged apolipoprotein B binds to negatively charged dextran sulfate
MONET: Lipid filtration	Series of filters eliminate lipoproteins based on size
TheraSorb: Apolipoprotein B antibodies	Plasma is passed through columns containing apolipoprotein B antibodies that bind lipoproteins
Lipopac: Apoprotein (a) antibodies (this is only used for research purposes)	Plasma is passed through columns containing apoprotein (a) antibodies that bind Lp(a)

LA methods

Filtration

Lipidfiltration

MONET

Adsorption

TheraSorb™ LDL

Liposorber D

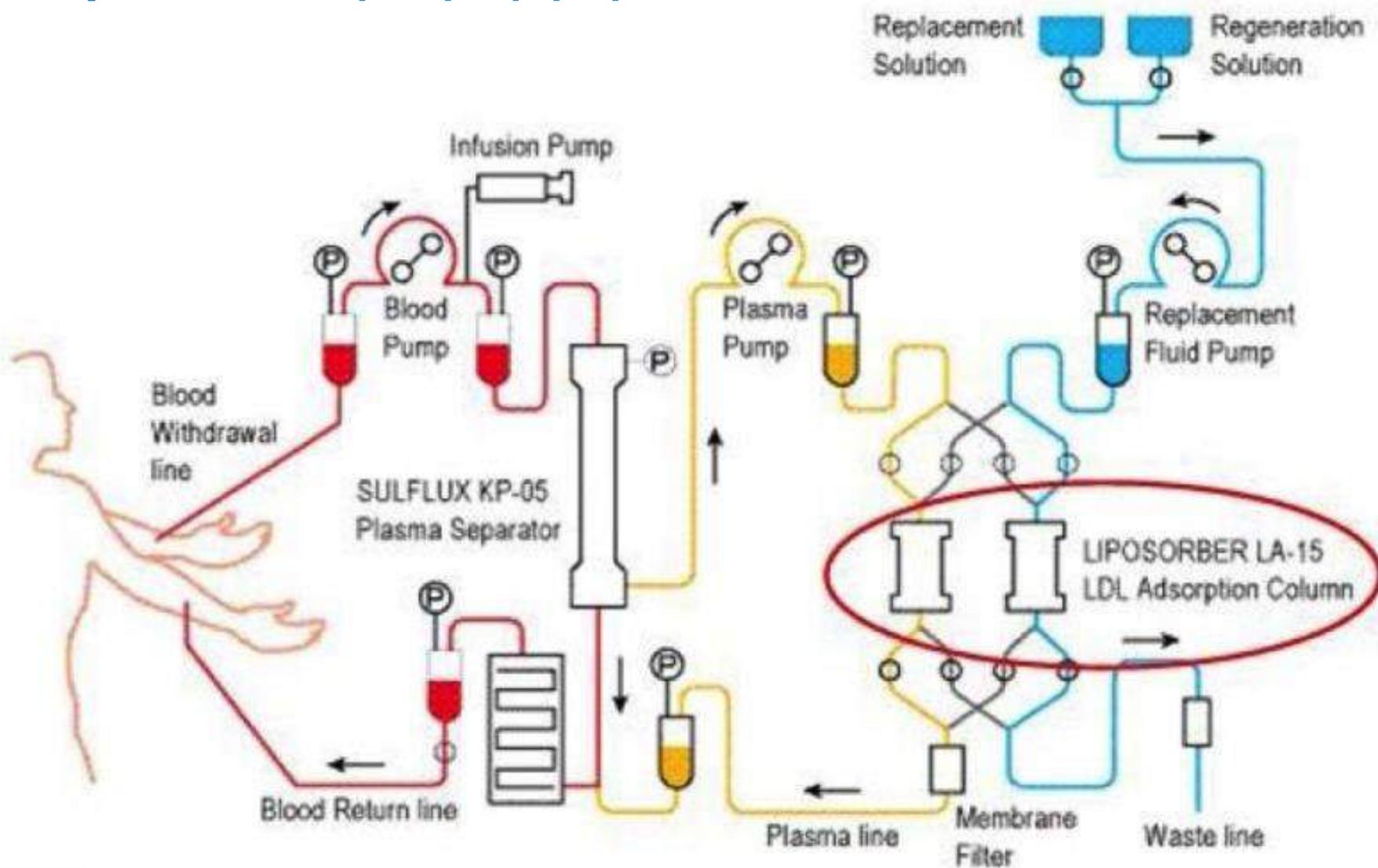
DALI

Specific columns for Lp(a)

Precipitation

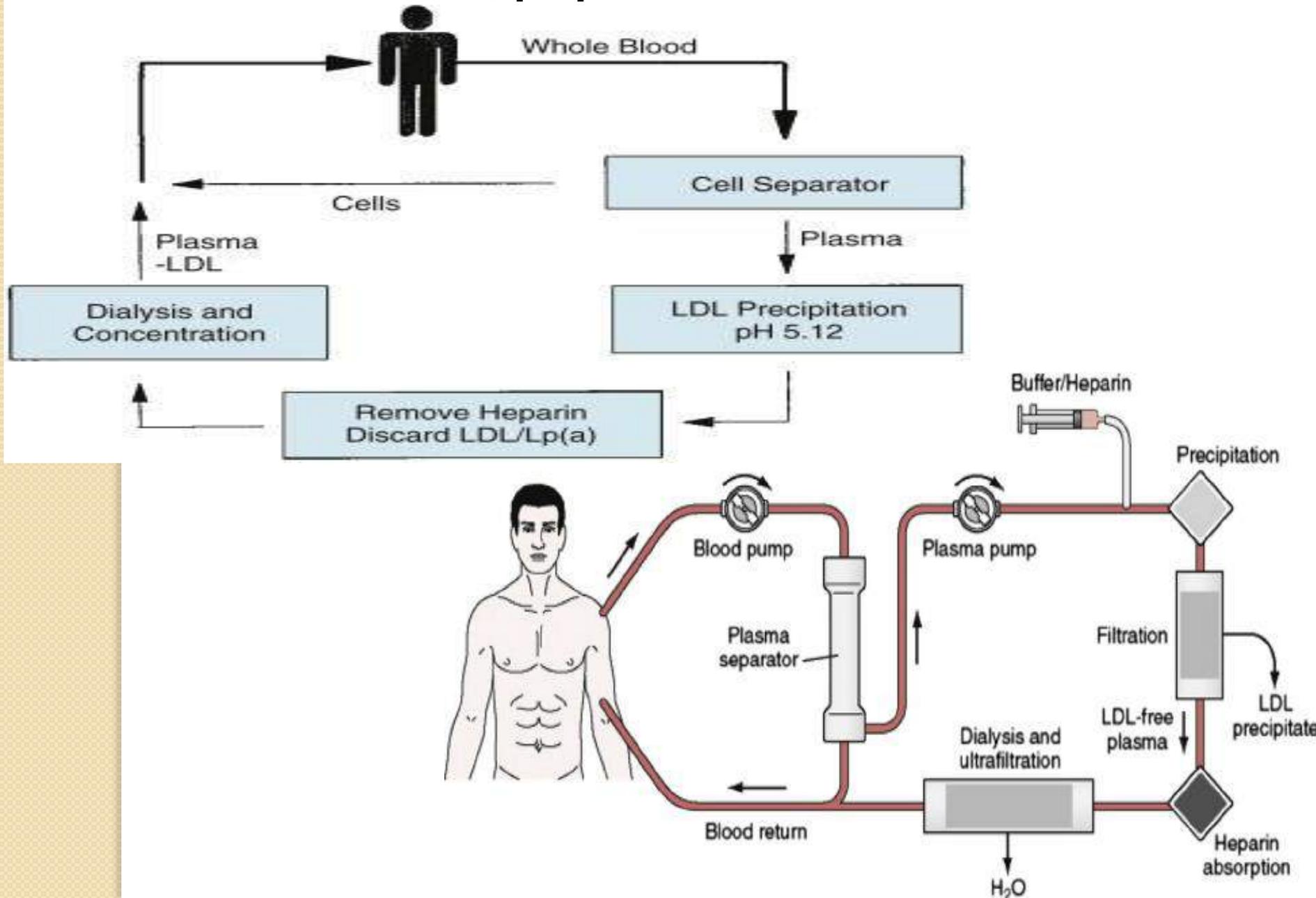
HELP

Liposorber (προσρόφηση)

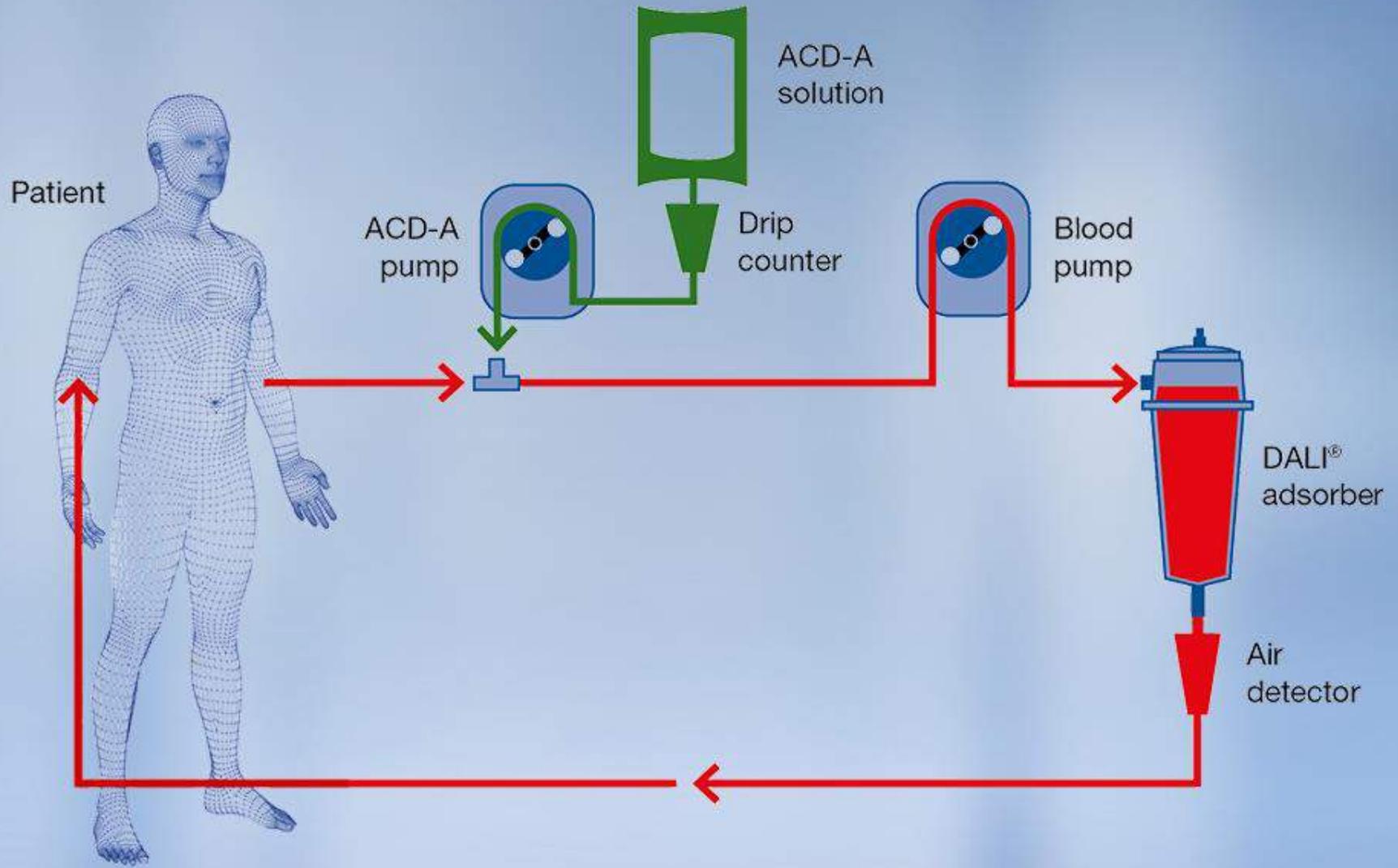


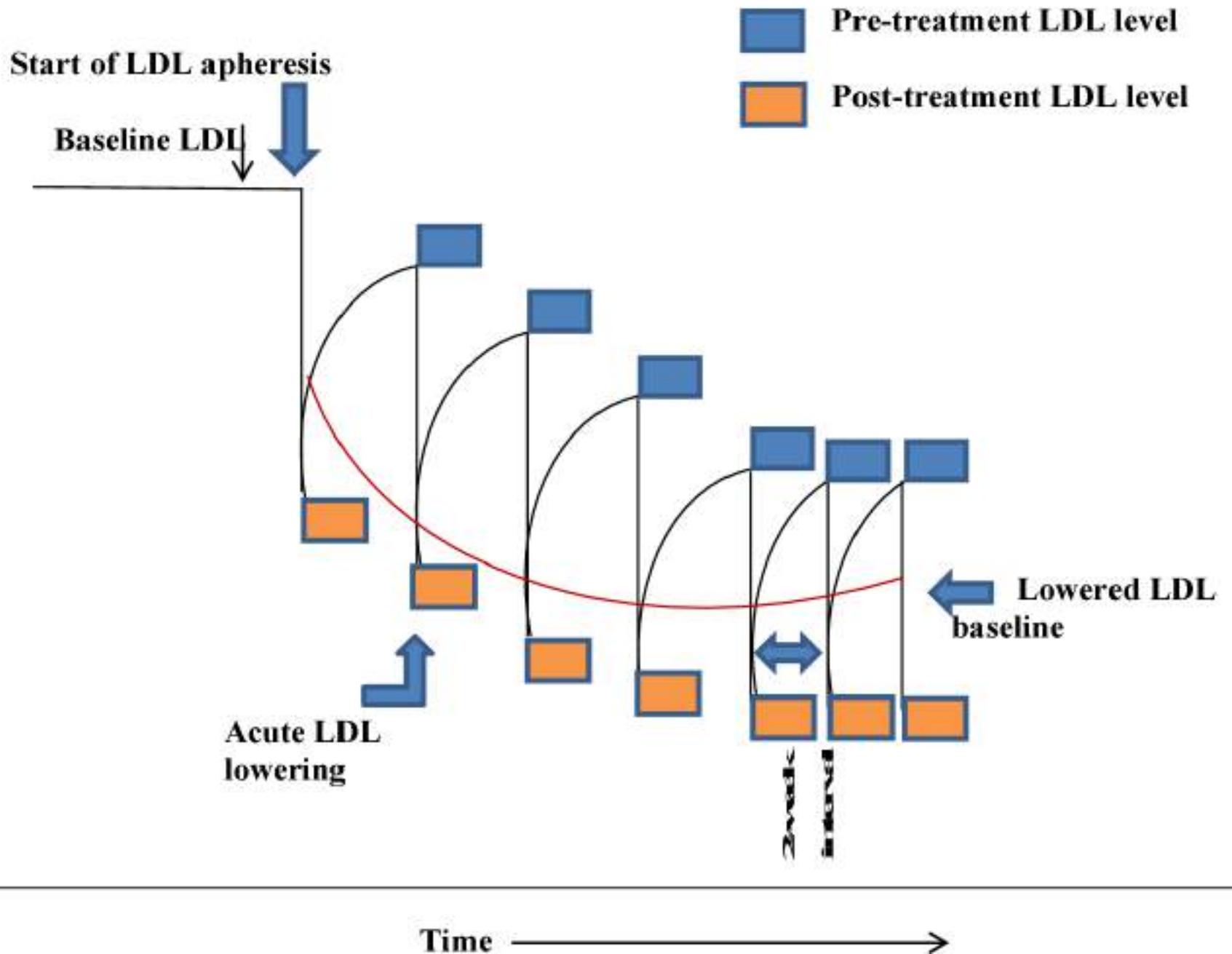
HELP System

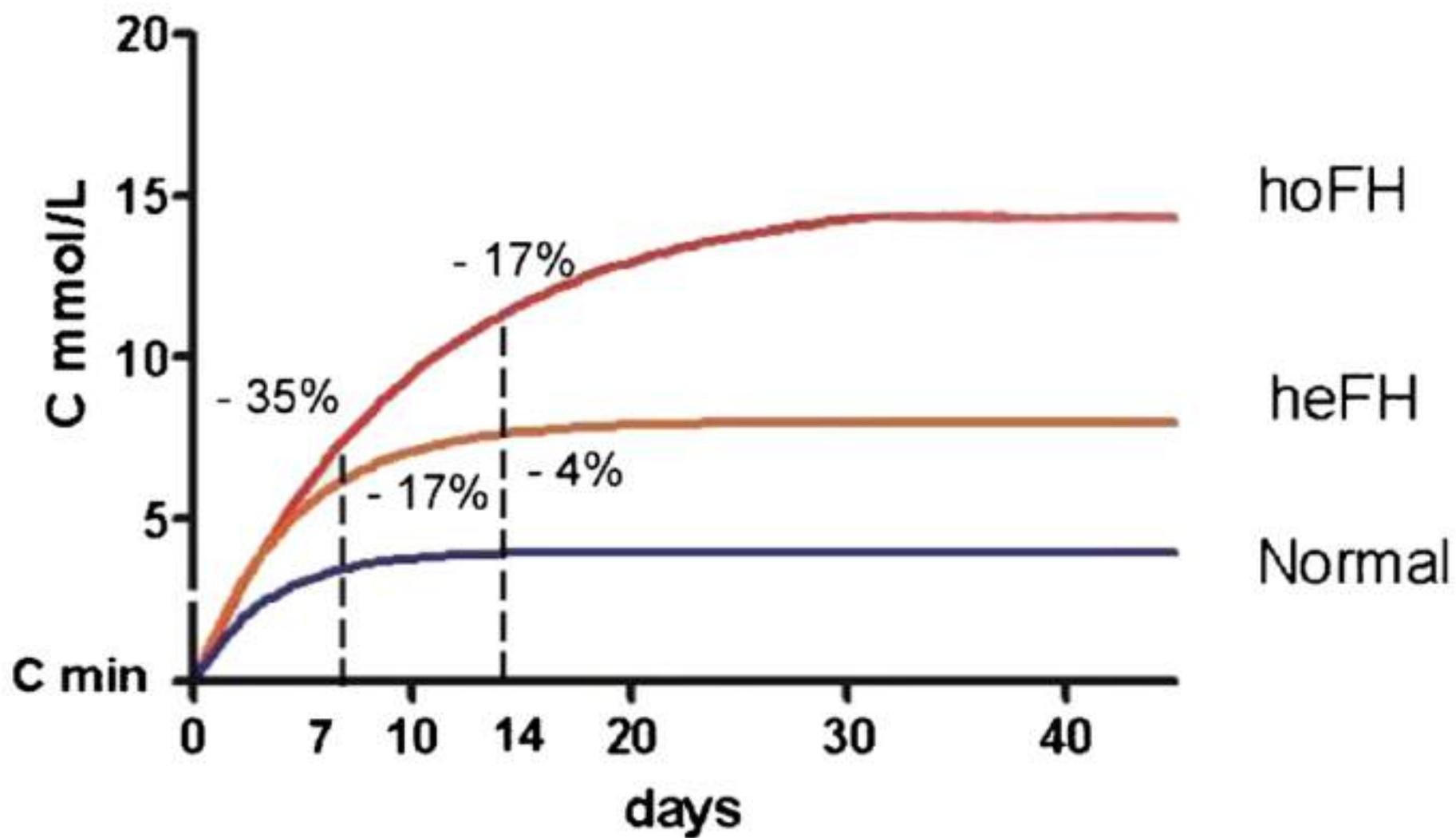
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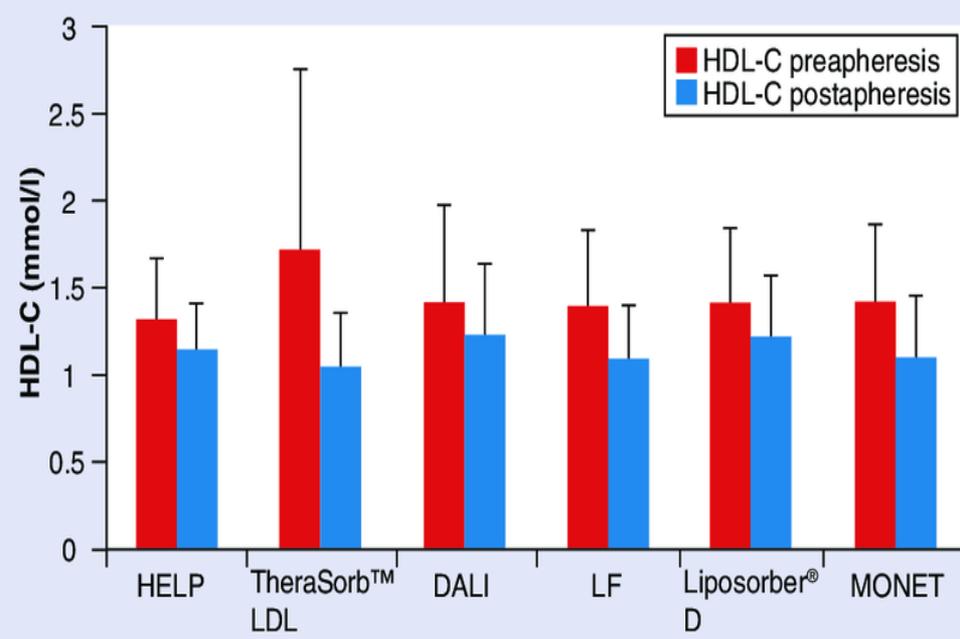
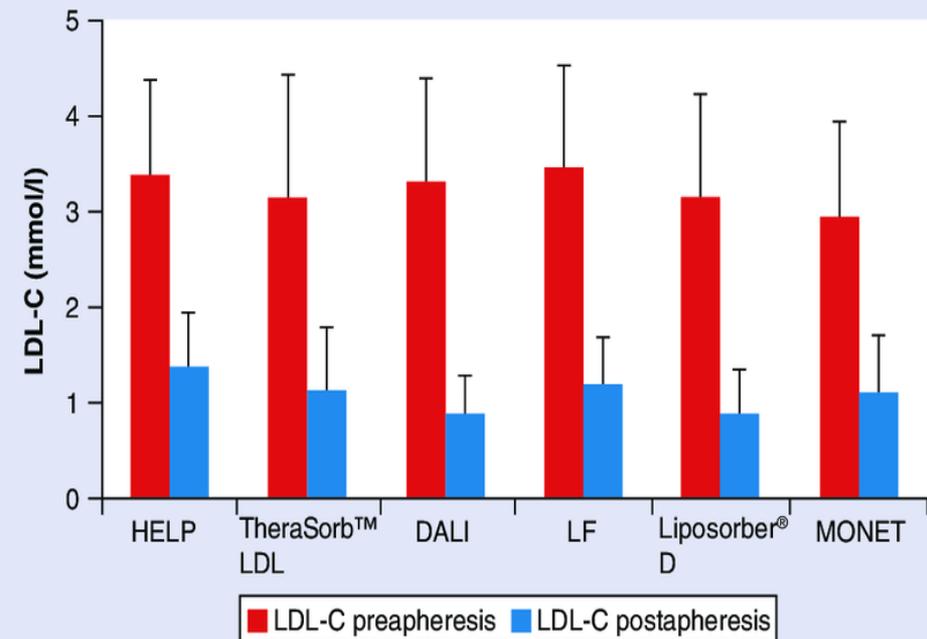
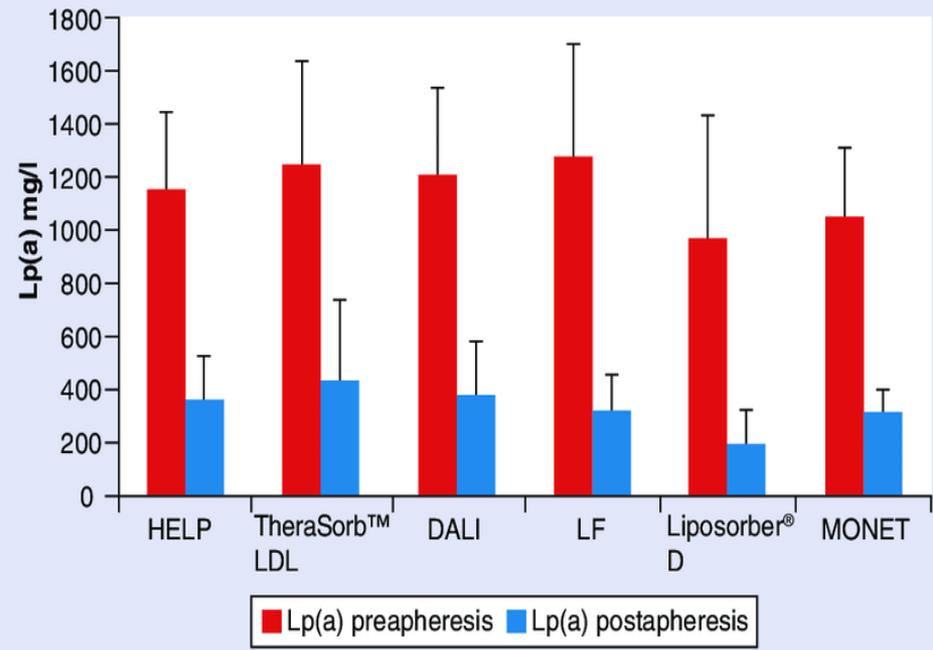
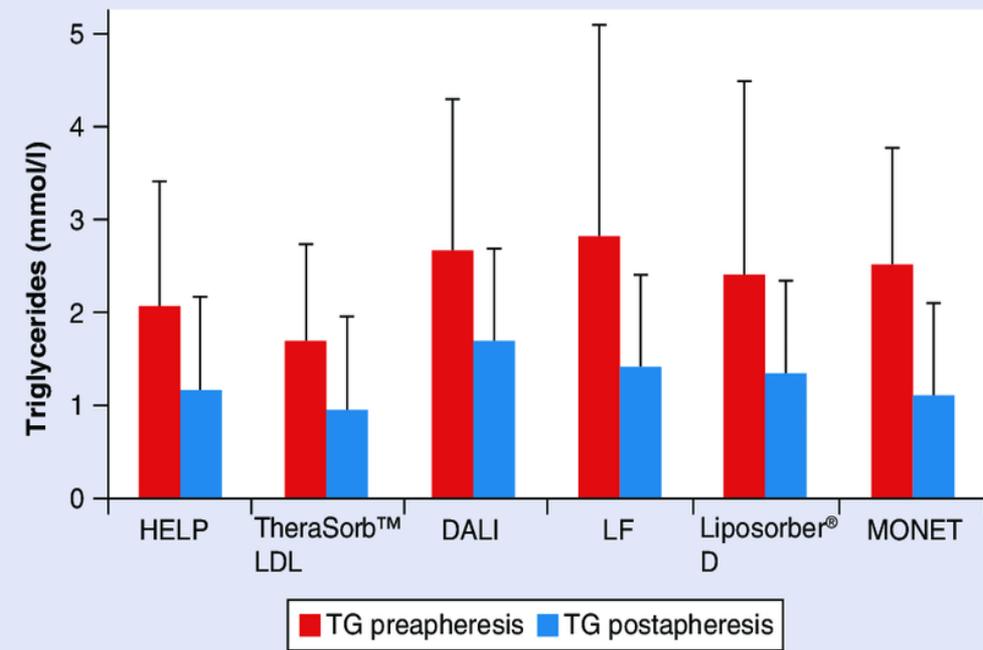


DAI (προσρόφηση)









Mean percentage reduction of plasma lipoproteins and fibrinogen with different methods of LDL apheresis [8]

	DFPP (%)	Thermofiltration (%)	HELP (%)	DALI (%)	DSA (%)	IA (%)
LDL cholesterol	56-62	61	55-61	53-76	49-75	62-69
HDL cholesterol	25-42	6	5-17	5-29	4-17	9-27
Lp(a)	53-59	61	55-68	28-74	19-70	51-71
Triglycerides	37-49	56	20-53	29-40	26-60	34-49
Fibrinogen	52-59	42	51-58	13-16	17-40	15-21

Review

Recommendations for the use of LDL apheresis

G.R. Thompson, HEART-UK LDL Apheresis Working Group¹

Angiographic changes according to treatment group in FH trials

Study (ref)	Group	n	Duration (years)	$\Delta\%$ LDL-C	% P ^a	% NC + R ^a
Type II [34]	Diet	57	5	-5	49	51
SCOR [35]	Diet	32	2	-12	41	59
		89	3.5	-7.5 ^b	46 ± 5 ^c	54 ± 5 ^c
Type II [34]	Drug	59	5	-26	32	68
SCOR [35]	Drug	40	2	-39	20	80
FHRS [30]	Drug	19	2	-44	21	79
LAARS [31]	Drug	21	2	-47	52	48
L-CAPS [33]	Drug	11	2	-34	64	34
		150	2.6	-35 ^b	33 ± 15 ^c	67 ± 15 ^c
Waidner [28]	Aph	25	3	-58	32	68
FHRS [30]	Aph	20	2	-53	10	90
LAARS [31]	Aph	21	2	-63	43	57
Richter [32]	Aph	23	4.6	-51	0	100
L-CAPS [33]	Aph	25	2	-43	8	92
		114	2.7	-53 ^b	18 ± 18 ^c	82 ± 18 ^c

Familial Hypercholesterolaemia Regression Study: a randomised trial of low-density-lipoprotein apheresis

Lancet 1995; 345: 811-16

G R Thompson, V M G Maher, S Matthews, Y Kitano, C Neuwirth, M B Shortt, G Davies, A Rees, A Mir, R J Prescott, P de Feyter, A Henderson

simva+Apheresis/2w vs simva+resin

	Apheresis	Drugs only	p
(A) Category*			
Progressor	2	4	} 0.62§
No change/mixed responder	13	11	
Regressor	5	4	
(B) Mean per patient¶			
Diameter stenosis (%)	-1.8 (4.0)	-2.2 (5.5)	0.83
Minimum lumen diameter (mm)	-0.01 (0.17)	0.05 (0.18)	0.29
Mean lumen diameter (mm)	-0.06 (0.22)	0.02 (0.16)	0.22

Low Density Lipoprotein Apheresis Improves Regional Myocardial Perfusion in Patients With Hypercholesterolemia and Extensive Coronary Artery Disease

J Am Coll Cardiol 1996;28:1696-704

The LDL-Apheresis Atherosclerosis Regression Study (LAARS)

WIM R. M. AENGEVAEREN, MD, ABRAHAM A. KROON, MD,
 ANTON F. H. STALENHOF, MD, PhD, GERARD J. H. UIJEN, PhD,
 TJEERD VAN DER WERF, MD, PhD, FACC

Nijmegen, The Netherlands

Table 2. Regional Hyperemic Mean Transit Time Values in the Three Myocardial Perfusion Areas

	LAD Region	LCx Region	RCA Region	All Regions
LDL apheresis group	n = 17	n = 18	n = 7	n = 42
Baseline HMTT (s)	3.28 (0.97)	2.93 (0.66)	4.61 (1.85)	3.35 (1.18)
Follow-up HMTT (s)	2.81 (0.86)	2.66 (0.67)	3.57 (0.79)	2.87 (0.82)
p value	0.02	0.08	0.11	0.001
Medication group	n = 15	n = 13	n = 7	n = 35
Baseline HMTT (s)	2.78 (0.78)	3.15 (1.22)	2.95 (1.35)	2.95 (1.06)
Follow-up HMTT (s)	2.75 (0.81)	3.11 (1.07)	3.14 (0.79)	2.96 (0.90)
p value	0.88	0.93	0.74	0.96
Difference (p value)*	0.17	0.51	0.14	0.04

Apheresis 2/w

Long-Term Efficacy of Low-Density Lipoprotein Apheresis on Coronary Heart Disease in Familial Hypercholesterolemia

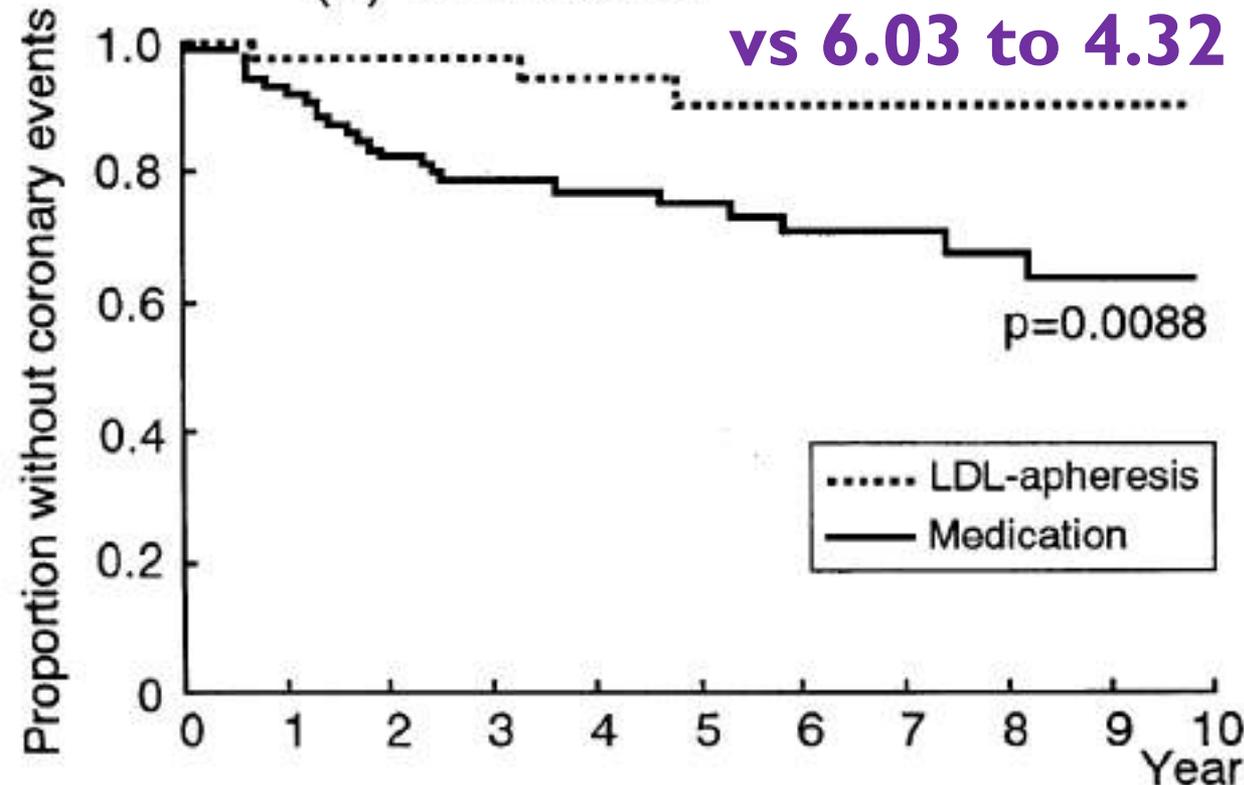
Hiroshi Mabuchi, MD, PhD, Junji Koizumi, MD, PhD, Masami Shimizu, MD, PhD, Kouji Kajinami, MD, PhD, Susumu Miyamoto, MD, PhD, Kousei Ueda, MD, PhD, and Tadayoshi Takegoshi, MD, PhD, for the Hokuriku-FH-LDL-Apheresis Study Group*

Am J Cardiol. 1998 Dec 15;82(12):1489-95. **Apheresis every 2 w for 6 years**

(A) All Patients

**LDL:7.42 to 3.13 mmol/L (58%)
vs 6.03 to 4.32 mmol/L (28%).**

mortality: NS



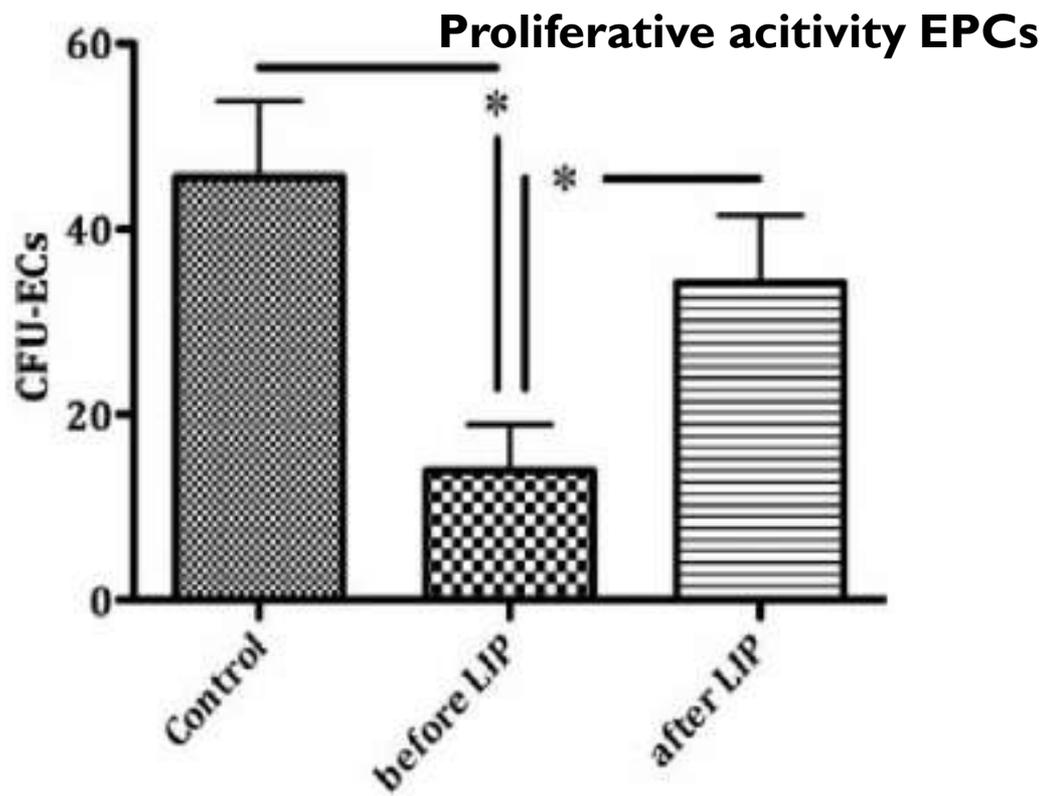
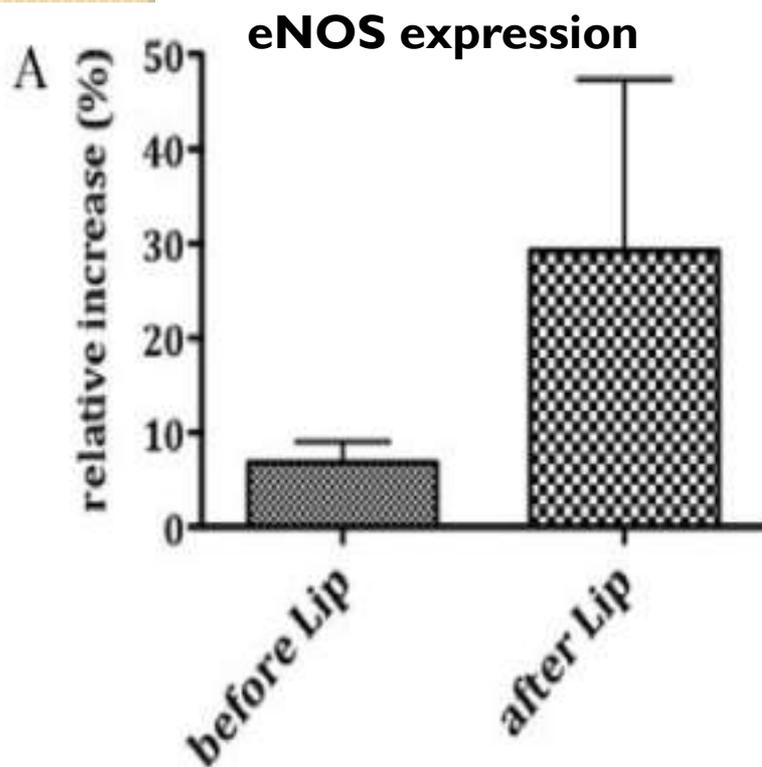
Impact of LDL-Apheresis on inflammation and microcirculation

Michael J. Koziol^{*,} Gerhard A. Mueller

Journal of Clinical Apheresis 24:180–185 (2009)

LDL Lipid Apheresis Rapidly Increases Peripheral Endothelial Progenitor Cell Competence

Daniel Patschan,^{1*} Susann Patschan,^{1†} Elvira Henze,¹ Johannes T. Wessels,^{1,2} Michael Koziol,¹ and Gerhard A. Müller¹



Retrospective Analysis of Long-term Lipid Apheresis at a Single Center

Michael J Koziolk,¹ Ulrich Hennig,¹ Antonia Zapf,² Carsten Bramlage,¹ Clemens Grupp,³
Victor W Armstrong,⁴ Frank Strutz,¹ and Gerhard A Müller¹

MACE (death, cerebrovascular accident, myocardial infarction, limb amputation, and renal vascular involvement) decreased from 7.02% events /patient/year at the start of lipid apheresis to 1.17% during lipid apheresis

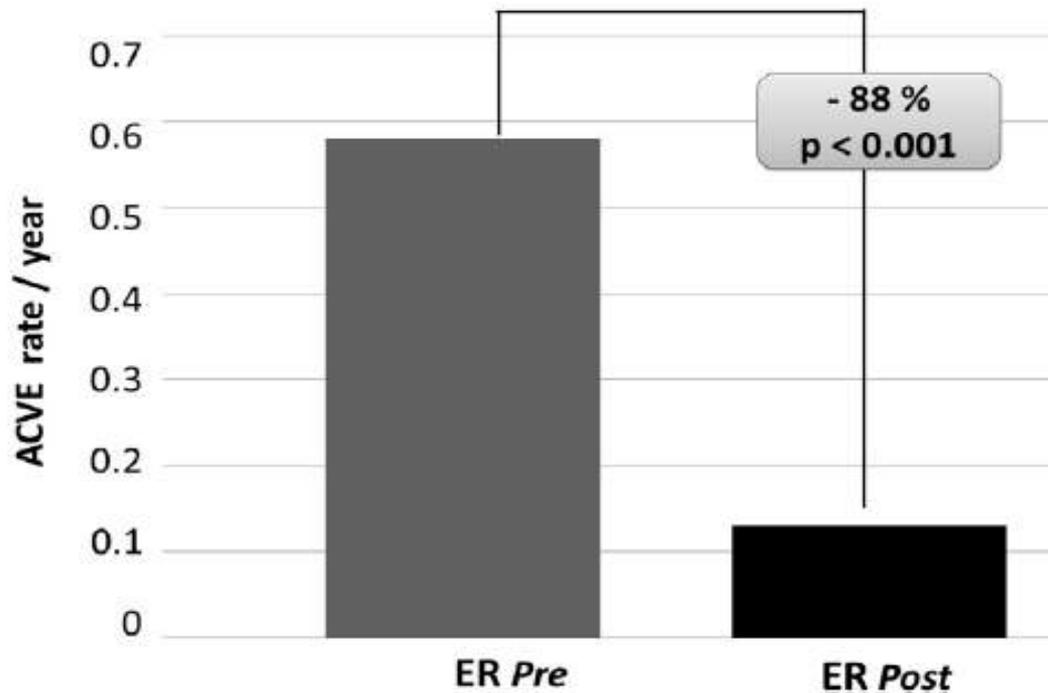
Myocardial revascularization decreased from 22.8% to 3.8% /patient/year.

Atherosclerosis Supplements 18 (2015) 268e272

The incidence of cardiovascular events is largely reduced in patients with maximally tolerated drug therapy and lipoprotein apheresis.
A single-center experience

T. Sampietro*, F. Sbrana, F. Bigazzi, A. Ripoli, B. Dal Pino, E.M. Pasanisi, C. Petersen,

Statins (73%) Fibrates (30%) Ezetimibe (33%) apheresis/2w





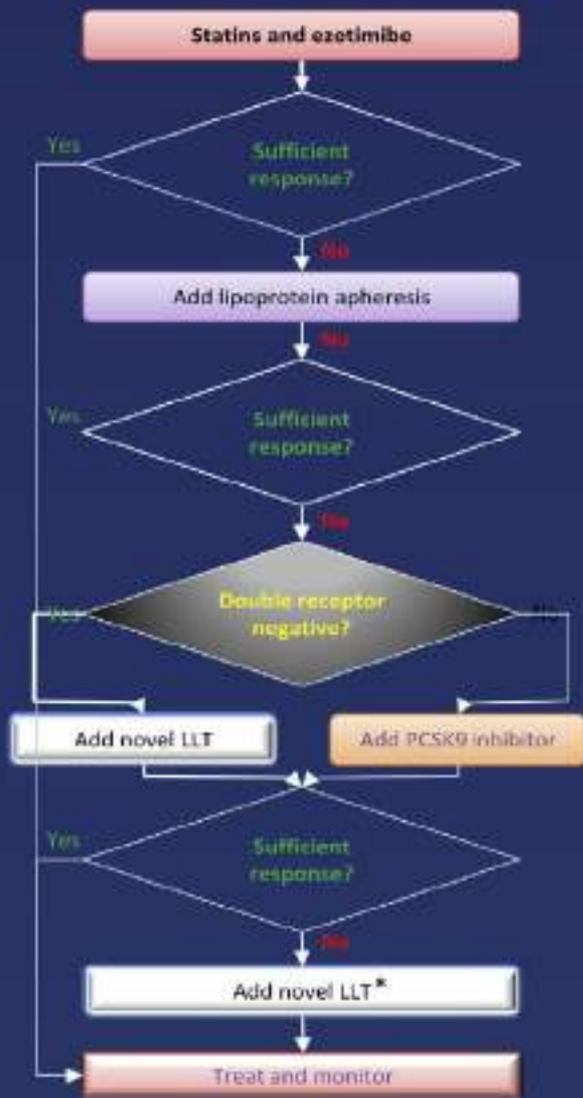
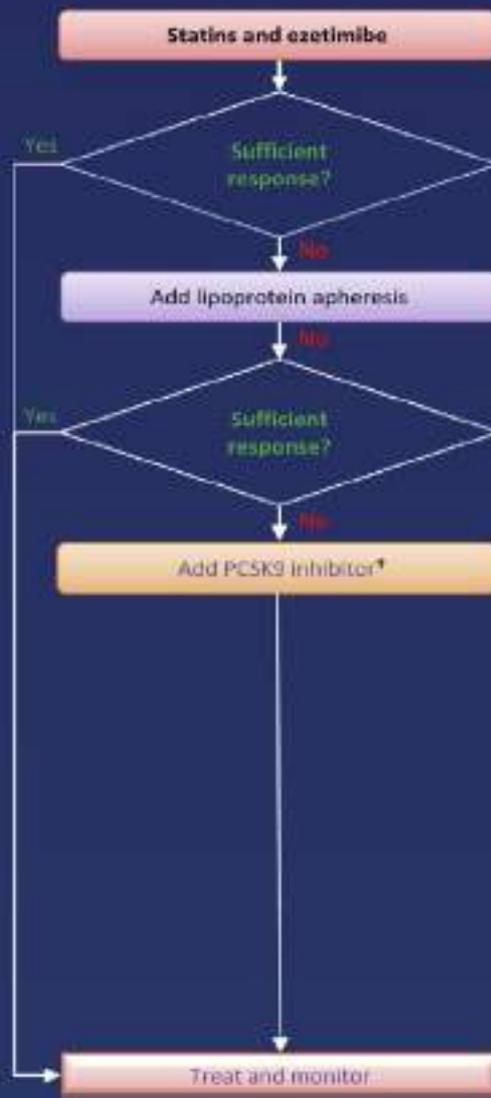
2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk

9.1.2.2 *Homozygous familial hypercholesterolaemia*. HoFH is a rare and life-threatening disease. The clinical picture is characterized by extensive xanthomas, marked premature and progressive CVD, and TC >13 mmol/L (>500 mg/dL). Myocardial infarction and aortic stenosis before the age of 20 years are common. The frequency of HoFH is estimated to be 1:250 in the general population. The early identification of the disease and referral to a specialized clinic is crucial. The mainstay of treatment is intensive LDL-lowering drug therapy and, when available, with lipoprotein apheresis. This treatment (every 1–2 weeks) can decrease plasma LDL-C levels by 55–70%. The procedure frequency may be adjusted for each patient as lipid levels, symptoms, and other disease-related parameters change. Maximally tolerated pharmacological therapy must be maintained.³⁶⁸ For a more detailed discussion on HoFH, see the EAS consensus statements.^{366,368}

specialized clinic is crucial. The patients should be treated with intensive LDL-lowering drug therapy and, when available, with lipoprotein apheresis. This treatment (every 1–2 weeks) can decrease plasma LDL-C levels by 55–70%. The procedure frequency may be adjusted

Box 6 Management of dyslipidaemia in women

Lipid-lowering drugs should not be given when pregnancy is planned, during pregnancy, or during the breastfeeding period. However, for severe FH patients, bile acid sequestrants (which are not absorbed) and/or LDL apheresis may be considered.

A**Homozygous familial hypercholesterolemia****B****Heterozygous familial hypercholesterolemia****C****Lp(a) hyperlipoproteinemia**

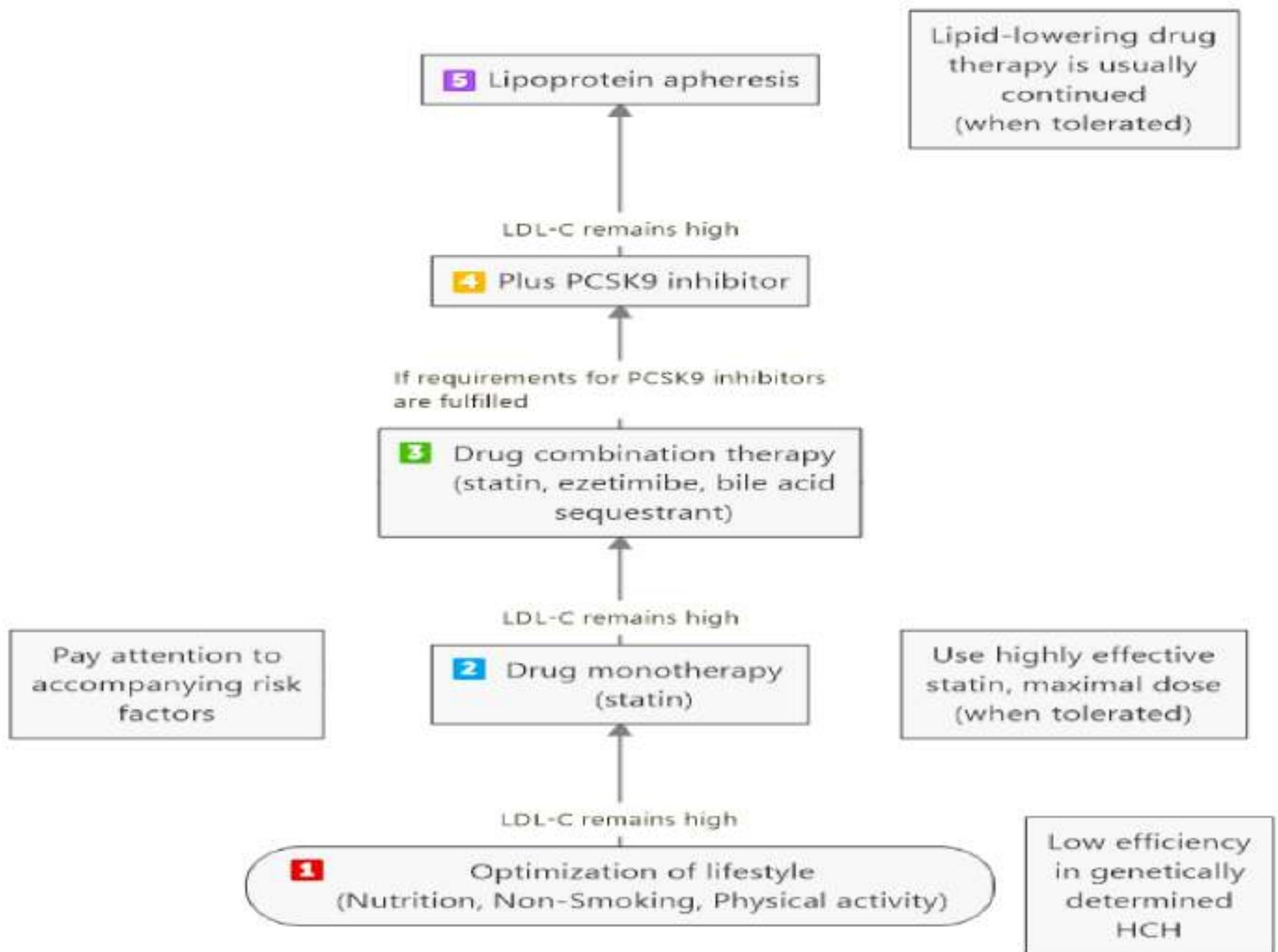


Table 1 - Results of the systematic review on the clinical studies evaluating the efficacy of lipoprotein apheresis to reduce increased lipoprotein(a) levels.

First author, year ^{ref}	Study design	Patients (n)	Mean duration (years±SD)	Concomitant lipid-lowering medications	Outcome	
					Laboratory	Clinical
Jager, 2009 ²⁴	Retrospective	120	5.0±3.6	Yes	Apheresis reduced mean Lp(a) levels by 73% (from 118±42 mg/dL to 33±16 mg/dL, p<0.0001)	Apheresis reduced the mean annual number of MACE by 86% (from 1.05 to 0.14, p<0.0001)
Koziolok, 2009 ²⁵	Retrospective	20	20	Yes	Apheresis reduced mean Lp(a) levels by 69.5-71.2%	Apheresis reduced the mean annual number of MACE by 84% (from 7.02 to 1.17)
Stefanutti, 2010 ²⁶	Randomised	21	1	Yes	Apheresis reduced mean Lp(a) levels by 58% (p<0.001)	No new cardiovascular events were observed during the study period
Leebman, 2013 ²⁷	Prospective	170	2	Yes	Apheresis reduced mean Lp(a) levels by 70% (from 111±47 mg/dL to 28±12 mg/dL, p<0.0001)	Apheresis reduced the mean annual number of MACE by 78% (from 0.41 to 0.09, p<0.0001)
von Dryander, 2013 ²⁸	Prospective	87	2	Yes	Apheresis reduced mean Lp(a) levels by 55-73%	Apheresis reduced the rate of cardiovascular events by 54-83.5%
Safarova, 2013 ²⁹	Prospective	30	1.5	Yes	Apheresis reduced mean Lp(a) levels by 73% (from 103±23 mg/dL to 29±16 mg/dL, p<0.0001)	Apheresis decreased median PDS (-2.0, p<0.01) and improved MLD (0.20, p=0.04)
Rosada, 2014 ⁴⁰	Retrospective	37	6.8	Yes	Apheresis reduced mean Lp(a) levels by 68% (from 112±34 mg/dL to 36±12 mg/dL, p<0.0001)	Apheresis reduced the mean annual number of MACE by 77% (from 0.35 to 0.08, p<0.0001)
Groß, 2015 ⁴¹	Prospective	59	5 (range, 0.5-22)	Yes	Apheresis reduced mean Lp(a) levels by 66-70%	Apheresis reduced the mean annual number of cardiovascular events by 83%
Schletter, 2015 ^{42,43}	Registry	688	4	Yes	Apheresis reduced mean Lp(a) levels by 72% (from 89 mg/dL to 25 mg/dL)	Apheresis reduced the mean annual number of cardiovascular events by 90%
Heigl, 2015 ^{44,45}	Retrospective	118	6.8	Yes	Apheresis reduced mean Lp(a) levels by 53% (from 127.2±67.3 mg/dL to 60.0±19.5 mg/dL, p<0.0001)	Apheresis reduced the mean annual number of cardiovascular events by 80% (p<0.0001)

SD: standard deviation; Lp(a): lipoprotein(a); MACE: major adverse coronary events; PDS: percent diameter stenosis; MLD: minimal lumen diameter.

Lipoprotein Apheresis in Patients With Maximally Tolerated Lipid-Lowering Therapy, Lipoprotein(a)-Hyperlipoproteinemia, and Progressive Cardiovascular Disease

Prospective Observational Multicenter Study

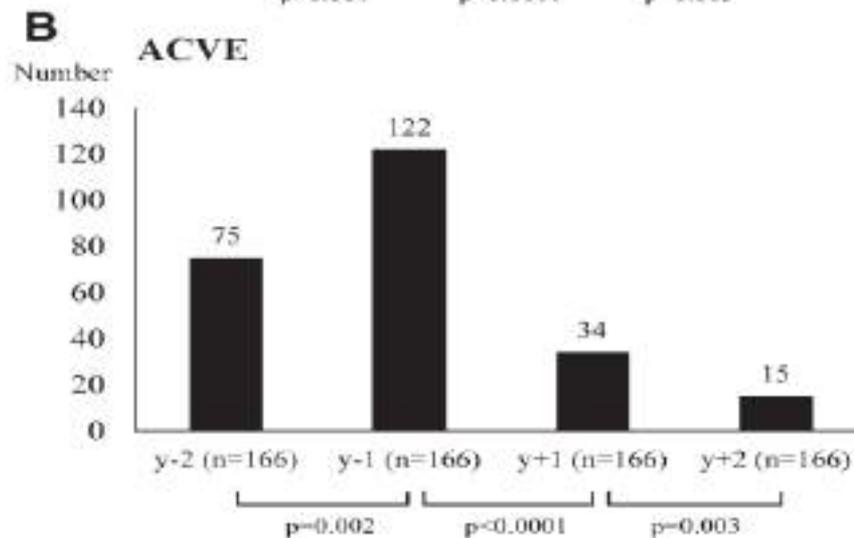
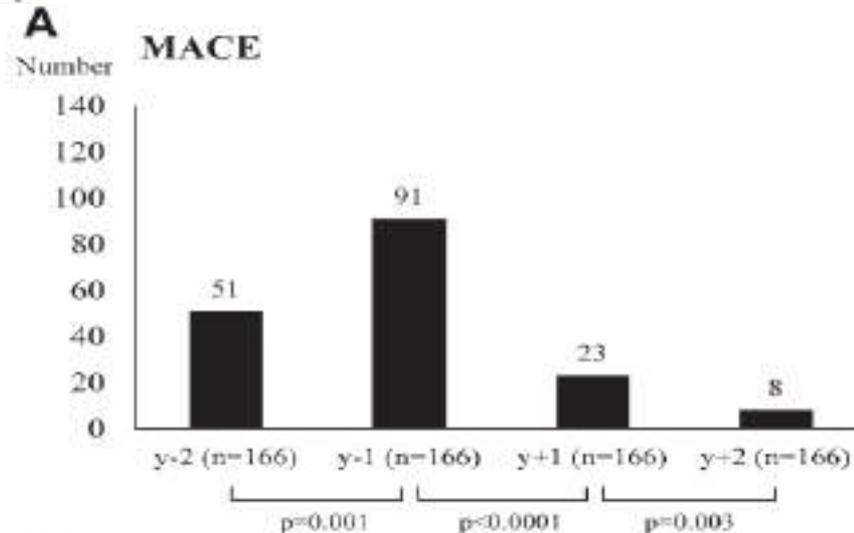
Josef Leebmann, MD; Eberhard Roeseler, MD; Ulrich Julius, MD; Franz Heigl, MD; Ralf Spithhoefer, MD; Dennis Heutling, MD; Paul Breitenberger, MD; Winfried Maerz, MD; Walter Lehmacher, PhD; Andreas Heibges, PhD; Reinhard Klingel, MD; for the Pro(a)LiFe Study Group*

Table 5. Mean Annual Rates for MACE, ACVE, MI, PCI, and CABG for 2 Years Before (y-2, y-1) and After (y+1, y+2) Commencing Chronic LA and Percentage Changes (Δ) Between Periods Before and During Apheresis

	(y-2 + y-1)	(y+1 + y+2)	Δ , %	PValue
MACE	0.41 \pm 0.45	0.09 \pm 0.22	-78.0	<0.0001
ACVE	0.58 \pm 0.53	0.14 \pm 0.31	-75.9	<0.0001
MI	0.14 \pm 0.24	0.02 \pm 0.10	-85.7	<0.0001
PCI	0.22 \pm 0.35	0.07 \pm 0.19	-68.2	<0.0001
CABG	0.05 \pm 0.15	0.01 \pm 0.05	-80.0	0.001

apheresis 2/w-1/3w

Circulation. 2013;128:2567-2576

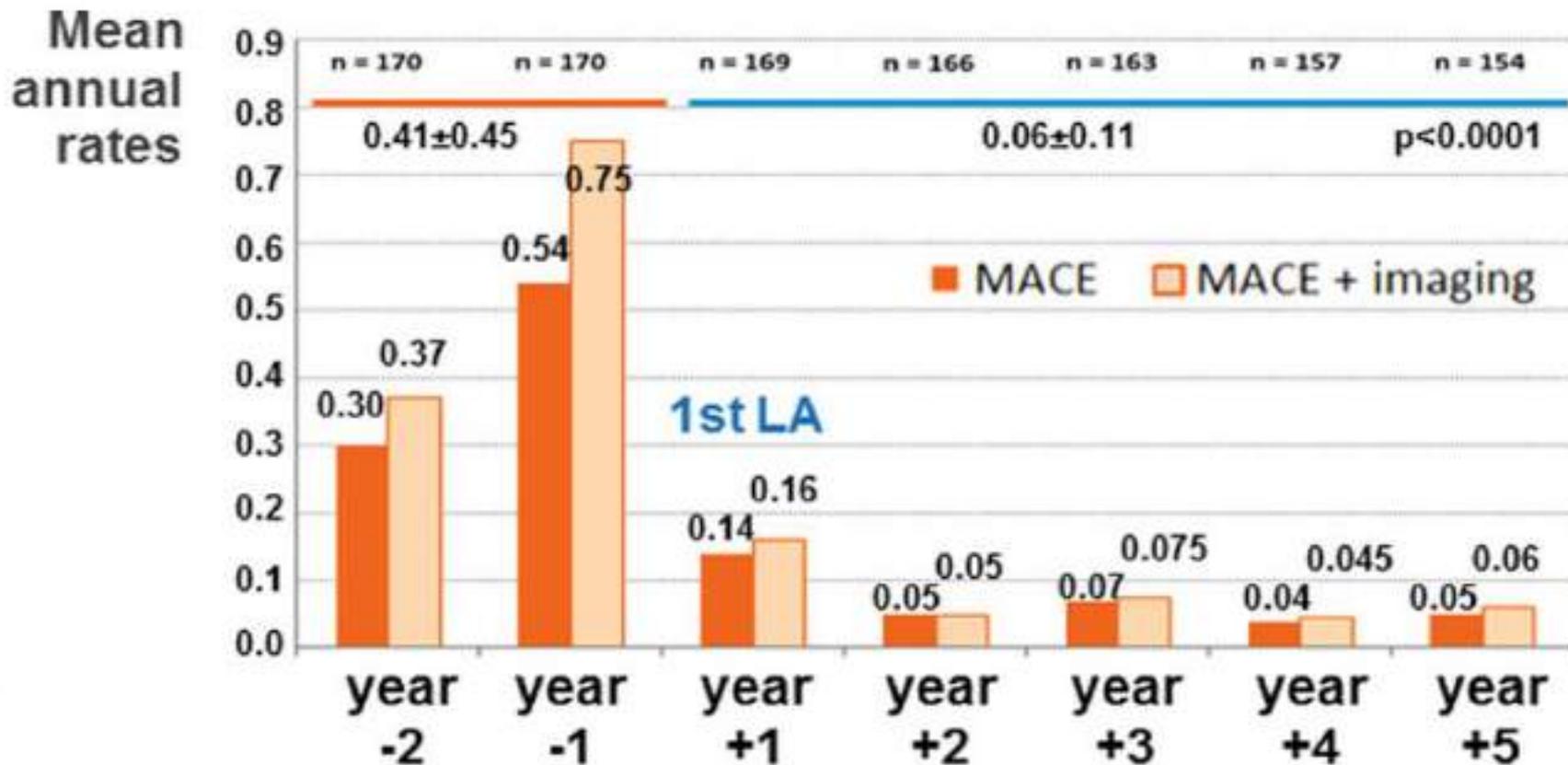


Prevention of cardiovascular complications in patients with Lp(a)-hyperlipoproteinemia and progressive cardiovascular disease by long-term lipoprotein apheresis according to German national guidelines

Reinhard Klingel^{1,2} · Andreas Heibges¹ · Cordula Fassbender¹ · Pro(a)LiFe-Study Group¹

170 patients commencing LA due to Lp(a)-HLP with Lp(a) > 60 mg/dl and progressive CVD

Apheresis I/w

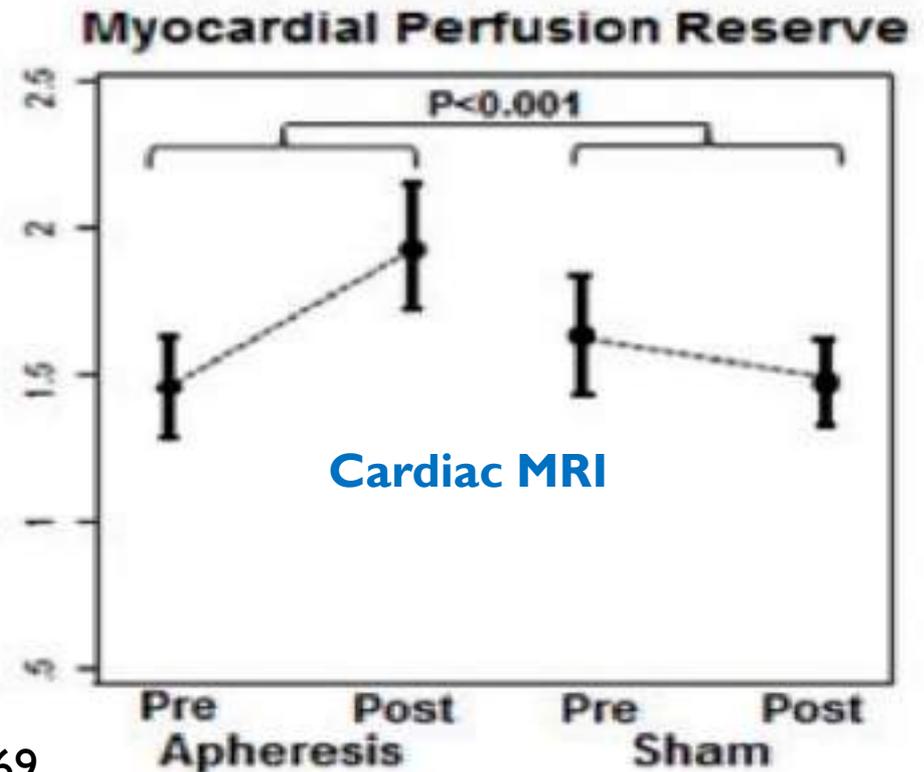


Apheresis as novel treatment for refractory angina with raised lipoprotein(a): a randomized controlled cross-over trial

Tina Z. Khan^{1,2}, Li-Yueh Hsu³, Andrew E. Arai³, Samantha Rhodes¹, Alison Pottle¹,

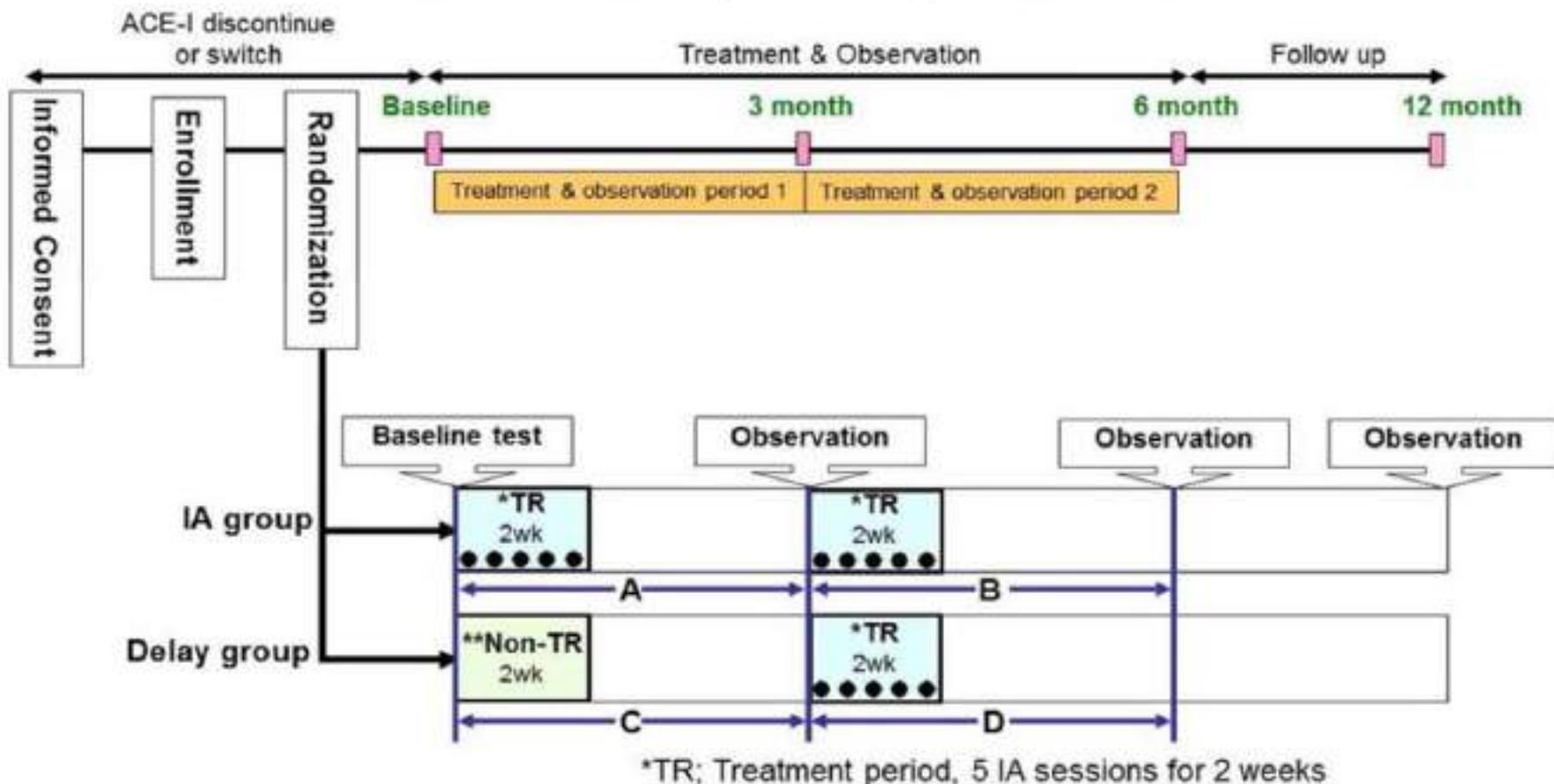
refractory angina for >3months
>1 episodes/week
previous MI, CABG, PCI
OMT with at least two anti-anginal drugs
Lp(a) >50mg/L and LDL < 4.0mmol/L,

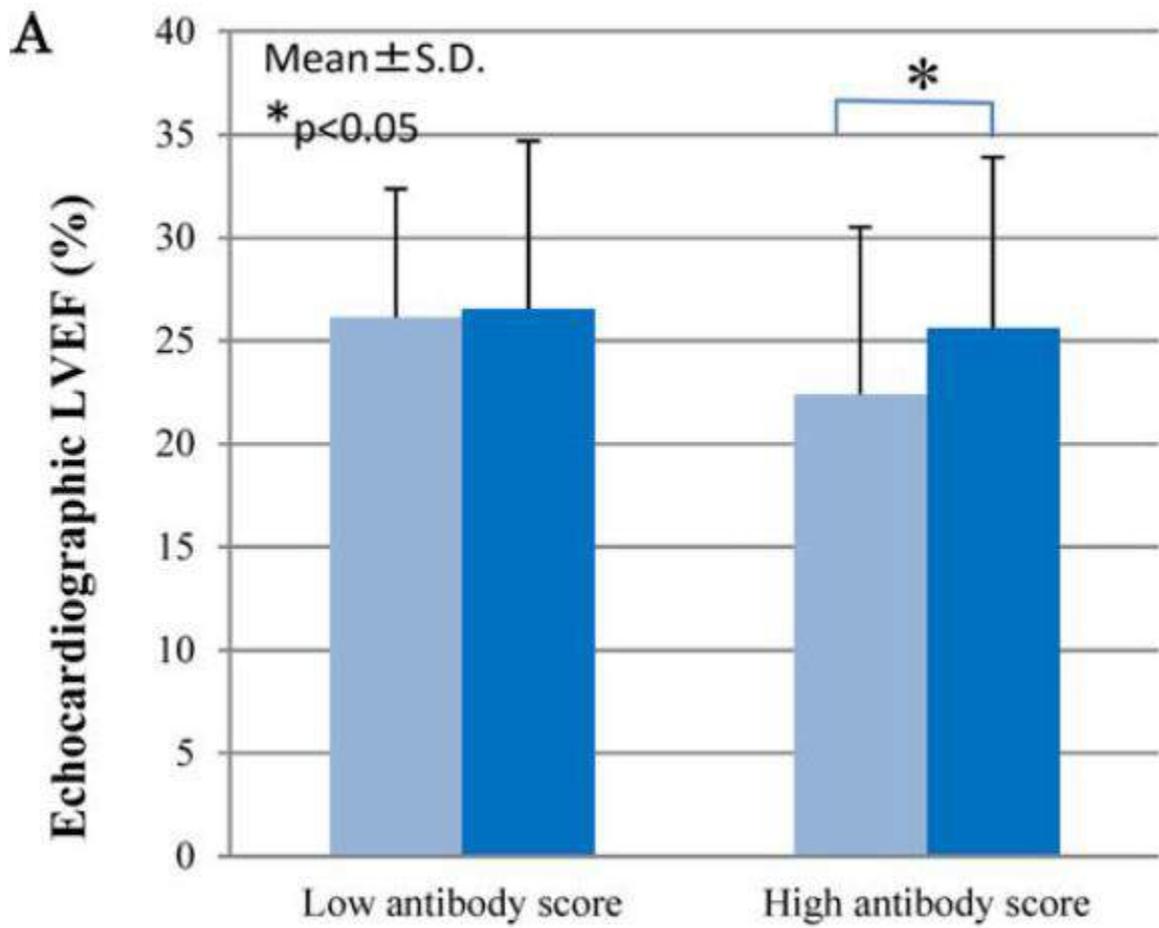
apheresis 1/w x 3 mo
1 mo wash out → crossover



Immunoadsorption Therapy for Dilated Cardiomyopathy Using Tryptophan Column—A Prospective, Multicenter, Randomized, Within-Patient and Parallel-Group Comparative Study to Evaluate Efficacy and Safety

Tsutomu Yoshikawa,^{1*} Akiyasu Baba,² Makoto Akaishi,² Yasuhisa Wakabayashi,³
Toshiaki Monkawa,⁴ Masafumi Kitakaze,⁵ Tohru Izumi,⁶ and Hitonobu Tomoike¹

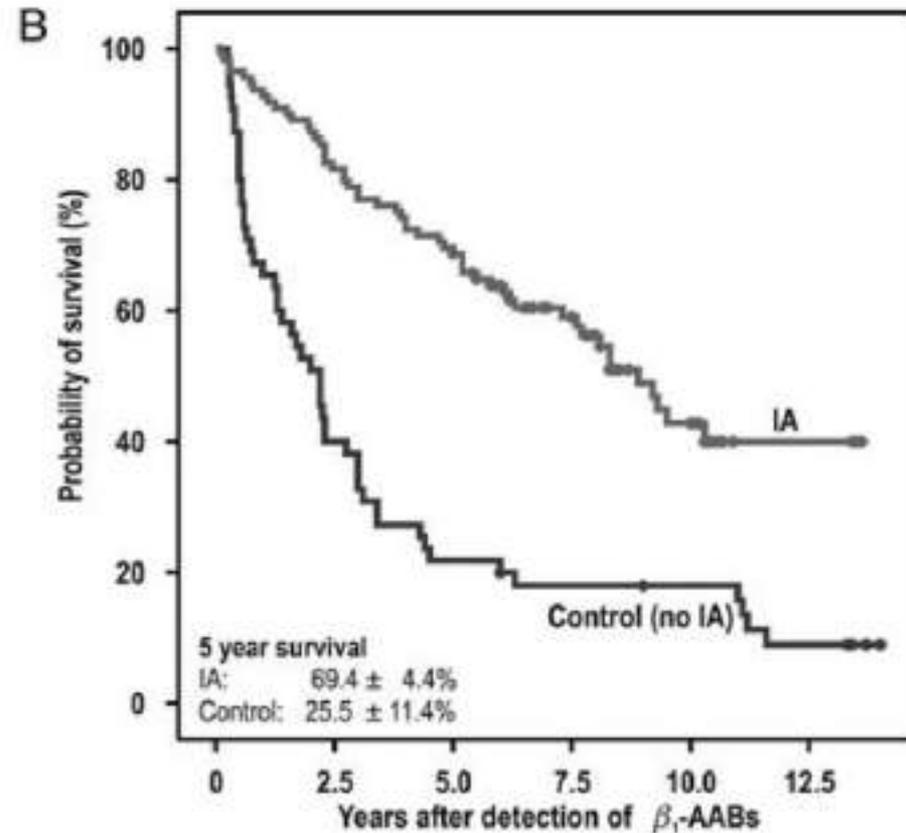
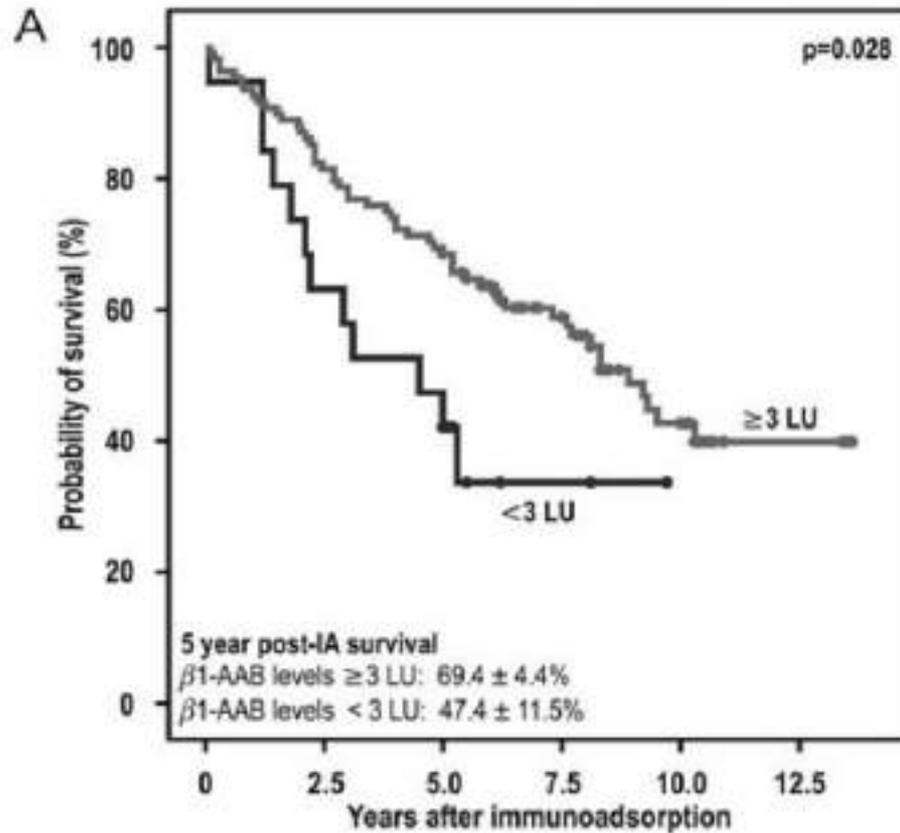




Long-term benefits of immunoadsorption in β_1 -adrenoceptor autoantibody-positive transplant candidates with dilated cardiomyopathy

Michael Dandel^{1*}, Gerd Wallukat², Angela Englert¹, Hans B. Lehmkuhl¹, Christoph Knosalla¹, and Roland Hetzer¹

VAD/Tx free survival



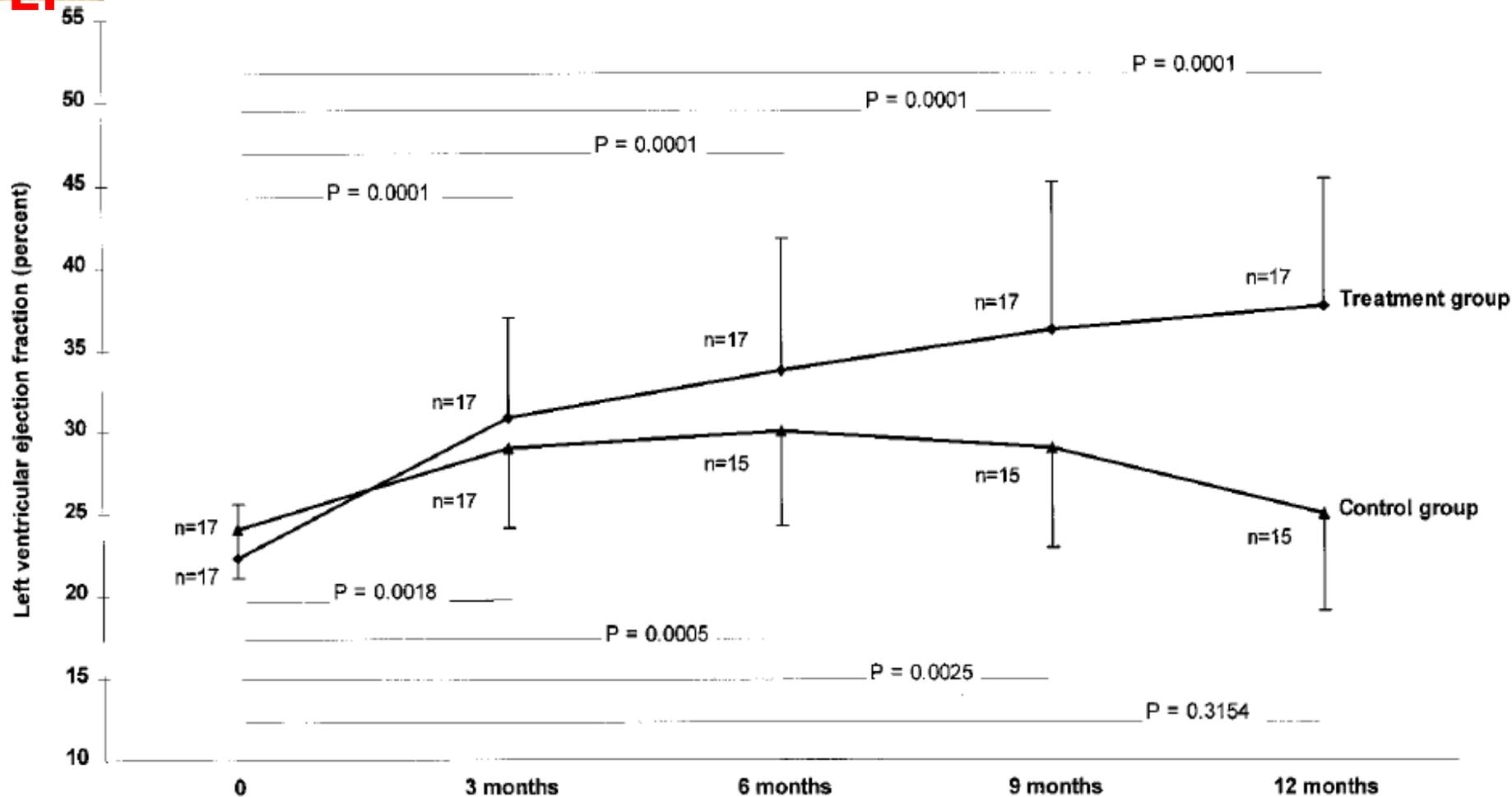
Immunoglobulin Adsorption in Patients With Idiopathic Dilated Cardiomyopathy **b1-AAB**

Johannes Müller, MD; Gerd Wallukat, PhD; Michael Dandel, MD; Heidrun Bieda, MD; Kersten Brandes, MD; Susanne Spiegelsberger, MD; Eberhard Nissen, PhD; Rudolf Kunze, PhD; Roland Hetzer, MD

Circulation. 2000;101:385-391

Immunoabsorption x 5 consecutive days

LVEF

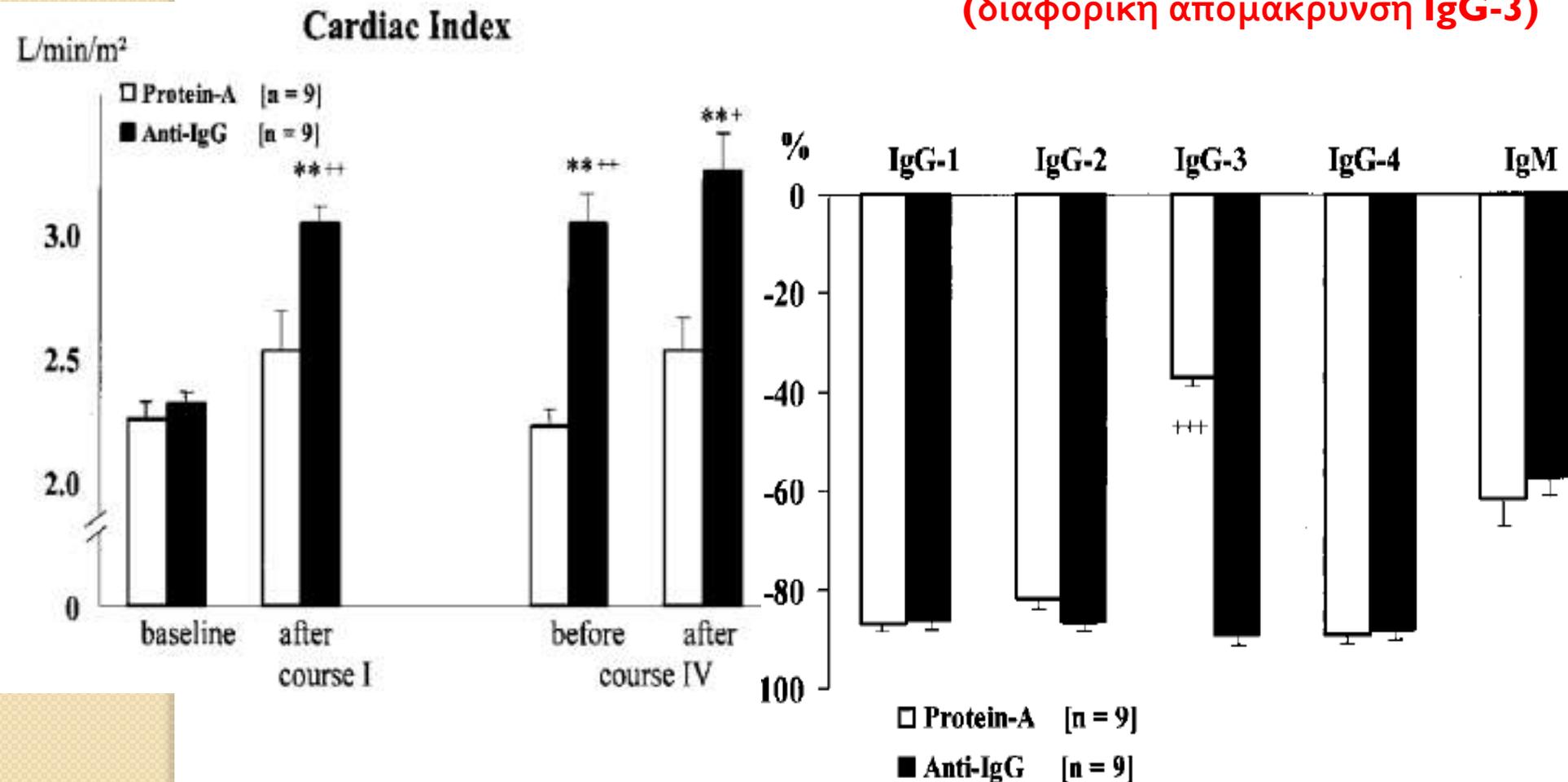


Potential Role of Autoantibodies Belonging to the Immunoglobulin G-3 Subclass in Cardiac Dysfunction Among Patients With Dilated Cardiomyopathy

Alexander Staudt, MD; Marko Böhm, MD; Fabian Knebel, MD; Yvonne Grosse, MD; Claudia Bischoff, MD; Astrid Hummel, MD; Johannes B. Dahm, MD; Adrian Borges, MD; Nicoline Jochmann, MD; Klaus D. Wernecke, PhD; Gerd Wallukat, PhD; Gert Baumann, MD; Stephan B. Felix, MD

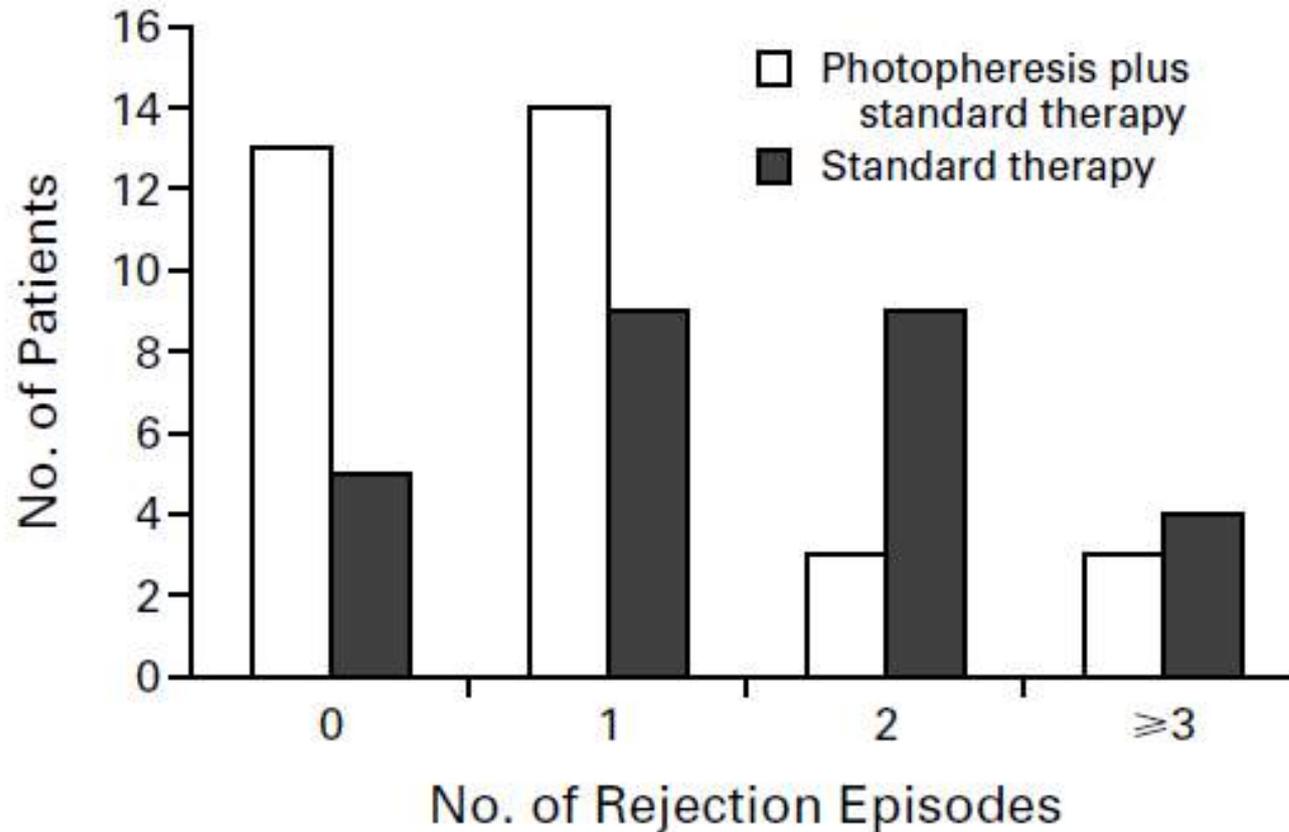
Circulation. 2002;106:2448-2453

IA x3 days/mo x 4 mo
(διαφορική απομάκρυνση IgG-3)



PHOTOPHERESIS FOR THE PREVENTION OF REJECTION IN CARDIAC TRANSPLANTATION

MARK L. BARR, M.D., BRUNO M. MEISER, M.D., HOWARD J. EISEN, M.D., RANDALL F. ROBERTS, M.D., UGOLINO LIVI, M.D., ROBERTO DALL'AMICO, M.D., PH.D., RICHARD DORENT, M.D., JOSEPH G. ROGERS, M.D., BRANISLAV RADOVANČEVIĆ, M.D., DAVID O. TAYLOR, M.D., VALLUVAN JEEVANANDAM, M.D., AND CHARLES C. MARBOE, M.D.,
FOR THE PHOTOPHERESIS TRANSPLANTATION STUDY GROUP



mortality: NS

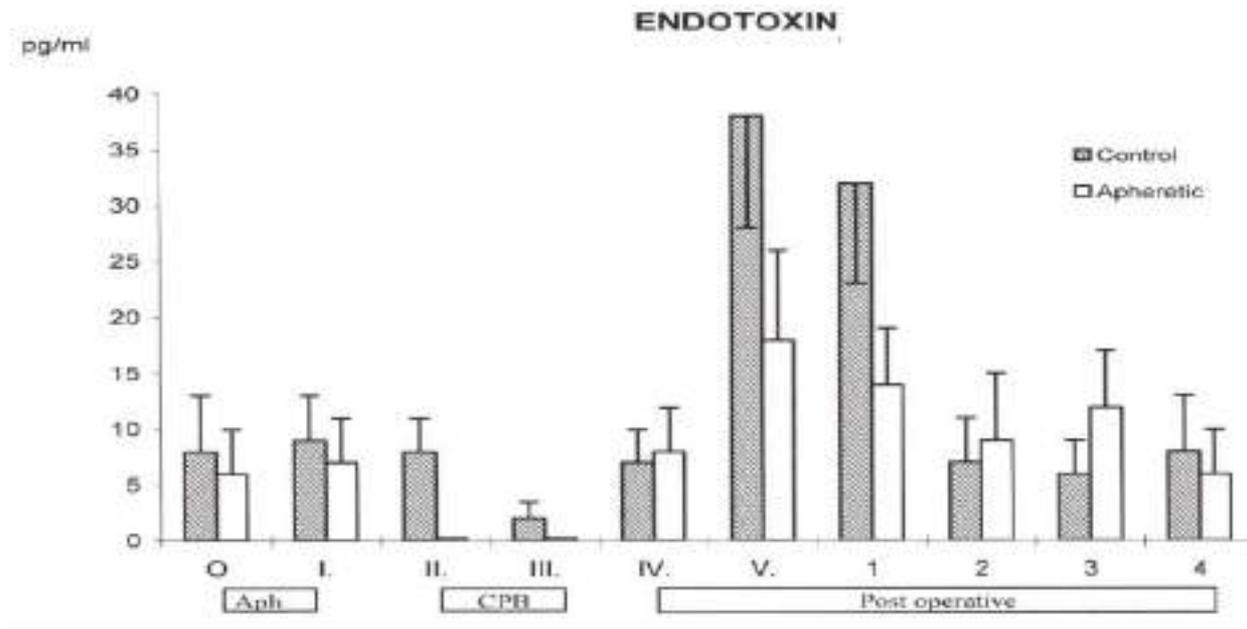
Systemic inflammatory response syndrome is reduced by preoperative plasma-thrombo-leukocyte aphaeresis in a pig model of cardiopulmonary bypass

Robert Wagner^{a,b}, Pavel Piler^{a,b}, Borivoj Uchytíl^a, Roman Halouzka^c, Hana Kovaru^d, Marie Bobkova^e, Petr Nemeč^{a,b}

Table 3. Early postop. data of animals.

	Parameter	Control n=10	Aphaeretic n=11	significance
PaO ₂	% of change	13±5	14±6	NS
Static. compliance	% of change	33±5	24±3	<i>P</i> < 0.05
Inspir. resistance	% of change	22±5	3±2	<i>P</i> < 0.003
Myocard. ischaemia	percentage	90	10	<i>P</i> < 0.003
Inotropic support	percentage	90	25	<i>P</i> < 0.007
Extubation	hours	6 ± 1.5	5 ± 1.2	NS
Blood loss	mL/18 h	375 ± 95	490 ± 150	<i>P</i> < 0.05

Biomed Pap Med Fac Univ
Palacky Olomouc Czech Repub.
2016; 160(3):399-406.



Case Report

“First in Man”: Case Report of Selective C-Reactive Protein Apheresis in a Patient with Acute ST Segment Elevation Myocardial Infarction

Wolfgang Ries,¹ Ahmed Sheriff,² Franz Heigl,³ Oliver Zimmermann,⁴
Christoph D. Garlichs,¹ and Jan Torzewski ⁴



Ευχαριστώ

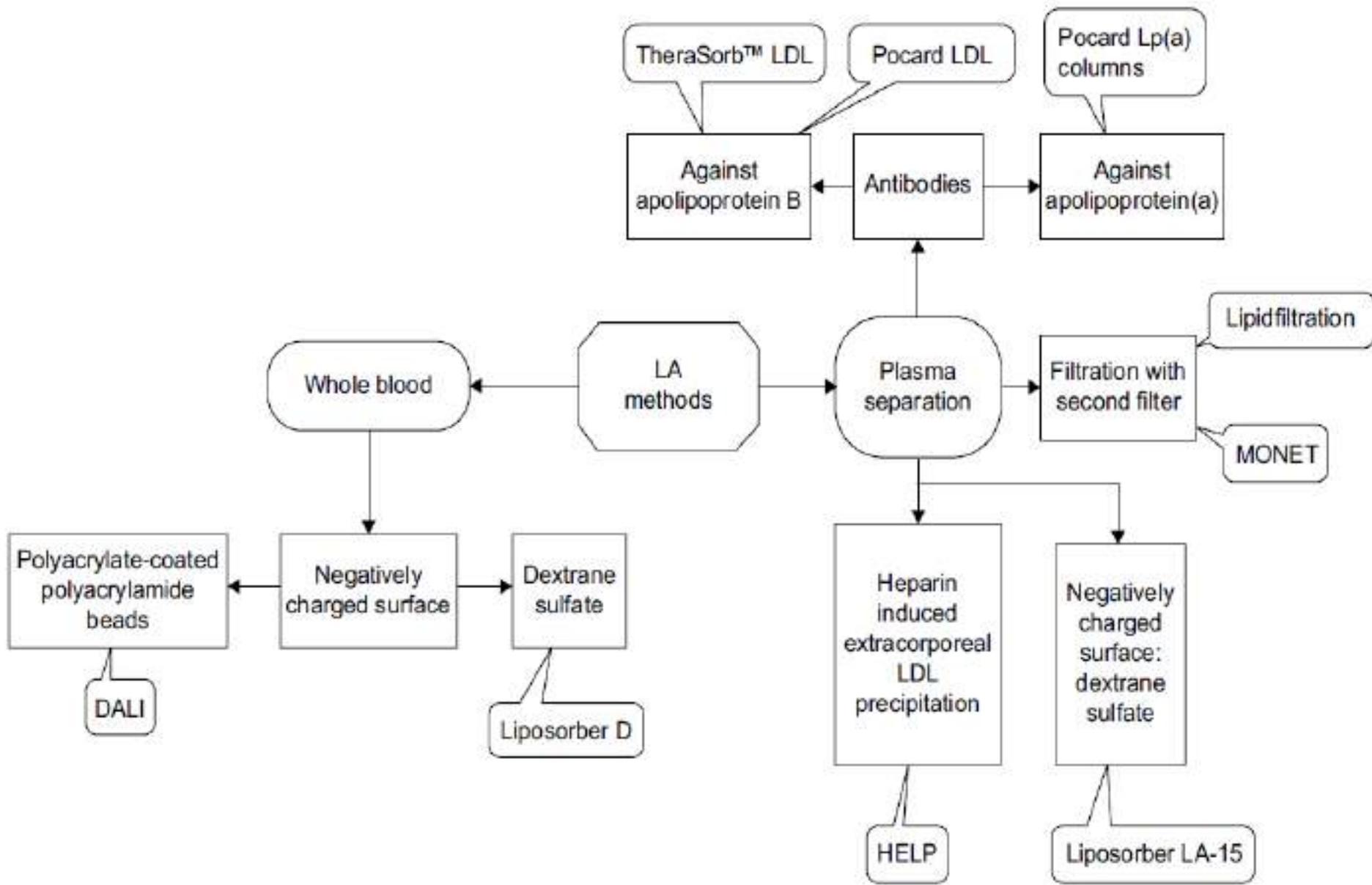


Figure 2 Principles of available LA methods.

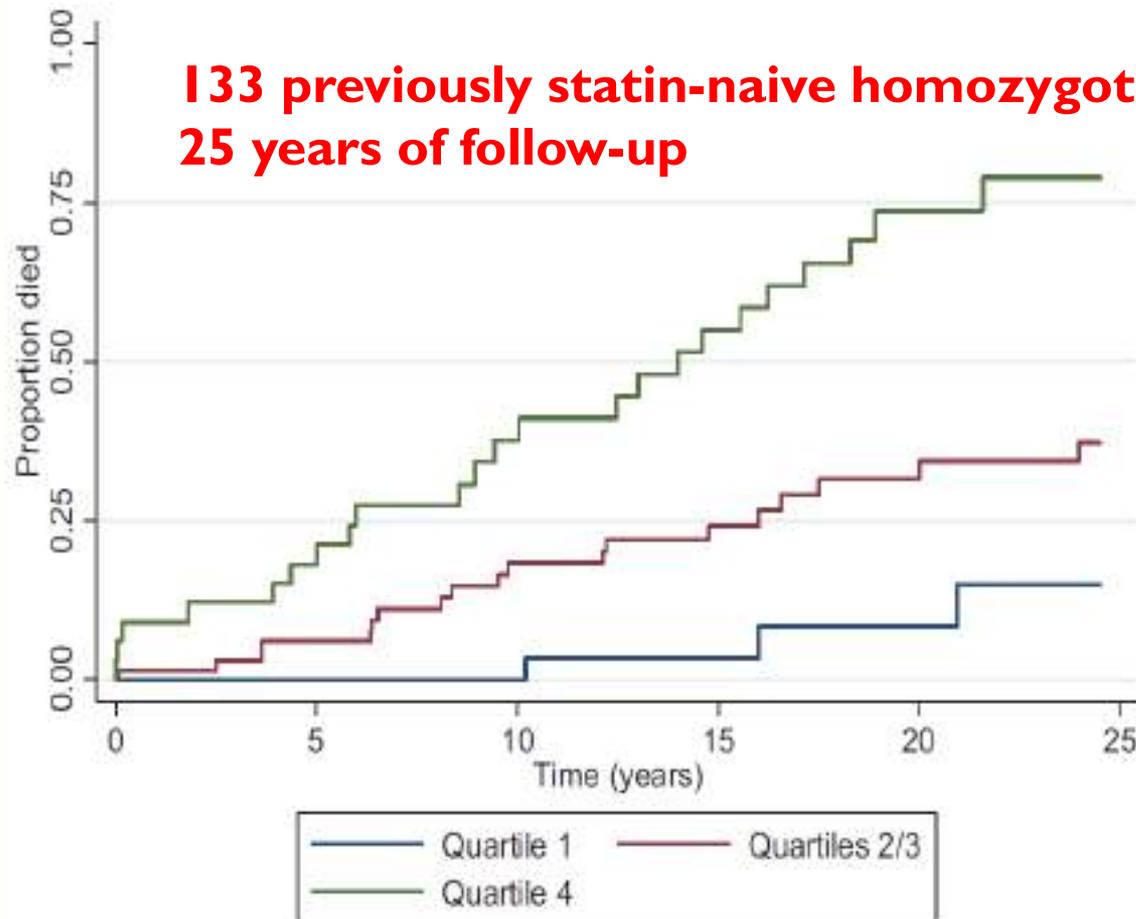
Reversal of Severe Late Left Ventricular Failure After Pediatric Heart Transplantation and Possible Role of Plasmapheresis Am J Cardiol 2000;85:735-739

Elfriede Pahl, MD, Susan E. Crawford, MD, Richard A. Cohn, MD,
Sherrie Rodgers, MSN, David Wax, MD, Carl L. Backer, MD,
Constantine Mavroudis, MD, and Samuel S. Gidding, MD

Survival in homozygous familial hypercholesterolaemia is determined by the on-treatment level of serum cholesterol

Gilbert R. Thompson^{1*}, Dirk J. Blom², A. David Marais³, Mary Seed⁴, Gillian J. Pitcher⁵, and Frederick J. Raal⁵

**133 previously statin-naïve homozygotes
25 years of follow-up**



Quartile 1, <8.1 mmol/L
Quartiles 2/3, 8.1–15.1 mmol/L
Quartile 4, >15.1 mmol/L

Table 4 Adverse effects during extracorporeal LA therapy (listed according to frequency)

- Puncture problems (24%)
 - Hypotension (20%)
 - Discomfort (11%)
 - Bleeding/hematoma (9%)
 - Hypocalcemia (8%)
 - Pain at the puncture site (6%)
 - Angina pectoris attack (4%)
 - Hypertension (3%)
 - Nausea (3%)
 - Edema (3%)
 - Vertigo (2%)
 - Other (6%)
-