
Dr Di Bernardo Stefano
La myocardite:
Un défi du diagnostic
à la guérison
canton de vaud
UNIL | Université de Lausanne
CHUV

UN CAS

Patiente de 6 ans adressée par sa pédiatre pour investigation d'une hépatomégalie:

- Asthénie depuis 5 mois
- Diminution de l'appétit
- Episodes de vomissements et de diarrhées intermittentes
- Episode fébrile il y a 6 mois: vomissement et diarrhées. Virose ?

Poids : 19.7kg (P25-50)

Taille : 113cm (P25-50)

TAS : 99mmHg ; TAD : 77mmHg

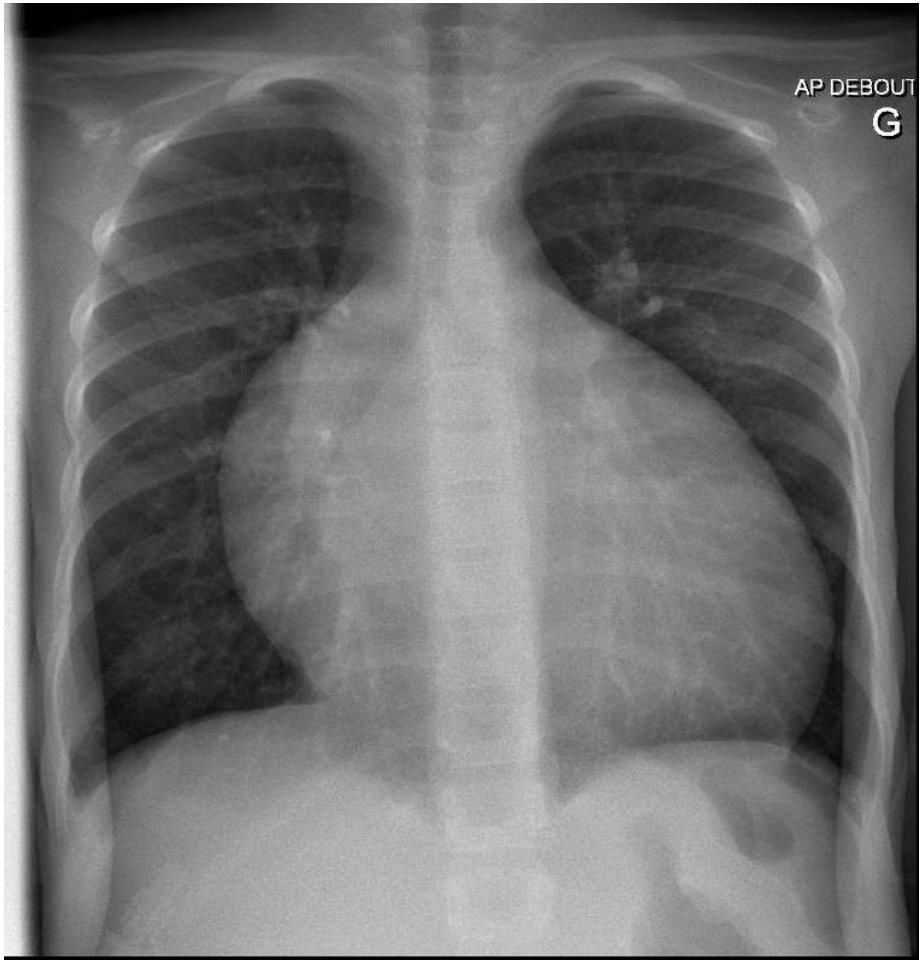
FC : 106/min

Pâle

Eupnéique

Pas de souffle audible

hépatomégalie à 6 cm du rebord costal, rate non palpable


AP DEBOUT
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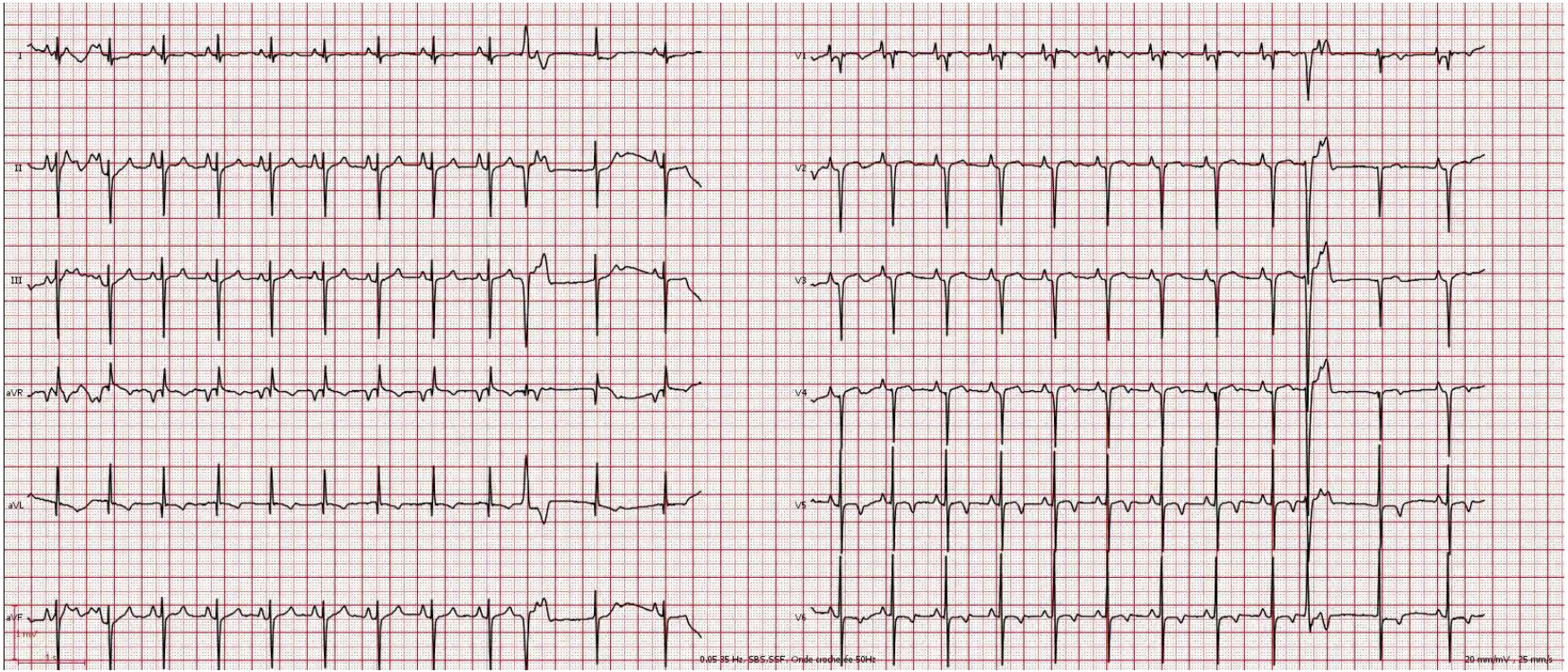
I
II
III
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aVL
aVF
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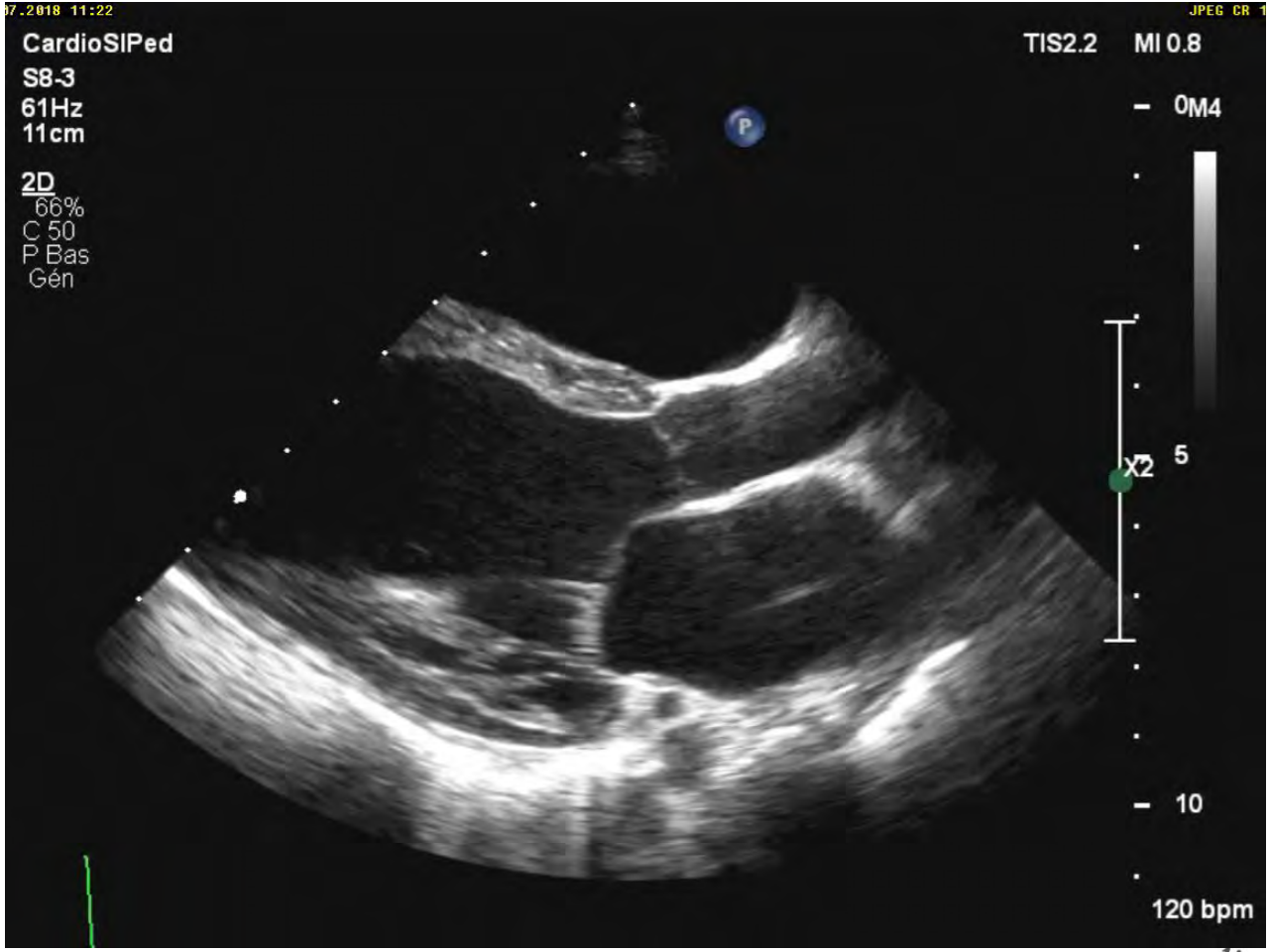
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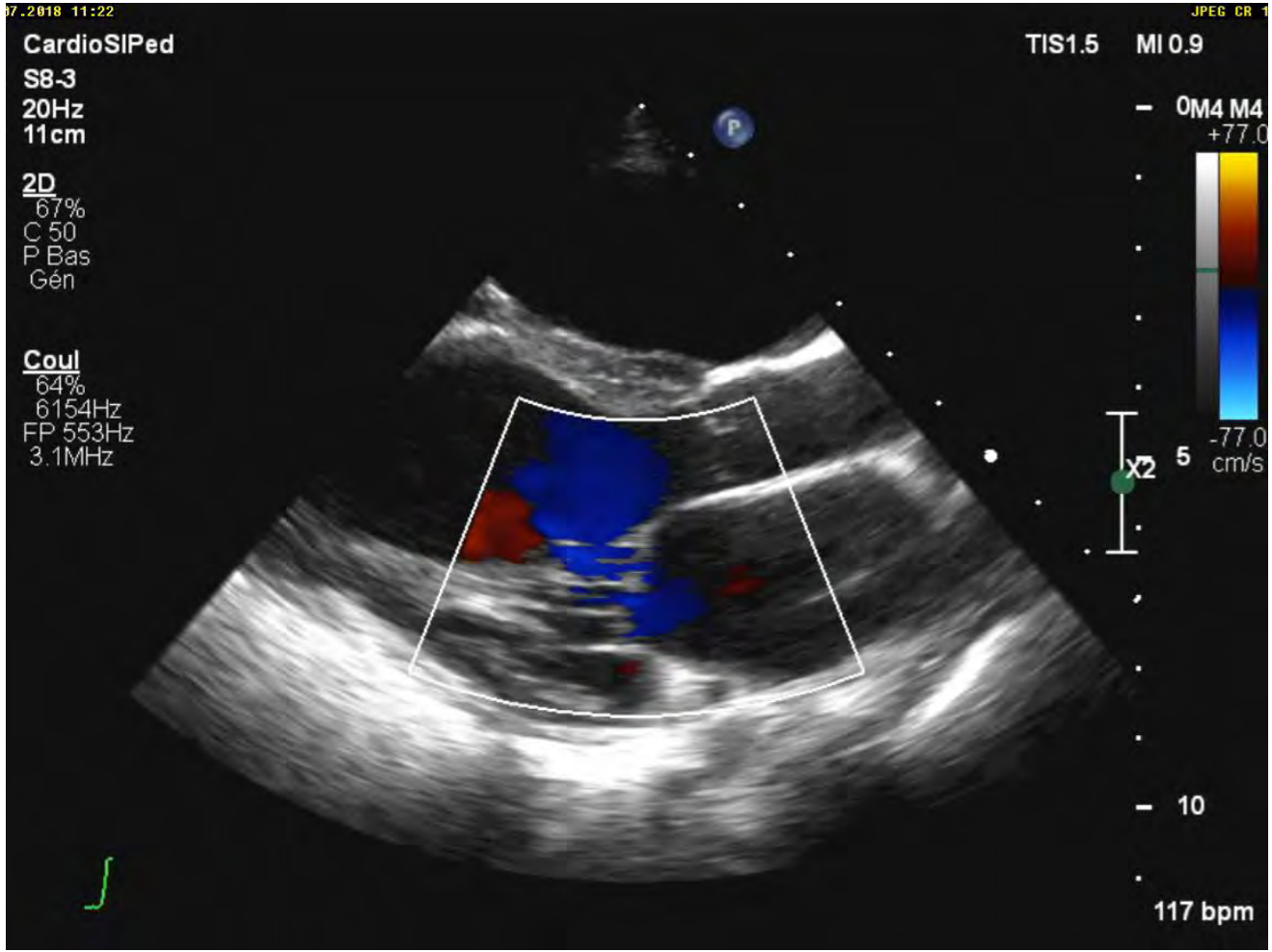
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cm/s
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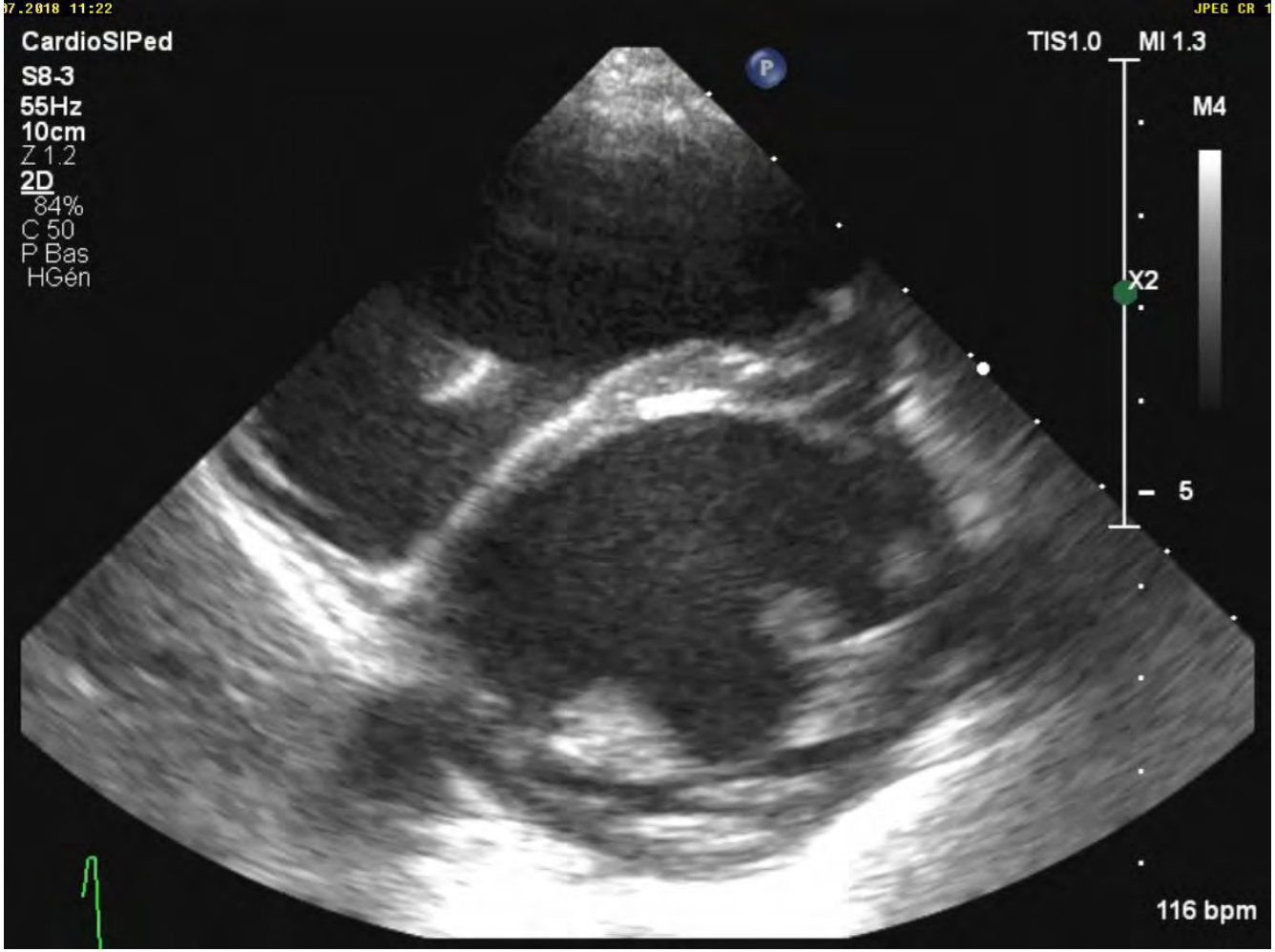
117 bpm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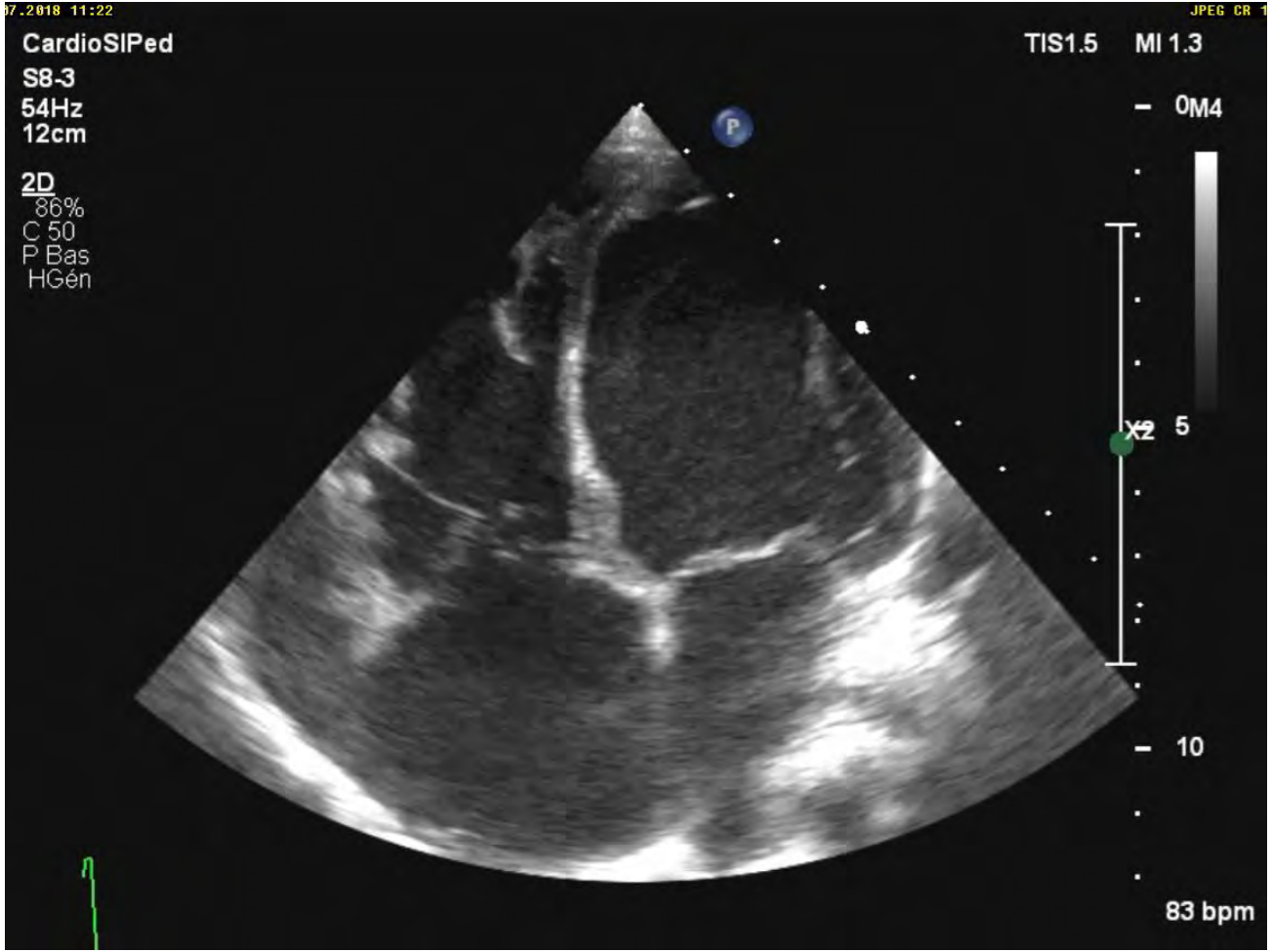
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5
10
83 bpm

European Heart Journal (2013) **34**, 2636–2648
doi:10.1093/eurheartj/eht210

ESC REPORT

Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases

Definitions

Myocarditis (WHO /ISFC[1]):

Inflammatory disease of the myocardium diagnosed by established histological, immunological and immunohistochemical criteria**.*

**N.B. established histological Dallas criteria[12] defined as follows:*

'histological evidence of inflammatory infiltrates within the myocardium associated with myocyte degeneration and necrosis of non-ischaemic origin [12]'.

***N.B. unspecified immunohistochemical criteria[1], we propose an abnormal inflammatory infiltrate to be defined as follows:*

'$\geq$ 14 leucocytes/mm^2 including up to 4 monocytes/mm^2 with the presence of CD 3 positive T-lymphocytes $\geq$ 7 cells/mm^2'.[15,18,19]

Inflammatory Cardiomyopathy (WHO /ISFC[1]):

Myocarditis in association with cardiac dysfunction.

N.B. Inflammatory cardiomyopathy, involved in the pathogenesis of DCM, includes idiopathic, autoimmune and infectious subtypes.[1]

Dilated Cardiomyopathy (ESC[13]; WHO /ISFC[1]):

DCM is a clinical diagnosis characterized by dilation and impaired contraction of the left or both ventricles that is not explained by abnormal loading conditions or coronary artery disease.

N.B. DCM includes idiopathic, familial/genetic, viral and/or immune, alcoholic/toxic subtypes.[1]

Table 1 Etiology of Myocarditis

Etiology	Subgroups Examples
Infectious	Bacterial: *Chlamydia, Corynebacterium diphtheria, Legionella, Mycobacterium tuberculosis, Mycoplasma, Staphylococcus, Streptococcus A, Streptococcus pneumoniae*
	Fungal: *Actinomyces, Aspergillus, Candida, Cryptococcus*
	Helminthic: *Echinococcus granulosus, Trichinella spiralis*
	Protozoal: *Toxoplasma gondii, Trypanosoma cruzi*
	Viral: Adenoviruses, Echoviruses, Enteroviruses (e.g., Coxsackieviruses), Herpes Viruses (Human Cytomegalovirus, Epstein-Barr virus, Human Herpesvirus 6), Hepatitis C Virus, Human Immunodeficiency Virus (HIV), Influenza A virus, Parvovirus B19
	Rickettsial: *Coxiella burnetti, Rickettsia typhi*
	Spirochetal: *Borrelia burgdorferi, Leptospira, Treponema pallidum*
Autoimmune diseases	Celiac disease, Churg-Strauss syndrome, Crohn's disease, dermatomyositis, giant cell myocarditis, hypereosinophilic syndrome, Kawasaki disease, lupus erythematodes, lymphofollicular myocarditis, rheumatoid arthritis, sarcoidosis, scleroderma, ulcerative colitis
Hypersensitivity reactions to drugs	Penicillin, ampicillin, cephalosporins, tetracyclines, sulfonamids, antiphlogistics, benzodiazepines, clozapine, loop and thiazide diuretics, methyldopa, smallpox vaccine, tetanus toxoid, tricyclic antidepressants
Toxic reactions to drugs	Amphetamines, anthracyclines, catecholamines, cocaine, cyclophoshamide, 5-fluorouracil, phenytoin, trastuzumab
Toxic	Ethanol
Others	Arsenic, copper, iron, radiotherapy, thyreotoxicosis

Kindermann et al. Update on Myocarditis; JACC 2012

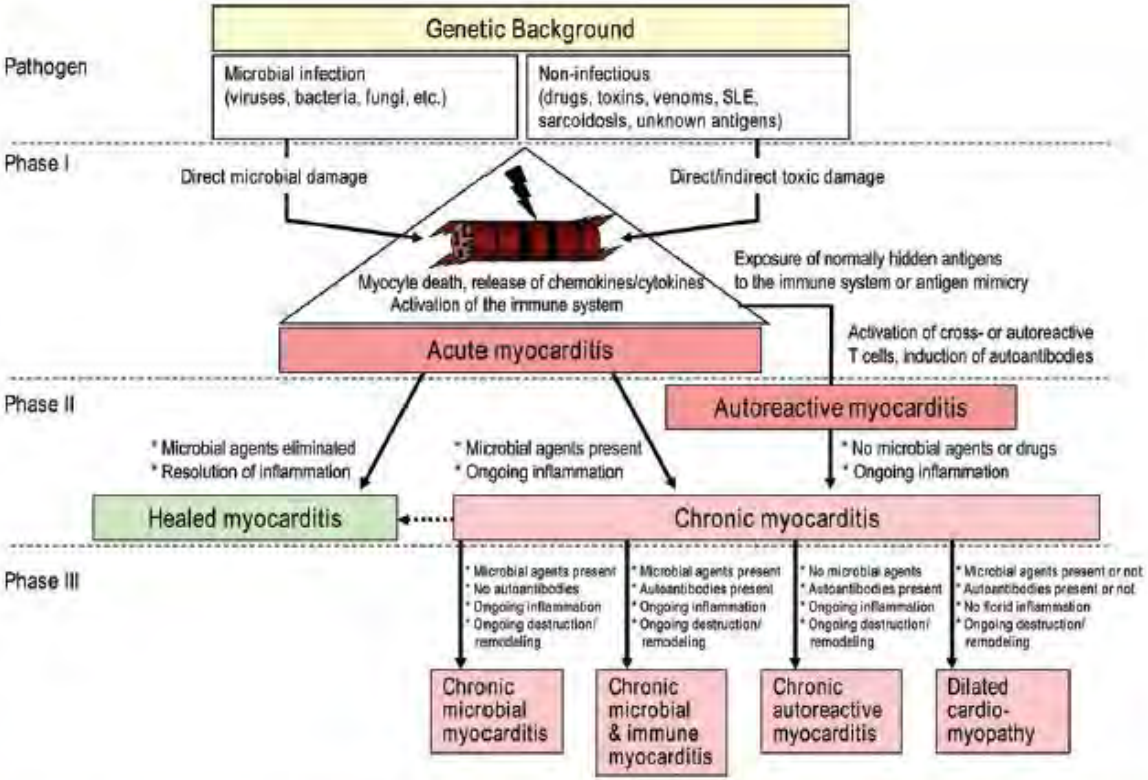


Figure 2 The picture shows the pathogenetic mechanisms involved in myocarditis and progression to dilated cardiomyopathy.

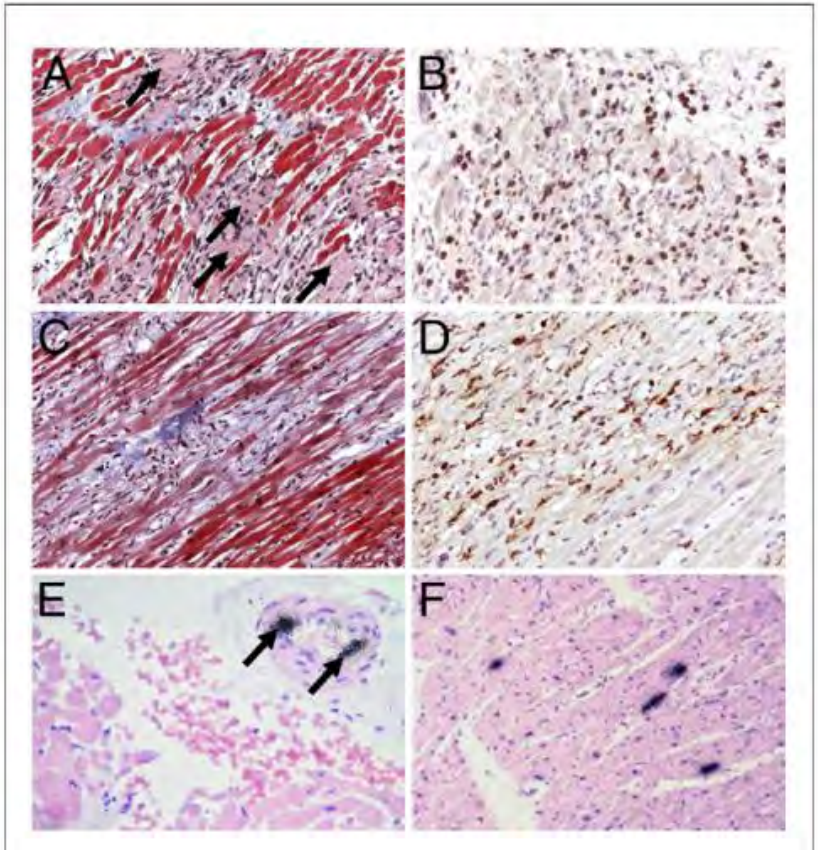


Figure 4 **Histopathological, Immunohistological, and Molecular Biological Findings in Hearts of Patients With Myocarditis**

Histology and immunohistology of **(A, B)** acute myocarditis and **(C, D)** chronic myocarditis. In acute myocarditis, numerous necrotic myocytes **(A, arrows)** are associated with mononuclear cell infiltrates including CD3+ T cells **(B)**, whereas in chronic myocarditis, inflammatory cells such as CD68+ macrophages **(D)** are mainly present in areas with fibrosis **(C, blue staining)**. **(E, F)** Radioactive in situ hybridization reveals PVB19 nucleic acid in endothelial cells of an arteriole in a patient with chronic myocarditis **(E)**, whereas enterovirus ribonucleic acid is detected in several myocytes **(F)**.

Table 2 Serum cardiac autoantibodies in autoimmune myocarditis/dilated cardiomyopathy: frequency in myocarditis/dilated cardiomyopathy, other cardiac disease (OCD) and normals

Cardiac autoantibody (Ab)	% aabs positive		%antibody positive		Functional effect/clinical relevance	References
	Myoc	DCM	OCD	Normal		
Muscle-specific ASA, (AFA,IFA,AMLA)	28–59*	9–41*	NT	0–25	Myocytolysis	72,77,57,64
Cardiac-specific						
AHA	41–56*,^,a	26–30*,^,a	1–4	3	Cardiac- and disease-specific early predictors; predict DCM development in relatives	9[a],50[a],35[a], 36[a],118[a],52[a]
AIDA	17*,^,a	16*,^,a	2–4	0		
Anti-Beta1-AR	33	40–51^	13–55	0–13	Negative predictors, pro-apoptotic and other *in vitro* effects[b]	48,55,61–63,66[a],72,74–76, 78,84,109,88,90,92,93,98
	NT	35*,^,a	16	7		
	73–96*,^,a	29–95*,^,a	8	0		
	NT	27–28	10	0		
Anti-Beta2-AR	NT	30–38^	33	15	Association with idiopathic arrhythmia	53^,62,69[a],89
	NT	13–14				
	NT	30–75[a]	37	18		
Anti-muscarinic acetylcholine receptor-2	11	30–77[c]	23[d]–61	8–13	Negative inotropic, muscarinic effects	47[d],48[c],54,58,59,70,74–75,88,94,98
	NT	83[e]			Association with atrial arrhythmia	
Cardiodepressant (Fy-gamma-receptor 2a)	NT	64			Negative inotropic effects in rat and human myocytes *in vitro*	56,66^,85–87,91^
Anti-Ky channel-interacting protein 2, KChIP2.6—ELISA)	NT	14^	8	4	Increased cell death in myocytes *in vitro*	
Anti-Alpha-MHC (cardiac-specific)	17–37*,^,a	20–46*,^,a	4–16	0–2.5	Negative predictors, pro-apoptotic	109,51[a],60[a], 118[a],140[a]
Anti-Beta-MHC (muscle-cross reactive)						68,95,96
Anti-MLC 1v	NT	17^–35	25	0–15		51,67^
Anti-tropomyosin	NT	55^	21	NT		67
Anti-non-myofibrillar	NT	46*,^,a	17	0		51[a]
Anti-MHC	NT	67^	42	NT		67
Anti-actin	NT	71^	21	NT		67
Anti-Troponin I ,T	NT	1.7^–20^	0^–18	0–4	Negative predictors	66,68,80

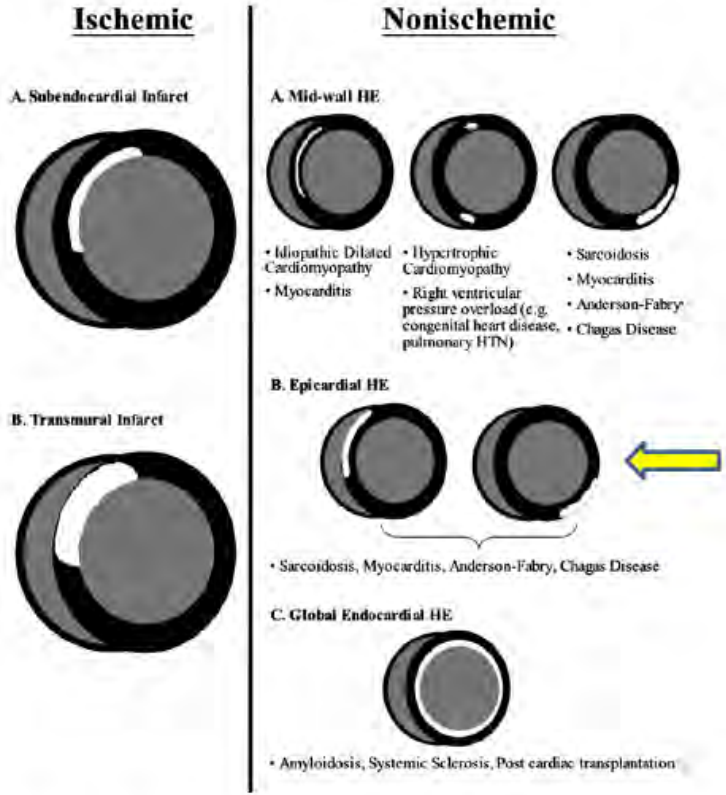
Table 4 **Diagnostic criteria for clinically suspected myocarditis**

Clinical presentations[a]
Acute chest pain, pericarditic, or pseudo-ischaemic
New-onset (days up to 3 months) or worsening of: dyspnoea at rest or exercise, and/or fatigue, with or without left and/or right heart failure signs
Subacute/chronic (>3 months) or worsening of: dyspnoea at rest or exercise, and/or fatigue, with or without left and/or right heart failure signs
Palpitation, and/or unexplained arrhythmia symptoms and/or syncope, and/or aborted sudden cardiac death
Unexplained cardiogenic shock
Diagnostic criteria
I. ECG/Holter/stress test features
Newly abnormal 12 lead ECG and/or Holter and/or stress testing, any of the following: I to III degree atrioventricular block, or bundle branch block, ST/T wave change (ST elevation or non ST elevation, T wave inversion), sinus arrest, ventricular tachycardia or fibrillation and asystole, atrial fibrillation, reduced R wave height, intraventricular conduction delay (widened QRS complex), abnormal Q waves, low voltage, frequent premature beats, supraventricular tachycardia
II. Myocardiocytolysis markers
Elevated TnT/TnI
III. Functional and structural abnormalities on cardiac imaging (echo/angio/CMR)
New, otherwise unexplained LV and/or RV structure and function abnormality (including incidental finding in apparently asymptomatic subjects): regional wall motion or global systolic or diastolic function abnormality, with or without ventricular dilatation, with or without increased wall thickness, with or without pericardial effusion, with or without endocavitary thrombi
IV. Tissue characterization by CMR
Oedema and/or LGE of classical myocarditic pattern (see text)

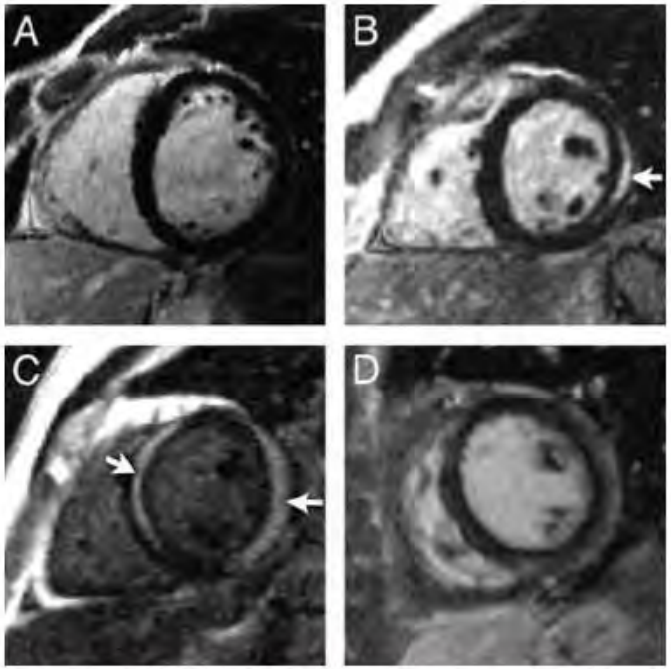
Clinically suspected myocarditis if ≥1 clinical presentation and ≥1 diagnostic criteria from different categories, in the absence of: (1) angiographically detectable coronary artery disease (coronary stenosis ≥ 50%); (2) known pre-existing cardiovascular disease or extra-cardiac causes that could explain the syndrome (e.g. valve disease, congenital heart disease, hyperthyroidism, etc.) (see text). Suspicion is higher with higher number of fulfilled criteria.

[a]If the patient is asymptomatic ≥2 diagnostic criteria should be met.

HYPERENHANCEMENT PATTERNS



Heiko Mahrholdt et al. Eur Heart J 2005



A: normal myocardium without myocite injury
B: regional sub-epicardial enhancement of the lateral wall (the most frequent)
C: sub-epicardial enhancement of the lateral wall and midwall enhancement of the septal wall
D: diffuse subepicardial enhancement

Friedrich et al. *JACC White Paper: CMR in Myocarditis*; JACC 2009

Table 5 Diagnostic cardiac magnetic resonance criteria for myocarditis

In the setting of clinically suspected myocarditis (*Tables 3–4*), CMR findings are consistent with myocardial inflammation, if at least two of the following criteria are present:

(1) Regional or global myocardial signal intensity increase in T2-weighted oedema images[a]
(2) Increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium-enhanced T1-weighted images[b]
(3) There is at least one focal lesion with non-ischaemic regional distribution in inversion recovery-prepared gadolinium-enhanced T1-weighted images (late gadolinium enhancement)[c]

A CMR study is consistent with myocyte injury and/or scar caused by myocardial inflammation if Criterion 3 is present

A repeat CMR study between 1 and 2 weeks after the initial CMR study is recommended if

- None of the criteria are present, but the onset of symptoms has been very recent and there is strong clinical evidence for myocardial inflammation
- One of the criteria is present

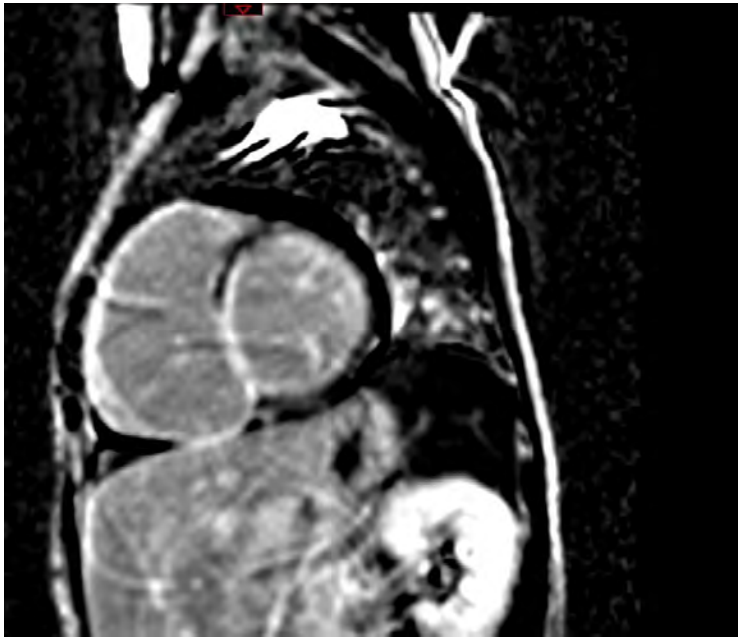
The presence of LV dysfunction or pericardial effusion provides additional, supportive evidence for myocarditis

Table reprinted with permission from (20).

[a]Global signal intensity (SI) increase has to be quantified by an SI ratio of myocardium over skeletal muscle of ≥ 2.0. If the edema is more subendocardial or transmural in combination with a colocalized ischaemic (including the subendocardial layer) pattern of late gadolinium enhancement, acute myocardial infarction is more likely and should be reported.

[b]A global SI enhancement ratio of myocardium over skeletal muscle of ≥ 4.0 or an absolute myocardial enhancement of $\geq 45\%$ is consistent with myocarditis.

[c]Images should be obtained at least 5 min after gadolinium injection; foci typically exclude the subendocardial layer, are often multifocal, and involve the subepicardium. If the late gadolinium enhancement pattern clearly indicates myocardial infarction and is colocalized with a transmural regional edema, acute myocardial infarction is more likely and should be reported.



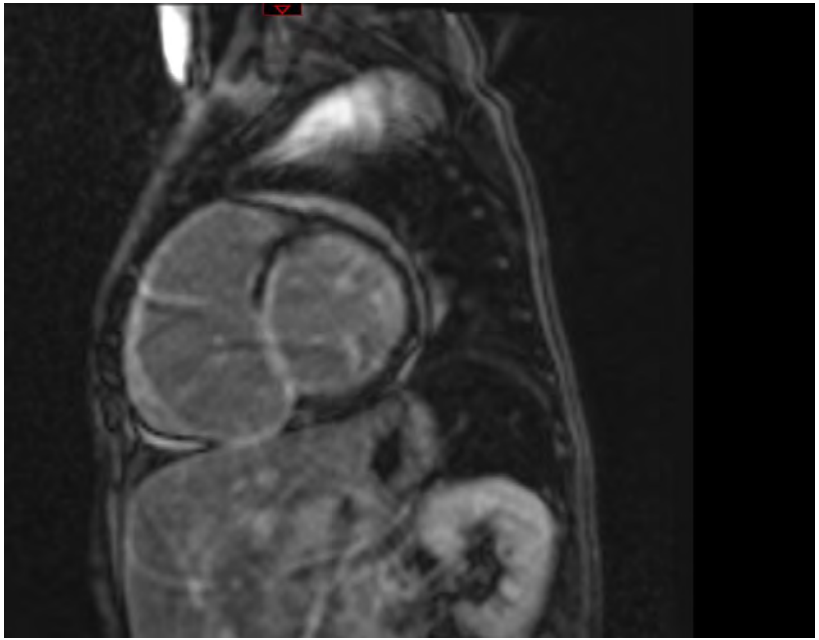


Table 6 Controlled immusuppression trials in myocarditis and dilated car

Trial	Year	Type	Pts (*n*)	Diagnosis	Primary e
Prednisone trial for DCM	1989	Randomized controlled trial (RCT): prednisone (PDN)	102	'Reactive' DCM ($n = 60$) 'Nonreactive DCM)' ($n = 42$)	Either highe fraction (or lower dimensio exercise
MTT	1995	RCT: PDN and cyclosporine or azathioprine	111	Acute biopsy-proven myocarditis (unknown aetiology)	LVEF at 6 m
Giant cell myocarditis treatment trial	2008	Prospective: PDN and cyclosporine	11	Giant cell myocarditis (autoimmune)	Survival at 1
	2003	Prospective: PDN and azathioprine	41	Active myocarditis and chronic heart failure (aetiology known in retrospect)	LVEF at 1 ye
	2001	RCT: PDN and azathioprine	84	Inflammatory DCM (unknown aetiology, increased HLA espression on EMB)	LVEF at 3 m 2 years
TIMIC	2009	RCT: PDN and azathioprine	85	Inflammatory virus-negative DCM	LVEF at 6 months

Favourable

Frustaci[101]

Recommendations

21. Immunosuppression should be started only after ruling out active infection on EMB by PCR.
22. Based on experience with non-cardiac autoimmune disease, the task group recommends consideration of immunosuppression in proven autoimmune (e.g. infection-negative) forms of myocarditis, with no contraindications to immunosuppression, including giant cell myocarditis, cardiac sarcoidosis, and myocarditis associated with known extra-cardiac autoimmune disease.[10,99]
23. Steroid therapy is indicated in cardiac sarcoidosis in the presence of ventricular dysfunction and/or arrhythmia and in some forms of infection-negative eosinophilic or toxic myocarditis with heart failure and/or arrhythmia.
24. Immunosuppression may be considered, on an individual basis, in infection-negative lymphocytic myocarditis refractory to standard therapy in patients with no contraindications to immunosuppression.
25. Follow-up EMB may be required to guide the intensity and the length of immunosuppression.

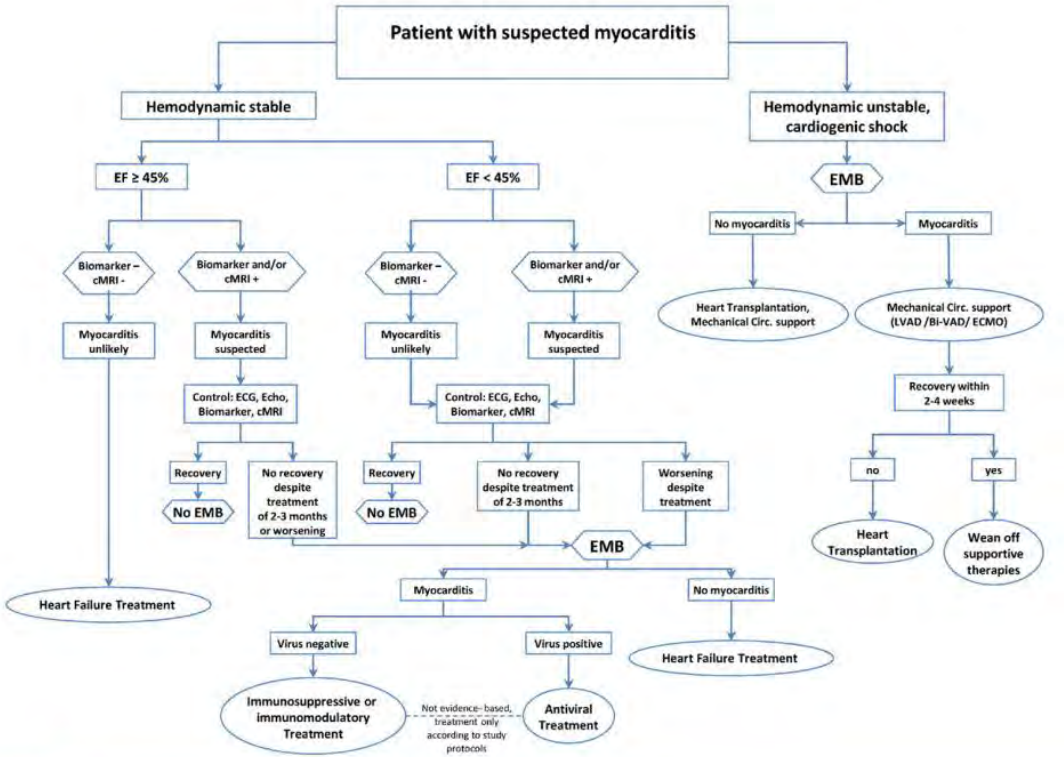


Figure 5 **Proposed Diagnostic and Therapeutic Algorithm for Suspected Myocarditis**

Proposed diagnostic and therapeutic algorithm for patients with suspected acute myocarditis considering biomarkers, cardiac magnetic resonance imaging (cMRI), and endomyocardial biopsy (EMB). Bi-VAD = biventricular assist device; Circ. = circulatory; ECMO = extracorporeal membrane oxygenation; LV = left ventricular; LVAD = left ventricular assist device.

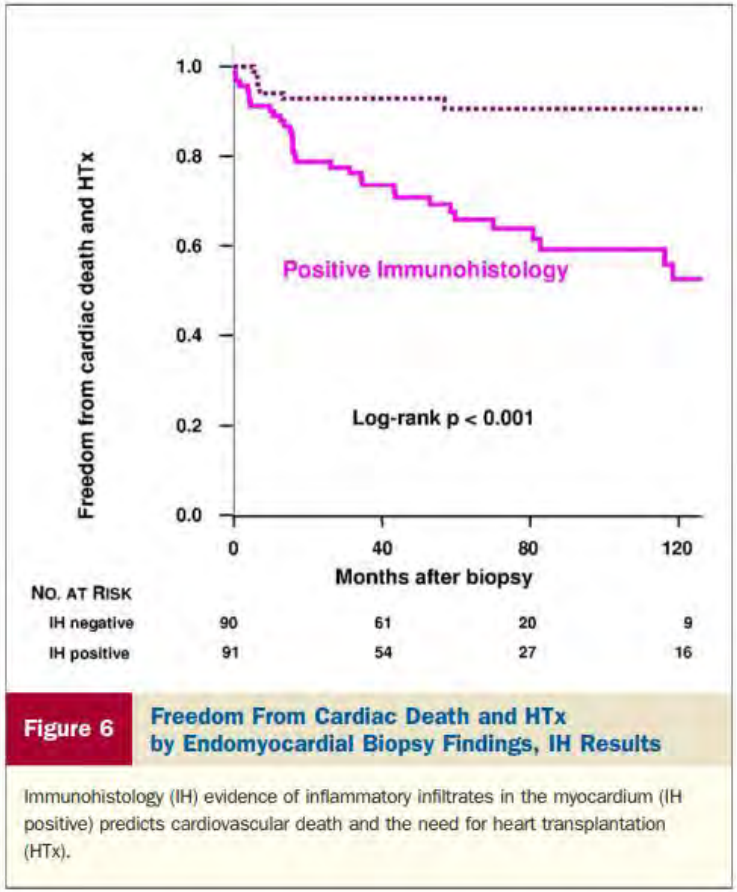


Figure 6 **Freedom From Cardiac Death and HTx by Endomyocardial Biopsy Findings, IH Results**

Immunohistology (IH) evidence of inflammatory infiltrates in the myocardium (IH positive) predicts cardiovascular death and the need for heart transplantation (HTx).

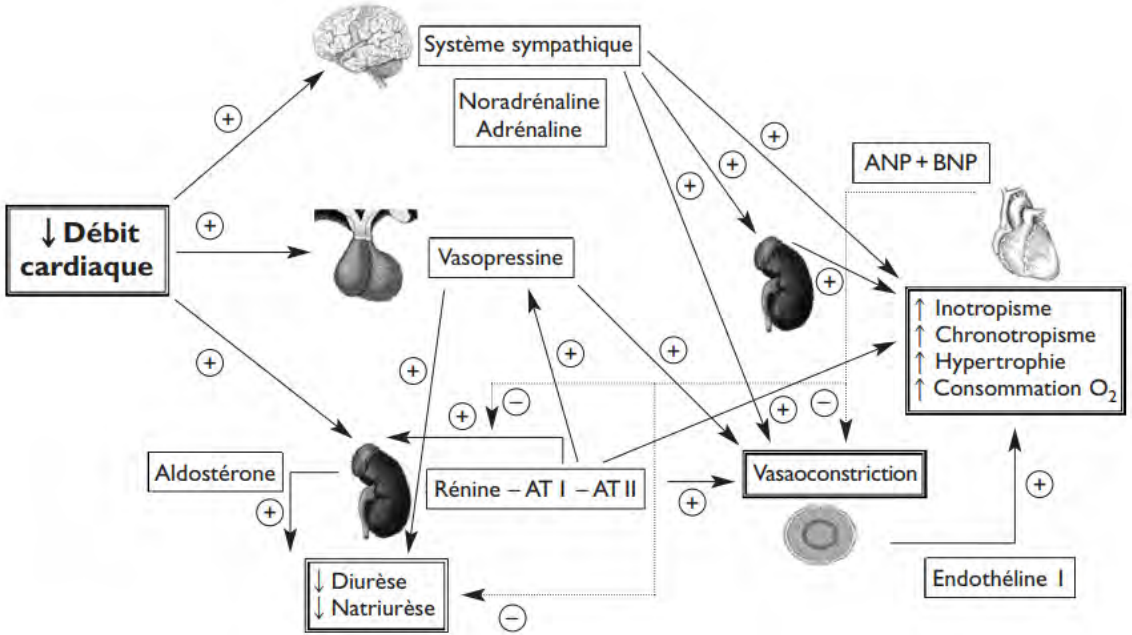


Figure 1. Mécanismes de régulation neurohormonaux de l'insuffisance cardiaque

La baisse du débit cardiaque va provoquer au niveau du système nerveux central la stimulation du système sympathique agissant directement sur le cœur, les vaisseaux périphériques et les surrénales avec une augmentation de production d'adrénaline et de noradrénaline endogène. L'effet sur le système nerveux central est complété via l'hypothalamus et l'hypophyse par la sécrétion de vasopressine qui agira par effet vasoconstricteur direct et par effet antidiurétique direct. Finalement, la perfusion rénale diminuant, le système rénine-angiotensine I-angiotensine II est activé provoquant des effets cardiaques directs, une vasoconstriction, une stimulation de la production de vasopressine et stimulant la production d'aldostérone ce qui mène à une rétention liquidienne. La vasoconstriction vasculaire est modulée par l'endothéline 1, qui a aussi des effets myocardiques. La seule contre-régulation vient de la production par les cavités cardiaques de peptides natriurétiques atrial (ANP) et cérébral (BNP), qui ont un effet vasodilatateur direct, un effet diurétique et un effet inhibiteur de la rénine.


Simdax®
12,5 mg/5 ml
Levosimendan
Konzentrat zur Herstellung
einer Infusionslösung /
Concentré pour solution
pour perfusion
i.v.
2,5 mg/ml
1 Durchstechflasche /
1 flacon à 5 ml
Simdax® 12,5
Levosimendan
2,5 mg/ml
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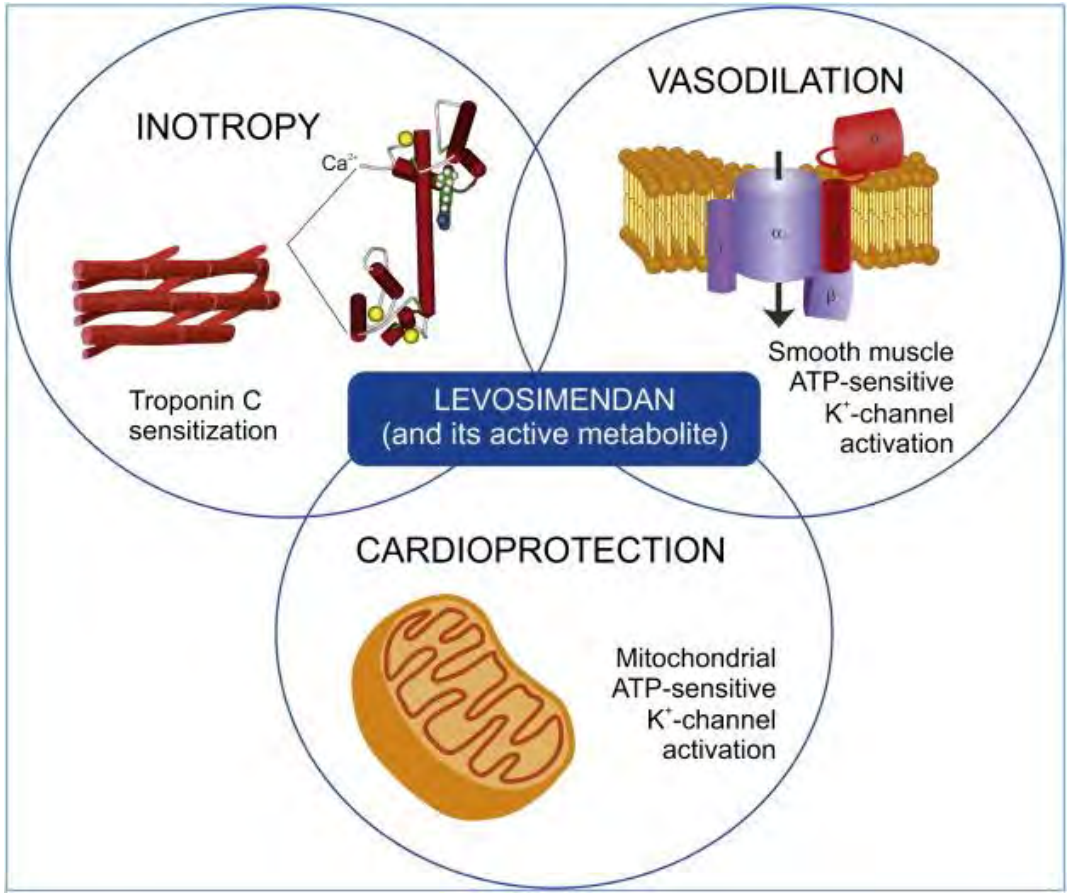
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Troponin C
sensitization
VASODILATION
Smooth muscle
ATP-sensitive
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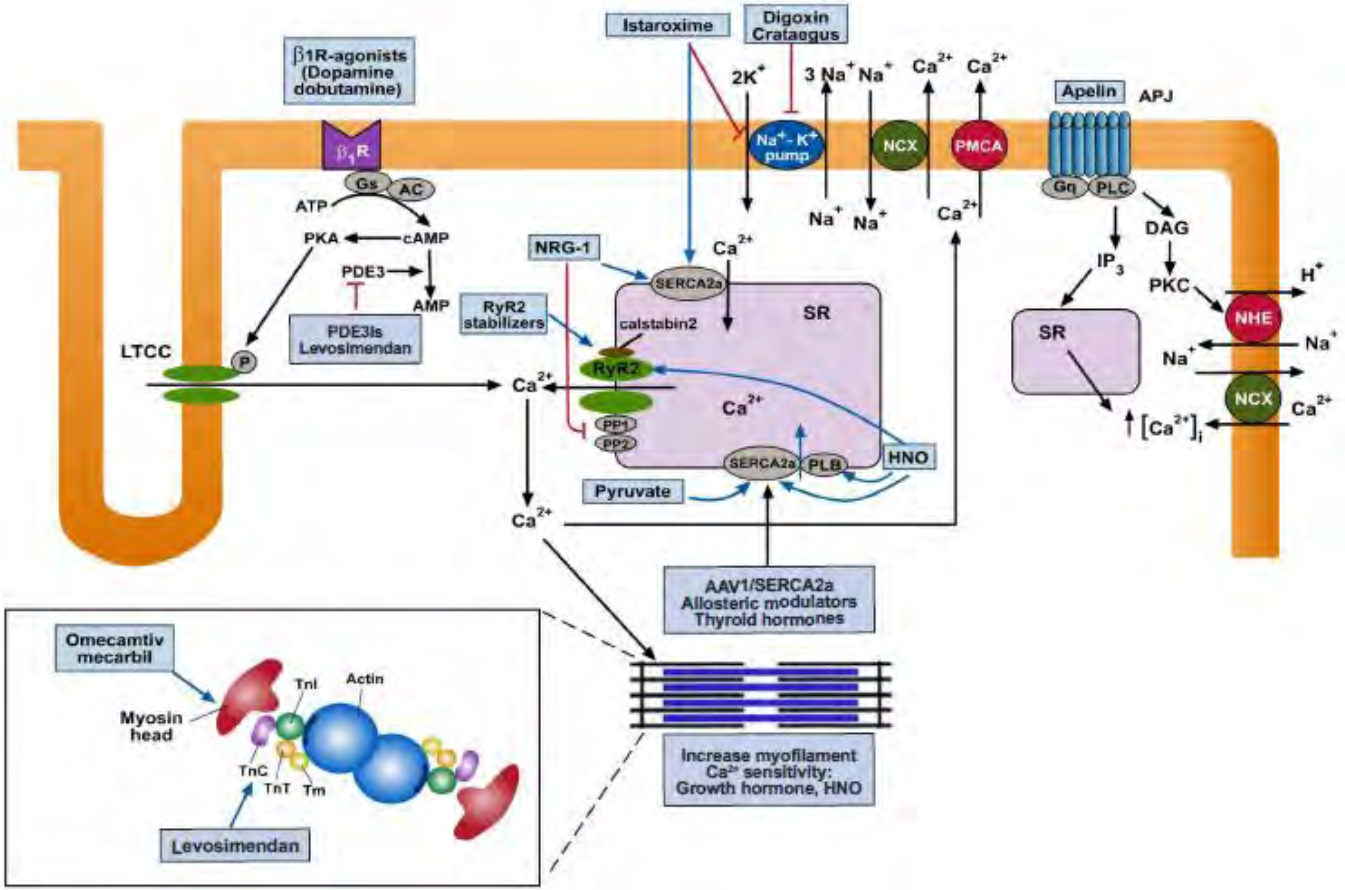
activation
LEVOSIMENDAN
(and its active metabolite)
CARDIOPROTECTION
Mitochondrial
ATP-sensitive
K+-channel
activation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Digoxin
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Na+
Ca2+
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Apelin
APJ
β1R
Na+- K+
pump
NCX
PMCA
Gs
AC
ATP
PKA
cAMP
PDE3
AMP
PDE3Is
Levosimendan
NRG-1
Ca2+
SERCA2a
SR
RyR2
stabilizers
calstabin2
RyR2
LTCC
P
Ca2+
PP1
PP2
SERCA2a
PLB
HNO
Pyruvate
Gq
PLC
DAG
IP3
PKC
H+
NHE
Na+
NCX
Ca2+
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AAV1/SERCA2a
Allosteric modulators
Thyroid hormones
Omecamtiv
mecarbil
Myosin
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TnI
Actin
TnC
TnT
Tm
Levosimendan
Increase myofilament
Ca2+ sensitivity:
Growth hormone, HNO

Original Article

The Hemodynamic Eff of Repetitive Levosime on Children With Dila

Pertti Suominen, MD, PhD[1], Nikla Olle Nyblom, MD[2], Paula Rautiair Maila Turanlahti, MD, PhD[2], and (

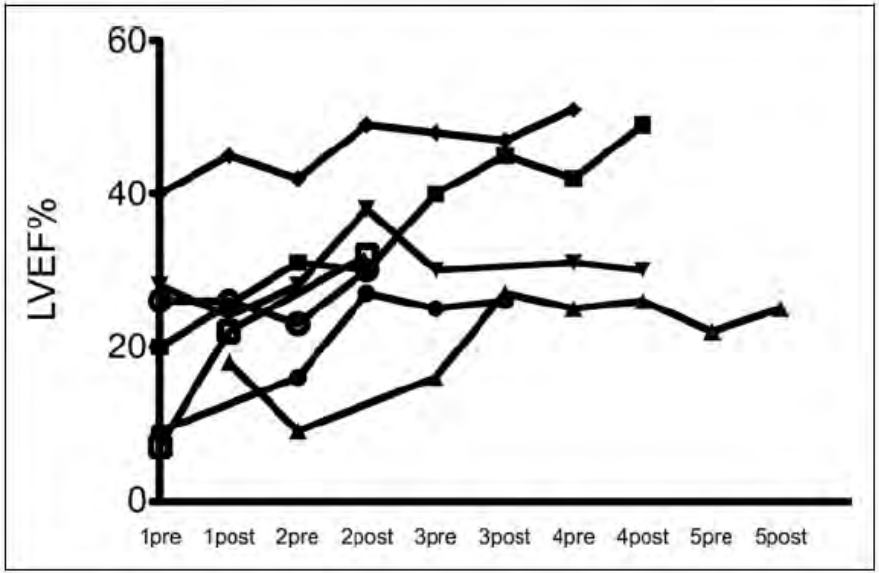


Figure 2. Seven patients with favorable responses as measured by increases in mean left ventricular ejection fraction after repetitive levosimendan infusion.

Pediatr Cardiol (20
https://doi.org/10.1

ORIGINAL

Use of Le
Repair of

Vivianne Amie
Julia Natterer[1]

Received: 25 July

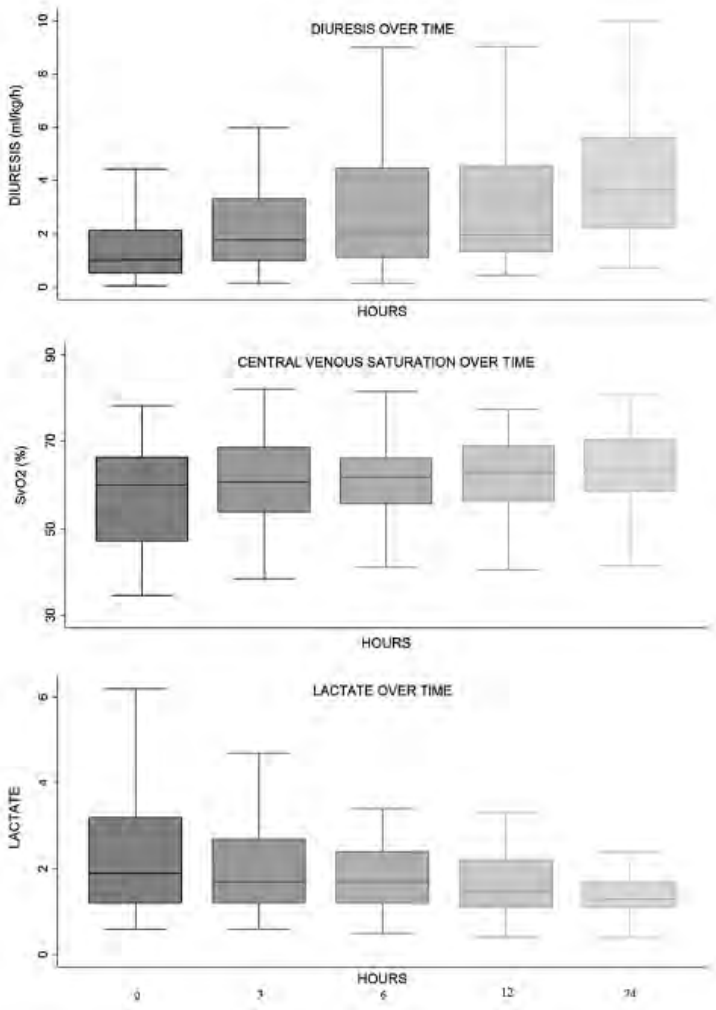


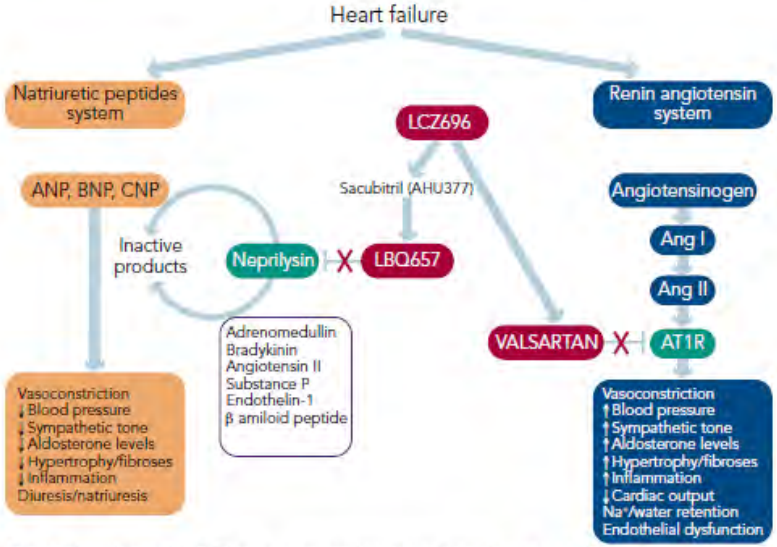
Fig. 1 Median with interquartile range (*box plot*) and minimum and maximum values (*lines*) of diuresis, central venous oxygen saturation, and lactate at time 0, before initiation of levosimendan infusion, and then at 3, 6, 12, and 24 h after the beginning of the infusion

Immunologique:
IVIG 1g / kg iv sur 24 h 2x
Schéma prednisone:
- 1 mg/kg 1x/j ad J5
- 0.5 mg/kg 1x/j ad J5
- 0.25 mg/kg 1x/j ad J5

Spironolactone 20 mg 2x/j
Captopril 7 mg 3x/j
Torasemide 2.5 mg 1x/j
Bisoprolol 2.5 mg 1x/j
Entresto /Enalapril

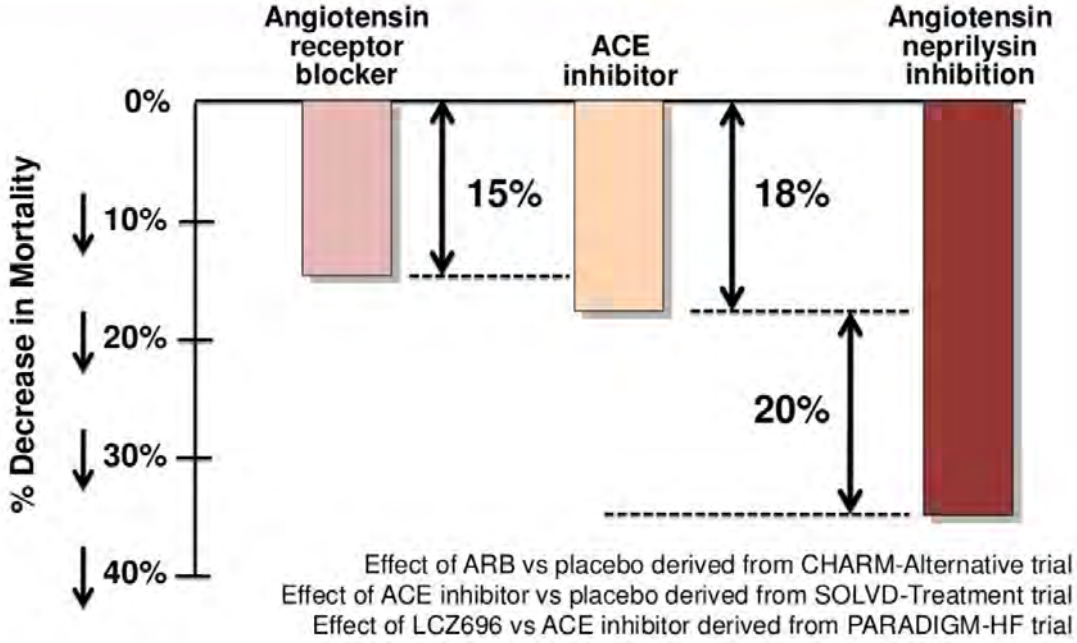
Entresto™
(sacubitril/valsartan) tablets
24/26mg · 49/51mg · 97/103mg

Figure 1: The Role of the Natriuretic Peptides in Heart Failure

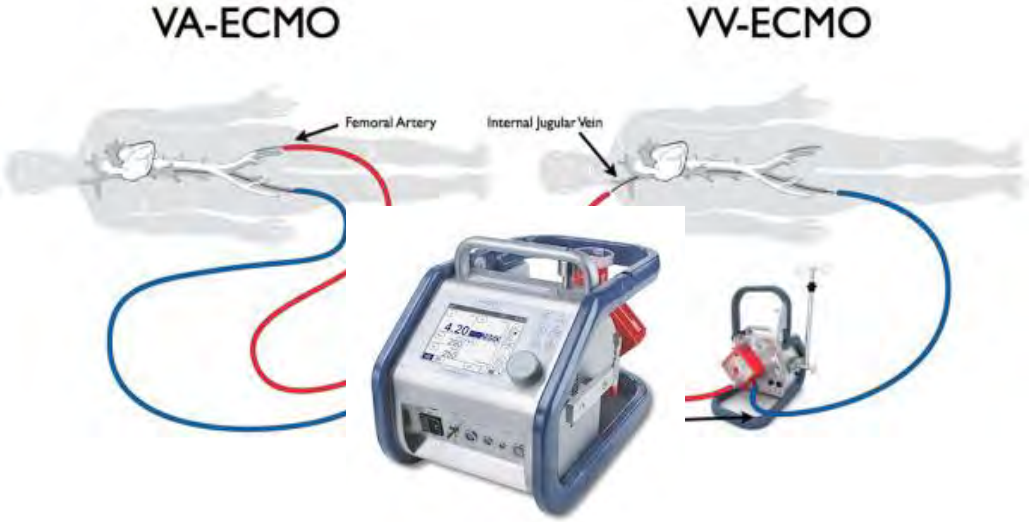


The natriuretic peptide (NP) system comprises three homologous peptides: atrial (ANP), brain (BNP) and C-type (CNP), and two biologically active receptors. ANP and BNP bind to the natriuretic receptor-A (NPR-A) and CNP specifically binds to the NPR-B. NPR-A and NRP-B are coupled to particulate forms of guanylyl cyclase (GC-A and GC-B) and catalyse the synthesis of cyclic guanosine (cGMP), which modulates the activity of cGMP-dependent protein kinase G (PKG) to exert its multiple cardiac, vascular and renal actions. The NP-cGMP-PKG signalling pathway is terminated by phosphodiesterases (PDEs) that hydrolyse cGMP to guanosine monophosphate (GMP). NPs are removed from the circulation and inactived by the clearance receptor (NPR-C) and degraded by several peptidases, including neprilysin (neutral endopeptidase) (NEP). In addition, the NPR-C mediates non-cGMP regulated biological actions. DAG: = diacilglicerol; GTP = guanosine triphosphate; IP3 = inositol 1,4,5-trifosfato; LTCC = L-type calcium channel; PLC = phospholypase C; RAAS = renin-angiotensin-aldosterone system; UROD = urodilatin.

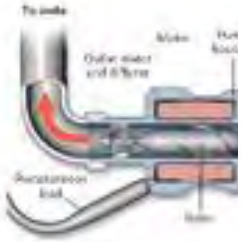
Angiotensin Neprilysin Inhibition With LCZ696 Doubles Effect on Cardiovascular Death of Current Inhibitors of the Renin-Angiotensin System

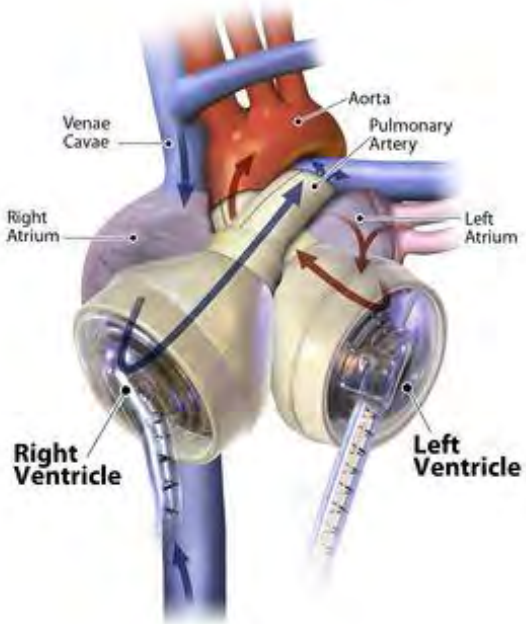


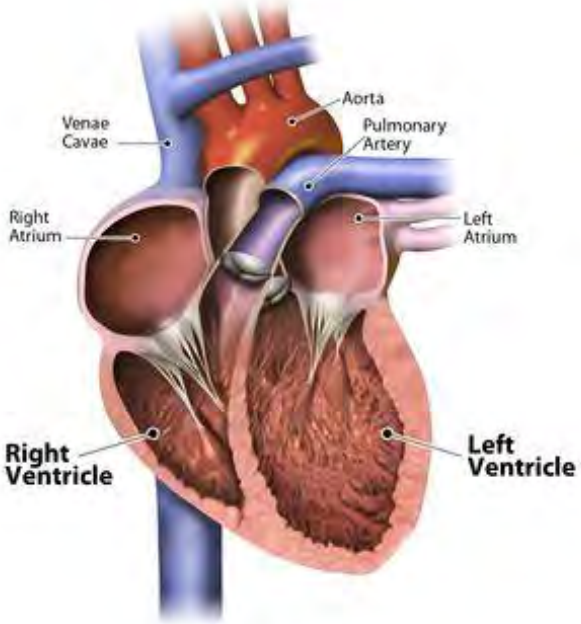
ET ENSUITE ?


VA-ECMO
VV-ECMO
Femoral Artery
Internal Jugular Vein

THORA
CORPORA







1901: Alexis Carrel (Lyon)

- Première greffe rénal animale expérimentale

1905: Charles Claude Guthrie (Chicago)

- Première greffe cardiaque hétérotopique animale, anastomose vasculaire sur le chien

1930: Franck C. Mann (Mayo Clinic)

- Héparine
- Description du rejet

1946: Vladimar Petrovich Demikhov (Moscou)

- Première greffe cardiaque intrathoracique animale

1960: Lower & Shumway (Stanford)

Cardioplégie, Greffon 4°C, technique de la coiffe des oreillettes

1964: James Hardy (Mississipi)

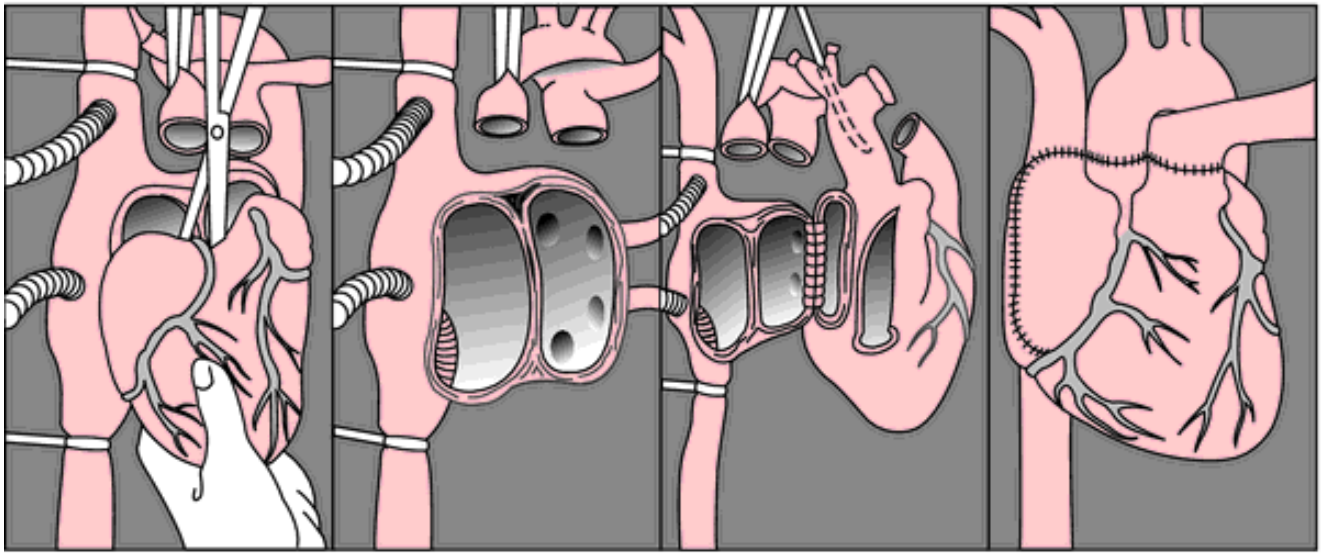
Première Xénogreffe cardiaque (Chimpanzé- humain)

1967: Christian Barnard (Cape Town)

Première greffe cardiaque adulte

1967: Adrian Kantrowitz (New York)

Première greffe cardiaque pédiatrique



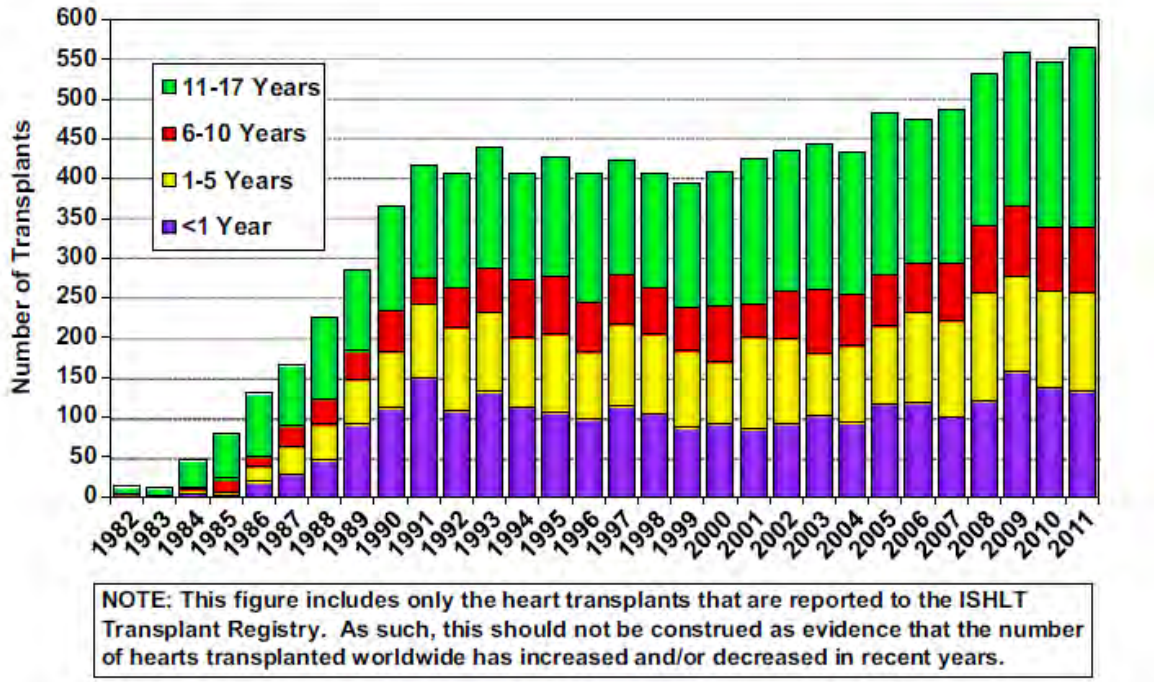
1. After the recipient is placed on cardiopulmonary bypass, the heart is removed.

2. The posterior walls of the recipient's left and right atria are left intact.

3. The left atrium of donor heart is anastomosed to the recipient's residual posterior atrial walls, and the other atrial walls, the atrial septum, and the great vessels are joined.

POSTOPERATIVE RESULT

En 2017, le recul sur la transplantation cardiaque pédiatrique a atteint 35 ans



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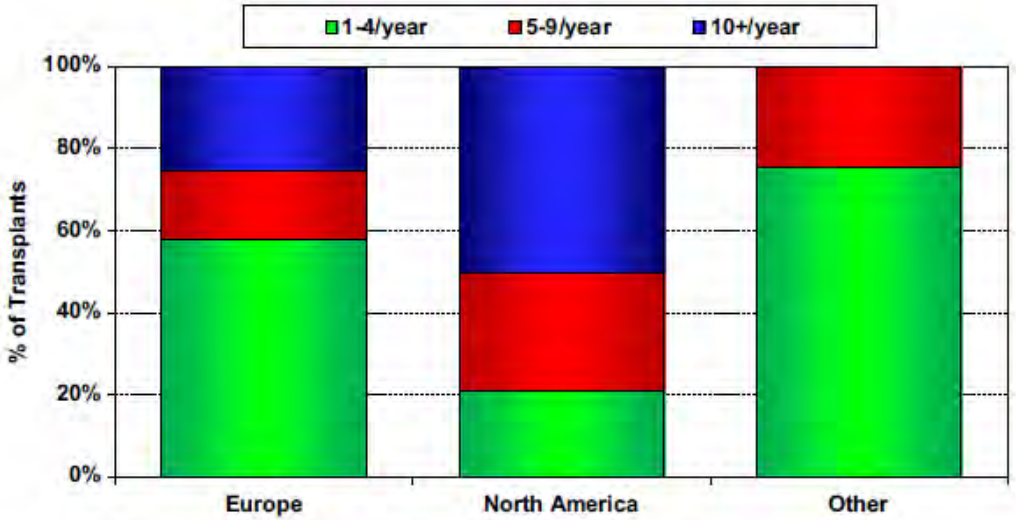

Figure 4 Distribution of transplants by location and average center volume (Transplants: January 2000–June 2012).

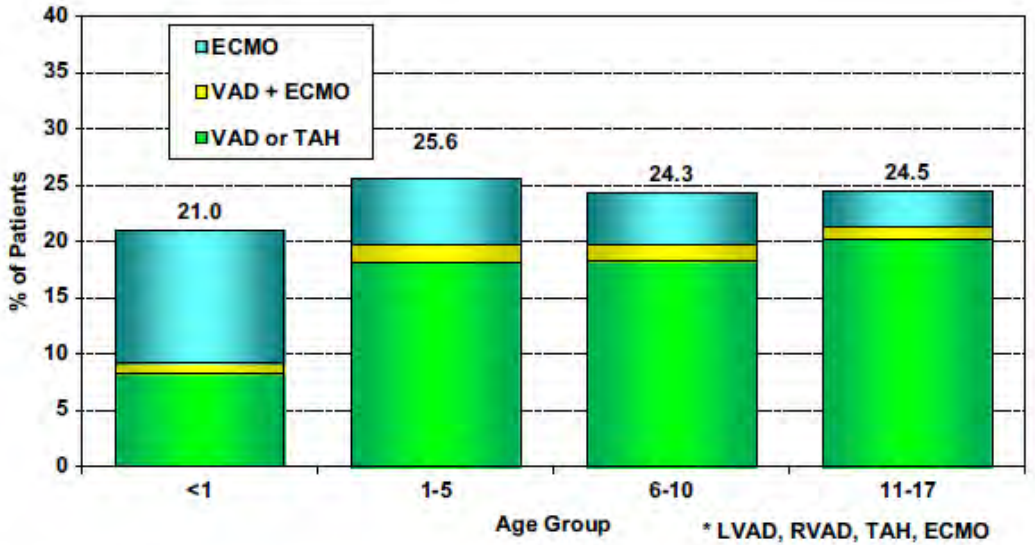

Figure 8 Percentage of patients bridged with mechanical circulatory support by age group (Transplants: July 2004–June 2012). ECMO, extracorporeal membrane oxygenation; LVAD, left ventricular assist device; RVAD, right ventricular assist device; TAH, total artificial heart; VAD, ventricular assist device.

Waiting List Mortality Among Children Listed for Heart Transplantation in the United States

Christopher S.D. Almond, MD, MPH*; Ravi R. Thiagarajan, MBBS, MPH*; Gary E. Piercey, BS; Kimberlee Gauvreau, ScD; Elizabeth D. Blume, MD; Heather J. Bastardi, NP; Francis Fynn-Thompson, MD; T.P. Singh, MD, MS

(*Circulation*. 2009;119:717-727.)

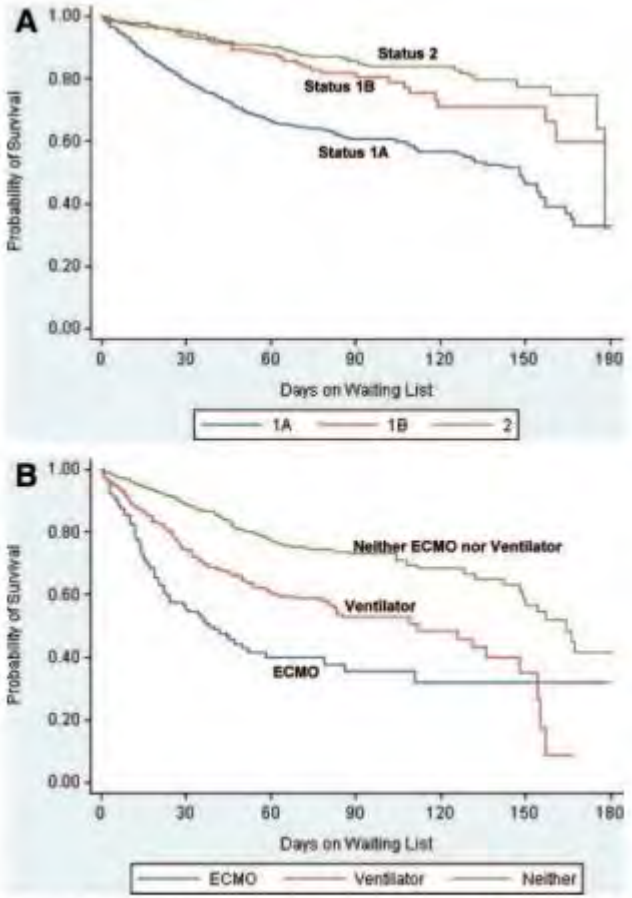

Figure 1. Kaplan-Meier survival for all children listed for heart transplant according to listing status (A) and for those children listed as status 1A according to invasive hemodynamic support (B).

Myocardite est d’abord une maladie infectieuse qui se transforme en maladie auto-immune

Son identification, son traitement et sa prise en charge nécessite des moyens techniques et humains importants