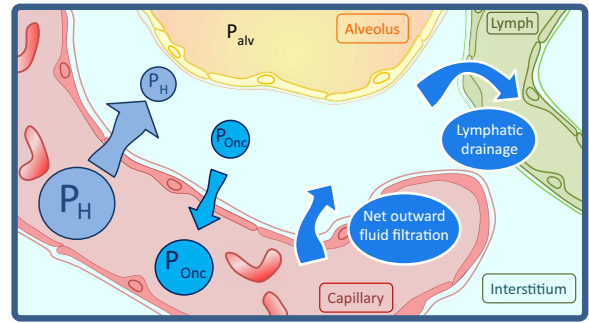


OAP hydrostatique

B Séguy PH USIC-Hémodynamique
Lacanau 11 juin 2016

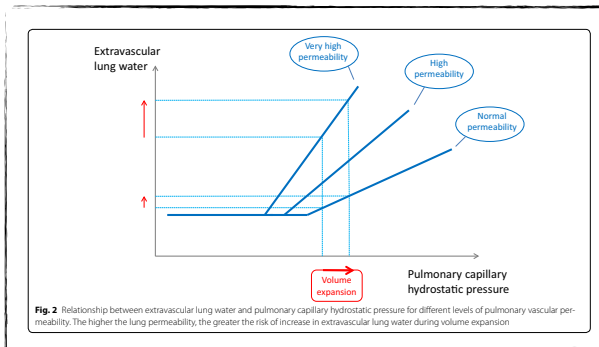
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$$\text{Flux sortant «net»} = K(P_{H \text{ capillaire}} - P_{H \text{ interstitielle}}) - K(P_{Onc \text{ capillaire}} - P_{Onc \text{ interstitium}})$$

Jozwiak & al ICM 2015

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Augmentation pression hydrostatique

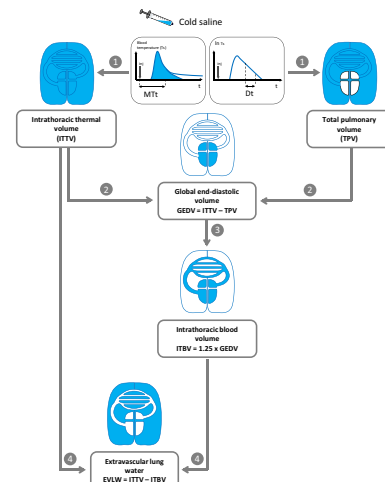
Diminution de la pression oncotique

Diminution de perméabilité capillaire

Jozwiak & al ICM 2015

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Mesure de l'eau pulmonaire extra vasculaire en pratique clinique



Jozwiak & al ICM 2015

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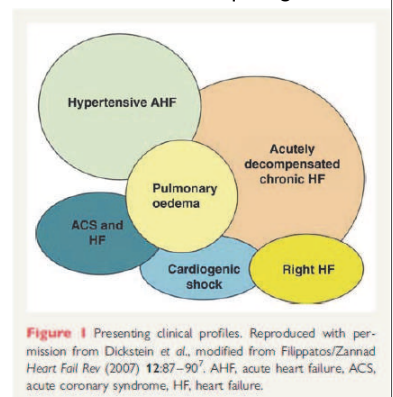
	Study	Number of patients	Type of EVLW indexation	Prognostic value
General critically ill patients	Sakka et al. [4]	373	Actual body weight	Independent predictor of ICU mortality
	Martin et al. [3]	29	Actual body weight	Higher EVLW in ICU non-survivors
	Chung et al. [75]	33	Actual body weight	Independent predictor of in-hospital survival
	Chung et al. [76]	67	Actual body weight	Independent factor for the development of MODS
	Chew et al. [73]	51	Actual and predicted body weight	Higher EVLW in ICU non-survivors
ARDS patients	Mallat et al. [78]	55	Actual and predicted body weight	Independent predictor of ICU mortality
	Philips [85]	59	Actual and predicted body weight	Good predictor of ICU mortality
	Craig et al. [45]	44	Predicted body weight	Independent predictor of ICU mortality
	Brown et al. [37]	59	Predicted body weight	Independent predictor of ICU mortality
	Jozwiak et al. [36]	200	Predicted body weight	Independent predictor of Day 28 mortality

Jozwiak & al ICM 2015

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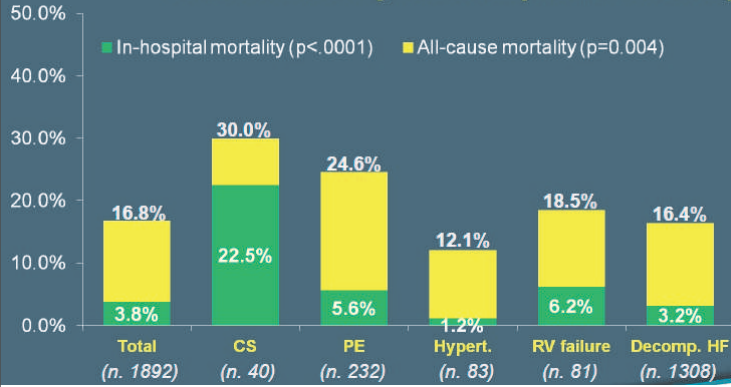
1° cause d'OAP «hydrostatique» = Insuffisance cardiaque aigue

- $\nearrow$ Ph Cap. = $\nearrow$ PTDVG
- 95% des patients avec ICA présentent des signes de surcharge pulmonaire



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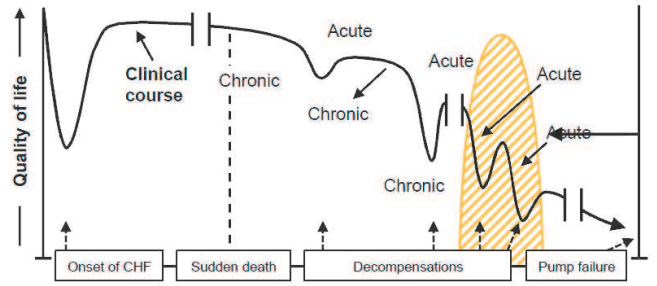
Acute HF: All-cause mortality*: Overall and by clinical profile at entry



European Journal of Heart Failure (2013) 15, 808–817

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A depiction of the clinical course of HF



A chaque décompensation, une dégradation des organes cibles qui aggrave le pronostic

Circulation. 2012;125:1928-1952

samedi 11 juin 16

L'examen clinique est rarement normal

L'ECG est rarement normal

Les BNP ont une bonne VPN (> 90%) mais la VPP est médiocre (66%)

Ponikowski & al Eur Heart J 2016

Madamanchi & al Int J cardiol 2014

Causes non cardiaques d'augmentation des peptides natriurétiques

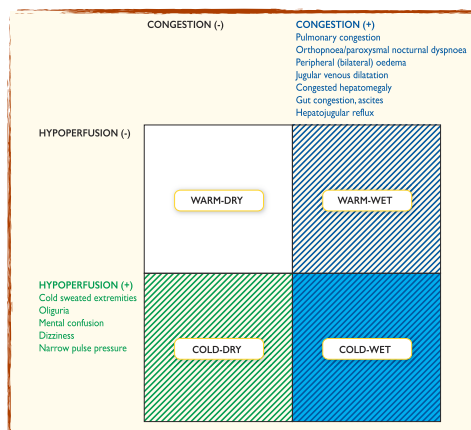
Cardiac	- Heart failure - Acute coronary syndromes - Pulmonary embolism - Myocarditis - Left ventricular hypertrophy - Hypertrophic or restrictive cardiomyopathy - Valvular heart disease - Congenital heart disease - Atrial and ventricular tachyarrhythmias - Heart contusion - Cardioversion, ICD shock - Surgical procedures involving the heart - Pulmonary hypertension
Non-cardiac	- Advanced age - Ischaemic stroke - Subarachnoid haemorrhage - Renal dysfunction - Liver dysfunction (mainly liver cirrhosis with ascites) - Paraneoplastic syndrome - Chronic obstructive pulmonary disease - Severe infections (including pneumonia and sepsis) - Severe burns - Anaemia - Severe metabolic and hormone abnormalities (e.g. thyrotoxicosis, diabetic ketosis)

Faux négatif : obèse

Ponikowski & al Eur Heart J 2016

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Diagnostic clinique

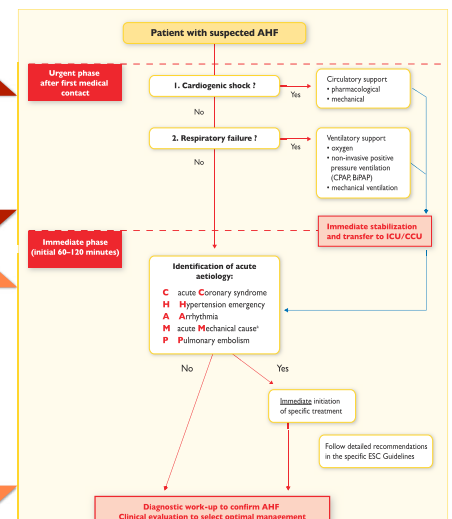


Ponikowski & al Eur Heart J 2016

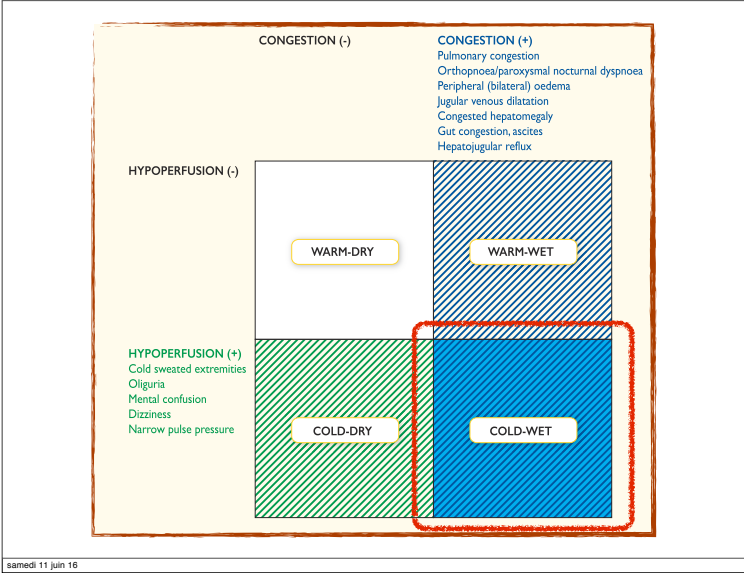
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Phase urgente

Phase immédiate



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Prise en charge respiratoire phase urgente

La VNI doit être considérée chez tout les patients en détresse respiratoire aigue et commencé dès que possible **IIa B**

La ventilation mécanique est recommandée pour les patients en échec de traitement non invasif **I C**

$O_2 > 90\%$ et $PO_2 > 8$ kPa **I C**

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Identification des patients instable hémodynamiquement

OAP + hypoTA/
hypoperfusion

Pronostic péjoratif

PEC en centre spécialisée

Diagram illustrating patient profiles based on Congestion and Hypoperfusion status (same as slide 1):

	CONGESTION (-)	CONGESTION (+) Pulmonary congestion Orthopnoea/paroxysmal nocturnal dyspnoea Peripheral (bilateral) oedema Jugular venous dilatation Congested hepatomegaly Gut congestion, ascites Hepatojugular reflux
HYPOPERFUSION (-)	WARM-DRY	WARM-WET
HYPOPERFUSION (+) Cold sweated extremities Oliguria Mental confusion Dizziness Narrow pulse pressure	COLD-DRY	COLD-WET

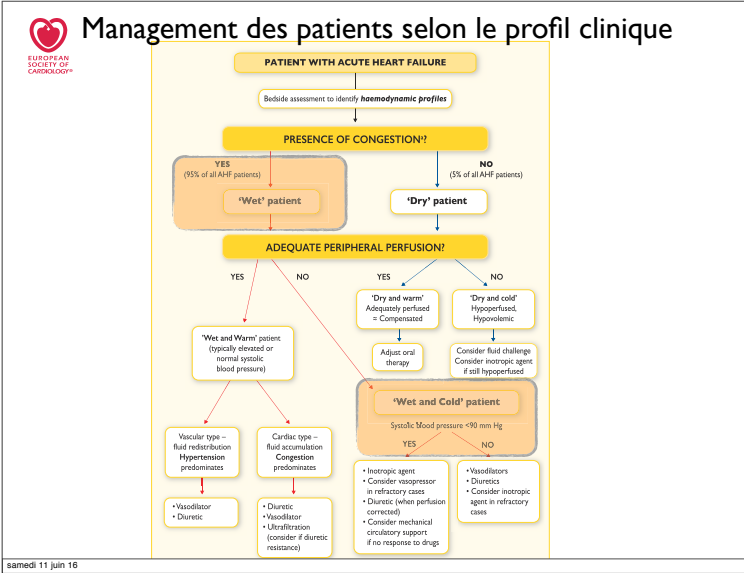
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Phase immédiate

- Identification des facteurs déclenchant nécessitant une PEC urgente (< 120mn)
- Sy coronarien aigue (Angio<2h)
- Complication mécanique aigue (IM rupture de cordage, Endocardite...)
- Urgence hypertensive
- Urgence rythmique

Role de l'ETT à la phase aigue surtout si instable hémodynamiquement **I**

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Prise en charge médicamenteuse

Diuretics

Intravenous loop diuretics are recommended for all patients with AHF admitted with signs/symptoms of fluid overload to improve symptoms. It is recommended to regularly monitor symptoms, urine output, renal function and electrolytes during use of intravenous diuretics.

In patients with new-onset AHF or those with chronic, decompensated HF not receiving oral diuretics the initial recommended dose should be 20–40 mg i.v. furosemide (or equivalent); for those on chronic diuretic therapy, initial i.v. dose should be at least equivalent to oral dose.

It is recommended to give diuretics either as intermittent boluses or as a continuous infusion, and the dose and duration should be adjusted according to patients' symptoms and clinical status.

Combination of loop diuretic with either thiazide-type diuretic or spironolactone may be considered in patients with resistant oedema or insufficient symptomatic response.

Vasodilators

i.v. vasodilators should be considered for symptomatic relief in AHF with SBP > 90 mmHg (and without symptomatic hypotension). Symptoms and blood pressure should be monitored frequently during administration of i.v. vasodilators.

In patients with hypertensive AHF, i.v. vasodilators should be considered as initial therapy to improve symptoms and reduce congestion.

Inotropic agents – dobutamine, dopamine, levosimendan, phosphodiesterase III (PDE III) inhibitors

Inotropic agents are not recommended unless the patient is symptomatically hypotensive or hypoperfused because of safety concern.

Other drugs

For acute control of the ventricular rate in patients with atrial fibrillation:

a. digoxin and/or beta-blockers should be considered as the first-line therapy.

b. amiodarone may be considered.

Recommendations:

Recommendation	Class	Level
Intravenous loop diuretics	I	C
Initial recommended dose of 20–40 mg i.v. furosemide	I	B
Combination of loop diuretic with thiazide-type diuretic or spironolactone	IIb	C
i.v. vasodilators for symptomatic relief	IIa	B
i.v. vasodilators for hypertensive AHF	IIa	B
Inotropic agents	III	A
digoxin and/or beta-blockers	IIa	C
amiodarone	IIb	B

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Peu de nouveauté !

Table 4 Short- and long-term novel therapies for acute heart failure syndromes

Short term	Long term	Both
Cinaciguat	Direct renin inhibitors	Adenosine antagonists
CD-NP	Macronutrients	Vasopressin antagonists
Relaxin	Micronutrients	Digoxin
Adenosine regulating agents	CRT/AICD	
Stresscopin		
Istaroxime		
Cardiac myosin activators		

European Heart Journal (2010) 31, 784–793

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Et l'EER /UF ?

Recommendations	Class ^a	Level ^b	Ref ^c
Ultrafiltration may be considered for patients with refractory congestion, who failed to respond to diuretic-based strategies.	IIb	B	578–580
Renal replacement therapy should be considered in patients with refractory volume overload and acute kidney injury.	IIa	C	

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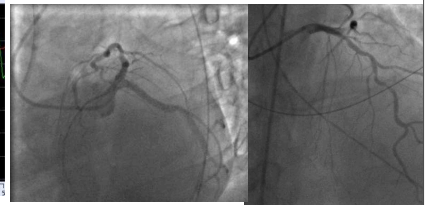
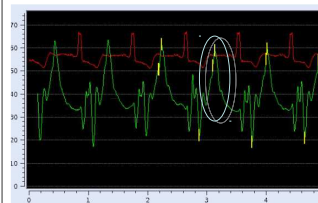
Medical History

- 43 year-old male with no past medical history
- Posterior STEMI
- Primary PCI at H12 in local hospital (no on site surgery)
- Proximal Cx TIMI 0 and Mild LAD stenosis
- Succesful Cx desobstruction (Thrombo-aspiration and BMS implantation)
- At d5 acute pulmonary oedema mechanical ventilation
- Ventilator acquired pneumoniae -Septic shock
- Weaning failure from ventilator the next 3 wks
- A TEE done during one failed weaning test demonstrated massive MR and EF% 35
- Transferred for urgent mitral valve surgery
- At arrival , patient was sedated,ventilated with low tidal volume - 100% O₂ (P/F ratio 90) and under 1 mg/h epinephrine



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Hemodynamic and TEE evaluation on arrival



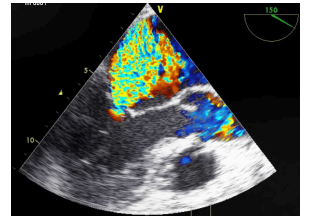
MeanPulmonary wedge pressure 38 mmHg

Mean pulmonary artery pressure 55 mmHg

Cardiac Index 2.3 l:mn:m²

Aortic pressure 85/50 mmHg

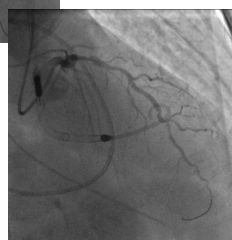
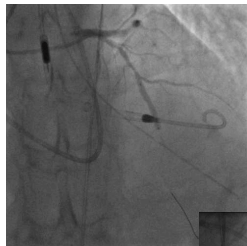
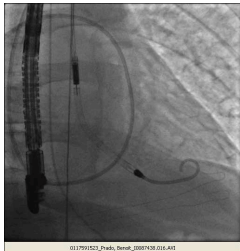
Left ventricular pressure 90/35 mmHg



Grade IV acute mitral regurgitation and EF % 35

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Impella LP 2.5 implantation and LAD PCI



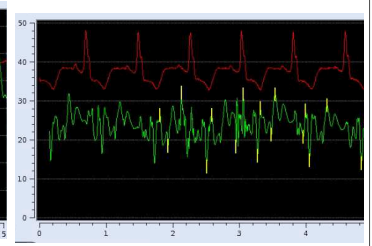
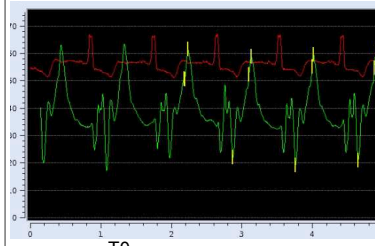
Succesful implantation of Impella 2.5 by right femoral artery

Significant mild LAD stenosis treated with BMS implantation

Pemeability of the Prox Circumflex stent

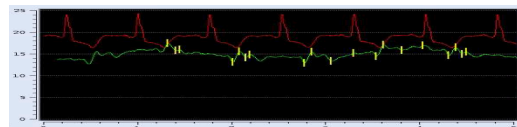
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Pulmonary wedge pressure evolution under Impella implantation



T0

T 15 mn

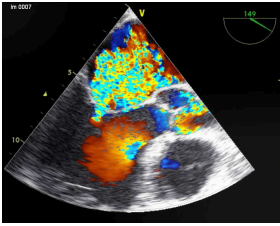


T30mn

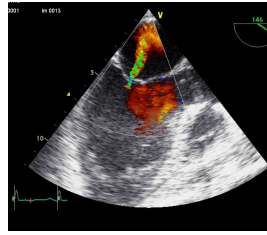
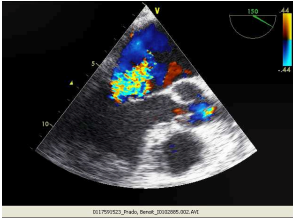
Mean Pulmonary wedge pressure 15 mmHg at 30 mn

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Confirmed by TEE ..



Before impella ...



After ...

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Conclusion

- OAP hydrostatique associé à une surmortalité
- La première cause est l'insuffisance cardiaque aigue
- La prise en charge est
 - «urgente» : PEC Ins Resp Aigue et identifier les chocs cardiogéniques
 - «immédiate» (<120mn): SCA , complications mécaniques
- Apport diagnostique et pronostic de l'ETT

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