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# Interest of lung ultrasound in the management of acute heart failure in post-emergency service

Elena Bel Alonso

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Année : 2022

**INTÉRÊT DE L'ÉCHOGRAPHIE PLEURO PULMONAIRE DANS LA  
PRISE EN CHARGE DE LA DÉCOMPENSATION CARDIAQUE EN  
POST URGENCE (EPPICA)**

THÈSE

POUR LE DIPLÔME D'ÉTAT DE DOCTEUR EN MÉDECINE  
SPÉCIALITÉ : MÉDECINE D'URGENCE

Par Mme Elena BEL ALONSO

*[Données à caractère personnel]*

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# RÉSUMÉ

## *Introduction*

La congestion pulmonaire est un facteur pronostique majeur dans l'insuffisance cardiaque aigue et donc une cible thérapeutique importante. L'échographie pleuropulmonaire s'est imposée dans la dernière décennie comme une méthode simple, rapide et non invasive dans l'évaluation dynamique de la surcharge pulmonaire au lit du patient. L'objectif de cette étude est de corrélérer les données échographiques, la posologie de diurétiques administrée, et l'évolution clinico-biologique des patients en service de médecine polyvalente.

## *Matériel et méthodes*

Dans cette étude prospective, 42 patients admis pour insuffisance cardiaque aigue dans le service de médecine interne du Centre Hospitalier Universitaire Grenoble Alpes ont été successivement inclus entre mai 2021 et juillet 2022. L'échographie pleuropulmonaire, l'examen clinique et la biologie sont recueillis quotidiennement. Le statut vital est recueilli 30 jours après la dernière échographie. Le critère de jugement principal étant l'évolution du nombre de lignes B en échographie pleuropulmonaire en fonction de l'évolution des doses de diurétiques, nous réaliserons un modèle de régression JoinPoint, suivi d'une analyse des tendances temporelles sur l'évolution des lignes B et des doses de diurétiques.

## *Résultats*

On observe une tendance à la baisse des lignes B qui est plus importante en début d'hospitalisation et qui stagne dans le dernier quart de celle-ci. La tendance est similaire pour la dose de diurétique administrée sans que les deux soient corrélés. La mortalité à 30 jours est quant à elle liée de façon significative au nombre de lignes B à la sortie ( $p < 0.001$ ). L'échographie pleuropulmonaire pourrait permettre l'optimisation de la décongestion dans les derniers jours de la prise en charge et l'amélioration du pronostic à 30 jours.

## *Conclusion*

L'absence de gold standard dans l'évaluation quantitative de la surcharge au cours d'un épisode d'insuffisance cardiaque aigue est un problème dont l'échographie pleuropulmonaire pourrait apporter une solution. Une étude interventionnelle est nécessaire afin d'évaluer l'intérêt d'une prise en charge thérapeutique échoguidée.

**MOTS CLES :** Insuffisance cardiaque aigue, Congestion pulmonaire, Echographie pleuropulmonaire, Diurétiques, Médecine interne

**FILIERE :** Médecine

## ABSTRACT

### INTEREST OF LUNG ULTRASOUND IN THE MANAGEMENT OF ACUTE HEART FAILURE IN POST-EMERGENCY SERVICE

#### *Introduction*

Pulmonary congestion is a major prognostic factor in acute heart failure and therefore an important therapeutic target. Lung ultrasound has emerged in the last decade as an easy-to-use, rapid and non-invasive method for the dynamic assessment of pulmonary congestion at the patient's bedside. The aim of this study is to correlate ultrasound data, diuretic dosage administered, clinical and biological evolution of patients in a multidisciplinary medical ward.

#### *Material and Methods*

In this prospective study, 42 patients admitted for acute heart failure in the internal medicine department of the Grenoble Alpes University Hospital were successively included between May 2021 and July 2022. Pleuropulmonary ultrasound, clinical examination and biology were collected daily. Vital status was collected 30 days after the last lung ultrasound. As the primary endpoint is the evolution of the number of B-lines in pleuropulmonary ultrasound according to the evolution of diuretic doses, we will perform a JoinPoint regression model, followed by a time trend analysis of the evolution of B-lines and diuretic doses.

#### *Results*

There is a downward trend in B-lines, which is greater at the beginning of the hospitalization and stagnates in the last quarter of the stay. The trend is similar for the dose of diuretic administered without the two being correlated. 30-day mortality was significantly related to the number of B-lines at discharge ( $p < 0.001$ ). Lung ultrasound could optimize decongestion in the last days of management and improve the 30-day prognosis.

#### *Conclusion*

The lack of a gold standard in the quantitative assessment of cardiac overload during an episode of acute heart failure is a problem for which lung ultrasound could provide a solution. An interventional trial is needed to assess the value of ultrasound-guided therapeutic management.

KEY WORDS : Acute heart failure ; Pulmonary congestion ; Lung ultrasound ; Internal medicine department

## ABBREVIATIONS

AHF : Acute Heart Failure

AKI : Acute Kidney Injury

ARDS : Acute Respiratory Distress Syndrome

CCI : Charlson Comorbidity Index

CKD : Chronic Kidney Disease

ED : Emergency Department

EF : Ejection Fraction

GFR : Glomerular Filtration Rate

HFrEF : Heart Failure Reduced Ejection Fraction

HEpEF : Heart Failure Preserved Ejection Fraction

HR : Hazard Ratio

LUS : Lung ultrasound

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EEPICA : Echographie pleuropulmonaire dans l'insuffisance cardiaque aigue



## INTRODUCTION

Heart failure is a major public health problem. In France, this frequent and growing pathology affects 2.3% of the adult population and is responsible for 70,000 deaths per year. Acute heart failure (AHF) is one of the leading cause of hospitalization in patients over 65 years old, which are often long and expensive (1).

Pulmonary congestion appears as a major prognostic factor in AHF (2,3), and therefore represents an important therapeutic target (4). However, it is poorly evaluated by the usual methods used in the medical ward : clinical evaluation including crackles and lower limb oedema, is not very sensitive for the detection of pulmonary congestion (5) even by experienced physicians. Moreover, pulmonary auscultation is poorly reproducible between different physicians. However, these clinical data represent the usual patient monitoring and are used to adapt diuretic therapy. This evaluation based on clinical signs and biology, is not standardized, and studies are currently undergoing to protocolize this care, such as the CARESS protocol within the PRODUCT HF study (6).

In intensive care units, different methods have shown its interest in monitoring pulmonary congestion, some of them invasive as thermodilution methodology and others non-invasive as cardiac ultrasound associated with lung ultrasound (LUS) (7). However, a large proportion of patients hospitalized for acute heart failure are not admitted to specialized units.

LUS is an easy-to-use, rapid, reproducible and non-invasive method that offers bedside evaluation of pulmonary congestion status (8). Minimum training is required and reproducible results are obtained after 30 minutes to 1 hour of practice (9). B-line or comet tail is an ultrasound artefact, a vertical hyperechogenic line starting from the pleural line, not fading, and

moving synchronously with the lung sliding. It reflects the presence of engorged interlobular septa, an area where air is in close contact with water, air content decreases and lung density increases because of their difference in acoustic impedance. The number of B-lines increases with the reduction in air content. They are correlated with the presence of an alveolar-interstitial syndrome, with a sensitivity and a specificity of 93% (10,11). Pleural effusion is another common finding in AHF, that can be also easily diagnosed by LUS.

The interest of LUS is already proven for the diagnosis of AHF, especially in patients admitted for acute dyspnoea in the emergency department (ED) (9–11). Moreover, LUS allows dynamic monitoring in pulmonary congestion because diuretic administration is followed by a rapid decrease in the number of B-lines (12). Monitoring of B-lines, a tool available for physicians, could be interesting to monitor pulmonary congestion and to adjust treatment for patients with acute heart failure.

It seems interesting to evaluate the relevance of lung ultrasound combined with the use of clinical and biological protocol to adapt diuretics in acute heart failure. Optimizing diuretic therapy could allow a decrease in adverse effects and in hospitalization time improving the patient's prognosis.

In this prospective observational study, we will correlate the B-lines evolution to diuretic posology and clinic and biologic signs of pulmonary congestion in patients admitted with cardiac heart failure in a polyvalent medicine unit where most of the patients come from the ED. We also evaluate the reliability and the feasibility of a daily LUS in a medicine ward.

## MATERIAL AND METHODS

### Study design

EPPICA is a single-centre prospective cohort study, conducted in the internal medicine department of Grenoble Alpes University Hospital.

The internal medicine department is partly dedicated to patients that need urgent hospitalization, directly from the ED of the Grenoble Alpes University Hospital. It takes care patients with any type of medical pathologies, usually with a short hospitalization time (mean stay is around five days). They can leave the service to return home or be transferred to another specialized ward if needed.

Senior doctors working in this department are emergency physicians, general practitioners, geriatrics and internists.

### Population

All patients admitted in internal medicine department between May the 18<sup>th</sup> of 2021 and July the 15<sup>th</sup> of 2022 are successively screened for inclusion in the study. Patients are eligible if they are over 18 years-old and present an acute heart failure at admission. Diagnosis is assessed by two senior physicians, according to international guidelines diagnosis criteria of the European Society of Cardiology (13). Patients with haemodialysis, pneumonectomy, lung fibrosis and patients under guardianship or deprived of freedom are excluded.

### Heart failure treatment and protocol

Patients are treated for AHF according to usual practices and with the help of a protocol created by the cardiologists of the Grenoble Alpes University Hospital that summarize the guidelines of the European Society of Cardiology (Appendix 1). It has been used for a few months before the start of the study and senior and junior physicians were encouraged to follow it. However, adaptation of diuretic posology were left to their discretion.

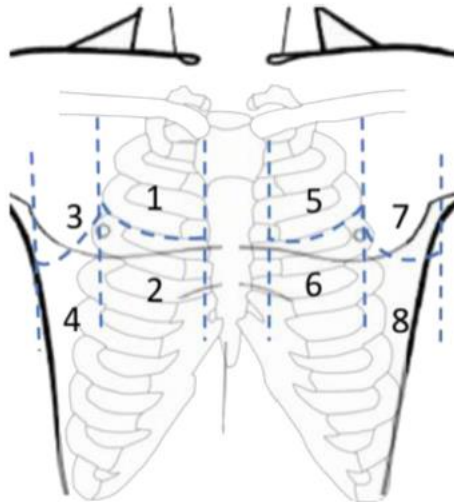
### Lung Ultrasound protocol

For each patient, LUS is performed at admission and daily by a senior physician from the department, who is not in charge of the patient and blinded to clinical and biological findings.

Patients are installed in a 30° supine position with adjustable beds, to perform an eight zone LUS at bedside. They should be in the same position during all of the follow-up ultrasound because the number of B-line may be influenced by patient position (8,14). Inspired by the BLUE-protocol of Lichtenstein (11,15) and according to the expert consensus document (16), four points on each hemithorax are analyzed : two anterior points, between the sternum and the anterior axillary line, one under the clavicle and one under the nipple ; and two lateral points between the anterior and the posterior axillary line, one superior and one inferior.

For each area the number of B-lines are counted and the presence of pleural effusion is registered. Results are reported on a paper form (Appendix 2), completed by the physician for each exam, and kept sealed.

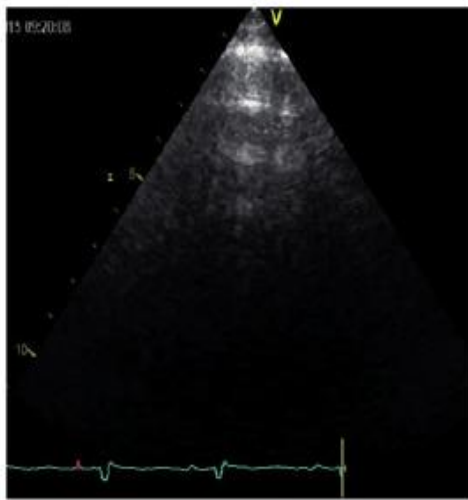
GE Healthcare Vivid T8 echograph device is used, with a 3.5-5 MHz phased array transducer at an imaging depth of four to six centimetres.



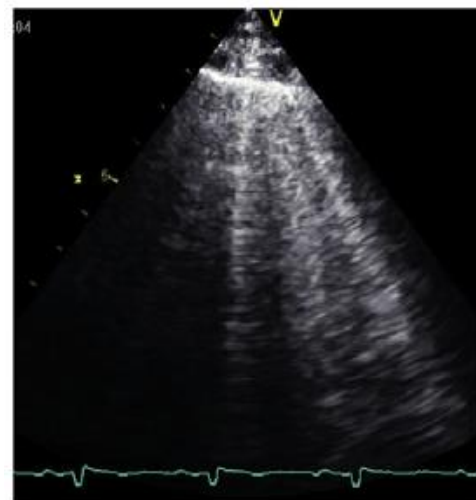
**8-zones LUS (17)**



**Pulmonary effusion in LUS (17)**



**Normal LUS (9)**



**Multiple B-lines in LUS (9)**

Doctors performing LUS received special training before starting inclusion. Most of them already had an echography certification and others followed a theoretical and practical course before starting the inclusions.

AHF therapy is not guided by the results of LUS as the physician implied in the patient's care is blinded from the results.

### Follow-up / Data collection

Medical information about the patient's status is recorded at admission, and every day by physicians in charge of the patient (Appendix 3) in a nominative data collection notebook (Appendix 4).

Demographic variables (age, sex), past medical history and comorbidities (Modified Charlson score), usual treatments, heart failure characteristics (aetiology, left ventricular ejection fraction), physical examination and clinical variables (height, weight, vital signs, heart failure signs and symptoms), electrocardiogram, chest radiography and biological variables (hemogram, electrolyte panel and renal function, martial assessment, liver function test, natriuretic peptides, troponin, and nutritional status) are recorded at admission.

Every day including the discharge day, clinical signs and symptoms of heart failure, vital parameters, diuretic treatment administered and electrolyte panel if available are recorded (Appendix 5).

On the day of discharge, a few items are collected to assess the compliance to the service protocol. Such as the triggering factor of cardiac decompensation, iron supplementation during hospitalization, nutritional and kinetic assessment, and planned follow-up (biological and medical). Patients remaining in hospital after resolution of AFH for a social or secondary medical issue are considered discharged after 48 hours of stable diuretic treatment or return to baseline posology.

Vital status and possible rehospitalization are enquired 30 days after the last LUS by review of hospital medical record and phone call to the patient, his family or his attending physician.

### Study outcomes:

The primary endpoint examined is the number of B-line in LUS measured daily, compared to the evolution of diuretic doses.

Many secondary outcomes are assessed : the evolution of the number of B-lines according to clinical signs of cardiac overload (crackles and lower limbs oedemas) and the agreement between them at the hospital discharge ; the evolution of the number of B-lines according to plasmatic creatinine level and the prognostic value of LUS discharge to predict readmission for AHF and mortality at 30 days.

We also aim to assess the feasibility of daily monitoring during working days of LUS in a medical ward as well as the interobserver reproducibility of results between different physicians by performing LUS on the same group of patients, on the same day, by two doctors.

### Statistical analysis

We calculated that with a sample size of 42, a two-sided 95% confidence interval for a single mean will extend one from the observed mean, assuming that the standard deviation is known to be 3,28 and the confidence interval is based on the large sample z statistic.

To perform the statistical analysis, we divide the duration of follow-up in hospitalization for each patient in four parts in which 100% is the total time of hospitalization.

A descriptive statistical analysis will be done on the baseline characteristics of the population. Continuous variables are reported as median (interquartile range [IQR]) or mean

value (standard deviation [SD]), as appropriate. Categorical variables are expressed as percentage (%).

In the analysis of the primary endpoint, we will first use a Join Point regression model describing B-lines and diuretics posology. Then, a temporal tendency analysis will be performed on the evolution of the number of B-lines and posology of diuretics. A linear regression model will be used to model linear tendency, and to compare tendencies according to time of hospitalization threshold defined as 0% (admission), 25%, 50%, 75% et 100% (discharge). Adjustment on the age of the patients will be done.

The assessment of evolution of the number of B-lines according to clinical signs of cardiac overload, the relationship between B-lines and crackles at discharge and the evolution of B-lines count according to plasmatic creatinine level, will be performed with same analysis as the primary endpoint.

The prognostic value of LUS discharge to predict readmission or all-causes mortality at 30 days will be analyzed by a linear regression model.

For the reproducibility study, we will perform a test to calculate a correlation coefficient : an intraclass correlation coefficient for the quantitative variable (B-lines) and a Kappa coefficient for the qualitative variable (pleural effusion).

Statistical significance was set at  $p < 0.05$ . And all analysis will be performed using the programs RStudio (Version 2022.07.1, PBC, Boston, MA) and Jamovi (Version 2.3.13.0, Sydney, Australia).



### Ethical aspects

The study protocol and its review were approved by an institution review board (Comité de Protection des Personnes Est III), on November the 4<sup>th</sup> of 2020 (Appendix 6) and on March the 3<sup>rd</sup> of 2022 (Appendix 7) respectively. This study was conducted in accordance with the principles of the Declaration of Helsinki.

Patients received oral and written information about the nature of the study, its objectives and expected advantage. Their non-opposition was recorded by one of the senior doctors implied in the research project, according with the French law for observational study with usual care. If patients were unable to decide for themselves, consent from the support person was collected.

## RESULTS

### General characteristics

From May 25<sup>th</sup> 2021 to July 14<sup>th</sup> 2022, 57 patients hospitalized for AHF were screened and 43 were included in the study. Finally, 42 patients analyzed due to the loss of a file (Figure 1). No patients were lost for follow-up.

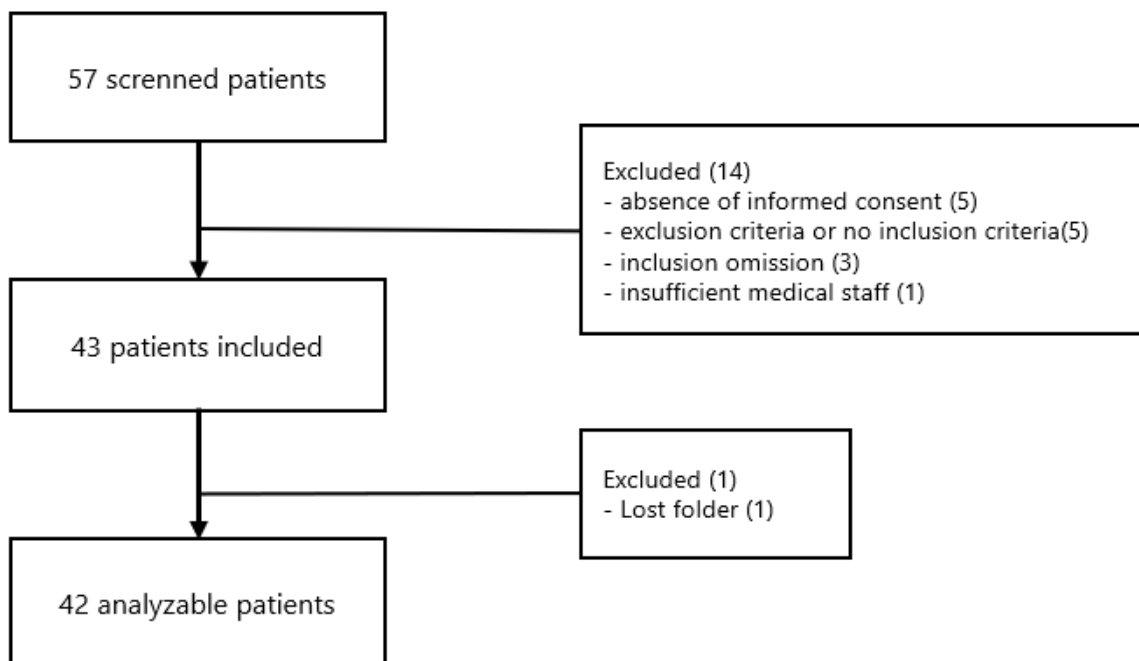


Figure 1. Flow chart

The population was elderly (average age,  $85.8 \pm 8.2$  years), mainly masculine (61.9%) and polypathological (Charlson Comorbidity Index  $8.5 \pm 2.3$ ). 73.8% of patients had a known heart disease and the daily mean *per os* furosemide posology was 132.5 mg before hospitalization (Table 1).

	No	Mean (SD) or No. (%)
<b>Baseline characteristics</b>		
Age, years (extremes)	42	85.8 (SD 8.2) (71.7 - 98.8)
Male gender	42	26 (61.9%)
BMI (kg/m <sup>2</sup> )	39	26.5 (SD 6.2)
Charlson Comorbidity Index	42	8.5 (SD 2.3)
Chronic kidney disease	42	22 (52%)
Hypertension	42	32 (76%)
Diabetes	42	18 (43%)
<b>Previous cardiac history</b>		
Know heart disease	42	31 (73.8%)
LVEF	25	49.8 (SD 13.5)
Daily <i>per os</i> furosemide posology (mg)	42	132.5 (SD 231.5)
<b>Triggering factor of AHF</b>		
Unknown	42	8 (19,0%)
Anaemia	42	9 (21,4%)
Pneumopathy	42	8 (21,4%)
Atrial fibrillation	42	5 (11,9%)
Other bacterial infections (pyelonephritis, erysipelas)	42	4 (9,5%)
Recent decrease in diuretic treatment	42	2 (4,8%)
Non-compliance in healthcare	42	2 (4,8%)
Pulmonary embolism	42	1 (2,4%)
Altitude hypoxemia	42	1 (2,4%)
Covid-19	42	1 (2,4%)
<b>Biological and radiography characteristics at admission</b>		
Haemoglobin (g/dL)	42	11.2 ( SD 1.9)
Ferritin (ng/ml)	30	167.1 (SD 199.2)
Transferrin saturation (%)	30	12.4 (SD 9.1)
Creatinine (µmol/L)	42	134.9 (SD 55.0)
Urea (mmol/L)	42	14.7 (SD 9.5)
Natremia (mmol/L)	42	139.8 (SD 3.8)
Kalemia (mmol/L)	41	4.2 (SD 0.7)
NT-pro BNP (ng/L)	41	9041 (SD 8770.6)
Albumin (g/L)	35	32.1 (SD 5.6)
Signs of pulmonary oedema on chest radiography	36	30 (83.3%)
<b>During hospitalization</b>		
Length of hospitalization (days)	42	8.0 (5.1)
Furosemide posology IV received in ED (mg)	42	122.4 (SD 214.5)
Furosemide posology IV/24h on admission (mg)	42	159.2 (SD 256.5)
Intra-venous iron supplementation	42	24 (57.1%)
Dietetic consultation	42	15 (35,7%)
Physiotherapy	42	25 (59,5%)
Therapeutic patient education	42	5 (11,9%)
<b>At discharge</b>		
NT-pro BNP (ng/ml)	11	3750 (SD 4038.5)
Daily <i>per os</i> furosemide posology (mg)	42	153.9 (SD 268.5)
Biological check-up at 7 days	42	22 (52,4%)
Consultation with attending physician at 7 days	42	18 (42,9%)
Consultation with cardiologist at 20 days	42	20 (47,6%)

BMI = body mass index ; LEVF = left ventricular ejection fraction ; AHF = acute heart failure ; NYHA = New York Heart Association ; BNP = brain natriuretic peptide ; ED = emergency department ; IV = intra venous

**Table 1. Characteristics of the study population**

The Table 2 reports the clinical exam, creatinine level as well as the furosemide posology and the number of B-lines during the different times of hospitalization (admission, 25%, 50% and 75% of hospitalization and discharge).

		Admission (0%)	25%	50%	75%	Discharge (100%)
<b>Weight</b>						
Mean (SD) [count]		73.6 (18.0) [41]	73.4 (16.0) [24]	73.6 (18.8) [22]	71.5 (18.4) [24]	75.6 (20.4) [21]
<b>Dyspnoea</b>						
Mean (SD) [count]		5.1 (3.2) [34]	3.3 (2.9) [25]	2.1 (2.5) [26]	1.8 (2.3) [25]	1.7 (2.2) [28]
<b>NHYA</b>						
Mean (SD) [count]		3.1 (1.0) [35]	2.3 (1.0) [27]	2.2 (1.0) [27]	2.1 (0.9) [23]	2.2 (0.9) [27]
<b>Orthopnoea</b>						
% of total [count]	<b>Yes</b>	36.6% [15]	14.7% [5]	9.7% [3]	7.4% [2]	2.6% [1]
	<b>No</b>	63.4% [26]	85.3% [29]	90.3% [28]	92.6% [25]	97.4% [37]
<b>Asthenia</b>						
Mean (SD) [count]		5.6 (3.0) [31]	4.4 (2.7) [23]	3.9 (3.0) [20]	3.3 (3.2) [18]	3.2 (2.6) [21]
<b>Crackles</b>						
% of total [count]	<b>No</b>	24.4% [10]	32.3% [10]	35.5% [11]	30.0% [9]	44.7% [17]
	<b>Base</b>	26.8% [11]	38.7% [12]	48.4% [15]	53.3% [16]	31.6% [12]
	<b>1/4 lung</b>	12.2% [5]	6.5% [2]	6.5% [2]	10.0% [3]	21.1% [8]
	<b>1/2 lung</b>	29.3% [12]	22.6% [7]	9.7% [3]	6.7% [2]	2.6% [1]
	<b>3/4 lung</b>	7.3% [3]	0.0% [0]	0.0% [0]	0.0% [0]	0.0% [0]
<b>VM abolition</b>						
% of total [count]	<b>No</b>	61.0% [25]	75.0% [24]	63.3% [19]	63.3% [19]	84.2% [32]
	<b>Base</b>	22.0% [9]	9.4% [3]	23.3% [7]	26.7% [8]	10.5% [4]
	<b>1/4 lung</b>	4.9% [2]	6.2% [2]	3.3% [1]	3.3% [1]	5.3% [2]
	<b>1/2 lung</b>	7.3% [3]	6.2% [2]	6.7% [2]	6.7% [2]	0.0% [0]
	<b>3/4 lung</b>	2.4% [1]	3.1% [1]	3.3% [1]	0.0% [0]	0.0% [0]
	<b>All lung</b>	2.4% [1]	0.0% [0]	0.0% [0]	0.0% [0]	0.0% [0]
<b>Lower limbs oedema</b>						
% of total [count]	<b>No</b>	33.3% [14]	50.0% [16]	14 (46.7%)	48.3% [14]	56.8% [21]
	<b>Ankle</b>	28.6% [12]	21.9% [7]	12 (40.0%)	44.8% [13]	45.1% [13]
	<b>Knee</b>	28.6% [12]	25.0% [8]	3 (10.0%)	6.9% [2]	5.4% [2]
	<b>Thigh</b>	9.5% [4]	3.1% [1]	1 (3.3%)	0.0% [0]	2.7% [1]
<b>Creatinine (<math>\mu\text{mol/l}</math>)</b>						
Mean (SD) [count]		133.7 (55.1) [42]	139.1 (51.8) [30]	139.8 (54.0) [26]	144.1 (58.5) [21]	142.8 (57.5) [30]
<b>Furosemide posology (IV, mg)</b>						
Mean (SD) [count]		162.1 (SD 259.0) [42]	147.4 (224.7) [35]	124.0 (220.2) [36]	80.5 (137.4) [34]	77.0 (134.2) [42]
<b>Pleural effusion</b>						
% of total [count]	<b>Yes</b>	61.5% [8]	60.0% [15]	48.0% [12]	46.2% [12]	35.7% [10]
	<b>No</b>	38.5% [5]	40.0% [10]	52.0% [13]	53.8% [14]	64.3% [18]
<b>B-lines left</b>						
Mean (SD) [count]		9.1 (7.5) [13]	6.8 (5.1) [25]	5.6 (5.1) [25]	4.8 (4.1) [26]	4.5 (3.5) [28]
<b>B-lines right</b>						
Mean (SD) [count]		9.8 (7.1) [13]	8.2 (6.9) [25]	6.3 (4.8) [25]	6.0 (4.2) [26]	3.9 (4.0) [28]
<b>Total B-lines</b>						
Mean (SD) [count]		18.1 (13.0) [13]	13.4 (10.2) [25]	11.9 (9.4) [25]	10.8 (7.8) [26]	8.4 (6.8) [28]

NYHA = New York Heart Association ; VM = vesicular murmur ; IV = intra venous

**Table 2. Characteristics evolution of the patients during hospitalization**

### Reproducibility and feasibility

After the comparison of 17 exams done by two different physicians, the intraclass correlation coefficient obtained for the total number of B lines is 0,809 (IC 95% : 0.556 – 0.955) and the Cohen's kappa coefficient for the presence of pleural effusion is 0,764 (IC 95% : 0.403 – 1.00).

The 42 patients analyzed add up 237 working days (Monday to Friday) of hospitalization. 188 LUS were performed, that is 79,3% of the working days. In other words, we have 20,7% of LUS missing during patients follow-up.

### LUS B-lines, posology diuretics, clinical and biological parameters

The number of B-lines decrease promptly and importantly at the beginning of hospitalization with the diuretic therapy, then stagnates at the middle of hospitalization until the discharge. Diuretic posology remains stable at beginning of hospitalization. Then, it decreases towards the middle of hospitalization until discharge (Figure 2, Table 3). The number of B lines and the diuretic posology progresses without relationship between them.

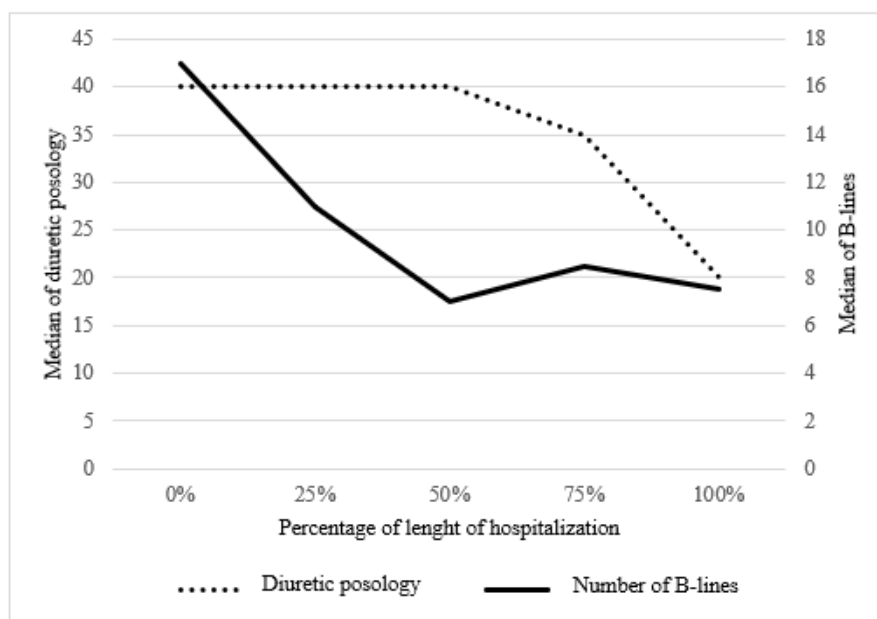


Figure 2. Evolution of median of B-lines and median diuretic posology according to time

Hospitalization time	Median of B-lines	Median of diuretic posology	B-lines tendency	Diuretic posology tendency	Correlation coefficient r	p-value univariate	p-value multivariate <sup>1</sup>
0%	17	40			0.023	0.941	0.869
25%	11	40	0.65	1	- 0.098	0,901	0.960
50%	7	40	0.64	1	- 0.256	0,216	0.396
75%	8,5	35	1.21	0.88	- 0.213	0,306	0.591
100%	7,5	20	0.88	0.57	- 0.182	0,305	0.737

<sup>1</sup> Adjustment on the age

**Table 3. Evolution of number of B-lines and diuretic posology according to time**

Cardiac overload signs (crackles, lower limb oedema) decreases also during the hospitalization, without any significant relationship to the number of B-lines (Table 4).

Hospitalization time	Clinic sign	Intensity of clinic sign	No.	B-lines mean (SD) [count]	p value <sup>1</sup>
Admission (0%)	Crackles	Absent	13	14.7 (9.1) [3]	0.295
		Moderate <sup>a</sup>		12.8 (14.4) [5]	
		Severe <sup>b</sup>		25.4 (12.3) [5]	
	Lower limb oedema	Absent	13	23.2 (15.5) [5]	0.281
		Moderate <sup>c</sup>		14.9 (11.1) [5]	
		Severe <sup>d</sup>		0.0 (NA) [0]	
25%	Crackles	Absent	31	10.3 (11.4) [10]	0.824
		Moderate <sup>a</sup>		8.7 (9.9) [14]	
		Severe <sup>b</sup>		11.7 (11.1) [7]	
	Lower limb oedema	Absent	32	12.5 (11.1) [16]	0.233
		Moderate <sup>c</sup>		7.1 (9.1) [15]	
		Severe <sup>d</sup>		0.0 (NA) [0]	
50%	Crackles	Absent	31	9.8 (11.8) [11]	0.539
		Moderate <sup>a</sup>		9.1 (8.3) [17]	
		Severe <sup>b</sup>		3.0 (3.6) [3]	
	Lower limb oedema	Absent	30	10.9 (10.5) [14]	0.451
		Moderate <sup>c</sup>		7.9 (8.5) [15]	
		Severe <sup>d</sup>		0.0 (NA) [1]	
75%	Crackles	Absent	30	12.8 (9.4) [9]	0.223
		Moderate <sup>a</sup>		7.4 (7.9) [19]	
		Severe <sup>b</sup>		4.5 (6.4) [2]	
	Lower limb oedema	Absent	30	10.0 (9.6) [14]	0.563
		Moderate <sup>c</sup>		8.2 (7.5) [15]	
		Severe <sup>d</sup>		1.0 (NA) [1]	
At discharge (100%)	Crackles	Absent	39	6.6 (7.1) [17]	0.055
		Moderate <sup>a</sup>		3.9 (6.4) [21]	
		Severe <sup>b</sup>		20.0 (NA) [1]	
	Lower limb oedema	Absent	35	5.4 (6.9) [18]	0.697
		Moderate <sup>c</sup>		6.2 (7;7) [16]	
		Severe <sup>d</sup>		0.0 (NA) [1]	

<sup>a</sup> Crackles in bases and 1/4 lung <sup>b</sup> Crackles in 1/2 , 3/4 and all lung <sup>c</sup> Lower limb oedema in ankle or knee <sup>d</sup> Lower limb oedema in thigh or all limb

<sup>1</sup> Linear Model ANOVA

**Table 4. Evolution of number of B-lines according to clinical signs of congestion**

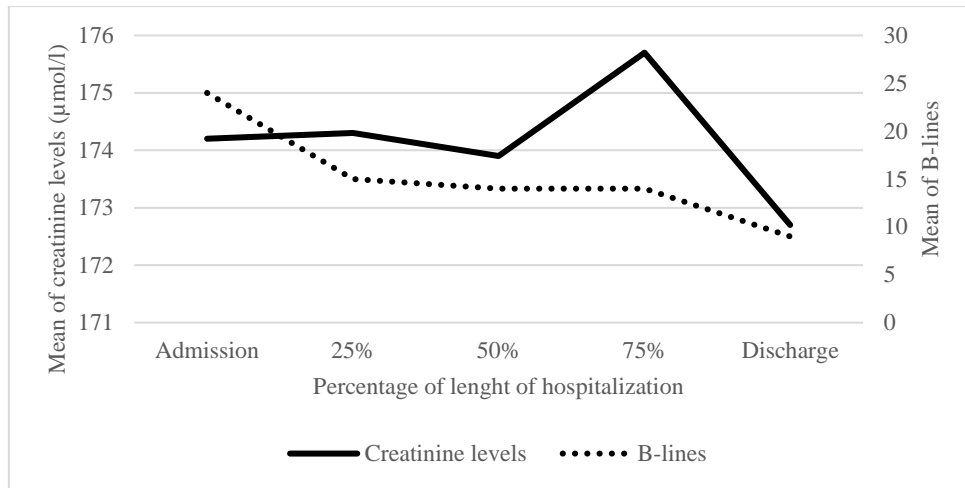
At discharge, there is no significant connection between the number of B-lines and the presence and the intensity of crackles. 22 of patients had crackles with an average number of B-lines of 7.6 (Table 5)

At discharge	No crackles	Bases crackles	¼ lung crackles	½ lung crackles	¾ lung crackles	All lung crackles	p-value <sup>1</sup>
<b>B lines</b> Mean (SD) [count]	6.6 (7.1) [17]	4.1 (7.2) [13]	3.6 (5.4) [8]	20.0 (NA) [1]	0.0 (NA) [0]	0.0 (NA) [0]	0.124

<sup>1</sup>Linear Model ANOVA

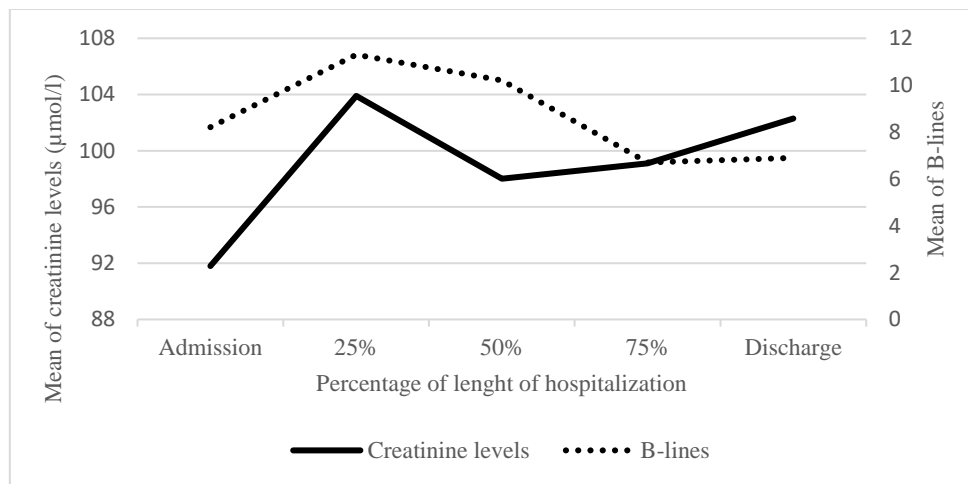
**Table 5. Number of B-lines according to crackles at discharge**

22 (52%) of our patients had a known history of chronic kidney disease (CKD) with a baseline average creatinine value of 155.7 µmol/l. Their average value was 174.2 µmol/l at hospital admission and tends to remain stable throughout the hospital stay (Figures 3). Among them, 15 (68%) suffered acute kidney injury (AKI). It was present for 11 (50%) of them at admission and 4 (18%) developed it during hospitalization. The other 20 (48%) patients of our study had not a CKD history, their average creatinine level at admission was 91.8 µmol/l and tended to remain stable too (Figures 4). Among them, seven (35%) suffered AKI. It was present for two (10%) of them at admission and the other five (25%) developed it during hospitalization. Table 6 shows that, at admission, patients with CKD history had a significantly higher number of B-lines than those without CKD history. There are no relationship between the evolution of the number of B-lines and that of creatinine levels.



CKD = Chronic kidney disease

**Figure 3. Evolution of number of B-lines and creatinine levels according to time in patients with CKD history**



CKD = Chronic kidney disease

**Figure 4. Evolution of number of B-lines and creatinine levels according to time in patients without CKD history**

Hospitalization time	CDK history	No.	Creatinine in µmol/l mean (SD) [count]	B-lines mean (SD) [count]	p value <sup>1</sup>
Admission (0%)	Yes	22	174.2 (43.1) [22]	24.2 (11.6) [8]	0.023
	No	20	91.8 (26.9) [20]	8.2 (8.8) [5]	
25%	Yes	22	174.3 (43.8) [15]	14.6 (9.4) [16]	0.450
	No	20	103.9 (31.4) [15]	11.3 (11.7) [9]	
50%	Yes	22	170.5 (49.8) [15]	13.7 (10.2) [12]	0.373
	No	20	98.0 (21.7) [11]	10.2 (8.7) [13]	
75%	Yes	22	171.8 (56.6) [13]	14.4 (8.5) [14]	0.008
	No	20	99.1 (23.3) [8]	6.7 (3.9) [12]	
At discharge (100%)	Yes	22	169.8 (57) [18]	9.2 (7.5) [18]	0.406
	No	20	102.3 (26.9) [12]	6.9 (5.2) [10]	

<sup>1</sup> Linear Model ANOVA

**Table 6. B-lines evolution according to creatinine measurement**



Within a month of hospitalization, 5 (12%) patients died for any causes and 7 (20%) were re-hospitalized for AHF. Only one of the deceased patients died after readmission for AHF. The relationship between the number of B-lines and the vital status at 30-days discharge is statistically significant with a *p-value* < 0.001 (Table 7)

At 30-days after discharge		B-lines mean (SD) [count] at discharge	p-value <sup>1</sup>
Vital status	Death	15.2 (10.1) [5]	<0.001
	Alive	3.9 (5.4) [37]	
Hospital status for AHF	Readmission	4.9 (5.4) [8]	0.469
	No readmission	4.1 (5.7) [30]	

<sup>1</sup> Linear Model ANOVA

**Table 7. B-lines at discharge according to vital and hospital status 30-days after discharge**

## DISCUSSION

In this prospective monocentric study conducted in a post-urgency ward, we are interested in the relationship between the number of B-lines at LUS, the diuretic posology administrated and the clinical and biological signs of cardiac overload in in-patients with AHF. We observed an important decrease in the number of B-lines in the first half of hospitalization and a stabilization trend then, whereas diuretics posology decreases during whole hospitalization. At the discharge some of our patients presented a sub-clinical pulmonary overload without crackles but with the persistence of B-lines. The presence of B-lines at discharge is significantly associated with mortality risk at 30-days.

Few previous studies have looked at the relation between LUS B-lines and the diuretic posology in in-patients outside cardiology and intensive care unit. Pang et al (18) lead to a pilot trial in which patients consulting for AHF and pulmonary overload in the ED are divided in two groups in order to compare usual standard care and LUS-guided strategy-of-care in the first six hours. Patients in the intervention group showed a consistent numeric reduction of B-lines during hospitalization suggesting that protocol driven therapy might facilitate a faster congestion reduction. For Panuccio et al (19) who conducted a pilot study in in-patients for AKI in nephrology ward, showed that LUS clearly document the superiority over clinical examination for detection of interstitial lung oedema, notably when congestion is in a pre-clinical stage. Cardiologic groups have assessed the value of LUS in the adaptation of diuretic treatment in out-patients. For example, Rivas-Lasarte et al. (20) used LUS for monitoring patients after a hospitalization for AHF and showed that patients with LUS-guided adaption of diuretic treatment had less urgent visit, hospitalization for AHF and death from any causes (HR = 0.518 [CI 95% : 0.268-0.998 ; p = 0.049]) than those with a standard follow-up. In the same way, in the Chinese randomized IMP-OUTCOME trial, Yunlong et al (21) are currently studying the

adaptation of diuretic treatment according to LUS (test group) in patients discharged after an AHF episode.

Our results agree with those previous studies that report a discrepancy between clinical pulmonary congestion, notably crackles, and LUS B-lines. This can be related to whether on admission or on discharge, some patients have no detectable clinical congestion but have subclinical pulmonary fluid overload. LUS can identify this clinically silent pulmonary oedema. At admission, in the study by Platz and al. (8), 35% of patients with acute heart failure didn't have crackles whereas only 6% didn't have B-lines. At discharge, Rivas-Lasarte and al. (22) have found that 40% of their patients "lung dry" to auscultation presented subclinical fluid overload detected by LUS and this seems to have similar poor prognosis to over clinical pulmonary congestion. The poor interobserver agreement of crackles at lung auscultation can also participate at its discrepancy. For Panuccio et al (19), in patients with AKI, the agreement between lung crackles and US-B lines was poor ( $\kappa = 0.02$ ,  $p = 0.63$ ). Ramos-Hernandez et al (23) compared the interobserver agreement of LUS and lung auscultation and found that the reproducibility is good for B-lines ( $\kappa = 0.81$  [CI 95% : 0.81 – 0.83]) but poor for crackles ( $\kappa = 0.18$  [CI 95% : 0.16 – 0.20]). Those findings suggest that LUS can be a complement to the other techniques (physical examination, chest-X-ray, natriuretic peptides) for the evaluation of pulmonary congestion.

During a AHF episode, AKI can arise in different cases (6,9,12). It can be a congestion marker due to hypervolemia and to an increment of central venous pressure secondary to a cardiac output reduction, it is the cardiorenal syndrome type 1. In this case fluid removal with diuretics might improve kidney function. AKI can also be the result of hypovolemia caused by diuretic therapy and be the result of a transient decrease in glomerular filtration rate (GFR) without

reflecting excessive depletion. Other factors as dehydration, nephroangiosclerosis and drug adverse effects can contribute to AKI. Therefore, it is difficult to attribute AKI to a single cause as well as to determine the exact mechanism. The evaluation of lung congestion in patients AKI is not an easy task. Panuccio et al (19) showed that patients with AKI hospitalized in nephrology ward, hidden lung congestion is much frequent in apparently euvolemic and hypovolemic patients, approximately 30% of their euvolemic and hypovolemic patients presented clear-cut cardiovascular congestion. For Ruggenti et al (24), patients with AHF and concomitant AKI are those that require an urgent and efficient therapy because the combination of both is associated with poor outcomes and appears to be a stronger predictor of mortality. For them, the priority must be the pulmonary decongestion without being limited by the alteration of renal markers. For Blair et al (25) AKI appearing in-hospital may reflect an aggressive decongestion rather than kidney injury because it is often associated with reduction in natriuretic peptides, blood pressure and body weight which are good prognostic factors. We did not find studies focused on LUS and clinical or biological signs of AKI during AHF episode. In our study, patients with a CKD history had more B-lines at admission than those with a healthy kidney. This relationship was only significant at admission, because during hospitalization creatinine levels remain steady in the two groups while the number of B-lines decrease.

Our results of 30-day all-cause mortality and readmission for AFH are consistent with the data of the American Heart Association who reports that in patients older than 65 years after an episode of AFH rates of 10% and 20-25% respectively (26). In the population of Freund et al (27), the average age was 87 years and 30-days mortality and readmission was similar to those mentioned earlier. The number of B-lines decreased from hospital admission to discharge, but a part of patients still have B-lines at discharge and the lack of clearance of it at this time is associated with a high risk of adverse events like ED consultation, AHF readmission and all-

causes death. Cogliati et al (3) who were interested in in-patients in internal medicine department found that the sonographic score was significantly associated with adverse events at 100-days with an hazard ratio (HR) equal to 1.19 (IC 95% : 1.05-1.14 ; p = 0.005). In same order, E. Platz et al (28) found that the relation between the risk of adverse event and an high number of B-lines at discharge is strong : unadjusted HR at 60 days was 3.30 (IC 95 % CI : 1.52-7.17 ; p = 0.002). In our study we found a significant relationship between the number of B-lines and 30-days mortality. However we did not find a connection of B-lines and re-hospitalization risk, which is discordant with literature. Gargani et al (4) and Miglioranza et al (29) conclude that absence or a mild degree of B-lines ( $\leq 15$ ) identify a subgroup at extremely low risk to be readmitted for AFH, perhaps our patients belong to this group. If we utilize this cut off of  $\geq 15$  B-lines at discharge to determine prognosis, we note that among our death patients four of five had  $\geq 15$  B-lines, but among the patients alive only two of 37 had  $\geq 15$  B-lines. Only Cogliati et al where interested in elderly patients (mean age 80 years). The average age in this studies mentioned above was 70, 53 and 71 years respectively. Other factors may contribute to explain this high mortality. The mean age of our patient was 85.6 years (rank : 71.7 – 98.8) and we know that age is a strong and independent predictor factor of mortality in patients with HF (30). Furthermore the past medical history should also be taken into account, the average Charlson Comorbidity Index (CCI) was 8.5 (rank : 5 – 17). CCI is an independent predictor of mid-term post-discharge mortality among elderly HF patients (31). A CCI  $\geq 5$  fits a HR age-adjusted of 77.3 (IC 95% 74.7-79.8) at 1-year mortality (32). The prognosis seems not driven only by pulmonary oedema but rather by triggering factor and underlying conditions (27,33)

### *Strengths*

Our study has some strengths. First, we performed 8-zones LUS according to recommendations by international consensus statement (16) and interpreted in real time. LUS were performed with patients in the same position, with the same ultrasound device and configuration. Other studies use a clip recording for the evaluation of B-lines but Platz et al (34) found that the length of the clip can have an impact in the count of B-lines.

Second, even if the extrapolation of our results can be limited by the small sample size and the single centre approach, we performed a large number (188) of LUS during the study. Moreover, our patients are mainly elder polypathological who can reflect a good part of the population in medical ward. The fragility of our population means that some patients had a longer than expected length of hospitalization because of social or secondary medical issues. To overcome this, we considered patients as discharged after 48 hours of stable diuretic treatment or return to baseline posology.

Third, most of the studies evaluating LUS are conducted in a cardiology or an intensive care unit and few are conducted in medicine ward. Our population is elderly and polypathological what brings it closer to geriatric population than to cardiology patients who are younger.

Fourth, practitioners who make LUS were blinded of clinical examination, and even if LUS is not performed every day by the same physician, the intra class coefficient correlation for the number of B-lines is excellent (0,809 [IC 95%: 0.556 – 0.955]) and close to that found in other studies. For instance, for Russel et al (35) this coefficient was 0,74 after a trial of approximately 11 exams and two hours of theoretical class. It was 0.98 (95% IC : 0.98-0.99 ;  $p < 0.0001$ ) for

Miglioranza et al (29) assessed in a set of 20 consecutive patients and 0.98 ( $p < 0.0001$ ) for M. Mazzola et al (36) in 50 LUS videos evaluated by an expert reader.

Fifth, even if doctors are free to prescribe treatment doses according to their assessment, our study had a positive impact in the implementation of protocolize care for AHF treatment in the department.

### *Limitations*

To our knowledge, our study has some limitations. First, the absence of a consensus definition of cardiac congestion and the lack of a gold standard to verify its presence are an important limitation for the diagnosis of AFH. Therefore, physicians based on a body of evidence to establish the diagnosis before including a patient, as recommend by the ESC (13). There is also no consensus on the definition of ultrasound pulmonary congestion, which is why we decided to count the number of B-lines and not to set a cut-off.

Second, a daily LUS on working days for the duration of the hospitalization was not possible in our study, 79,3% of the LUS are performed. Some reasons can explain this, as the high workload or the need for sufficient senior doctors to hold the blind. A solution can be to train the residents for LUS. We don't found other studies that performed daily LUS, it is a originality of our study. Gargani et al (4) performed LUS at admission and at discharge hospitalization with a feasibility of 100%, Mazzola et al (36) realized LUS at admission, 24 hours and 48 hours after in-hospital and before the discharge.

Third, during their stay in the ED, our patient received furosemide (average of 122 mg IV), some of them stayed several hours and we don't know if they had a LUS. B-lines are dynamic

and are quickly impacted by diuretic treatment (8), it is possible consequently that our first clinical and ultrasound examination showed fewer signs of congestion than when patients arrived at ED. We were interested in post-emergency care but the data at initial intake could have an impact. Pang et al (18) shows that patients with a LUS-guided therapy at ED have a significantly greater B-lines reduction in the first 48 hours compared with those receiving usual care ( $p = 0.04$ ).

Fourth, the detection of B-lines doesn't necessarily imply the cardiogenic origin. Other pathologies can be a source of B-lines like pulmonary fibrosis, acute respiratory distress syndrome (ARDS), pneumopathies bacterial and viral. None of our patients presented an ARDS and we excluded patients with fibrosis and pneumonectomy. Nevertheless we included patients with an infectious pneumonia which is a frequent triggering factor of AHF and we used the total number of B-lines for our analysis that can be increased in this cases and constitute a confounding factor. Even if pneumonia represent a confounding factor for the evaluation of B-lines in AHF, as shown by Mazzola et al (36) LUS is able to monitor pulmonary decongestion in patients with AHF whether they have associated pneumonia or not.

Fifth, we conducted the study regardless of if ejection fraction (EF) is reduced (HFrEF) or preserved (HFpEF). This information is not always easy to obtain and few of our patient had an echocardiography during their hospitalization. Yang et al (37) did not find significant differences in the B-lines between HFpEF and HFrEF groups. Unlike, Palazzuoli (38) et al found that patients with HErEF had more B-lines compare to them with HEpEF at admission ( $p$ -value = 0.04) and discharge ( $p$ -value = 0.009). Studies are therefore discordant, further need to be carried out.



## CONCLUSION

Acute heart failure is a major public health problem and pulmonary congestion is the main therapeutic target. Pleuropulmonary ultrasound has emerged in the last decade as an additional tool in the diagnostic aid and follow-up of these patients.

In this prospective observational study conducted in a multidisciplinary medical ward, we observed a trend towards stagnation in the number of B-lines at the end of hospitalization while the given diuretics posology administered continues decreasing. At the time of discharge, a number of our patients presented a clinical examination without signs of overload despite the presence of B lines. And at the same time, we found a significant relationship between the number of B-lines at discharge and the risk of death at 30-days.

In the absence of a gold standard in the quantitative assessment of cardiac overload, LUS is an interesting tool that could help optimizing the management of congestion, particularly when it becomes subclinical, and thus improve the prognosis at 30 days. A randomized interventional study is needed to assess the value of echo-guided therapeutic management in conventional non-cardiology hospitalization.

THÈSE SOUTENUE PAR : Elena BEL ALONSO

TITRE :

Intérêt de l'échographie pleuro pulmonaire dans la prise en charge de la décompensation cardiaque en post-urgence

CONCLUSION :

L'insuffisance cardiaque aiguë est un problème de santé publique majeur et la congestion pulmonaire est sa cible thérapeutique principale. L'échographie pleuropulmonaire a émergé dans la dernière décennie comme un outil supplémentaire dans l'aide diagnostique et le suivi de ces patients.

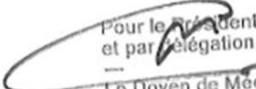
Nous avons observé dans cette étude observationnelle prospective conduite en service de médecine polyvalente, une tendance à la stagnation du nombre de lignes B en fin d'hospitalisation alors que la posologie de diurétiques administrés continu de diminuer. Au moment de la sortie, un certain nombre de nos patients présentait un examen clinique sans signes de surcharge malgré des lignes B encore présentes. Parallèlement, nous avons mis en évidence une relation significative entre le nombre de lignes B à la sortie d'hospitalisation et le risque de décès à un mois.

En absence de gold standard dans l'évaluation quantitative de la surcharge cardiaque, l'échographie pleuropulmonaire est un outil intéressant qui pourrait aider à optimiser la prise en charge de la congestion notamment quand celle-ci devient subclinique, et ainsi améliorer le pronostic à 30 jours. Une étude interventionnelle randomisée est nécessaire afin d'évaluer l'intérêt d'une prise en charge thérapeutique échoguidée en hospitalisation conventionnelle non-cardiologique.

VU ET PERMIS D'IMPRIMER

Grenoble, le :

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et par délégation  
Le Doyen de Médecine  
Pr. Patrice MORAND

Pr. Patrice MORAND

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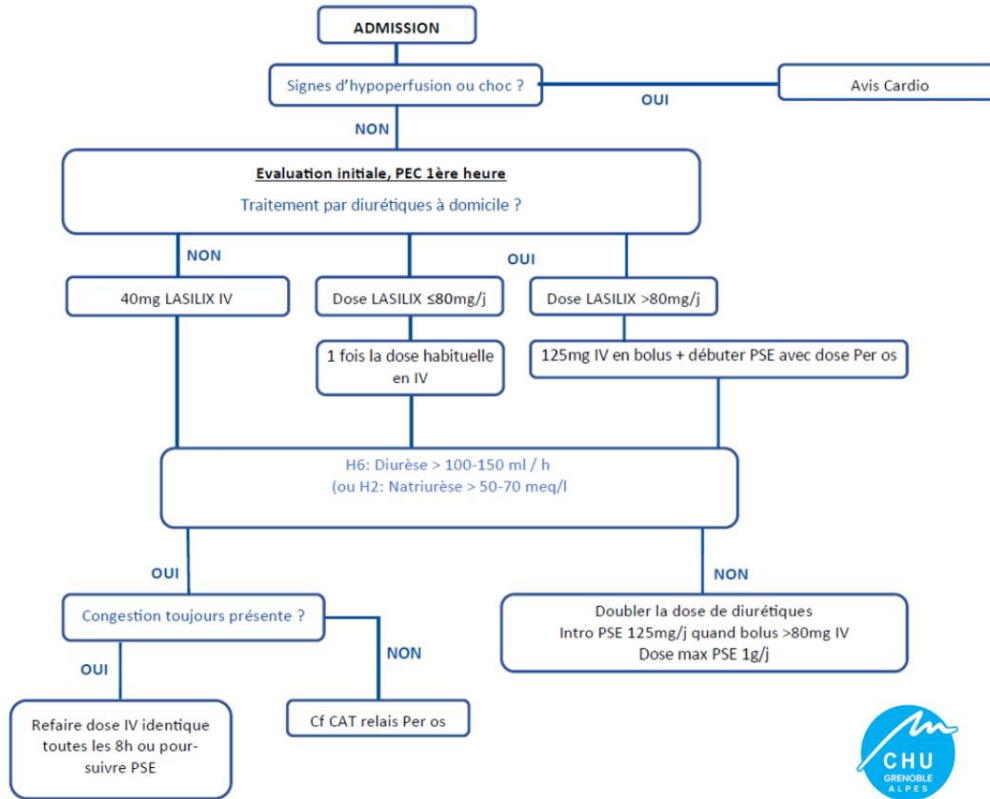
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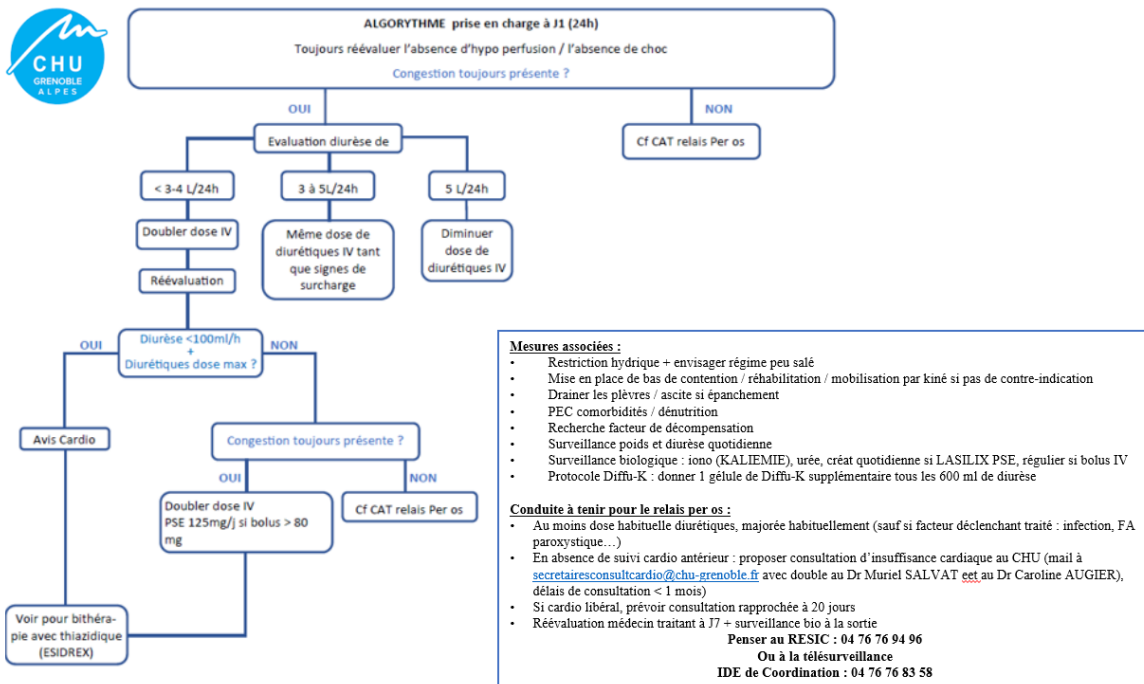
# APPENDICES

## Appendix 1. Diuretic adaptation protocol

- At admission :



- 24h after admission :



**Appendix 2. Paper form to report LUS results**

EPPICA

Version 2 du 14/12/2021

**Feuille de recueil de données**

**Thèse Elena**

Numéro patient EPPICA :

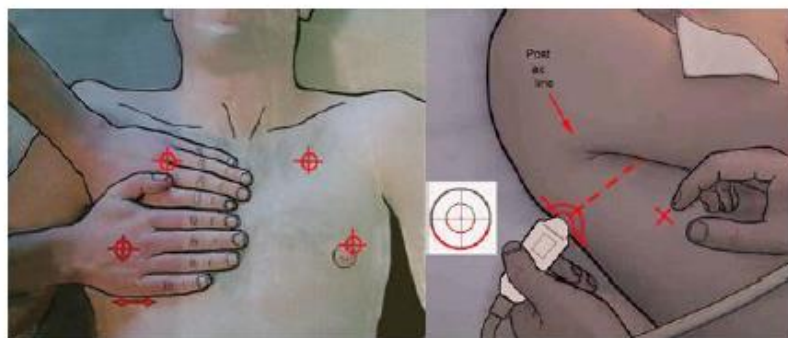
Nom du réalisateur :

**A COMPLETER TOUS LES JOURS**

DATE : .../.../...

HEURE DE REALISATION : ...

<b>Upper Blue Point Gauche</b>	<b>Upper Blue Point Droit</b>
Nombres de lignes B : ...	Nombres de lignes B : ...
<b>Lower Blue Point Gauche</b>	<b>Lower Blue Point Droit</b>
Nombres de lignes B : ...	Nombres de lignes B : ...
<b>Axillaire Gauche</b>	<b>Axillaire Droit</b>
Nombres de lignes B : ...	Nombres de lignes B : ...
<b>Base postérieure Gauche</b>	<b>Base postérieure Droite</b>
Nombres de lignes B : ...	Nombres de lignes B : ...
Epanchement pleural : <input type="checkbox"/> Oui <input type="checkbox"/> Non	Epanchement pleural : <input type="checkbox"/> Oui <input type="checkbox"/> Non



Deux mains (de la taille de celle du patient) se posent ainsi, à partir de la clavicule. Le milieu de la main supérieure détermine l'upper BLUE-point. Le milieu de la paume inférieure, le lower BLUE-point. Partant transversalement du lower BLUE-point, une ligne croise la ligne axillaire postérieure. Ce croisement, ou le plus postérieurement possible, définit le PLAPS-point (base postérieure).

### Appendix 3. Follow-up table

Descriptif des données collectées	Pré inclusion J0 (entrée dans le service)	Inclusion T0	Quotidiennement durant l'hospitalisation	Visite de suivi 30 jours (+/- 3 jours) après la dernière échographie
Information des sujets		✓		
Vérification des critères d'éligibilité	✓	✓		
Recueil des données cliniques		✓	✓	
Recueil des données biologiques		✓	✓*	
Echographie		✓	✓	
Recueil des réhospitalisations ou statut vital				✓
Revue du dossier médical				✓

\* récolte des données toutes les semaines et à la sortie



## Appendix 4. Data collection notebook

EPPICA

N° Patient :  
Version 3.0 du 14/12/2021

# CAHIER de recueil de données

## Étude EPPICA

### Thèse Elena

0645068249 - ebelalonso@chu-grenoble.fr

Numéro patient EPPICA :

Date, heure et service d'ENTREE :

.../.../... ..:.. ..

Date et heure SORTIE :

.../.../... ..:.. ..

Lieu SORTIE :

**A COMPLETER A L'ADMISSION**

DATE : .../.../...

**Antécédents (médicaux et chirurgicaux) :**

<input type="checkbox"/> Diabète <input type="checkbox"/> Complicé <input type="checkbox"/> HTA <input type="checkbox"/> BPCO <input type="checkbox"/> Insuffisance rénale chronique <input type="checkbox"/> Insuffisance hépato cellulaire <input type="checkbox"/> SAOS <input type="checkbox"/> AOMI, pathologie vasculaire périphérique <input type="checkbox"/> Autres :	Antécédents permettant de calculer l'index de comorbidité de Charlson : <input type="checkbox"/> Démence <input type="checkbox"/> Infarctus du myocarde <input type="checkbox"/> Pathologie pulmonaire chronique <input type="checkbox"/> AVC / AIT <input type="checkbox"/> Hémiplegie séquellaire <input type="checkbox"/> Tumeur solide <input type="checkbox"/> Avec métastases <input type="checkbox"/> Leucémie <input type="checkbox"/> Lymphome <input type="checkbox"/> SIDA
---	--

Cardiopathie :     Non     Oui : (préciser)

Dernière FEVG (%) : ...

**Traitements habituels :**

- Furosémide ☐ Posologie : ...
- Autres diurétiques : ...
- Béta bloquant
- IEC / ARA 2
- Inhibiteur calcique
- Entresto (sacubitril + valsartan)
- Dapaglifozide
- Autres traitements : .....

Constantes				
Taille	Poids (après pesée)	Fréquence cardiaque	Fréquence respiratoire	Pression artérielle
_____ cm	_____ kg	_____ bpm	_____ /min	___ / ___ mmHg

Examen clinique	
Dyspnée (évaluée par patient)	0 – 1 – 2 – 3 – 4 – 5 – 6 – 7 – 8 – 9 – 10
Stade NYHA	1 – 2 – 3 – 4
Orthopnée	Oui / Non
Asthénie (évaluée par patient)	0 – 1 – 2 – 3 – 4 – 5 – 6 – 7 – 8 – 9 – 10
Balancement thoraco-abdominal	Oui / Non
Tirage	Oui / Non
Crépitants	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total
Abolition murmure vésiculaire	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total
Œdèmes des membres inférieurs	Non – Chevilles – Genou – Racine de cuisse
Turgescence jugulaire	Oui / Non
Reflux hépato-jugulaire	Oui / Non
Hépatomégalie	Oui / Non

ECG	<input type="checkbox"/> Normal <input type="checkbox"/> ACFA <input type="checkbox"/> Bloc de branche G <input type="checkbox"/> Bloc de branche D <input type="checkbox"/> Signes d'hypertrophie ventriculaire
Radio pulmonaire	<input type="checkbox"/> Lignes B Kerley <input type="checkbox"/> Opacités périhiliaires floconneuses bilatérales symétriques (en ailes de papillon) <input type="checkbox"/> Epanchements pleuraux <input type="checkbox"/> Redistribution vasculaire apex <input type="checkbox"/> Cardiomégalie

<b>ETT à l'admission</b>	<input type="checkbox"/> Non faite <input type="checkbox"/> Oui : FEVG = ... %
--------------------------	--

Biologie			
Na (mmol/L)		TP (%)	
K (mmol/L)		TSH (mUI/L)	
Bicarbonates (mmol/L)		Troponines (ng/L)	
Créatinine (µmol/L)		NT pro BNP (ng/L)	
Urée (mmol/L)		Albumine (g/L)	
Hémoglobine (g/L)		Préalbumine (g/L)	
Hématocrite (%)		HbA1c (%)	
Ferritine (µg/L)		Bilirubine (mmol/L)	
Coefficient de saturation			

**FUROSEMIDE reçu aux urgences :**

Posologie :                      mg

Voie administration :

**Autres traitements notables reçus aux urgences :****FUROSEMIDE prescrit à l'entrée dans le service :**

Posologie prescrite :                      mg

Voie administration :

**Autres traitements notables prescrits à l'entrée dans le service :**

**A COMPLETER TOUS LES JOURS**

<b>DATE</b>	.../.../...	.../.../...	.../.../...
<b>TA (mmHg)</b>	.../...	.../...	.../...
<b>FC (bpm)</b>			
<b>FR (/min)</b>			
<b>Poids (kg) après pesée</b>			
<b>Dyspnée 0 à 10</b>			
<b>NYHA 1 à 4</b>			
<b>Orthopnée</b>	Oui / Non	Oui / Non	Oui / Non
<b>Asthénie 0 à 10</b>			
<b>Crépitants</b>	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total
<b>Abolition MV</b>	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total	Non – Bases – ¼ champs – ½ champs – ¾ champs – Total
<b>OMI</b>	Non – Chevilles – Genou – Racine de cuisse	Non – Chevilles – Genou – Racine de cuisse	Non – Chevilles – Genou – Racine de cuisse
<b>Diurèse / 24h</b>			
<b>Biologie</b>	Na : K : Bicarbonates : Créatinine :	Na : K : Bicarbonates : Créatinine :	Na : K : Bicarbonates : Créatinine :
<b>Furosémide (préciser voie d'administration)</b>	Posologie prescrite (/24h) :	Posologie prescrite (/24h) :	Posologie prescrite (/24h) :
	Modif : □□□	Modif : □□□	Modif : □□□
<b>Restriction hydrique (préciser)</b>	Oui / Non ...	Oui / Non ...	Oui / Non ...

**A COMPLÉTER A LA SORTIE**

Facteur déclenchant d'insuffisance cardiaque aiguë (si identifié) : ....

NT-proBNP de sortie : ....

Posologie Furosémide prescrite : ....

Bas de contention  Oui  NonCarence martiale mise en évidence au cours du séjour :  Oui  NonSi oui : supplémentation en fer :  Oui IV ou PO  NonConsultation diététicienne :  Oui  NonKinésithérapie :  Oui  NonÉducation thérapeutique :  Oui  NonSurveillance bio à J7 prescrite  Oui  NonConsultation MT J7  Oui  NonConsultation cardio J20  Oui  Non

EPPICA

N° Patient :  
Version 3.0 du 14/12/2021

***A COMPLETER à J30 de la sortie d'hospitalisation***

Date de l'appel : .../.../...

Statut du patient :  Vivant  Décédé

Réhospitalisation précoce pour insuffisance cardiaque :  Oui  Non

**Appendix 5. Biology follow-up**

<b>EBP RÉALISÉ EN LOCAL → Prise en Charge de Soins courants du patient (PCS)</b>			
	A l'entrée J0	Toutes les semaines	A la sortie
Ionogramme : natrémie, kaliémie, bicarbonates	PCS	PCS	PCS
Créatinine, urée	PCS	PCS	PCS
Hémoglobine, hématocrite, TP	PCS		
Ferritine, coefficient de saturation transferrine	PCS		
NT-pro BNP	PCS		PCS
Troponines	PCS		
TSH	PCS		
Albumine, préalbumine	PCS		
HbA1c	PCS		
Bilirubine	PCS		



**COMITÉ de PROTECTION des PERSONNES EST-III**

Hôpital de Brabois, Rue du Morvan - 54511 VANDŒUVRE-LES-NANCY Cedex  
 Téléphone : 03 83 15 43 24 - Télécopie : 03 59 62 06 02 - Courriel : [eppest.3@chu-nancy.fr](mailto:eppest.3@chu-nancy.fr)

Projet de recherche envisagé  
 Sous les références  
 N°ID-RCB: 2020-A0213-36  
 N° CNRPH: 20/09/07/37902  
 N° CPP: 20.10.05  
 N°2030

Le Comité a été saisi le 28 octobre 2020 par Mr **Michele SCRIBENTINO**, Directeur du CHU Grenoble Alpes, représentant le CHU de Grenoble Alpes, promoteur, pour l'examen des modifications apportées suite à une première séance le 6 octobre 2020 du projet de recherche mentionné au 3° de l'article L1121-1 du Code de Santé Publique, intitulé :

**Intérêt de l'échographie pleuro pulmonaire dans la prise en charge thérapeutique des patients hospitalisés pour décompensation cardiaque aiguë en service de médecine conventionnelle (EPPICA).**

Dont l'investigateur est Mme le Dr **Ferrine DUMANIER** - Unité Past Urgences - CHU Grenoble Alpes  
 Le Comité a examiné ces modifications lors de sa séance du 3 novembre 2020. Ont participé aux délibérations :

- les membres du Collège n°1 :
- Catégorie 1 : Mme Lupercin (Thalaise), Mr Petit (Thalaise), Mr Perrin (Thalaise),
- Catégorie 3 : Mr Bureau (Suppléant),
- Catégorie 4 : Mr Pédler (Thalaise),
- les membres du Collège n°2 :
- Catégorie 5 : Mr Marinet (Thalaise),
- Catégorie 6 : Mme Humbert (Suppléante),
- Catégorie 7 : Mme Batt (Thalaise),
- Catégorie 9 : Mme Boulet (Suppléante), Mr Girz (Suppléant)

Le Comité a adopté la délibération suivante : **AVIS FAVORABLE** pour :

Courrier de demande d'avis (PDF signé + Document WORD)	01.09.2020
Formulaire de demande d'avis + page de signature	01.09.2020
Protocole + page de signature	1.0 du 25.08.2020
Résumé	1.0 du 25.08.2020
Lettre d'information et de non opposition	1.0 du 25.08.2020
Liste des investigateurs	1.0 du 25.08.2020
CVs des investigateurs	NA
Bon niveau d'enregistrement RCB	21.08.2020
CRF	1.0 du 25.08.2020

mercredi 4 novembre 2020

La Vice-Présidente  
 Martine BATT



**COMITÉ de PROTECTION des PERSONNES EST-III**

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 Téléphone : 03 83 15 43 24 - Télécopie : 03 59 62 06 02 - Courriel : [eppest.3@chu-nancy.fr](mailto:eppest.3@chu-nancy.fr)

Mme Catherine PORTAL  
 ABC promoteur  
 DRU - cellule de promotion  
 CHU Grenoble Alpes

Projet de recherche envisagé  
 Sous les références  
 N°ID-RCB: 2020-A0413-36  
 N° CNRPH: 20/09/07/37902  
 N° CPP: 20.10.05  
 N°2030

Madame,

Je vous prie de bien vouloir trouver ci-joint l'avis du Comité concernant le protocole intitulé :

**Intérêt de l'échographie pleuro pulmonaire dans la prise en charge thérapeutique des patients hospitalisés pour décompensation cardiaque aiguë en service de médecine conventionnelle (EPPICA).**

Veuillez agréer, Madame, l'assurance de ma stricte considération.

La Vice-Présidente

Martine BATT  


**NB :** Le comité demande au promoteur d'apporter les modifications dans un délai de 12 jours à partir du téléchargement de l'avis sur le portail CNRPH. Veuillez, si il vous plaît, veiller à ce que les documents ayant fait l'objet de modifications soient correctement datés, versionnés et téléversés sur le site SI CN RPH, complét « réponses ». Il convient également de prévenir par mail le CPP contenant la liste des documents transmis avec dates et numéros de version.



# Le serment d'Hippocrate

Texte revu par l'Ordre des médecins en 2012

“ **Au moment d'être admis(e) à exercer la médecine, je promets et je jure d'être fidèle aux lois de l'honneur et de la probité.**

Mon premier souci sera de rétablir, de préserver ou de promouvoir la santé dans tous ses éléments, physiques et mentaux, individuels et sociaux.

Je respecterai toutes les personnes, leur autonomie et leur volonté, sans aucune discrimination selon leur état ou leurs convictions. J'interviendrai pour les protéger si elles sont affaiblies, vulnérables ou menacées dans leur intégrité ou leur dignité. Même sous la contrainte, je ne ferai pas usage de mes connaissances contre les lois de l'humanité.

J'informerai les patients des décisions envisagées, de leurs raisons et de leurs conséquences.

Je ne tromperai jamais leur confiance et n'exploiterai pas le pouvoir hérité des circonstances pour forcer les consciences.

Je donnerai mes soins à l'indigent et à quiconque me les demandera. Je ne me laisserai pas influencer par la soif du gain ou la recherche de la gloire.

Admis(e) dans l'intimité des personnes, je tairai les secrets qui me seront confiés. Reçu(e) à l'intérieur des maisons, je respecterai les secrets des foyers et ma conduite ne servira pas à corrompre les mœurs.

Je ferai tout pour soulager les souffrances. Je ne prolongerai pas abusivement les agonies. Je ne provoquerai jamais la mort délibérément.

Je préserverai l'indépendance nécessaire à l'accomplissement de ma mission. Je n'entreprendrai rien qui dépasse mes compétences. Je les entretiendrai et les perfectionnerai pour assurer au mieux les services qui me seront demandés.

J'apporterai mon aide à mes confrères ainsi qu'à leurs familles dans l'adversité. Que les hommes et mes confrères m'accordent leur estime si je suis fidèle à mes promesses ; que je sois déshonoré(e) et méprisé(e) si j'y manque.

”