

## Introduction

The incidence of supraventricular arrhythmias (SVA) in intensive care is high, and new-onset atrial fibrillation (NOAF) in critically ill patients is among the most frequent tachyarrhythmias [1–5]. NOAF is not only a marker of disease severity, but it also directly worsens haemodynamics through loss of atrial contraction, shortening of diastolic time, decrease in cardiac output, and increase in filling pressures [1]. In the context of sepsis and septic shock, NOAF is associated with higher short-term morbidity and mortality, as well as with an unfavourable long-term prognosis in patients who survive and are discharged from the intensive care [2–4].

The aim of this review is to summarise the incidence of NOAF, its clinical consequences, options for prevention and treatment, and to demonstrate how bedside echocardiography and key functional indices, particularly the left atrial (LA) parameters, enable individualised decision-making between rhythm and rate control in the management of NOAF.

## Epidemiology

NOAF denotes atrial fibrillation that develops during hospitalisation in a patient with no previous history of persistent or permanent atrial fibrillation (AF); definitions of NOAF and methods of detection vary across the literature [5, 6]. According to the European Society of Cardiology (ESC) guidelines for atrial fibrillation, the minimum episode duration required for diagnosis is 30 seconds; only episodes of this length are classified as sustained atrial fibrillation [7]. The reported incidence of NOAF in sepsis varies depending on the definition used and the monitoring strategy. In cohorts of critically ill patients, it ranges from 7–14% [3–6] and further increases with disease severity, reaching as high as 46% in septic shock [5]. Continuous ECG monitoring additionally shows that some episodes are subclinical and only detectable on a more detailed analysis of recorded data; different thresholds and detection methods therefore substantially contribute to the heterogeneity of the reported incidences [6, 8].

The occurrence of NOAF is associated with adverse short-term and long-term clinical outcomes. In critically ill patients, it is linked to a higher risk of ischaemic stroke, longer hospital stay, and increased mortality [2, 5, 9]. In the prospective multicentre AFIB-ICU study, patients with AF, compared with those without AF, had a higher incidence of ischaemic thromboembolic events (13.6% vs. 7.9%), more episodes of major bleeding (5.9% vs. 2.1%), and higher in-hospital mortality (41.2% vs. 25.2%) [4]. After discharge from the intensive care, patients who developed NOAF during sepsis remain at a substantial risk of AF recurrence (as many as 55% over 5 years) and experience unfavourable long-term outcomes (ischaemic stroke, heart failure, and mortality) [10].

## Pathophysiology: NOAF and the left atrium in septic shock

The development of NOAF in critically ill patients is multifactorial and has not yet been fully elucidated. Risk factors overlap with those in the general population (advanced age, obesity, male sex, arterial hypertension, coronary artery disease, heart failure, genetic predisposition), but

in the intensive care setting, sepsis, overall disease severity including organ failure, and ARDS requiring mechanical ventilation play a particularly important role [3, 8]. The relationship between hypertension, coronary artery disease, and NOAF in patients with sepsis is inconsistent compared with the general population [11]. Reversible triggers include systemic inflammation, excessive sympathetic activation and elevated catecholamine levels, hypoxia, acidosis, hypo-/hypervolaemia, anaemia, and electrolyte disturbances (especially  $Mg^{2+}$  and  $K^+$ ) [6, 12, 13].

Outside the intensive care setting, the arrhythmogenic substrate of paroxysmal AF is in most cases located in the region of the pulmonary vein orifices entering the left atrium [14]. In critically ill patients, accelerated electrical and structural changes also occur elsewhere in the myocardium and contribute to atrial remodelling. Acute conditions such as myocardial ischaemia and systemic inflammation in sepsis promote remodelling and may initiate atrial myocardial fibrosis, thereby increasing the susceptibility to triggers and the development of SVA [15]. During septic shock, vasoplegia leads to a reduction in systemic vascular resistance and marked fluctuations in preload. At the same time, excessive  $\beta_1$ -adrenergic stimulation and chronotropic dysregulation are present, and systolic as well as diastolic dysfunction of the left ventricular (LV) and right ventricular (RV) myocardium may develop as part of septic cardiomyopathy [16].

The LA fulfils three functions: a reservoir in systole (accumulating blood from the pulmonary veins); a conduit in early diastole (passively transferring blood to the LV after mitral valve opening); and, in sinus rhythm (SR), active atrial contraction in late diastole (“atrial kick”, i.e. active completion of LV diastolic filling). In diastolic dysfunction, loss of atrial contraction is clinically tolerated depending on the heart rate, ventricular elastance, and filling pressures in the LA/LV [17]. Irregular RR intervals during AF reduce cardiac output and increase filling pressures independently of the mean heart rate [1]. Patients with essential hypertension, diabetes, and chronic kidney disease frequently have diastolic dysfunction, which leads to structural remodelling and left atrial dilatation, and may create an arrhythmogenic substrate in later stages [18]. Left atrial size has a high predictive value for both the development of NOAF and its recurrence [19].

## Echocardiographic assessment in critically ill patients with NOAF

According to the 2024 ESC guidelines, echocardiographic examination is indicated in every patient with NOAF, and TTE should be repeated with any clinically significant change in their condition [7]. The indications, optimal timing, and frequency of repeat TTE in critically ill patients with SVA are not yet standardised [13]. Transthoracic echocardiographic examination (TTE) at the bedside in intensive care is an integral part of the assessment of any circulatory instability [20]. Point-of-care ultrasonography (POCUS) is used for rapid targeted screening according to recommended protocols; this is followed by comprehensive TTE for detailed quantification [21]. Comprehensive assessment includes evaluation of left and right ventricular systolic function, estimation of cardiac output, estimation of pulmonary artery pressure, pericardial status, left ventricular diastolic function, which includes examination