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The Missing Risk Factor in Cardiovascular Care

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Obstructive sleep apnea (OSA) is increasingly recognized as an independent cardiovascular risk factor. Yet most health systems have been slow to act on this. This article traces the growing, and still underutilized, role of sleep in cardiovascular care: The biological pathways linking OSA to heart disease, the evolving frameworks for understanding it, and the gap between scientific evidence and clinical implementation.

Cardiovascular disease (CVD) prevention has long focused on the familiar: Diet, physical activity, smoking, blood pressure, and cholesterol. Sleep has barely featured. That is beginning to change.

Sleep disorders, and obstructive sleep apnea (OSA) in particular, are increasingly recognized as important contributors to cardiovascular disease development and progression. The American Heart Association (AHA) has highlighted that OSA is both common and frequently underdiagnosed in cardiovascular patients. It is now understood not merely as a coexisting condition but as an independent and modifiable cardiovascular risk factor.[1-4]

What is less clear is whether health systems have caught up. The international evidence suggests they have not.

How OSA affects the heart and vasculature

The biological case for OSA as a cardiovascular risk factor is well established. Repeated breathing interruptions during sleep cause intermittent hypoxia, sympathetic nervous system activation, systemic inflammation, and endothelial dysfunction. Together, these pathways place sustained stress on the cardiovascular system and may actively drive disease progression rather than simply accompany it.

Longitudinal and clinical studies support this picture. Untreated severe OSA is associated with higher rates of fatal and non-fatal cardiovascular events; patients treated with positive airway pressure (PAP) exhibit reduced risk in some studies. OSA severity and nocturnal hypoxemia have also been identified as significant predictors of sudden cardiac death.

The relationship appears partly bidirectional. Heart failure, for example, can worsen sleep-disordered breathing through fluid redistribution and altered ventilatory control, but the balance of evidence positions OSA primarily as a contributing and compounding factor in cardiovascular disease.

One important nuance: The evidence on PAP's effect on major cardiovascular events remains mixed across randomized controlled trials. PAP reliably improves symptoms and intermediate outcomes such as blood pressure, but its impact on hard endpoints like myocardial infarction or stroke has been variable. This has influenced, and in some cases constrained, how health systems respond.

Rethinking how OSA is classified

The way OSA is conceptualized is also evolving. Traditionally, OSA severity has been defined almost exclusively by the apnea-hypopnea index (AHI), a basic count of breathing interruptions per hour. But this measure, while useful, does not capture the full clinical picture.

The Baveno classification,[5] an emerging framework in the field, proposes moving beyond AHI-centered definitions toward a broader assessment that incorporates symptoms, cardiovascular and metabolic comorbidity, and end-organ consequences when stratifying disease burden and treatment need. Crucially, cardiovascular disease plays a central role in defining clinically significant OSA under this framework reinforcing a shift away from viewing OSA as a purely sleep-specific disorder toward understanding it as a chronic disease and risk-management issue with cardiovascular dimensions at its core.

The implementation gap

Despite this growing clinical evidence, health systems have not systematically translated it into policy or routine practice. In most countries, OSA remains largely confined to sleep medicine rather than integrated into cardiovascular prevention and chronic disease management. Screening tends to occur selectively in hypertension clinics, heart failure services, or arrhythmia care rather than as part of standardized cardiovascular risk assessment.

The populations most affected are precisely those already placing the greatest burden on cardiovascular services: Patients with obesity, hypertension, heart failure, and metabolic disease. As obesity rates rise, populations age, and multimorbidity becomes more common, the burden of unidentified sleep-disordered breathing is likely to grow.

At the same time, several enabling factors are coming together. Home sleep testing, ambulatory monitoring, and digital screening technologies are making earlier identification increasingly feasible outside specialist sleep laboratories. The remote care infrastructure that expanded rapidly during the pandemic has lowered practical barriers to decentralized assessment. And as populations age and multimorbidity becomes more common, the evidence base for OSA's role across the cardiovascular risk profile is maturing, with long-term outcomes data now available that simply did not exist a decade ago. The conditions for more integrated care are more favorable than they have ever been.

What the international picture shows

A comparison examines how this gap manifests across healthcare systems in five major markets: France, Germany, the United Kingdom, the United States, Australia, and Japan. It reviews to what extent each has integrated OSA into cardiovascular prevention, screening, and treatment frameworks.

The international picture reveals significant variation. Japan stands out as the most advanced model, with OSA systematically embedded across cardiovascular prevention and disease management from routine screening through to treatment. Most other systems occupy a middle ground: Acknowledging the OSA-CVD link, incorporating it selectively in areas like resistant hypertension or atrial fibrillation, but stopping short of treating it as a core cardiovascular prevention target. The UK sits at the more limited end, with OSA largely confined to sleep medicine guidance and largely absent from mainstream cardiology pathways.

What this means for cardiovascular policy

The gap between the strength of scientific evidence and its limited operationalization in most health systems is substantial — but not insurmountable. Tools for earlier identification, including home sleep testing, ambulatory monitoring, and digital screening, are improving feasibility. Emerging classification frameworks like Baveno explicitly position OSA within cardiovascular risk management rather than as a purely sleep-specific disorder. And the evidence base is maturing, with long-term outcomes data now available that supports a more central role for OSA in cardiovascular care.

Whether health systems close this gap will depend less on additional biological evidence — that case is well established — and more on whether cardiovascular prevention frameworks, quality measures, and care pathways are updated to reflect what is already known.

References

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