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Implications of Pacemaker Implantation After Aortic Valve Surgery for Endocarditis: a SWEDEHEART Study

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Background:Infective endocarditis is associated with a high risk of atrioventricular block and surgery adds to the risk of receiving a permanent pacemaker. The clinical impact of pacemaker implantation in endocarditis patients is poorly understood.

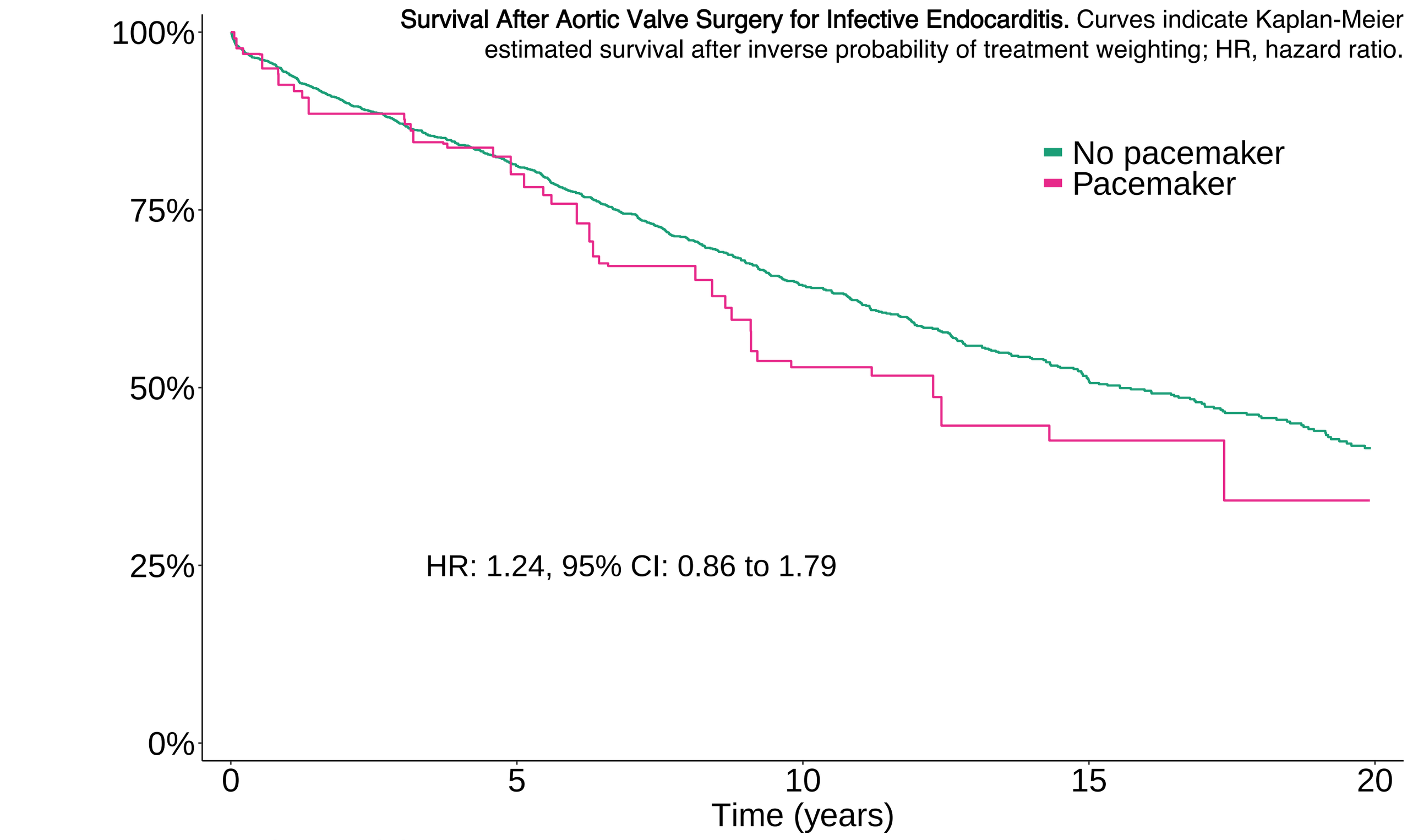
Objective:To analyze all-cause mortality, heart failure and prosthetic valve endocarditis in patients who receive a permanent pacemaker after surgery for native aortic valve IE.

Methods:We conducted a nationwide population-based, observational cohort study, based on the SWEDEHEART register, including all patients who underwent aortic valve replacement for infective endocarditis in Sweden 1997-2022. Mean follow-up was 8.5 years. Inverse probability of treatment weighting accounted for intergroup differences. Flexible parametric models were used to estimate hazards and cumulative incidences.

Results:Among 1778 patients, 119 (7%) received a permanent pacemaker. The mean age was 58 years; 335 (19%) were female. During follow-up, 683 patients (38%) died. At 20 years, the cumulative incidence of all-cause mortality in the pacemaker versus the no pacemaker group, respectively, was 66% versus 59%. For heart failure hospitalization, it was 25% versus 18%. After adjustment, we found no association of pacemaker implantation with mortality (HR 1.24, 95%;CI: 0.86-1.79), heart failure (HR 1.61, 95%;CI: 0.9-2.87), or prosthetic valve endocarditis (HR 0.90, 95%;CI: 0.48-1.69).

Conclusion:Permanent pacemaker implantation after surgery for native aortic valve endocarditis carried no increased long-term risk of death, heart failure, or prosthetic valve endocarditis. These results are reassuring and suggest that factors other than pacemaker implantation are more important for the long-term prognosis in endocarditis patients.

Survival After Aortic Valve Surgery for Infective Endocarditis. Curves indicate Kaplan-Meier estimated survival after inverse probability of treatment weighting; HR, hazard ratio.



Number at risk

No pacemaker	1659	1054.9	594.8	303.8	120.9
Pacemaker	119	67.9	31.9	16.5	4

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Long-term outcomes after permanent pacemaker implantation in low-risk surgical aortic valve replacement: an observational cohort study of 19,576 Swedish patients

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Background: Permanent pacemaker implantation is associated with an increased risk of mortality and heart failure after surgical aortic valve replacement (SAVR). We analyzed long-term prognosis of permanent pacemaker implantation following SAVR in low-risk patients.

Materials and methods: This nationwide, population-based, observational cohort study included all patients who underwent SAVR in Sweden between 2001-2018 with low surgical risk, defined as logistic EuroSCORE I<10% or EuroSCORE II<4%. Patients received a permanent pacemaker implantation within 30 days after SAVR. Main outcomes were all-cause mortality, heart failure hospitalization and endocarditis. Regression standardization addressed confounding.

Results: We included 19,576 patients with low surgical risk. Of these, 732 (3.7%) patients received a permanent pacemaker within 30 days after SAVR. The mean age was 68 years and 33% were women. We found no difference in all-cause mortality between patients who received a pacemaker compared to those who did not (absolute survival difference at 17 years: 0.1% (95% CI: -3.6%–3.8%). After 17 years, the estimated cumulative incidence of heart failure in patients who received a pacemaker was 28% (95% CI: 24%–33%) vs. 20% (95% CI: 19%–22%) in patients who did not (absolute difference 8.2% (95% CI: 3.8%–13%)). We found no difference in endocarditis between the groups.

Conclusions: We found an increased incidence of heart failure in patients with low surgical risk who received a permanent pacemaker after SAVR. Permanent pacemaker implantation was not associated with all-cause mortality or endocarditis. Efforts should be made to avoid the need for permanent pacemaker following SAVR.

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Survival after PCI or CABG for multivessel disease in NSTE-ACS: a report from the SWEDEHEART registry

Elmir Omerovic

The authors have chosen not to publish the abstract

1-4

Long-term mortality after PCI or CABG in patients with diabetes and multivessel coronary artery disease: A SWEDEHEART study

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Background

Randomized studies indicate that coronary artery bypass grafting (CABG) is superior to percutaneous coronary intervention (PCI) for coronary revascularization in patients with diabetes and multivessel disease (MVD). Real world studies are sparse. We compared all-cause and cardiovascular (CV) mortality after CABG and PCI in a large nationwide cohort.

Material and methods

The SWEDEHEART registry was used to identify 26,166 patients with diabetes and MVD who underwent PCI or CABG in Sweden from 2006 to 2020. Mortality was compared using inverse probability of treatment weighting to adjust for confounding effects in Cox regression models. The median follow-up time was 5.5 (interquartile range 2.5-9.2) years.

Results

The majority of patients were treated with PCI (64%). All-cause mortality [adjusted hazard ratio (aHR) 0.79 (95% CI 0.75-0.83)] and CV mortality [aHR 0.71 (95% CI 0.67-0.76)] risks were lower after CABG compared to PCI. The lower mortality risk with CABG was consistent across subgroups based on sex, age, insulin treatment, and comorbidities. The adjusted median survival time was 0.9 (95% CI 0.4-1.3) years longer after CABG compared with PCI, and markedly longer in patients with left main stem stenosis or three vessel disease [4.3 (95% CI 3.4-5.1) and 3.4 (95% CI 2.8 -4.0) years, respectively].

Conclusions

CABG was associated with significantly lower all-cause and cardiovascular mortality risk and longer survival compared to PCI, particularly in those with extensive coronary artery disease. The results support current guidelines favoring CABG in patients with diabetes and multivessel disease.

1-5

Acute Type A Aortic Dissection mimicking Acute Coronary Syndrome: does it matter?

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Background: The impending risk of rupture makes misdiagnosis of acute type A aortic dissection (ATAAD) critical. Antiplatelet-therapy for suspected acute coronary syndrome (ACS) escalates the risk of perioperative bleeding and might render ATAAD-patients for increased morbidity and mortality. ATAADs can mimic ACS clinically, but occasionally affect the coronary arteries and cause myocardial ischemia. We aimed to investigate the consequences of ACS-mimicking ATAAD.

Materials and methods: Medical records of 259 consecutive emergent ATAAD-surgeries at Oslo University Hospital from 2018-2022 were retrospectively reviewed.

Results: 79.3% experienced chest pain, while ECG-changes were present in 20.4% of cases. Elevated Troponin-T(>14 ng/L) was seen in 60.2%, while 23.5% had a value >50 ng/L. Acetylsalicylic acid was given in 68 cases (26.4%), dual antiplatelet-therapy (DAPT) in 29 (11.3%) and DAPT supplemented by Heparin in 20 (7.8%). Coronary angiography was performed in 22 (8.5%) cases. 14 (5.4%) patients had true coronary compromise - with seven requiring CABG. The validated group had higher 30-day-mortality (53.9% vs. 13.3%, p=0.001). In a ACS-suspicion-group (DAPT or angiography) of 35 patients (13.5%), only eight had true coronary compromise. This group did not present higher mortality (22.9% vs. 14.2%, p=0.185), but had increased durations of operation (p=0.003), extracorporeal circulation (p=0.004) and cardiac ischemia (p=0.020), and increased postoperative bleeding (p=0.014). Furthermore, more transfusion of SAG (p=0.008) and plasma (p<0.001) was required.

Conclusion: Coronary artery involvement in ATAAD is rare and severe, while ACS-mimicking clinical features are common. ACS-misdiagnosis may increase the perioperative ATAAD-risk as the burden of bleeding complicates the surgical treatment.

1-6

Causes and clinical impact of initial misdiagnosis of acute type A aortic dissection

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Background

The high mortality in untreated acute type A aortic dissection (ATAAD) stresses the need for prompt diagnosis and immediate surgical treatment. The aim of this study was to evaluate the frequency and clinical impact of misdiagnosis and delayed diagnosis of ATAAD.

Methods

This was a single-center, retrospective, observational study including all ATAAD patients in our region with available admission charts between 2001-2021. The primary endpoints were initial misdiagnosis, delayed diagnosis and 30-day mortality. Surgical treatment was a secondary endpoint. Independent predictors of misdiagnosis and 30-day mortality were identified by multivariable logistic regression and subgroup analyses by severity of clinical presentation were performed.

Results

556 patients were included in the study (418 surgically treated and 138 non-surgically treated), and 45.3% were initially misdiagnosed. Misdiagnosed patients were more often female (47.6% vs 35.9%; p=0.005) and demonstrated significantly lower rates of syncope, hypotensive shock, malperfusion or cardiac arrest. Misdiagnosed patients with end-organ malperfusion exhibited higher 30-day mortality (44.4% vs 23.3%; p=0.022) and were less likely to receive surgical treatment (69.6% vs 88.7%; p=0.013). Female sex was an independent predictor of misdiagnosis (OR: 1.635; 95% CI 1.069-2.500; p = 0.023) but neither misdiagnosis nor delayed diagnosis were independent predictors of 30-day mortality.

Conclusions

Misdiagnosis occurred in nearly half of ATAAD-cases and significantly more often in female patients. Misdiagnosis or delayed diagnosis did not influence overall 30-day mortality likely due to misdiagnosed patients presenting in a more favorable clinical state.

1-7

Coronary artery bypass grafting with and without preoperative physiological flow assessment

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The authors have chosen not to publish the abstract

1-8

Long-term outcome after aortic valve replacement for patients with bicuspid aortic valve and severe aortic stenosis

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The authors have chosen not to publish the abstract

1-9

Clinical outcomes of small postoperative creatinine changes after surgical aortic valve replacement: an observational cohort study of 16, 766 Swedish patients.

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Background: The impact of small increases in serum-creatinine after surgical aortic valve replacement (SAVR) that fail to meet the acute kidney injury stage (AKI) 1 criteria is unknown. We investigated the prognosis after primary SAVR in patients with small increases in postoperative serum-creatinine.

Materials and methods: In this observational cohort study, we included all adult patients who underwent primary SAVR in Sweden 2009–2022. The main outcome was all-cause mortality. Secondary outcomes were chronic kidney disease and heart failure. Regression standardization addressed confounding.

Results: In 16,766 patients, 4,074 (24.2%) had no change in postoperative serum-creatinine, 5,764 (34.3%) had a small increase in postoperative serum-creatinine (5µmol/L≤ Δserum-creatinine <26.5µmol/L), and 2,753 (16.4%) fulfilled the KDIGO AKI stage 1 criteria. The mean age was 67 years and 31% were female. No significant difference in long-term all-cause mortality was observed in the no-change group at 13 years compared with the small-increase group (absolute survival difference: 2.3% (95%CI: 0–4.6)). A stepwise increase in the risk of 30-day mortality was observed with increasing change in serum-creatinine. At 13 years, there was a significant difference in the risk of chronic kidney disease (absolute difference: 2.8% (95%CI: 1.0–4.5)) and heart failure (absolute difference: 3.5% (95%CI: 1.3–5.7)) between the no-change and small-increase groups.

Conclusions: A small increase in postoperative serum-creatinine after SAVR was associated with increased risk of adverse outcomes. The AKI definition may benefit from including specific biomarkers together with small creatinine increases to detect kidney injury.

1-10

Dilatation of thoracic aorta and chronic obstructive pulmonary disease

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Background: Thoracic aortic dilatation is associated with increased susceptibility to aortic events. Chronic obstructive pulmonary disease (COPD) may add to tissue degeneration and inflammation associated with the risk of increased aortic dilatation. We studied whether COPD is associated with aortic wall degeneration during thoracic aortic dilatation.

Materials and methods: We enrolled 35 consecutive patients who underwent elective surgery for aortic dilatation between February 2016 and November 2016 due to increased ascending aortic dilatation. The patients were grouped according to the presence of COPD. Extensive aortic wall histopathology was performed and compared with the aortic diameter measured from the aortic root, sinotubular junction, ascending aorta, aortic arch, and descending aorta.

Results: The aortic wall of COPD patients (n=7) had more mucoid extracellular matrix accumulation, medial degeneration, elastic fiber fragmentation and loss, and adventitial fibrosis than those without COPD (5.7±1.1 vs 4.3±1.0; point score unit (PSU), P=0.006; 2.7±0.5 vs 1.7±0.8; PSU, P=0.006; 4.5±1.2 vs 3.3±1.2; PSU, P=0.034 and 0.5±0.5 vs 0.1±0.3; PSU, P=0.023, respectively). Only the distal ascending aortic diameter differed in patients with COPD vs without (36.5±3.3 vs 32.2±3.8; mm, P=0.011), though increased aortic dilatation was otherwise present in both groups.

Conclusions: The extent of ascending aortic wall degeneration is increased in COPD patients with increased thoracic aortic dilatation. Risk stratification of aortic events in patients with COPD warrants clarification.