

Alcohol Consumption Patterns and Vascular Properties: The Value of Repeatedly Measured Longitudinal Data

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In this issue of *JAHA*, O'Neill et al demonstrate the utility of longitudinal data in illustrating the relation between alcohol consumption and arterial stiffness.¹ Recent reports from various parts of the world detail a disturbing stagnation and decline in peace-time population life expectancy.^{2,3} Among the leading attributed causes is substance abuse, including alcohol use. Globally, alcohol use accounts for nearly 6% of deaths and 5% of disability-adjusted life years.⁴ Cardiovascular consequences of alcohol use include cardiovascular events from acute ingestion, longer-term risk of events such as atrial fibrillation and stroke; complex relations with lipids; and hypertension. Some studies conceptualize the relation between alcohol consumption and cardiovascular consequences as a J-shaped relationship, with moderate consumption of alcohol being least deleterious compared to low or high consumption.⁵

Noninvasively measured aortic stiffness has emerged as a strong predictor of future cardiovascular events such as stroke, heart failure, and coronary disease as well as a precursor to hypertension.^{6–8} Previous studies have shown a cross-sectional association between alcohol use and aortic stiffness, parallel to other observed alcohol and cardiovascular disease relations.^{5,9} O'Neill and colleagues relate findings from other investigators demonstrating that higher alcohol consumption was prospectively associated with worsening aortic stiffness.^{10,11} Another study with repeat alcohol assessments found that while heavy drinkers with hypertension had worsening aortic stiffness over time, moderate drinkers without hypertension had better aortic stiffness over time compared to nondrinkers.¹² This counterintuitive result

regarding nondrinkers being at higher risk dovetailed with recent work suggesting that results may be critically sensitive to misclassification, especially with the co-mingling of never drinkers with the recently abstaining.¹³ Since alcohol use patterns may vary over time, understanding the change in aortic function from alcohol use may require detailed longitudinal specificity in alcohol exposure assessment.

O'Neill and colleagues attempted to disentangle the conflation of nondrinkers from recent abstainers and other alcohol use patterns in the well-described Whitehall II cohort. This sample of the cohort had carotid femoral pulse wave velocity measured as the “gold standard” of aortic stiffness assessment at 2 separate examination visits. Self-reported, long term alcohol use was then reported in 6 categories by stable versus unstable use and by nondrinker, moderate, or heavy use and short term patterns by heavy use, moderate use, or no use. When examining recent drinking patterns, only men who were recent nondrinkers appeared to have a higher aortic stiffness, after adjustment for many covariates that may or may not be along the “causal chain” such as lipid levels and body mass index. There was no association in recent heavy drinkers, undercutting the idea of a J-shaped relation. Intriguingly, unadjusted aortic stiffness did not appear to show an easily discernible trend across long term alcohol use patterns. Indeed, unstable heavy drinkers were comparable to the reference group stable moderate drinkers, while stable nondrinkers were among the most stiff, although none of these differences were reported as significant. Also interesting was that overall long term alcohol use patterns did not show many consistent associations with either baseline or change in aortic stiffness. Stable heavy drinkers in both sexes appeared to have stiffer aortae at baseline compared to stable moderate drinkers. Women who were unstable moderate drinkers also had higher stiffness at baseline. To disambiguate the recent low-consumption group, recently former drinkers had more aortic stiffening over time, while stable nondrinkers did not. This result in 2 types of recent nondrinkers may correspond to clinical intuition that recent abstainers may include persons with worsening health status. Therefore, misclassification of recent nondrinkers is epidemiologically relevant and should be addressed through repeated measures collected over time.

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The highlighted findings around the vascular consequences of alcohol use are useful to investigators and perhaps even clinicians. The analysis is well supported by the large cohort size, although some subgroup analyses were hampered by small numbers. Future work would do well to examine mechanisms behind sex differences in associations and sex-specific aging interactions, given previously described age- and sex-specific patterns in vascular properties.¹⁴ O'Neill and colleagues are to be commended on a worthy contribution on the role of long-term alcohol use and aortic stiffening.

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Disclosures

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