between acute coronary syndrome (ACS) and stable angina (SA), males and females, and males and females with SA and ACS. In our analysis, 121,066 patients were included (26% INOCA, 31% female, mean age 62 years). Patients with INOCA were more likely to be female and associated with stable angina and atrial fibrillation as compared to patients with obstructive CAD (OR=3.29, 95% CI: 3.01, 3.59, p< 0.001; OR=2.62, 95% CI: 2.4, 2.86, p< 0.001; OR=2.21, 95% CI: 1.77, 2.76, p< 0.001, respectively). The percent of INOCA to obstructive CAD ranged between 24.2% and 26.7% in all patients (p < 0.001), 19.4% and 21.4% in patients with ACS (p=0.002), and 30.6% and 37.5% in patients with SA (p < 0.001). Additionally, women had a higher prevalence of INOCA than men, with the percentage of INOCA to obstructive CAD ranging between 17.4% and 20.2% in males and 37.9% and 40.9% in females (p < 0.001; p=0.011, respectively). A similar trend was observed in both males and females with ACS and SA, with the percent of INOCA to obstructive CAD ranging between 14.1% and 15.7% in males with ACS and 31.4% and 33.5% in females with ACS and 27.3% and 29.5% in males with stable angina and 49.7% and 54.6% in females with stable angina. Overall, there was no substantial difference in the percentage of INOCA to obstructive CAD over time.

CONCLUSION: The results from this indicate that there remains a high prevalence of non-obstructive coronary arteries detected during invasive CAG, and this prevalence remains stable over time for the entire population and the sub-groups evaluated. This demonstrates an opportunity to exclude obstructive coronary disease with less invasive testing, particularly in females.

Table 1. Comparison of baseline characteristics between patients with INOCA and obstructive CAD

Characteristic	ALL (n = 121066)	INOCA (n = 30886)	CAD (n = 90180)	P value
Age, mean years	62.3 ± 11.8	59.0 ± 12.1	63.4 ± 11.5	<0.001
Female (%)	37900 (31)	15024 (49)	22876 (25)	<0.001
Normal coronary arteries (%)	14943 (12.4)	14943 (48)	0 (0)	N/A
Systolic BP, mean	121.1 ± 24.5	122.4 ± 23.4	121.0 ± 24.9	<0.001
Diastolic BP, mean	67.9 ± 12.5	69.1 ± 11.8	67.5 ± 12.7	<0.001
BMI, mean	29.3 ± 6.0	29.8 ± 6.7	29.1 ± 5.7	<0.001
ACS (%)	76252 (63)	15496 (50)	60756 (67)	<0.001
SA (%)	44814 (37)	15390 (50)	29424 (33)	<0.001
Comorbidities (%)				
Hypertension	63257/96429 (66)	15547/25392 (61)	47710/71037 (67)	<0.001
Dyslipidemia	70361/93826 (75)	16924/24500 (69)	53437/69326 (77)	<0.001
Diabetes	23467 (19)	4648 (15)	18819 (21)	<0.001
Current smoker	29139/90964 (32)	6105/23703 (26)	23034/67261 (34)	<0.001
Previous smoker	30554/90964 (34)	7840/23703 (33)	22714/67261 (34)	<0.052
FhxCAD	26840/59593 (45)	7446/16189 (46)	19394/43404 (45)	0.004
Malignancy	3330/85178 (4)	869/22654 (4)	2461/62524 (4)	0.505
Atrial fibrillation	707/12790 (6)	262/3620 (7)	445/9170 (5)	<0.001
CKD	772/32923 (2)	152/8743 (2)	620/24180 (3)	<0.001
CEVD	4470/92107 (5)	960/24514 (4)	3510/67593 (5)	<0.001
HF	3758/91556 (4)	908/24562 (4)	2850/66994 (4)	<0.001
Renal disease	3336/86445 (4)	633/22851 (3)	2703/63594 (4)	<0.001
Liver disease	559/66856 (1)	199/18115 (1)	360/48741 (1)	<0.001
PAD	3778/85477 (4)	611/22641 (3)	3167/62836 (5)	<0.001
Medications at Time of Cath (%):				
Aspirin	84762/104499 (81)	20306/26221 (77)	64456/78278 (82)	<0.001
Beta-blockers	63810/104499 (61)	15001/26221 (57)	48805/78278 (62)	<0.001
Statins	57882/104499 (55)	13492/26221 (52)	44390/78278 (57)	<0.001
CCBs	13686/104499 (13)	3476/26221 (13)	10210/78278 (13)	0.376
ACE-inhibitor	42526/104499 (41)	9297/26221 (36)	33225/78278 (42)	<0.001
Long-acting nitrates	17697/104499 (17)	4128/26221 (16)	13569/78278 (17)	<0.001
Insulin	4603/104499 (4)	825/26221 (3)	3778/78278 (5)	<0.001
Lab Results				
Total Cholesterol (mmol/L)	4.6 ± 1.2	4.5 ± 1.1	4.6 ± 1.2	<0.001
HDL (mmol/L)	1.2 ± 0.4	1.3 ± 0.4	1.2 ± 0.4	<0.001
LDL (mmol/L)	2.6 ± 1.0	2.5 ± 1.0	2.7 ± 1.0	<0.001
Triglyceride (mmol/L)	1.7 ± 1.2	1.6 ± 1.0	1.8 ± 1.3	<0.001
Random glucose (mmol/L)	7.4 ± 5.1	6.6 ± 4.3	7.6 ± 5.3	<0.001
Creatinine (μmol/L)	90.4 ± 51.3	85.4 ± 50.6	92.2 ± 51.4	<0.001
HbA1c (%)	6.4 ± 1.5	6.1 ± 1.3	6.4 ± 1.6	<0.001
Index PCI	7508 (6)	0	7508 (8)	N/A
Index CABG	6488 (5)	0	6488 (7)	N/A

Data presented in mean ± SD or count (%). P-value assessed by student's t-test or χ^2 test, as appropriate, and significance was set at p < 0.05.

Figure 1.

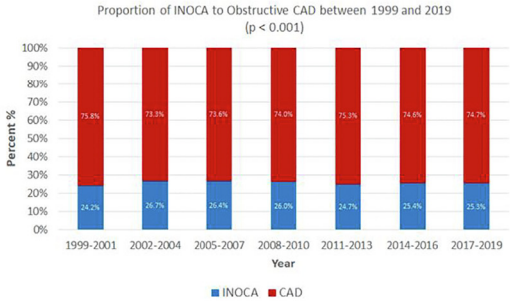


Figure 2A.

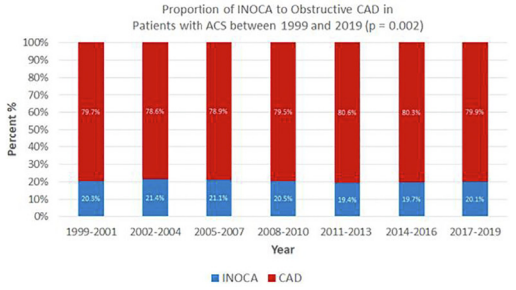
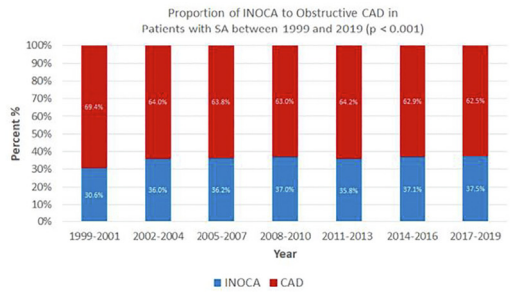


Figure 2B.



P041
THE IMPACT OF STATIN ON POST-OPERATIVE ATRIAL FIBRILLATION AFTER DISCHARGE FROM CARDIAC SURGERY: SECONDARY ANALYSIS OF THE SEARCH-AF CARDIOLINK-1 RANDOMIZED TRIAL

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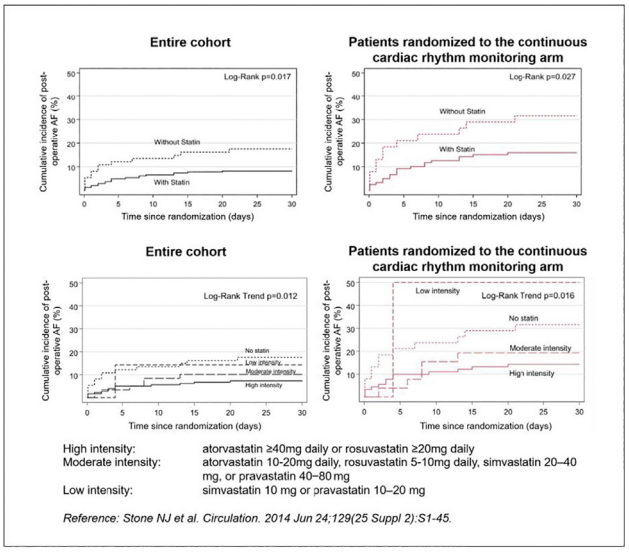
BACKGROUND: There is conflicting evidence regarding the use of statins to reduce the risk of post-operative atrial fibrillation (POAF) in patients undergoing cardiac surgery.

METHODS AND RESULTS: We sought to determine the effects of statin use on the incidence of post-discharge POAF in the Post-Surgical Enhanced Monitoring for Cardiac Arrhythmias and Atrial Fibrillation (SEARCH-AF) CardioLink-1 randomized controlled trial. This trial randomized 336 patients with risk factors for stroke (CHA2DS2-VASc score ≥2) and no history of preoperative AF were randomized to usual care or continuous cardiac rhythm monitoring for 30 days after discharge from cardiac surgery with a wearable, patched-based device. The primary endpoint was the occurrence of cumulative AF/AFL lasting for ≥6 minutes detected by continuous monitoring or AF/AFL documented by a 12-lead electrocardiogram within 30 days of randomization. We evaluated the association

between the risk of post-operative AF in relation to statin use and dosing intensity (low, moderate, high) at the time of discharge. We excluded patients who experienced post-operative AF during hospitalization in this analysis. In the overall cohort (n=336), 260 (77.4%) patients were treated with statins at the time of hospital discharge. There were 18 (5.4%) patients who experienced post-operative AF during hospitalization. Patients prescribed with statins were more likely to be male (p=0.018), had lower CHA2DS2-VASc scores (p=0.011), and were more likely to undergo isolated coronary artery bypass grafting (CABG) (p=0.083). Patients treated with statins at discharge had a 2-fold lower rate of POAF than those who were not treated with statins in the overall cohort (17.6% vs. 8.2%, Log-Rank p=0.017) and in those randomized to continuous cardiac rhythm monitoring (31.6% vs. 16.0%, Log-Rank p=0.027) (Figure). After adjusting for surgery type (CABG vs. valve surgery) and the CHA2DS2-VASc score, statin use at discharge was associated with a lower risk of post-operative AF within 30 days after surgery (hazard ratio 0.48, 95% CI 0.24-0.97). Increased intensity of statin therapy was associated with lower risk of POAF (P=0.0012 for trend). (Figure)

CONCLUSION: High-intensity statin could reduce the risk of POAF among cardiac surgical patients with risk factors for stroke. This merits further study.



BMS/Pfizer Alliance, Boehringer Ingelheim Canada Ltd., Canadian Stroke Prevention Intervention Network (C-SPIN), Heart and Stroke Foundation of Canada, Medtronic

P042
THE NON-INVASIVE ASSESSMENT OF PERIPHERAL MICROVASCULAR AND ENDOTHELIAL FUNCTION IN WOMEN WITH NON-OBSTRUCTIVE CORONARY ARTERY DISEASE

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BACKGROUND: Myocardial ischemia with non-obstructive coronary arteries (INOCA) is a disease disproportionately affecting women that is associated with a reduced quality of life and an increased risk of adverse cardiovascular events. Coronary microvascular and endothelial abnormalities are suspected to be the underlying causes of INOCA in the majority of patients. Currently, there is no widely accepted noninvasive test for diagnosing coronary microvascular and endothelial dysfunction in affected patients. To that end, recent evidence suggests microvascular function in the peripheral vascular circulation may correspond to coronary endothelial dysfunction. In this study, we tested the hypothesis that women with INOCA have attenuated peripheral microvascular and endothelial function compared to healthy controls.

METHODS AND RESULTS: We utilized three measures of peripheral arterial function, (1) flow-mediated dilation (FMD), (2) pulse arterial tonometry (PAT) and (3) velocity time integral (VTI), to understand the prevalence of microvascular and endothelial dysfunction in women with INOCA. Our study sample consisted of 32 perimenopausal women presenting with persisting chest pain and a diagnosis of INOCA following catheterization (mean age= 55 $\pm$ 6), and we compared them with 46 healthy age-matched healthy women (mean age= 51 $\pm$ 5). We found a significant difference in small-vessel endothelial function between the two groups as assessed by PAT, with patients demonstrating reduced function (RHI 2.08 $\pm$ 0.72 vs 2.54 $\pm$ 0.69, p=0.007) (Figure 1). This difference was statistically significant after correcting for confounders correlated with PAT, including age, body mass index, and hypertension (p=0.027). Small attenuations in brachial vasodilatory function as measured by FMD (Patients, 7.9 $\pm$ 3.9 vs controls, 9.3 $\pm$ 3.4, p=0.192) and in hyperemic flow velocity as measured by VTI (patients 109 $\pm$ 47m vs controls, 128 $\pm$ 42m/s, p=0.138) were not significant.

CONCLUSION: We demonstrated that patients with INOCA have significantly attenuated peripheral microvascular endothelial function compared to healthy controls as assessed by pulse arterial tonometry. Our findings suggest that peripheral and coronary microvascular dysfunction coincide and thus may reflect a systemic nature of vascular dysfunction in women with INOCA.