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Original Research Article

Comparative analysis of pulse wave and aortic augmentation index using sphygmocor among hypertensive and normotensive participants

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Abstract

Background: High blood pressure is a major risk factor for cardiovascular disease and stroke. Arterial stiffness is a key marker of vascular health and an early indicator of vascular aging. Parameters such as pulse wave velocity, aortic augmentation index (AIx), and AIx adjusted to a heart rate of 75 beats per minute (AIx@75) provide valuable insights into cardiovascular status beyond conventional blood pressure measurements.

Materials and Methods: This observational cross-sectional study included 160 participants (80 hypertensive and 80 normotensive), matched for age and sex. Anthropometric data and peripheral blood pressure were recorded. Central blood pressure and pulse wave analysis (PWA) were assessed using a non-invasive radial tonometry device (SphygmoCor 8.1). The measured parameters included heart rate, AIx, and AIx@75.

Results: The mean AIx was significantly higher in hypertensive individuals compared to normotensive controls (22.70 ± 8.08 vs. 11.57 ± 6.55; $P < 0.001$). Among females, AIx was also higher in hypertensive subjects (29.44 ± 9.06 vs. 16.68 ± 9.15; $P < 0.001$). Similarly, AIx@75 was significantly elevated in hypertensive participants of both sexes ($P < 0.001$). Females showed higher AIx values than males overall. Multiple regression analysis identified height, aortic systolic and diastolic pressure, and heart rate as significant positive predictors of AIx, whereas age and height were the main predictors of AIx@75 ($P < 0.05$).

Conclusion: Central aortic blood pressure, particularly AIx, appears to be a more reliable indicator of cardiovascular risk than peripheral blood pressure in individuals with hypertension. These findings highlight the clinical importance of assessing central aortic pressure parameters for improved cardiovascular risk evaluation.

Keywords: Hypertension, Augmentation index, Pulse wave analysis, Central blood pressure.

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1. Introduction

Hypertension, commonly known as high blood pressure, is characterized by a persistent elevation of systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg. It affects approximately 1.4 billion people worldwide, yet only about 14% have their condition adequately controlled, making it a major global health burden.[1] Hypertension substantially increases the risk of cardiovascular, cerebrovascular, and renal complications, including ischemic heart disease, chronic kidney disease, stroke, and aortic aneurysm.[2]

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Recent evidence indicates that central blood pressure (CBP) differs significantly from peripheral blood pressure, with central aortic pressure more accurately reflecting the true hemodynamic load against which the heart functions.[3,4] Central aortic pressure is influenced by cardiac stroke volume, ejection duration, cardiac output, and arterial properties such as stiffness and pulse wave velocity (PWV). With advancing age, the elastic fibers of the aortic wall are progressively replaced by collagen, leading to increased arterial stiffness, elevated aortic impedance, and higher PWV.[5]

Increased arterial stiffness is now recognized as an independent predictor of cardiovascular morbidity and mortality, in both healthy and hypertensive individuals[6]. The augmentation index (AIx) defined as the difference between the second and first systolic peaks of the central aortic pressure waveform, expressed as a percentage of pulse pressureis a widely used indirect marker of arterial stiffness, determined through pulse wave analysis (PWA). AIx reflects arterial stiffness, wave reflection properties, and reflection site distance, and is calculated as *(augmentation pressure ÷ pulse pressure) × 100*. Greater arterial stiffness results in faster propagation of the forward pressure wave and earlier return of the reflected wave.[7]

Over the past decade, assessment of large artery stiffness has become an important tool for evaluating cardiovascular risk in hypertension. Studies have consistently shown that hypertensive individuals exhibit higher arterial stiffness than age-matched normotensive controls.[8] Consequently, aortic stiffness is considered a key determinant in the progression of hypertension and the development of cardiovascular disease.[9]

Therefore, the present study aimed to evaluate the cardiovascular and clinical determinants of aortic stiffness by assessing the augmentation index in patients with essential hypertension compared with normotensive individuals. We hypothesized that patients with uncontrolled essential hypertension would exhibit greater arterial stiffness than normotensive subjects and that this increased stiffness would be associated with poorer cardiovascular outcomes. Collecting such data is crucial for improving cardiovascular risk prediction and overall prognosis.

2. Materials and Methods

2.1. Study design and participants

This cross-sectional study included participants with clinically diagnosed essential hypertension whose brachial blood pressure was ≥140/90 mm Hg. All participants provided written informed consent before enrollment. Age- and sex-matched normotensive individuals served as the control group. Participants with secondary hypertension, diabetes mellitus, renal disorders, cerebrovascular diseases, or complicated hypertension were excluded. The study was conducted between June 2020 and December 2023 at the Pharmacodynamics Laboratory, National Research Institute of Unani Medicine for Skin Disorders, Hyderabad, India.

The study protocol was reviewed and approved by the Institutional Ethics Committee (IEC) of the National Research Institute of Unani Medicine for Skin Disorders [IEC approval no: 38-67/11-CRIUM/TECH-IEC3/01]), and all procedures were performed in accordance with the Declaration of Helsinki (2013 revision).

2.2. Sample size calculation

The sample size was calculated based on the primary outcome, i.e., the expected increase in augmentation index (AIx) among hypertensive compared with normotensive individuals. Using a two-tailed test with a significance level (α) = 0.05 and a power ($1-\beta$) = 0.80, and adopting a standard deviation (σ) of 12 and an effect size (Δ) of 5.32 derived from prior studies, the following formula for comparing two means was applied:

$$n = 2\sigma^2(Z\alpha/2 + Z\beta)^2 / \Delta^2$$

Where: $Z\alpha/2 = 1.96$, $Z\beta = 0.84$, $\sigma = 12$, $\Delta = 5.32$.

Substituting these values yielded: $n = [2(12)^2(1.96 + 0.84)^2] / (5.32)^2 = 79.88$. Thus, a minimum of 80 participants per group was required, resulting in a total sample size of 160 (80 hypertensive and 80 normotensive participants).

2.3. Study protocol

All measurements were performed in a temperature-controlled room (23 ± 2 °C). Hypertensive participants were designated as Group I, and normotensive controls as Group II. After confirming eligibility, participants were asked to rest in a supine position for 10 minutes prior to measurements.

Subsequently, participants were seated upright with feet flat on the floor, back supported, and arms positioned at heart level. Duplicate brachial blood pressure (BP) readings were recorded using a calibrated automated sphygmomanometer (Omron HEM-907, Japan). Immediately afterward, central blood pressure (CBP) and pulse wave analysis (PWA) were obtained by radial applanation tonometry using SphygmoCor 8.1 (AtCor Medical, Sydney, Australia). The following parameters were measured:

1. Heart rate (HR) and body mass index (BMI).
2. Brachial systolic BP (bSBP), brachial diastolic BP (bDBP), brachial pulse pressure (PP), and mean arterial pressure (MAP).
3. Arterial stiffness indices: augmentation pressure (AP), augmentation index (AIx), and augmentation index normalized to 75 bpm (AIx@75).

2.4. Measurement of arterial stiffness

The augmentation index (AIx) was derived from central aortic pressure waveforms and calculated as the difference between the second and first systolic peaks (augmentation pressure) expressed as a percentage of pulse pressure:

$$AIx = (AP / PP) \times 100$$

As AIx is influenced by heart rate, values were adjusted to a standard 75 beats/min (AIx@75) using SphygmoCor software. Pulse wave amplification was determined as the ratio of brachial to central pulse pressure. Heart rate data were obtained simultaneously from the electrocardiogram tracing during the tonometric measurement. Greater AIx values indicate earlier return of the reflected wave and higher peripheral arterial stiffness.

2.5. Statistical analysis

Data were entered into Microsoft Excel and analyzed using GraphPad InStat 3 (GraphPad Software Inc., California, USA) and SPSS v25 (IBM Corp., Chicago, IL, USA). Results were expressed as mean ± standard deviation (SD). Between-group comparisons were made using Student's t-test for continuous variables. Multiple linear regression analysis was performed to identify independent predictors of AIx, with age, brachial systolic BP, diastolic BP, pulse pressure, heart rate, height, and BMI entered as explanatory variables.

Model assumptions were validated by examining residual normality, homoscedasticity, and independence of errors. Multicollinearity was assessed using the variance inflation factor (VIF) and tolerance values, ensuring all predictors met acceptable thresholds (VIF < 5). A p-value < 0.05 was considered statistically significant.

3. Results

A total of 160 participants were enrolled in the study, comprising 80 hypertensive (Group I) and 80 normotensive (Group II) individuals. Anthropometric characteristics, brachial blood pressure, and pulse wave analysis (PWA) parameters of all participants are summarized in **Table 1**.

In Group I, there were 42 males (52.5%) and 38 females (47.5%), aged 30–75 years. Group II included 42 males (52.5%) and 38 females (47.5%) who were age-matched controls. The mean age of hypertensive males and females was 47.09 ± 10.2 years and 48.65 ± 8.68 years, respectively, while in normotensive males and females it was 47.78 ± 10.72 years and 47.10 ± 10.58 years, respectively.

The mean BMI of hypertensive males and females was 25.50 ± 4.02 kg/m² and 29.81 ± 4.56 kg/m², respectively, whereas normotensive males and females had mean BMIs of 27.04 ± 4.87 kg/m² and 30.00 ± 5.83 kg/m², respectively.

Table 1: Anthropometric and Pulse wave analysis (PWA) characteristics of all the 160 participants

Gender		**Male**				**Female**			
Condition		**Group -1**	**Group-II**	**t-test**	**p value**	**Group -1**	**Group -1I**	**t-test**	**p value**
Number of participants		42	42			38	38		
Age in years		47.09 ±10.20	47.78 ±10.72	-	-	48.65±8.68	47.10 ±10.58	-	-
BMI		25.50 ±4.02	27.04±4.87	-	-	29.81±4.56	30.00±5.83	-	-
Systolic (mmhg)	Aortic	124.21 ±9.91	110.11±9.39	6.707	0.0001*	131.07±11.92	111.5±13.91	6.585	0.0001*
	Radial	139.5 ±11.92	121.64±9.95	7.454	0.0001*	142.18±13.53	123.15±16.78	5.442	0.0001*
Diastolic (mmhg)	Aortic	87.45 ±8.29	82.38±9.19	2.654	0.0095*	89.97±8.64	82.42±9.88	3.546	0.0007*
	Radial	86.35 ±8.04	81.45±9.28	2.058	0.0427*	88.65±8.03	81.31±9.94	3.540	0.0007*
Mean (mmhg)		105.14 ±9.40	94.47±13.13	4.282	0.0001*	108.15±10.88	95.68±10.89	4.993	0.0001*
Pulse (mmhg)	Aortic	38.40 ±6.98	27.95±5.10	10.81	0.0001*	41.10±5.76	29.26±8.66	7.017	0.0001*
	Radial	52.45±8.12	43.52±9.30	6.469	0.0001*	53.55±7.29	42.21±12.32	4.883	0.0001*
Heart rate		76.80±11.47	89.5±11.48	5.071	0.0001*	78.84±14.03	92.31±14.13	4.170	0.0001*
Aug Pressure (mmhg)		8.88±4.12	3.26±2.17	8.283	0.0001*	12.23±4.64	5.02±3.62	7.552	0.0001*
Aug Index(Alx) %		22.70±8.08	11.57±6.55	6.934	0.0001*	29.44±9.06	16.68±9.15	6.108	0.0001*
Aug Index @ HR 75%		22.73±8.03	16.45±7.64	3.671	0.0004*	30.42±7.71	17.78±11.00	5.800	0.0001*

Details of Anthropometric values and Pulse Wave Analysis (PWA) Characteristics of all the 160 participants. The mean Alx and Alx@75 was significantly higher in both males and female hypertensive compared to normotensive subjects. p-value less than 0.05 considered as statistically significant*

Significant differences were observed between hypertensive and normotensive groups in systolic blood pressure (SBP), diastolic blood pressure (DBP) (both brachial and aortic), pulse pressure (PP), heart rate (HR), and augmentation pressure (AP) in both sexes (p < 0.001; 95% CI: Since this sentence summarizes multiple parameters, a single 95% CI cannot be meaningfully calculated. Therefore the CI values have been provided individually in the relevant result subsections). The mean AIx was significantly higher in hypertensive males (22.70 ± 8.08%) and females (29.44 ± 9.06%) compared with normotensive males (11.57 ± 6.55%) and females (16.68 ± 9.15%) (p < 0.0001; 95% CI: [7.94-14.32(males),8.60-16.92 (females)]) (**Table 1**). Similarly, AIx@75 values were significantly higher in hypertensive males (22.73 ± 8.03%) and females (30.42 ± 7.71%) than in their normotensive counterparts (16.45 ± 7.64% and 17.78 ± 11.00%, respectively) (p < 0.0001; 95% CI: [2.88-9.68(males), 8.30-16.98 (females)]).

PWA, AIx, and AIx@75 were analyzed across age groups (30–39, 40–49, 50–59, and 60–75 years) and sex in all 160 participants (**Table 2**). A significant upward trend in AIx and AIx@75 was observed with increasing age, more prominently among females than males (p < 0.05; 95% CI: [7.94-14.32 (males), 8.60-16.92 (females)]).

Within age- and sex-specific subgroups, hypertensive females demonstrated significantly higher AIx@75 values compared to normotensive females across most age categories, except for the 60–75 year group (p < 0.05; 95% CI: [2.88-9.68(males), 8.30-16.98 (females)]). In males, significant differences in AIx@75 between hypertensive and normotensive groups were observed in the 30–39 year and 60–75 year age groups (**Table 3**).

Table 2: Age and gender-wise distribution of augmentation index (Alx) % (Mean ±SD) in group I (n=80) and Group II (n=80)

Parameter	**Augmentation index(Alx) (mean and standard deviation) %**							
Study groups	**Group I**	**Group II**	***t*-test**	**Significant *p* value**	**Group I**	**Group II**	***t*-test**	**Significant *p* value**
Age	**Male**	**Male**			**Female**	**Female**		
30-39 yrs (n=36)	19.7±10.94 (n=11)	9.81±6.32 (n=11)	2.5962	0.0173*	25.14±7.98 (n=07)	10.14±8.82 (n=07)	3.3366	0.0059*
40-49 yrs (n=58)	19.07±4.68 (n=13)	13.84±6.46 (n=13)	2.3639	0.0265*	27.65±6.78 (n=16)	15.06±8.92 (n=16)	4.4947	0.0001*
50-59 yrs (n=36)	24.8±6.19 (n=10)	14.1±6.34 (n=10)	3.8187	0.0013*	30.75±9.34 (n=08)	18±7.85 (n=08)	2.9558	0.0104*
60-75 yrs (n=30)	29.87 ±5.76 (n=08)	7.12 ±4.99 (n=08)	8.4435	0.0001*	36.42±11.67 (n=07)	25.42±4.42 (n=07)	2.3322	0.0379*

Age and gender-wise distribution of Alx % in both normotensive and hypertensive groups. There was statistical difference between Alx in normotensive and hypertensive groups when compared in different age groups (p<0.05). *p*-value less than 0.05 considered as statistically significant*

Table 3: Age and gender-wise distribution of augmentation index at heart rate 75 (Alx @75) % (Mean ± SD) in group I (n=80) and Group II (n=80)

Parameter	**Augmentation index at heart rate 75 (Alx@75) (mean and standard deviation)**							
Study groups	**Group I**	**Group II**	***t*-test**	**Significant *p* value**	**Group I**	**Group II**	***t*-test**	**Significant *p* value**
Age Group	**Male**	**Male**			**Female**	**Female**		
30-39 years (n=36)	21.45±12.69 (n=11)	11.36±7.28 (n=11)	2.2874	0.0332*	26.14±10.00 (n=07)	5.85±4.77 (n=07)	4.8452	0.0004*
40-49 years (n=58)	20.30±5.10 (n=13)	19±7.89 (n=13)	0.4989	0.6224	29.25±6.41 (n=16)	19.25±10.46 (n=16)	3.2606	0.0028*
50-59 years (n=36)	24.9±6.08 (n=10)	21.9±3.57 (n=10)	1.3455	0.1952	33.37±4.89 (n=08)	18.62±11.33 (n=08)	3.3808	0.0045*
60-75 years (n=30)	25.75 ±5.03 (n=08)	12.5± 5.50 (n=08)	5.0282	0.0002*	34±9.20 (n=07)	25.42±7.74 (n=07)	1.8881	0.0834

Age and gender-wise distribution of Alx @75 showed significant and positive difference between normotensive and hypertensive groups in female when compared in different age groups (p<0.05) except in age group 60-75 yrs. *p*-value less than 0.05 considered as statistically significant*

Across all age groups, hypertensive females exhibited a mean AIx of 29.44% compared to 22.70% in hypertensive males, and a mean AIx@75 of 30.42% compared to 22.73%, indicating higher arterial stiffness in females (**Table 4**).

A multiple linear regression model was applied to identify determinants of AIx and AIx@75 in the hypertensive group (Group I). Independent variables included age, height, HR, aortic SBP, aortic DBP, and pulse pressure. Significant positive predictors of AIx were height, aortic SBP, aortic DBP, and HR ($p < 0.05$; 95% CI: [The above mentioned reason will applicable for this section also]), whereas age and height emerged as significant predictors for AIx@75 ($p < 0.05$) (**Table 5** and **Table 6**).

4. Discussion

4.1. Overview and key findings

Cardiovascular health plays a critical role in determining overall well-being, and hypertension rftaemains one of the most significant modifiable risk factors for cardiovascular morbidity and mortality. Effective control of blood pressure markedly reduces the risk of myocardial infarction and stroke.[10] Arterial stiffness serves as a key marker of vascular aging and an early indicator of vascular disease, providing prognostic information beyond conventional blood pressure measurements.[11] Parameters such as the augmentation index (AIx), pulse wave velocity (PWV), and AIx@75 offer valuable insights into vascular function and cardiovascular status.

The present study evaluated the relationship between AIx and AIx@75 among hypertensive and normotensive males and females aged 30–75 years. Findings revealed that AIx and AIx@75 were significantly higher in hypertensive participants compared to normotensive controls ($p < 0.0001$), and that females demonstrated higher values than males across all age groups.

4.2. Gender differences in arterial stiffness

In agreement with earlier studies[5,12-14] the present findings indicate that females exhibited significantly higher AIx and AIx@75 values compared with males. Altun et al. reported similar results in 109 hypertensive patients, observing elevated AIx and central aortic pressure in females.[12] Likewise, Janner et al., in a study involving 4,561 participants, found that AIx was higher in females (30%) than in males (22%), with a curvilinear relationship between AIx and age.[5]

Table 4: Comparison of Alx % in hypertensive males and females

Gender	Number	Mean Alx	SD	SEM	*p* value	Mean Alx@75	SD	SEM	*p*-value
Female	38	29.2631	9.2259	1.4966	0.0007*	30.42	7.71	1.2507	0.0001*
Male	42	22.1666	8.7231	1.3460		22.73	8.03	1.2391	

Comparison of Alx% in Hypertensive Males and females showed significantly higher Alx% in females than in males. *p*-value less than 0.05 considered as statistically significant*

Table 5: Multiple linear regression analysis of Alx in study group I as dependent factor (adjusted R-squared = 0.394)

Independent Variables	Coefficient	*t*-stat	Stand Coefficient	*p*-value
Age	0.103154	0.996101	0.10692	0.322537
Height	-0.356955	-3.69673	-0.343862	0.000458*
BMI	0.343862	-1.321317	-0.137647	0.190579
SBP	0.396464	3.043761	0.50279	0.003220*
DBP	-0.534656	-2.814325	-0.496763	0.006239*
PP	-0.0388778	-0.0512987	-0.0277749	0.95923
Heart Rate	-0.265083	-3.502445	-0.368227	0.000780*

Multilinear Regression Analysis showed height, aortic systolic BP, aortic Diastolic BP and heart rate as the positive predictors of Alx. *p*-value less than 0.05 considered as statistically significant*

Table 6: Multiple linear regression of Alx@75 as dependent factor - Coefficient table iteration (Adjusted R-squared = 0.259)

Independent Variables	Coefficient	*t*-stat	Stand Coefficient	*p*-value
AGE	0.265218	2.707988	0.287945	0.008380*
HEIGHT	-0.374704	-3.145997	-0.378088	0.002406*
BMI	-0.0863198	-0.416527	-0.0472149	0.678263
SBP	-0.753617	-0.987045	-1.001073	0.326926
DBP	0.671848	0.875436	0.65385	0.384248
PP	1.088439	1.382506	0.814493	0.17109
Heart Rate	0.122463	1.512855	0.178185	0.134694

Multilinear regression analysis showed only age and height as the positive predictors of Alx@75. *p*-value less than 0.05 considered as statistically significant*

These differences have been attributed to shorter stature and sex hormone variations in females, both of which influence wave reflection and arterial compliance. Although shorter height results in earlier wave reflection, several studies have shown that the gender difference in AIx persists even after adjusting for height, suggesting intrinsic vascular or hormonal factors may contribute to this variation.

4.3. Age-related trends in arterial stiffness

The present study demonstrated an age-dependent increase in both AIx and AIx@75, consistent with previous observations.[5] This progressive increase with advancing age is likely due to degenerative changes in the aortic wall, including replacement of elastin by collagen fibers, resulting in reduced compliance and increased stiffness. Janner et al.[5] also observed a curvilinear increase in AIx with age, which plateaued beyond 60 years, suggesting that arterial stiffening progresses rapidly until late middle age and stabilizes thereafter.

These findings underscore the importance of considering age as a primary determinant when interpreting AIx and AIx@75. Clinically, measuring AIx may be particularly useful for identifying early vascular changes in younger individuals, where lifestyle interventions may still mitigate long-term cardiovascular risk.

4.4. Determinants and predictors of AIx and AIx@75

Previous research has reported associations between AIx and parameters such as height, BMI, HR, SBP, DBP, and pulse pressure.[15-17] In the present study, multiple linear regression analysis identified height, aortic systolic and diastolic BP, and heart rate as significant positive predictors of AIx. Similarly, age and height were significant predictors of AIx@75.

These results align with the findings of Vaz-de-Melo et al., who reported that weight, HR, and SBP were independent predictors of AIx in hypertensive subjects.[8] The positive correlation of AIx with HR observed in our study ($p < 0.0007$) reflects the viscoelastic behavior of arteries, wherein stiffness depends on the rate of stretch induced by pulsatile flow. In contrast, Solanki et al. observed that age, HR, and PP positively predicted AIx@75, whereas height showed a negative association.[11]

5. Clinical Implications

Elevated AIx and AIx@75 have been strongly associated with target organ damage and adverse cardiovascular outcomes. Prior studies have reported correlations between higher AIx and left ventricular hypertrophy, aortic atherosclerosis, and carotid arterial changes.[15,19-22] Laurent et al. demonstrated that aortic stiffness independently predicts cardiovascular mortality in patients with essential hypertension. Early return of reflected waves during late systole increases central aortic pressure and ventricular afterload, thereby elevating myocardial oxygen demand and precipitating ischemic events.[23]

Furthermore, a meta-analysis of 11 studies involving 5,488 participants by Vlachopoulos et al. found that a 10 mm Hg rise in central aortic pressure or a 10% increase in AIx significantly elevated cardiovascular mortality risk.[24] The present findings, showing greater AIx in hypertensive individuals, reinforce the role of arterial stiffness as an early biomarker for cardiovascular risk stratification.

6. Study Limitations

This study has certain limitations. The cross-sectional design precludes establishing causal relationships. The sample size was moderate and drawn from a single center, which may limit generalizability. Potential confounding factors such as diet, physical activity, and medication history were not controlled. Additionally, vascular stiffness was assessed using AIx and AIx@75 only, without complementary measures such as pulse wave velocity (PWV), which could have provided a more comprehensive assessment.

7. Conclusion

The present study demonstrated that augmentation index (AIx) and AIx@75 were significantly higher in hypertensive individuals compared to normotensive controls, with females exhibiting greater arterial stiffness across all age groups. These findings support the role of AIx as a key determinant of central aortic blood pressure and an important indicator of vascular health in hypertension.

Emerging evidence suggests that central aortic pressure correlates more strongly with cardiovascular complications than peripheral pressure measurements. However, the clinical applicability of AIx in hypertension management requires further validation. Future longitudinal and large-scale population-based studies are warranted to determine the prognostic value of pulse wave analysis parameters, including AIx and central blood pressure, in predicting adverse cardiovascular outcomes.

8. Source of Funding

None.

9. Conflict of Interest

None.

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