# Interatrial Block: A Novel Risk Factor for Embolic Stroke?

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**Background:** Interatrial block (IAB; P wave ≥110 ms) is highly prevalent and is strongly associated with atrial tachyarrhythmias and left atrial dysfunction, making it a potential embolic risk.

**Methods and Results:** Among 293 neurological admissions over 2 years, 85 patients were diagnosed with embolic strokes and 208 with nonembolic strokes. Patients were then matched for stroke risk factors and evaluated for IAB. Eighty-eight percent of probable embolic stroke patients showed sinus rhythm, demonstrating a 61% IAB prevalence. Only hypertension ($P < 0.001$; $r = 0.3$) and IAB ($P < 0.006$; $r = 0.2$) were significant and directly correlated.

**Conclusion:** IAB could indeed be a novel risk for embolic strokes and further investigation is warranted.

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interatrial block, left atrial dysfunction, atrial fibrillation, embolic stroke, risk

## BACKGROUND

Interatrial block (IAB) signifies delayed impulse conduction between the right and left atrium (LA).[1] (On the electrocardiogram [ECG], time = duration of conduction; excessive time or delay = block).

While normal P-wave duration, as classified by the World Health Organization/International Society and Federation of Cardiology Task Force, is <110 ms,[2] P waves in IAB are prolonged (≥110 ms) with a resultant LA-activation lag; notched in ≥1 ECG leads.[1] Besides being a potent predictor of atrial tachyarrhythmias, especially atrial fibrillation (AF),[3,4] IAB is also correlated with LA enlargement (LAE) with 88% of its patients depicting this echocardiographically.[1,5,6] Not surprisingly, IAB is associated with significant LA electromechanical dysfunction (8.5% LA emptying fraction and 19.8 kdyne/cm per second LA kinetic energy compared to 24.6% and 64.7 kdyne/cm per second, respectively, in LA size-matched controls; $P < 0.0001$).[7] Therefore, blood stasis in a sluggish, poorly contractile LA is hypothesized to be a thrombotic risk that may manifest as embolic strokes. However, while exact mechanisms of potential IAB-mediated stroke are unclear, Lorbar et al.[8] recently observed an 80% IAB prevalence (almost twice the hospital prevalence) among such patients.

## METHODS

We reviewed medical records of patients from the neurology unit (ages 30–102 years; mean = 73.6 years) of a tertiary care hospital (Saint Vincent Hospital, Worcester, MA) who had been investigated between January 2003 and December 2004 via computer-assisted tomography (CT) or magnetic resonance imaging (MRI) for suspicion of stroke. Of 293 consecutive patients identified with ischemic (nonhemorrhagic) strokes, only 85 (mean age = 73.8 years; 47% female) had definitive new (acute or subacute) cerebral infarcts of probable embolic origin diagnosed via such imaging after neurologists' evaluation consistent with

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American Heart Association (AHA) guidelines and per the outlines of the Lausanne Stroke Registry and modified Oxfordshire Community Stroke Project criteria.[9,10] Embolism was preliminarily cited as probable cause in each patient based on clinical history and presentation (sudden onset, initial maximal symptoms, and/or subsequent rapid recovery, among others) as well as newly evidenced lesion size, numbers, anatomical cerebrovascular location, and exclusion of deep-penetrating vessel involvement in the absence of other etiologies prior to further investigation, such as Doppler ultrasonographic evaluations[9] (Table 1).

Seventy-five of the 85 patients with probable embolic stroke and 153 of the 208 patients with nonembolic stroke (atherothrombotic and small-vessel disease) who had 12-lead ECGs recorded within 14 days (0.04–11.6 days; mean = 1.8 days) of their respective CT or MRI evaluations formed our study sample. Both groups were then matched for common medical comorbidities and most of the well- and less well-documented stroke risk factors (nonmodifiable, modifiable, or potentially modifiable) per AHA Stroke Council classification (Table 2).[9] Comorbidites and risk factors had been documented in patients' records by physicians involved in their care and were consistent with classifications and guidelines for disease definition and diagnosis outlined by American College of Cardiology/AHA/American College of Physicians–American Society of Internal Medicine Task Force on Practice Guidelines.[11]

ECGs were recorded from a Marquette-2000 electrocardiograph (Marquette Electronics Incorporated, Milwaukee, WI) standardized at 25 mm/s and 10 mm/mV. IAB was diagnosed on a single read by each investigator using the greatest P-wave duration among all 12 leads measured with standard calipers under 10-fold magnification (reproducibility and interobserver concordance >95%).[12] To increase specificity and because 1 mm represents 40 ms under such ECG standardization, 120 ms (which is also the mode duration)[13] was used as our diagnostic criterion for IAB. The P wave onset was defined as the junction between the isoelectric T-P baseline and the beginning of the P deflection while the offset was defined as the junction between the end of the P deflection and PR segment.

Group differences were assessed with chi-square statistics for categorical variables and analysis of variance for continuous variables on SPSS Version 10.0 statistical software (SPSS Incorporated, Chicago, IL). Odds ratios (ORs), 95% confidence intervals (CIs), P values (<0.05 considered significant), and Pearson correlation coefficients (r values) were calculated.

## RESULTS

From the sample (N = 228; mean age ± SD = 73.8 ± 13.87 years; female 48.2%), 49 patients (21.5%) had history of prior strokes (with infarcts on previous CT or MRI) or transient ischemic attacks (TIAs); none hemorrhagic. Of those, 10 were patients with probable embolic strokes (13.3%; 3 had old lesions in the anatomical cerebrovascular region of current infarcts, 6 had old lesions at unrelated sites, and 1 had past TIA) and 39 were patients with nonembolic strokes (25.5%; 25 had old lesions in the anatomical cerebrovascular region of current infarcts, 5 had old lesions at unrelated sites, and 9 had past TIAs).

Overall, hypertension (HTN), coronary artery disease (CAD), diabetes mellitus (DM), hypercholesterolemia, dilated cardiomyopathy, chronic obstructive pulmonary disease (COPD), and history of prior strokes or TIAs were prevalent comorbidities between the groups for all rhythms (Table 2). Among these, only HTN was significant for embolic strokes in this cohort while COPD and history of TIAs or strokes were not (Table 2). Sixty-six of the 75 patients with probable embolic stroke (88%) and 133 of the 153 patients with nonembolic stroke (86.9%) showed sinus rhythm on respective ECGs. The prevalence of IAB among probable embolic stroke patients was 60.6% and among nonembolic stroke patients was 39.8% (46.7% overall hospital prevalence; Table 3). Only HTN (OR 3.235, 95% CI 1.73–6.03; $P < 0.0001$; $r = 0.3$) and IAB (OR 2.32, 95% CI 1.27–4.24; $P = 0.006$; $r = 0.2$) were direct correlates and significant risks for embolic strokes during sinus rhythm (Table 3).

## DISCUSSION AND LIMITATIONS

Our investigation of probable embolic stroke patients preliminarily diagnosed based on MRI and/or CT findings by both attending radiologists and neurologists before subsequent evaluations suggests that IAB could be a risk for such events. However, while IAB's correlation indeed appears weak ($r = 0.2$), it should be noted that HTN, an AHA established, well-documented, and potent stroke risk factor, is similarly depicted here ($r = 0.3$).[9] HTN is

**Table 1.** Common Criteria Used for Preliminary Diagnosis of Probable Embolic Stroke by Radiologists and Neurologists in Study Sample[a]

| |
|---|
| (1) Nature of presentation on admission and/or hospitalization history: |
| Patient demographics (increasing age, female sex, Caucasian race, etc.)[b] |
| Symptoms (headache, dizziness, blurring or decreased vision in one or both eyes, loss of balance or coordination and/or in combination with other symptoms, weakness, numbness, paralysis of the face, arm, or leg, commonly confined to one side of the body, etc.)[c] |
| Sudden onset of symptoms, with severity maximal at onset with or without subsequent rapid recovery |
| Decreased level of consciousness at onset of event or upon presentation to hospital |
| Association of seizure in relation to supratentorial cortical involvement and focal neurological presentation (although infrequent at onset and more common after gliotic scar formation)[d] |
| Symptoms corresponding to the involvement of different/multiple focal cerebral areas |
| Clinical evidence or signs established with neurologic testing suggesting focal neurological involvement consistent with lesions in multiple cereberovascular territories |
| Symptomatic or clinical findings of cortical or subcortical lesion that may be localized to specific territories supplied by branches of the anterior, middle, and posterior cerebral arteries and absence of evidence of proximal vascular stenosis/occlusion |
| Specific middle cerebral artery neurologic syndromes (opercular, brachial or hand plegia, isolated Broca's or Wernicke's aphasia and left visual neglect syndromes) |
| Rapid recovery from major hemispheric deficits ("spectacular shrinking deficit" phenomenon, common with fibrinous thrombi or posttreatment/intervention) |
| Absence of lacunar syndrome |
| (2) Presence of major risk factors and/or correlates in medical history: |
| AF or atrial flutter at time of presentation[d] |
| History of atrial tachyarrhythmias |
| Past TIA |
| Past stroke |
| History of blood dyscrasias[d] |
| Predisposing coronary vascular risk factors (CAD, HTN, etc.) |
| Myocardial infarction (recent or at time of presentation) |
| Known LVD or severe congestive heart failure during event |
| Iatrogenic (cardiac or great vessel surgery, angiography, endovascular manipulation, intravascular device implantation)[d] |
| Periprocedure risk of embolism (Valsalva-provoking activity in patients with a patent foramen ovale and other right-to-left shunting causes)[d] |
| (3) Presence of new infarct on CT and/or MRI: |
| Infarct consistent with embolic lesion and/or able to be differentiated from a nonembolic ischemic lesion with or without known presenting history |
| Lesion size and extent of involvement (unlikely involving both gray and white matter; large embolic infarct however may be suggestive of cardiac source) |
| Distinguishable cortical or subcortical lesion localization to the territory supplied by the branches of the anterior, middle, and posterior cerebral arteries in the absence of evidence of proximal vascular stenosis/occlusion (beyond main branches) |
| Radiographic evidence of embolic infarcts pertaining to multiple vascular territories or to the gray–white matter junction |
| In the lenticulostriate or thalamogeniculate territories, lesions $>1$ cm suggestive of embolism |
| Medial subthalamic and thalamic involvement suggestive of basilar artery lesion with corresponding symptoms (sudden sleepiness, impaired upward gaze, and bilateral ptosis) |
| Cerebellar hemispheric infarction consistent with presenting symptoms |
| Sparing involvement of the deep-penetrating arteries (although microemboli from valvular sources can occlude small penetrating arteries)[d] |

[a]Prior to the identification of a possible embolic source and in the absence of other etiologies at admission.
[b]Per outlines of the Lausanne Stroke Registry criteria.
[c]Modified Oxfordshire Community Stroke Project criteria.
[d]Excluded from study or were not factors among selected patients in sinus rhythm.

also closely correlated with AF[11] and less surprisingly, IAB;[14] the former already a known culprit in embolic strokes.[9,11] The arrhythmogenic propensity of IAB was noted in one 16-month follow-up investigation where IAB was shown to be prevalent in 52% of those patients who subsequently developed AF compared to 18% among controls ($P < 0.0001$).[3] Indeed, the unremarkable prevalence of a history of

AF or atrial flutter in this sample therefore appears unusual. However, it is uncertain if IAB patients sporadically flit in and out of atrial tachyarrhythmias, i.e., IAB is a precursor of both paroxysmal and persistent AF. This confers some credence to the hypothesis of such arrhythmia-mediated embolic phenomena in IAB despite an overall low AF prevalence.

Furthermore, we had grouped AF and atrial flutter as a single variable and this could have contributed to the insignificant numbers collectively. We had also not noted premature atrial contractions on our ECGs. Holter monitoring would surely have been the best method to detect such events and, particularly, the presence of any potentially contributive supraventricular tachyarrhythmias. In left ventricular dysfunction (LVD), especially during acute congestive heart failure or postmyocardial infarction, a poorly motile ventricle is often the harbinger for thrombus and subsequent embolism.[9,15] In parallel, given IAB's association with LAE and LA dysfunction, IAB may therefore act as a nidus for LA thrombosis and emboli propagation.[7,8] However, presently neither one of the IAB's modi operandi for embolism is clear.

We studied preliminarily evaluated neurological patients before subsequent investigations such as echocardiography, Doppler ultrasonography, and magnetic resonance angiography (Table 1). Our objective thus was clearly neither to unmask the underlying cause-and-effect mechanisms of how IAB contributes as a risk nor to trace embolic sources.[9] Furthermore, regardless if thrombi and/or spontaneous contrast ("smoke") are noted in such dysfunctional, enlarged LA echocardiographically, due to its arrhythmogenic predilection itself, IAB could still contribute to such strokes via tachyarrhythmia-mediated extracardiac embolism (precerebral vessels).[9,11] Failure to identify a high-risk source of embolism does not exclude an embolic mechanism; up to 40% of all ischemic strokes remain cryptogenic.[9,10] While variables such as passive second-hand smoke, genetics, family history, obesity, blood dyscrasias, and LVD are also recognized risks, factors that we evaluated were those with the highest reported risk for strokes, especially of embolic origin (Tables 2 and 3).[9] The high prevalence of COPD among nonembolic stroke patients is a challenge to interpret, despite the absence of a significant correlation (Table 2). While there may be a potential risk of hemorrhagic stroke from steroid abuse and there is a known association of negative vascular effects with glucocorticoid use, such a relationship between steroid-dependent

**Table 2.** Embolic versus Nonembolic Stroke in All Patients (n = 228)

| Variables | Embolic Stroke (n = 75) | Nonembolic Stroke (n = 153) | P |
|---|---|---|---|
| Age (mean ± SD; years) | 73.5 ± 13.32 | 73.9 ± 14.17 | 0.827 |
| Sex | | | |
| Male | 40 (53.3%) | 78 (51.0%) | 0.779 |
| Female | 35 (46.7%) | 75 (49.0%) | |
| Mitral stenosis | 3 (4.0%) | 2 (1.3%) | 0.192 |
| Mitral insufficiency | 4 (5.3%) | 3 (2.0%) | 0.165 |
| Dilated CM | 18 (24%) | 42 (27.5%) | 0.578 |
| Restrictive CM | 3 (4%) | 3 (2%) | 0.366 |
| Hypertrophic CM | 3 (4%) | 6 (3.9%) | 0.977 |
| Currently in nonsinus rhythms | 9 (12%) | 20 (13.1%) | 0.819 |
| Current smoker | 8 (10.7%) | 12 (7.8%) | 0.479 |
| Ex-smoker (quit ≥5 years) | 5 (6.7%) | 12 (7.8%) | 0.751 |
| Hyperthyroidism | 2 (2.7%) | 4 (2.6%) | 0.982 |
| Hypothyroidism | 6 (8.0%) | 10 (6.5%) | 0.684 |
| Hypercholesterolemia | 30 (40%) | 64 (41.8%) | 0.792 |
| HTN | 53 (70.7%) | 71 (46.4%) | 0.001 |
| COPD | 18 (24.0%) | 57 (37.3%) | 0.045 |
| CAD | 26 (34.7%) | 48 (31.4%) | 0.618 |
| DM | 21 (28%) | 35 (22.9%) | 0.398 |
| Past AF/flutter | 8 (10.7%) | 16 (10.5%) | 0.961 |
| Past strokes/TIAs | 10 (13.3%) | 39 (25.5%) | 0.036 |

**Table 3.** Embolic versus Nonembolic Stroke among Sinus Rhythm Patients (n = 199)

| Variables | Embolic Stroke (n = 66)[a] | Nonembolic Stroke (n = 133)[a] | P | OR | 95% CI | r |
|---|---|---|---|---|---|---|
| Age (mean ± SD; years) | 74.1 ± 13.77 | 73.2 ± 14.19 | 0.675 | | | |
| Sex | | | 0.574 | | | |
| Male | 39 (59.1%) | 73 (54.9%) | | | | |
| Female | 27 (40.9%) | 60 (45.1%) | | | | |
| Mitral stenosis | 2 (3.0%) | 2 (1.5%) | 0.470 | | | |
| Mitral insufficiency | 2 (3.0%) | 2 (2.3%) | 0.742 | | | |
| Dilated CM | 15 (22.7%) | 31 (23.3%) | 0.927 | | | |
| Restrictive CM | 3 (4.5%) | 2 (1.5%) | 0.197 | | | |
| Hypertrophic CM | 3 (4.5%) | 4 (3.0%) | 0.579 | | | |
| Current smoker | 7 (10.6%) | 9 (6.8%) | 0.348 | | | |
| Ex-smoker (quit ≥5 years) | 5 (7.6%) | 10 (7.5%) | 0.989 | | | |
| Hyperthyroidism | 2 (3.0%) | 3 (2.3%) | 0.742 | | | |
| Hypothyroidism | 6 (9.1%) | 8 (6%) | 0.424 | | | |
| Hypercholesterolemia | 24 (36.4%) | 53 (39.8%) | 0.635 | | | |
| IAB* | 40 (60.6%) | 53 (39.8%) | 0.006 | 2.32 | 1.27–4.24 | 0.2 |
| HTN | 45 (68.2%) | 53 (39.8%) | <0.0001 | 3.235 | 1.73–6.03 | 0.3 |
| COPD | 15 (22.7%) | 47 (35.3%) | 0.076 | 0.538 | 0.27–1.06 | |
| CAD | 20 (30.3%) | 36 (27.1%) | 0.633 | 1.171 | 0.61–2.24 | |
| DM | 19 (28.8%) | 30 (22.6%) | 0.337 | 1.388 | 0.71–2.71 | |
| Past AF/flutter | 2 (3%) | 4 (3%) | 0.993 | 1.008 | 0.18–5.65 | |
| Past strokes/TIAs | 9 (13.6%) | 33 (24.8%) | 0.069 | 0.478 | 0.27–1.07 | |

*The total frequency for IAB is 93 (46.7%).
TIA = transient ischemic attack; DM = diabetes mellitus; SD = standard deviation; IAB = interatrial block; HTN = hypertension; CI = confidence intervals; CAD = coronary artery disease; CM = cardiomyopathy; OR = odds ratio; AF = atrial fibrillation; COPD = chronic obstructive pulmonary disease; CT = computer-assisted tomography; MRI = magnetic resonance imaging.

COPD patients and nonhemorrhagic strokes is not known.[16] However, prospectively evaluated data from 12,878 patients in the Copenhagen City Heart Study revealed an inverse relationship between forced expiratory volume in one second (FEV1) with stroke (38% ischemic and 51% unspecified strokes) and a 30% higher risk in the group with the lowest compared to the highest FEV1.[17]

The 60.6% IAB prevalence among probable embolic stroke patients is high and particularly significant when compared to the study conducted at this very hospital in 2001, where among 1000 hospitalized patients, IAB was more common in those aged >60 years and had an overall prevalence of 41.1%.[12] Although that study used P wave ≥110 ms for IAB definition, our study may have in fact underestimated this cohort's true IAB prevalence. Both studies used ECGs with comparable presentations but since our selection criteria endpoints were patients with CT- or MRI-evidenced infarcts, initial presentations, i.e., dyspnea, fall, TIA, or even stroke may or may not necessarily be representative of cerebrovascular outcomes from embolic events.[10] When compared to the study by Lorbar et al.,[8] our methods differed considerably. We avoided establishing criteria based on International Classification of Diseases (ICD) codes.[18] This weeded out embolic strokes from causes classified as transient cerebral ischemia, acute but ill-defined cerebrovascular disease, other and ill-defined cerebrovascular disease, and late effects of cerebrovascular disease (ICD codes 435, 436, 437, and 438 respectively), thus eliminating potentially missed, uncertain or inconclusive diagnoses. Also, because only CT- and MRI-evidenced infarcts were the basis of our study, where embolism was the leading cited etiology, most patients who could have had TIAs that may not necessarily be evident radiologically were inadvertently excluded.[10] This defined our specific target population and partly explains our lower IAB prevalence. While CT and MRI imaging indeed use different spatial radiological resolutions, the usage of these modalities is consistent with current AHA/American College of Cardiology guidelines and Saint Vincent Hospital policies to aid in the diagnosis of such cerebral infarcts. Also, although MRI and CT have different sensitivities and specificities, they are both high-yield imaging modalities with high sensitivities for detection of ischemic lesions of most clinical

stroke subtypes.[10,19] Choosing one modality over the other could have avoided possible selection bias but may instead have dramatically reduced the study sample size and further allowed for underestimation of the true impact of IAB among these patients.

## CONCLUSION

IAB could be a novel risk factor for embolic stroke and if prospective investigations indeed prove this to be so, implication for anticoagulation use should be promptly evaluated.

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