Relationship of Paroxysmal Atrial Tachyarrhythmias to Volume Overload

Assessment by Implanted Transpulmonary Impedance Monitoring

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Background—Clinical experience suggests that atrial tachyarrhythmias (ATs) are a frequent comorbidity in heart failure patients with left ventricular systolic dysfunction and that volume overload may increase AT susceptibility. However, substantiating this apparent relationship in free-living patients is difficult. Recently, certain implantable cardioverter-defibrillators provide, by measuring transpulmonary electric bioimpedance, an index of intrathoracic fluid status (OptiVol index [OI]). The goal of this study was to determine whether periods of greater intrathoracic fluid congestion (as detected by OI) correspond with increased AT event frequency.

Methods and Results—This analysis retrospectively assessed the relation between AT events and OI estimate of volume overload in patients with left ventricular systolic dysfunction and OI-capable implantable cardioverter-defibrillators. OI values were stratified into 3 levels: group 1, <40; group 2, 40 to 60; and group 3, >60. An OI threshold-crossing event was defined as OI$\geq$60, a value previously associated with clinically significant volume overload. Findings in 59 patients (mean left ventricular ejection fraction, 24%) with 225 follow-up visits (mean, 3.8 visits per patient) were evaluated. AT prevalence was 73%. AT frequency (percent of patients visits with at least 1 episode of AT since previous device interrogation) was greater in group 3 versus group 1 ($P=0.0342$). Finally, in terms of temporal sequence, AT episodes preceded OI threshold-crossing event in 43% of incidences, followed threshold-crossing event in 29%, and was simultaneous or indeterminate in the remainder.

Conclusions—These findings not only support the view that worsening pulmonary congestion is associated with increased AT frequency in patients with left ventricular dysfunction but also suggest that AT events may be responsible for triggering episodic pulmonary congestion more often than previously suspected. **(*Circ Arrhythmia Electrophysiol.* 2009;2:488-494.)**

Key Words: bioimpedance ■ heart failure ■ atrial tachyarrhythmias ■ volume overload

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Atrial tachyarrhythmias (AT), particularly atrial fibrillation (AF), are a frequent comorbidity in patients with left ventricular (LV) systolic dysfunction and heart failure.[1–5] Further, clinical experience suggests that AT may be triggered by periods of volume overload in such patients.[1–5] In part, AT episodes in this setting may be the consequence of increased atrial wall stress and/or neurohumoral changes that often accompany periods of volume overload. Alternatively, however, it is possible that in certain cases, spontaneous AT events may be the trigger, by initiating a period of hemodynamic deterioration and ensuing volume overload.

Clinical Perspective on p 494

The close association of volume overload secondary to LV systolic dysfunction and AT susceptibility has been established primarily during follow-up of patients with chronic disease[1,6,7]; documenting such a relationship in acute settings in free-living individuals is much more difficult. Recently, however, certain implantable cardioverter-defibrillators (ICDs) are capable of not only tracking onset and duration of AT episodes but also monitoring transpulmonary electric bioimpedance (Z) as an index of intrathoracic fluid volume status. In the setting of increased intrapulmonary fluid accumulation, transpulmonary bioimpedance tends to fall (ie, due to the electrolyte-containing fluid exhibiting a lesser impedance to electric current flow than does air).[8,9] Conversely, a reduction of intra-alveolar fluid causes transpulmonary electric bioimpedance to increase.

At the present time, ICDs capable of assessing transpulmonary electric bioimpedance use a computed index value

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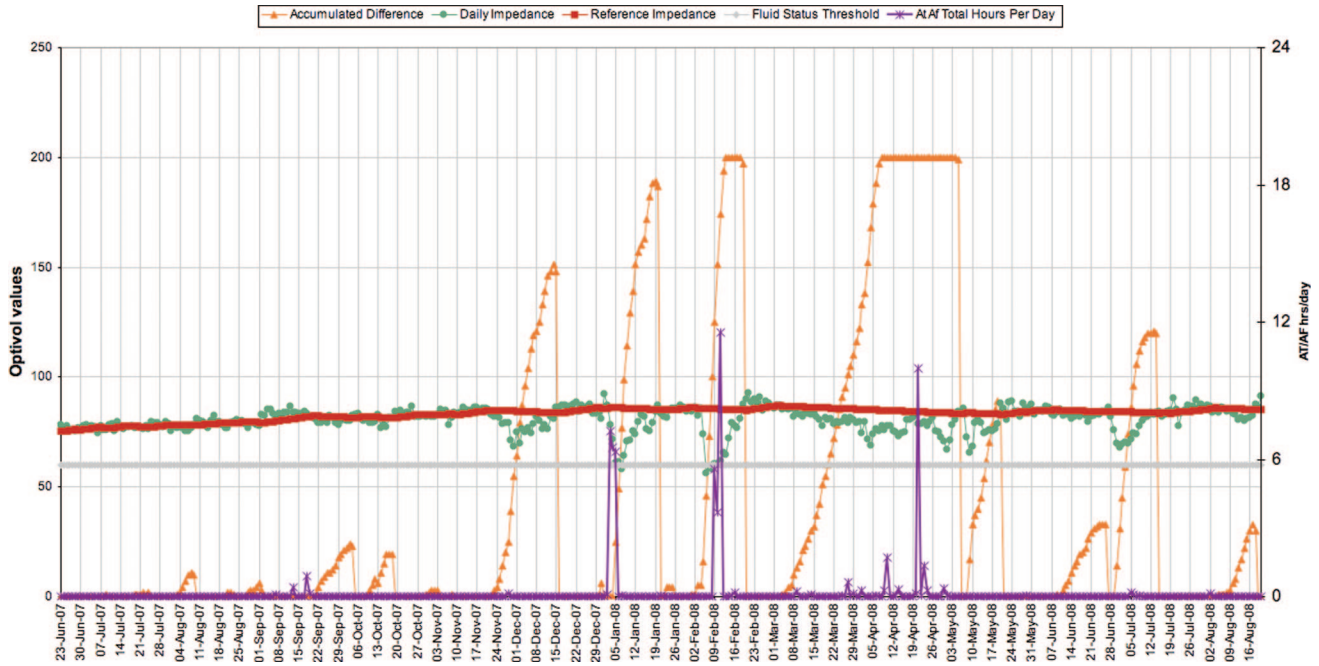


Figure 1. A representative tracing with expanded time base acquired from the OI-capable ICD that illustrates approximate coincidence of AT exacerbations (purple) with abrupt increases of OI values (orange) crossing the threshold of normal (horizontal line) and indicating pulmonary fluid accumulation in a patient with heart failure. The green line depicts the bioimpedance value (Z0); the red line provides average baseline reference impedance. Starting from the left of the figure, there are 5 transitions of OI above threshold. The first transition occurred without AT. AT preceded or coincided with OI crossing threshold in the 2nd and 3rd examples. In the 4th case, the AT burst occurs after OI increase above threshold. The final OI crossing occurred without AT.

(OptiVol index [OI], Medtronic Inc, Minneapolis, Minn) that corresponds to the inverse of a slowly moving average of the transpulmonary bioimpedance value.[8] Thus, a progressively increasing OI value suggests an increase of intrapulmonary fluid accumulation.

The purpose of this study was to examine the relationship between periods of increasing OI, suggesting greater pulmonary congestion, and the occurrence of ICD-documented AT events in patients with LV systolic dysfunction. We sought to determine whether AT events tended to be more frequent during those time periods during which the OI marker suggested increasing pulmonary vascular overload in patients with severe LV systolic dysfunction. We further sought to assess the temporal relation between AT and volume overload events.

Methods

This study examined retrospectively findings in consecutive patients with systolic LV dysfunction who are followed either at University of Minnesota Medical Center (Minneapolis, Minn) or Central Minnesota Heart Center (St Cloud, Minn) and who had implanted either a conventional or cardiac resynchronization ICD capable of measuring transthoracic electric bioimpedance between the coil of the right ventricular lead and the metallic shield of the implanted device. Further, to provide for an adequate follow-up observation period, findings were restricted to patients with ≥6 months of follow-up. Ethics committees at each of the participating institutions approved the study protocol.

The electric bioimpedance algorithm, summarized by the OI (units ohms-days), recorded the accumulation of consecutive day-to-day differences between the current daily and reference transpulmonary bioimpedance values. Because biological fluid is a better conductor of electricity than is air, greater intrapulmonary fluid accumulation reduces transpulmonary electric bioimpedance (Z).[9] However, as noted earlier, for purposes of clinical application a reduced transpulmonary bioimpedance Z is transposed to provide an increasing OI value. Thus, increasing OI is indicative of increasing pulmonary congestion and volume overload.[10–13]

Study observations, obtained in 59 ambulatory patients, comprised documenting the correspondence between both AT frequency (percent of visits during which device interrogation showed any episodes of AT since the immediate previous patient visit with device interrogations) and AT burden (presented as hours per month as percentage of total recorded time), and pulmonary congestion (as assessed by OI value). OI values were stratified (suggesting gradations of pulmonary fluid accumulation), as follows: group 1, <40 (normal range); group 2, 40 to 60 (borderline elevated); and group 3, >60 (markedly elevated). Additionally, we analyzed the temporal relationship between onset of AT events and the time when OI value crossed a threshold >60 (threshold crossing event [TCE]). TCE has been deemed indicative in prior studies of clinically significant volume overload with regard to hospitalizations for heart failure (Figure 1).[10–13]

Patients underwent clinical assessment and device interrogation at a minimum of every 3 months. Data were obtained from review of device interrogation recordings for each visit and included duration of AT episodes and corresponding OI values. All records were examined independently by 3 of the authors (R.J., G.A.T., D.G.B.). AT events were those detected by the implanted device and for study purposes were defined as episodes of duration >5 beats and with atrial rates ≥180 bpm. When intra-atrial electrograms showed an irregular atrial rate >180/min, atrial fibrillation was diagnosed. However, electrograms were available in only a minority (approximately 40%) of episodes; consequently, the more general term "AT" is used.

Data Analysis

The first goal of the study was to assess the correlation between peak OI (as a marker of volume overload and pulmonary congestion), AT frequency, and AT burden (defined as the percentage of time a patient exhibited AT since last follow-up). The second goal of the

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study was to evaluate the temporal relationship between OI >60 as a surrogate of marked volume overload and AT episodes.

The patient population was described using descriptive statistics. Generalized estimating equations (GEEs) with an autoregressive correlation structure were used to analyze AT frequency (binary outcome) and AT burden (continuous outcome) to account for possible correlation between visits within a patient. Univariate and adjusted analyses were performed. The following covariates were considered in the adjusted GEE models: group (OI<40, 40≤OI≤60, and OI>60), age, sex, ejection fraction, cardiomyopathy type, baseline atrial fibrillation, diabetes mellitus, hypertension, atrial tachycardia, hyperlipidemia, β-blocker therapy, antiarrhythmic drug therapy, diuretic therapy, statin therapy, angiotensin-converting enzyme/adrenergic receptor blocker therapy, aspirin therapy, and follow-up time.

Patient visits with missing data (outcome or covariates) were excluded from the analysis. No imputation of missing data was performed. Finally, the authors note that the AT burden outcome is a skewed variable because there is a large number of follow-ups in which patients did not have AT; thus, AT burden was zero.

In the adjusted analyses, backward selection was used and covariates with probability values <0.10 were retained in the model. Probability values <0.05 were considered statistically significant. Statistical analysis was performed using SAS version 9.1.3 (SAS Institute Inc, Cary, NC).

Results

Patient Population

A total of 69 patients with OI-capable devices were followed during the time frame of the study. Ten patients were excluded because of 1 or more of the following: incomplete data, presence of permanent AT, or ejection fraction ≥45%. Table 1 provides demographic data on the remaining 59 patients.

Median follow-up among all patients was 8.6 months (range, 0.8 to 29.5). The relationships between peak OI and AT frequency and AT burden were analyzed in 225 patient visits/device interrogations (mean, 3.8 visits per patient). In 3 instances, the follow-up visits were incomplete with missing values. These follow-up visits were excluded from analysis. Total patient visits for each of the 3 OI groups were: group 1, 120; group 2, 61; and group 3, 44. Temporal relationships between AT and OI were assessed in 41 of 225 patient visits in which OI was detected to have crossed the threshold of 60 (ie, TCE), and at least 1 episode of AT/AF was observed.

Prevalence of AT

Before device implantation, 30.5% of patients had had prior AT or AF episodes documented. However, during the course analysis of the data from device interrogations, 73% of patients had at least 1 episode of AT or AF, thereby substantially raising the documented prevalence of AT/AF in this cohort.

AT Susceptibility and Baseline Features

Clinical factors associated with ICD-documented AT occurrences were evaluated. Not surprisingly, a history of atrial fibrillation was closely associated with greater AT frequency during follow-up ($P=0.0200$); risk ratio was 2.81 times higher in the presence of versus the absence of AT history. On the other hand, whereas female sex was associated with borderline increased AT odds ($P=0.0653$), other clinical features including age, LV ejection fraction, and type of cardiomyopathy were not found by univariate analysis to be associated with increased AT frequency ($P=0.5523$, $P=0.7673$, $P=0.2502$, respectively) (Table 2).

OI Versus AT Frequency

The maximum OI was found by univariate analysis to be significantly associated with greater AT frequency ($P=0.0142$). AT risk increased as OI increased (odds ratio [OR]=1.007 per 1-U increase; 95% CI, 1.001, 1.013) (Figure 2). In only 20 of 225 or 8.9% of patient visits did TCE >60 occur without a recorded AT episode.

After adjusting for sex, baseline AT history, and use of antiarrhythmic drugs, and use of diuretic drugs the AT risk was 2.48 times higher with OI>60 compared with OI<40 ($P=0.0425$) (Figure 2). OI>60 compared with OI between 40 and 60 and OI between 40 and 60 compared with OI<40 did not differ significantly with respect to AT risk ($P=0.2145$ and $P=0.2608$, respectively) (Table 3).

Similar results were obtained without including aforementioned baseline variables in the model (OI>60 versus OI<40, OR=2.21, $P=0.0342$; OI>60 versus OI between 40 and 60, OR=1.78, $P=0.1278$; OI between 40 and 60 versus OI<40, OR=1.24, $P=0.4102$).

OI Versus AT burden

Peak OI (coefficient=−0.03; $P=0.0110$) and use of a β-blocker (coefficient=4.7; $P=0.0399$) were found to be associated with AT burden by univariate analysis from the GEE model (Figure 3). Other covariates, including OI groups, were not found to be associated with AT burden.

Temporal Relationships Between AT Events and OI

The temporal relationship between AT episodes and TCE for OI (ie, OI>60) was examined whenever the 2 occurred within 2 weeks of each other. This 2-week time period was chosen to account for the relatively slow cumulative manner in which OI changes in response to alterations of transpulmonary Z. This concordance was observed in 34 of 225 (15%) follow-up visits in 21 of 59 (35.6%) of patients.

In most patients, there were examples of all 3 possible scenarios (Figure 1): AT occurring before TCE (ie, before presumed clinically significant pulmonary congestion), AT occurring simultaneous with TCE, and AT episodes occurring after a TCE. However, AT episode(s) preceding TCE was the single most frequent finding (43% of incidences or 15 patient visits). AT episodes after TCE occurred in 29% of episodes or 10 patient visits, and both occurred essentially simultaneously in 22% or 7 patient visits. In the remaining 6% of instances of OI excursion above threshold, the temporal relation to AT events could not be determined with certainty.

Discussion

This study provides 3 principal findings regarding the temporal relationship between episodes of increasing OI (ie, decreased transpulmonary electric bioimpedance), a marker of worsening pulmonary congestion, and apparent susceptibility to temporally concordant AT events in patients with LV systolic dysfunction. First, higher OI values were associated

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Table 1. Demographic Information

	Statistic	n=59
Sex		
Female	n (%)	21 (35.6)
Male	n (%)	38 (64.4)
Age, y	Mean	64.4
	Median	66
	SD	12.54
	Minimum–maximum	37–87
LV ejection fraction	Mean	0.24
	Median	0.20
	SD	0.10
	Minimum–maximum	0.10–0.45
		1 missing
Cardiomyopathy		
Ischemic	n (%)	35 (59.3)
Nonischemic	n (%)	24 (40.7)
At least 1 AT episode		
Yes	n (%)	43 (72.9)
No	n (%)	16 (27.1)
Prior AT		
Yes	n (%)	18 (30.5)
No	n (%)	41 (69.5)
Diabetes mellitus		
Yes	n (%)	16 (28.6)
No	n (%)	40 (71.4)
		3 missing
Hypertension		
Yes	n (%)	29 (51.8)
No	n (%)	27 (48.2)
		3 missing
AT		
Yes	n (%)	16 (28.6)
No	n (%)	40 (71.4)
		3 missing
Hyperlipidemia		
Yes	n (%)	26 (46.4)
No	n (%)	30 (53.6)
		3 missing
β-Blocker		
Yes	n (%)	52 (92.9)
No	n (%)	4 (7.1)
		3 missing
Antiarrhythmic		
Yes	n (%)	15 (26.8)
No	n (%)	41 (73.2)
		3 missing
Diuretic		
Yes	n (%)	35 (62.5)
No	n (%)	21 (37.5)
		3 missing
		(*Continued*)

Table 1. Continued

	Statistic	n=59
Statin		
Yes	n (%)	37 (66.1)
No	n (%)	19 (33.9)
		3 missing
ACE/ARB		
Yes	n (%)	54 (96.4)
No	n (%)	2 (3.6)
		3 missing
Aspirin	n (%)	
Yes	n (%)	43 (76.8)
No		13 (23.2)
		3 missing

ACE/ARB indicates angiotensin-converting enzyme/adrenergic receptor blocker.

with greater likelihood of having an AT event since the most recent prior patient visit/device interrogation, that is, higher AT frequency. Second, higher peak OI values were associated with greater AT duration, that is, higher AT burden.

Table 2. Univariate Associations With AT Frequency (GEE Model)

	OR	95% CI		*P* Value
Sex, male/female	0.49	0.23	1.05	0.0653
Prior AT, yes/no	2.81	1.18	6.72	0.0200
Age	1.01	0.98	1.05	0.5523
LV ejection fraction	0.55	0.01	29.7	0.7673
Cardiomyopathy, ischemic/nonischemic	0.63	0.28	1.39	0.2502
Diabetes mellitus, yes/no	1.07	0.44	2.59	0.8759
Hypertension, yes/no	1.02	0.45	2.28	0.9680
AT, yes/no	1.92	0.78	4.71	0.1551
Coronary artery disease, yes/no	1.50	0.66	3.41	0.3377
Hyperlipidemia, yes/no	0.63	0.28	1.40	0.2535
β-Blocker, yes/no	1.40	0.36	5.41	0.6262
Antiarrhythmic, yes/no	2.48	1.01	6.10	0.0473
Diuretic, yes/no	0.62	0.28	1.38	0.2386
Statin, yes/no	1.78	0.78	4.06	0.1731
ACE/ARB, yes/no	0.98	0.18	5.27	0.9775
Aspirin, yes/no	0.87	0.32	2.31	0.7721
OI	1.007	1.001	1.013	0.0142
Unadjusted GEE model				
$40 \leq OI \leq 60$ vs $OI < 40$	1.24	0.74	2.08	0.4102
$OI > 60$ vs $OI < 40$	2.21	1.06	4.62	0.0342
$OI > 60$ vs $40 \leq OI \leq 60$	1.78	0.85	3.75	0.1278
Adjusted* for prior AT, sex, use of antiarrhythmic, and diuretic				
$40 \leq OI \leq 60$ vs $OI < 40$	1.40	0.78	2.51	0.2608
$OI > 60$ vs $OI < 40$	2.48	1.03	5.96	0.0425
$OI > 60$ vs $40 \leq OI \leq 60$	1.77	0.72	4.37	0.2145

ACE/ARB indicates angiotensin-converting enzyme/adrenergic receptor blocker.
*Covariates were identified using backward selection, and *P* values <0.10 were retained in the model.

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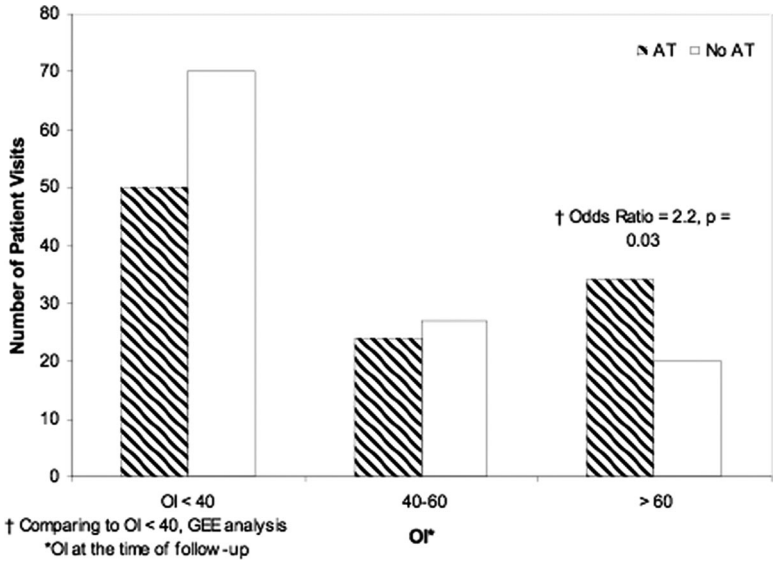


Figure 2. Graphic illustration of number of patient visits with and without AT (AT frequency) in the 3 categories of OI patient groups prespecified before the start of the study. Hatched bars show the patient visits in which episodes of AT were present. White bars show the patient visits in which no episodes of AT were found. Note that with increasing OI or increasing volume overload, there are more patient visits with AT relative to patient visits without AT.

Finally, in a subset of patients in whom OI exceeded 60, a value previously found to be consistent with clinically significant heart failure exacerbation,[10,–13] AT episodes preceded (43%) or occurred almost simultaneously (22%), with the threshold crossing in more than half the patients. Although timing could not be determined precisely (because OI is a slowly moving marker of impedance changes), the latter observation supports the notion that AT may be a relatively frequent trigger for fluid retention and pulmonary congestion in patients with LV dysfunction.

It has been generally accepted that in chronic cardiac disease, LV dysfunction and resultant chronic fluid retention may predispose individuals to arrhythmias in general and AT/AF in particular. Several mechanisms have been proposed, including activation of renin-angiotensin-aldosterone system that promotes fibrosis,[14–17] increased atrial stretch,[17–19] and elevated circulating catecholamine levels.[17] On the other hand, it is now generally agreed that over time, sustained AT/AF may adversely affect cardiac function by cellular and extracellular remodeling, alteration of ion channel properties,[20] and loss of atrial transport.[21,22] Whether the same relationships between volume overload and AT/AF holds for acute situations is less certain. However, concordant with clinical experience,[1–5] the findings reported here suggest that acute volume overload may be closely associated with initiation of AT episodes.

The prevalence of AT in our study population of heart failure patients with LV dysfunction was thought to be 30.5%, based on medical history obtained before data analysis. However, based on device interrogations, 73% of our patients exhibited AT/AF during follow-up. This value is greater than that found in several large previous clinical trials (14.4% in the V-HeFT group,[23] 15.4% in the CHF-STAT group,[24] and 25.8% in DIAMOND CHF group[25]). Consequently, the true prevalence of AT/AF in patients with LV dysfunction may be much higher than previously thought. On the other hand, our patients may have been subject to more frequent ventricular pacing than would have been the case in the comparator studies; ventricular pacing has been associated with greater AT susceptibility. However, the more likely explanation for the greater AT frequency in this study is that the implanted devices probably detected many AT events, as well as shorter AT events, that would have gone undocumented in previous studies in which recordings from implanted devices were not routinely available. For instance, modern ICDs are capable of documenting arrhythmia events, despite the fact that patients may be asymptomatic; such events may be missed during conventional clinical follow-up

Table 3. AT Burden, AT Hours, and Follow-Up Duration in the 3 OI Groups

	Statistic	OI<40 (n=120)	OI 40–60 (n=51)	OI>60 (n=54)
AT burden, %	Mean (SD)	3.8 (17.0)	6.2 (19.8)	7.6 (20.9)
	Median (IQR)	0 (0–0.02)	0 (0–0.1)	0.01 (0–3.0)
	Minimum–maximum	0–100	0–98.5	0–97.2
AT, h	Mean (SD)	37.5 (178.3)	113.5 (366.8)	154.0 (419.0)
	Median (IQR)	0 (0–0.2)	0 (0–3.2)	0.18 (0–44)
	Minimum–maximum	0–1480	0–2010	0–2570
Follow-up duration, h	Mean (SD)	1292.8 (1117.2)	6.2 (19.8)	2939.1 (2032.9)
	Median (IQR)	840 (444–2160)	2208 (1680–2928)	2376 (2112–3480)
	Minimum–maximum	96–6384	48–4848	336–11616

IQR indicates interquartile range.

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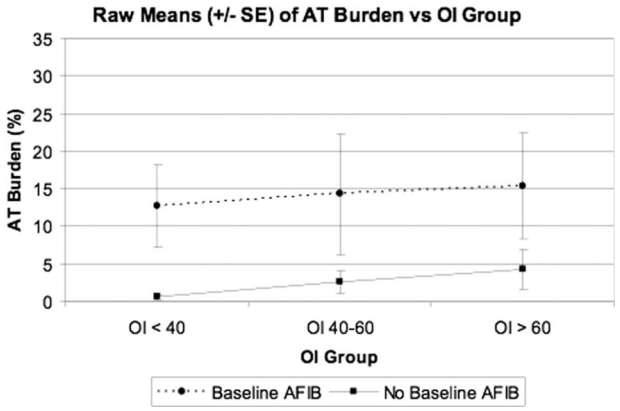


Figure 3. Graph illustrating the relationship of AT burden among the OI patient groups. There appeared to be a trend toward higher AT burden with higher OI, although differences were not statistically significant. See text for details.

visits, or if only a relatively brief ambulatory ECG monitoring period is relied on (eg, a 48-hour ambulatory ECG).

The observation that 35.6% of our patients had episodes of AT/AF and clinically significant volume overload (OI>60) occurring within 2 weeks of each other suggest the close relationship of these 2 clinical conditions in the acute/subacute setting. In nearly one-half of such episodes of pulmonary congestion (defined as OI>60), AT may have been the trigger or at least a complicating factor aggravating the clinical picture. Our observations suggest that acute volume overload is often heralded by or closely associated with AT. In this regard, the factors that trigger AT episodes and may lead to acute volume overload are not well understood and may be different from those factors associated with chronic AT (eg, atrial fibrosis, hypertensive heart disease, valvular dysfunction, and so on).[26–30] Further study of the relation between acute fluid retention and AT events is warranted. Indeed, if a close relationship proves to be the case in larger patient populations, there may be a window of opportunity (identified by warning from an appropriately programmed implanted pulse generator or implanted loop recorder) to address AT episodes and prevent their potential adverse hemodynamic consequences in patients with LV dysfunction.

Limitations

Several important limitations to this study warrant consideration. First, the correspondence between electric bioimpedance changes (such as OI) and clinical heart failure must be studied more thoroughly in large patient populations. However, although other technologies are being actively assessed to detect heart failure episodes at an early stage, electric bioimpedance is to date the only available surrogate measure of pulmonary fluid accumulation for use in free-living patients. Second, this is a retrospective analysis in which we observed an apparently greater incidence of atrial tachyarrhythmias than was suggested by the medical history of prior AT. This may be explained, as indicated earlier, by the presence of an implanted monitoring device, but could also have been influenced by our choosing to classify episodes ≥5 beats as an AT event. Further, the patient population was small and as such, we are unable to discern in this sample whether pulmonary fluid accumulation alone was responsible for de novo AT. Similarly, the subset analysis examining temporal relationships between AT and pulmonary congestion was limited by our using the previously suggested OI>60 threshold for diagnosing clinically significant volume overload[10–13] and by the fact that OI is a slowly moving average of impedance measurements collected periodically (a necessary limitation in current systems due to the low level nature of the signal); we can only state whether OI values and AT events occurred within 2 weeks of each other. Confirmation of the observation that AT tended to precede and perhaps trigger volume overload in many cases must be revisited in a larger population with more precise time estimates and correlation with emergency room and/or hospital admission data.

Conclusions

In ambulatory patients with LV dysfunction, our findings suggest that there is a high frequency of AT events and a potentially clinically important association between diminishing transpulmonary electric bioimpedance (ie, increasing OI suggesting worsening pulmonary fluid volume status) and increased susceptibility to AT events. Further, our findings document in free-living individuals that AT episodes may be an underappreciated but possibly important trigger for acute pulmonary congestion/volume overload. Potentially, if larger studies substantiate the relationships observed here, a treatment strategy designed to reduce AT risk and/or detect and foreshorten AT episodes may prove beneficial for reducing susceptibility to pulmonary congestion in patients with LV systolic dysfunction.

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Disclosures

Drs Benditt and Lurie have equity in Medtronic Inc, and Dr Benditt is a consultant to Medtronic Inc, the manufacturer of the ICD used in these studies.

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CLINICAL PERSPECTIVE

Atrial tachyarrhythmias (AT), particularly atrial fibrillation (AF), occur frequently in patients with heart disease and left ventricular dysfunction. Clinical experience suggests that AT/AF susceptibility is increased during periods of worsening heart failure, a condition typically associated with excess fluid volume in the lungs. However, the apparently close association between heart failure and AT/AF has been established primarily during follow-up of patients with chronic disease; documenting such a relationship in acute settings of heart failure exacerbation in free-living individuals is much more difficult. Recently, however, certain implantable cardioverter-defibrillators are capable of not only tracking onset and duration of AT/AF episodes but also estimating transpulmonary electric bioimpedance as an index of intrathoracic fluid volume status. Using this recently introduced technology, findings in this study support the view that there is a clinically important association between worsening pulmonary fluid volume status (as assessed by diminishing transpulmonary electric bioimpedance) and increased susceptibility to AT/AF events in free-living individuals. In addition, our findings suggest that AT/AF episodes may be an underappreciated but an important trigger for acute pulmonary congestion/volume overload. Potentially, if larger studies substantiate the relationships observed here, a treatment strategy designed to reduce AT risk and/or detect and foreshorten AT episodes may prove beneficial for reducing susceptibility to pulmonary congestion in certain patients with heart failure.

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