Supraventricular arrhythmias (SVA) are the most frequently encountered cardiac rhythm disturbances (1,2). Most often these arrhythmias manifest as intermittent or sustained atrial fibrillation/flutter, which are fairly common after cardiac and thoracic surgery, but also occur after other types of surgery and in nonsurgical intensive care unit (ICU) patients (3–7). These arrhythmias may result in prolonged ICU and hospital stays (8,9). Especially problematic are new-onset atrial arrhythmias in surgical ICU patients during and after noncardiothoracic surgery. These have been associated with increased mortality (4).

The associated demographic and clinical factors, as well as the consequences, of new-onset SVAs during an ICU stay among patients who have undergone noncardiac, nonthoracic surgery or who have sustained noncardiac or nonthoracic trauma have not been fully identified (2,3,10). Therefore, this study aimed to discern the preadmission and intra-ICU factors associated with SVAs developing in these patients. In addition, the effects of such arrhythmias on the length of ICU stay, in-hospital mortality, and long-term (4-yr) mortality were examined.

**METHODS**

We studied consecutive patients admitted over 1 yr to the 11-bed General ICU of the Hadassah-Hebrew University Medical Center (Ein Kerem Campus). Most admissions were postoperative patients and trauma victims, although hematology/oncology, medical and neurology patients were also admitted. Patients were excluded if they had undergone recent thoracic surgery or sustained thoracic trauma because they had a known frequent incidence of SVAs attributable in many cases to direct atrial irritation (3,9,10).

Patients were prospectively followed during their ICU stay for evidence of SVAs (excluding sinus tachycardia/bradycardia). Their medical records were reviewed daily and evidence of arrhythmias was collected from the bedside physiological monitors and 12-lead electrocardiograms. All observed episodes of
SVAs lasting more than 30 s were included in this study. The electrocardiograms were examined to determine the type of SVA (e.g., atrial fibrillation/flutter, paroxysmal atrial tachycardia). The patients were divided into three groups: no arrhythmias, prehospitalization history of SVAs, and new-onset SVAs. A history of SVAs was gleaned from the patient, treating physicians, and medical records. An \textit{a priori} decision was made to include these patients as a separate group, based on previous observations that such patients differed demographically and clinically from those with no arrhythmias and with new-onset arrhythmias (4).

Data were collected about potential etiologies of the new-onset SVAs, while the consequences of the SVAs were examined. The differences between the lengths of ICU and hospital stays of the three groups were compared, as were the ICU and hospital mortality rates. In addition, long-term survival rates (48 mo from the date of hospitalization) were determined using data from the Population Registry of the Israeli Interior Ministry.

The IRB of the Hadassah Medical Organization approved this study. Informed consent was waived because of the study’s observational nature.

**DATA ANALYSIS**

The demographic and clinical features of the three groups (no SVAs, new-onset arrhythmias, and a history of arrhythmias) were analyzed using descriptive statistics. Continuous parametric variables are express as mean ± sd and nonparametric variables as medians. Intergroup differences were examined using $\chi^2$ analysis for categorical variables and analysis of variance with suitable post hoc tests for continuous variables. Significance was defined as $P < 0.05$.

Logistic regression analyses were performed to identify the pre-ICU admission risk factors associated with the development of new-onset SVAs. Similar analyses identified factors associated with the triggering of new-onset arrhythmias during ICU stay. In addition, this analysis was used to examine the association of death with various pre-ICU admission and intra-ICU factors. The logistic regression analysis was rerun while controlling for sepsis, acute renal failure, diabetes, hypertension, and hemorrhagic shock. Only variables with a significance of $P < 0.05$ on univariate analysis were included in the multivariate analysis. In the multivariate logistic regression, variables are presented as odds ratio and 95% confidence intervals and those with $P < 0.05$ were considered statistically significant.

The differences between the lengths of ICU and hospital stays of the three groups were compared using Cox regression analysis. The long-term mortality data are the actual 4-yr survival, and thus do not include censored data. This permitted the use of the Wilcoxon’s ranked sum test to compare the survival curves.

**RESULTS**

The features of the 611 patients studied are found in Tables 1 and 2. Fifty-two patients developed new-onset SVAs lasting more than 30 s were included in this study. The electrocardiograms were examined to determine the type of SVA (e.g., atrial fibrillation/flutter, paroxysmal atrial tachycardia). The patients were divided into three groups: no arrhythmias, prehospitalization history of SVAs, and new-onset SVAs. A history of SVAs was gleaned from the patient, treating physicians, and medical records. An \textit{a priori} decision was made to include these patients as a separate group, based on previous observations that such patients differed demographically and clinically from those with no arrhythmias and with new-onset arrhythmias (4).

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**RESULTS**

The features of the 611 patients studied are found in Tables 1 and 2. Fifty-two patients developed new-onset
SVAs (38 atrial fibrillation, 2 atrial flutter, and 12 other SVAs [e.g., paroxysmal atrial tachycardia]). Among the 75 patients with a history of SVAs, 29 were in sinus rhythm on ICU admission and remained so during their hospital stays. Another 26 had SVAs upon ICU admission which continued throughout their hospitalization. Twenty patients were admitted to the ICU in sinus rhythm and then redeveloped SVAs (14 had atrial fibrillation, 2 atrial flutter, and 4 SVAs) while hospitalized.

Pre- and post-ICU admission factors associated with an increased likelihood of developing new-onset SVAs and pre-ICU admission factors that characterized patients as having histories of SVAs were identified (Table 3). Central venous or pulmonary artery catheters were located (on chest radiograph) in the right atrium in 41% of patients with central venous/pulmonary artery catheters in the group without arrhythmias and in 45% (NS) of the group with new-onset arrhythmias. Most patients in the new-onset (45/52, 87%) and history of SVAs (70/75, 93%) groups were older than 50 yr. Only 59% of the no SVAs group was older than 50 yr. Analysis of data from the patients older than 50 yr found few differences in the demographic and clinical variables, as well as odds ratios for the various determinants, when compared with analysis of the data from all age groups. In those older than 50 yr in the no SVA group, 18% died during their hospitalization and another 26% died after hospitalization. In the new-onset group 55% died while hospitalized and another 16% afterwards. However, in the history of SVA group, 33% died while in hospital, and another 33% died afterwards.

Cox regression analysis showed that the lengths of ICU stay with new-onset and history of SVAs were significantly longer than without arrhythmias ($P < 0.01$). There was no difference between the lengths of ICU stay of the new-onset and history of arrhythmias groups. The factors associated with mortality are shown in Table 4. The logistic regression analysis performed while controlling for sepsis, acute renal failure, diabetes, hypertension and hemorrhagic shock revealed no differences in the factors associated with mortality. In all three groups, over 90% of the deaths were ascribed to sepsis. The in-hospital mortality rate of patients with sepsis in the new-onset arrhythmias group (56%) was similar to the mortality rate (58%) of the entire group. However, the mortality rates of the patients with sepsis were higher in the no arrhythmias (35%) and history of arrhythmias (43%) groups than the mortality rate of the entire group, which were 16% and 25%, respectively. When patients with the same APACHE II scores were compared, those in the new-onset group had higher inpatient mortality, especially at lower scores (Fig. 1). The treatment of the new-onset arrhythmias was not a factor in survival in the new-onset group. Of the 52 patients in the group, 2 were not treated (1 alive, 1 died while hospitalized), 5 reverted to sinus spontaneously (3 alive, 2 died), and 45 were treated (20 alive, 25 died). Forty-three patients were treated successfully, i.e., either rate was controlled or sinus restored.

Long-term survival was especially poor in the new-onset SVAs group, with only 35% of the patients alive after 1 yr (Fig. 2a). Survival of patients with histories

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### Table 2. Intensive Care Unit (ICU) Data

<table>
<thead>
<tr>
<th>Groups</th>
<th>No SVA</th>
<th>New onset SVA</th>
<th>History SVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>$N$</td>
<td>484 (79%)</td>
<td>52 (9%)</td>
<td>75 (12%)</td>
</tr>
<tr>
<td>Apache II</td>
<td>16 ± 8</td>
<td>23 ± 8*</td>
<td>19 ± 6</td>
</tr>
<tr>
<td>Sepsis/SIRS</td>
<td>112 (23%)</td>
<td>37 (75%)*</td>
<td>32 (42%)*†</td>
</tr>
<tr>
<td>Ischemia</td>
<td>27 (6%)</td>
<td>5 (10%)</td>
<td>7 (9%)</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>34 (7%)</td>
<td>24 (46%)*</td>
<td>12 (16%)*†</td>
</tr>
<tr>
<td>Hemorrhagic shock</td>
<td>14 (3%)</td>
<td>5 (10%)*</td>
<td>4 (5%)</td>
</tr>
<tr>
<td>Dopamine</td>
<td>12 (3%)</td>
<td>25 (48%)*</td>
<td>16 (21%)*†</td>
</tr>
<tr>
<td>Other catechols</td>
<td>17 (4%)</td>
<td>15 (29%)*</td>
<td>15 (29%)*</td>
</tr>
<tr>
<td>Inhaled $\beta$-agonist</td>
<td>17 (4%)</td>
<td>1 (2%)</td>
<td>5 (7%)</td>
</tr>
<tr>
<td>$\beta$-Blocker</td>
<td>15 (3%)</td>
<td>2 (4%)</td>
<td>5 (7%)</td>
</tr>
<tr>
<td>ICU-LOS (in days) (median)</td>
<td>11 ± 17 (4)</td>
<td>15 ± 13 (11*)</td>
<td>12 ± 19 (6)</td>
</tr>
<tr>
<td>HOSP-LOS (in days) (median)</td>
<td>21 ± 21 (14)</td>
<td>34 ± 36 (25*)</td>
<td>37 ± 29 (28*)</td>
</tr>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICU stay</td>
<td>81 (16%)</td>
<td>28 (54%)*</td>
<td>19 (25%)*†</td>
</tr>
<tr>
<td>HOSP stay</td>
<td>87 (18%)</td>
<td>29 (56%)*</td>
<td>24 (32%)*†</td>
</tr>
<tr>
<td>After hospital discharge</td>
<td>83 (17%)</td>
<td>8 (16%)</td>
<td>23 (31%)*†</td>
</tr>
<tr>
<td>Total—four years</td>
<td>170 (35%)</td>
<td>37 (71%)*</td>
<td>47 (63%)*†</td>
</tr>
<tr>
<td>Mortality rate after hospital discharge</td>
<td>20%</td>
<td>36%*</td>
<td>45%*†</td>
</tr>
</tbody>
</table>

* Ischemia—evidence of myocardial ischemia and/or infarction.
† Dopamine infusion.
‡ Other catecholamines, e.g., norepinephrine.
§ Mortality after hospital discharge until 48 mo after hospitalization.
* Versus no SVA ($P < 0.05$).
† Versus new onset SVA ($P < 0.05$).
of SVAs was poorer than those without SVAs ($P < 0.01$). The in-hospital survival of the former patients was better than those with new-onset arrhythmias, although after 4 yr the survival rates converged (Table 2). The differences in the mortality patterns was not age related (Fig. 2b).

**DISCUSSION**

This study confirms the significant incidence of new-onset SVAs among ICU patients. It extends previous observations by identifying many of the factors associated with the development of new-onset SVAs. The in-hospital survival of patients with new-onset SVAs was better than those without SVAs ($P < 0.01$). The survival rates converged after 4 yr. The differences in the mortality patterns were not age related.
and their high mortality rate. Moreover, unlike any previous study, this study examined long-term (4-yr) mortality patterns. The 4-yr survival data were notable for the striking observation that almost all the deaths among those developing new-onset SVAs were during their hospitalization, and that mortality over the next 3 yr was minimal. Fifty-six percent of these patients died while hospitalized, but only another 16% died after they were released from the hospital leading to a 71% 4-yr mortality rate. This contrasts with a lower in-hospital mortality rate of 32% and a higher posthospitalization rate of 31% among those with histories of SVAs, leading to a 63% 4-yr mortality rate. This progressive decrease in survival is expected, since chronic atrial fibrillation in the general population is associated with an increase in the relative risk of death (1.3–2 times) (11). The differences in the survival patterns between the new-onset and history of SVAs groups lend further credence to the supposition that the new-onset of SVAs in ICU patients is a unique entity. This difference is also demonstrated by the greater mortality rates at lower APACHE II scores in new-onset group (Fig. 1). These two groups differ from the group without SVAs in which 18% died during hospitalization and 14% died after hospitalization leading to a 32% 4-yr mortality rate.

The present study excluded patients who underwent cardiothoracic surgery or sustained cardiac or thoracic trauma because of their propensity to develop SVAs (10,12). This was highlighted in a report showing that surgical ICU patients with thoracic trauma had an increased likelihood of developing new-onset atrial fibrillation (13). In the present study, 9% of patients developed new-onset SVAs, a rate similar to that described by others. Brathwaite and Weissman (4) reported a 10% incidence in ICU patients after noncardiac, nonthoracic surgery, while Reinelt et al. (14) and Valentine et al. (5) found similar incidences in a medical-surgical ICU and after elective aortic operations, respectively. Of 226 colorectal surgical patients admitted to a high dependency unit, 13% had ventricular or SVAs, with atrial fibrillation being the most common arrhythmia (15). Kirton et al. (6) reported a 5% incidence of paroxysmal atrioventricular nodal reentry tachycardia, a subtype of supraventricular tachycardia, in consecutive trauma and surgical ICU admissions (including those with chest injuries). Studies performed in broader postoperative populations that included non-ICU patients showed that 51 (0.37%) of 13,696 patients developed atrial fibrillation after noncardiac, nonthoracic surgery (16). In a population limited to patients >50 yr, who were admitted postoperatively to either an ICU or floor after major (expected hospital stay of ≥2 days) nonemergency, noncardiac procedures, the incidence of new-onset SVAs was 7.6% (17). SVAs are thus frequently encountered in postoperative and critically ill patients and are more common than ventricular arrhythmias.

It is not surprising that the majority of SVAs in the current study were atrial fibrillation/flutter, since these arrhythmias are the most frequently encountered in the general population. Their incidence increases with age, occurring in 1% of 50-yr-olds and 11% of 80-yr-olds. This is consistent with the current study in which the average age of those with a pre-ICU history of SVAs was 73 ± 11 yr (18). The prevailing theory is that, in the elderly, atrial fibrillation occurs in the setting of dysfunctional atrial electrical pathways that provide favorable conditions for arrhythmias to begin and continue. This has been ascribed to structural changes in the atria, causing electrophysiological dysfunction, and to the consequences of cardiac and extracardiac disease. Therefore, the current findings of an association of cardiac disease, hypertension, and diabetes mellitus with pre-ICU SVAs are consistent with previous investigations. For example, hypertension is present in at least half the individuals with chronic atrial fibrillation, as was observed in the present study. These associated chronic diseases, along with advanced age, are thought to be the reasons for increased perioperative mortality among these patients. Interestingly, in the present study only diabetes mellitus was an associated risk for increased mortality. Others have also observed that preoperative atrial fibrillation is a risk factor for postoperative mortality (19).

The new-onset SVAs were not associated with pre-ICU admission cardiac disease. Instead, only a history of chronic pulmonary disease and hypothyroidism were associated with the development of these arrhythmias. At first glance, this is surprising because hyperthyroidism, and not hypothyroidism, is traditionally associated with SVAs. However, hypothyroidism has been linked to atrial fibrillation in the elderly (20). Alternately, the thyroid replacement these patients received may have made them vulnerable to developing atrial arrhythmias, although studies have not shown this to be true (21). It is more likely that the incidence of hypothyroidism was associated with these patients’ advanced age, and is not directly related to the development of the arrhythmias. This is supported by the analysis of patients >50 yr in which

**Figure 1.** In-hospital mortality per APACHE II Score for the three groups is shown. Note that the new-onset supraventricular arrhythmia (SVA) group had a higher mortality than the other two groups at lower APACHE II scores. *Significantly different than the no-SVA and history of SVA groups (P < 0.05). #Significantly different than the no-SVA group.
the incidence of hypothyroidism was the same in the new-onset (8/45, 18%) and history of arrhythmia (12/70, 17%) groups. An additional observation was that the new-onset arrhythmias occurred equally in males and females, unlike the male prevalence of atrial fibrillation in the general population and in the history-of-arrhythmia group.

The present study identified possible triggering mechanisms for the new-onset arrhythmias. Hyperthyroidism, valvular disease, and electrolyte disturbances (hypomagnesium, hypocalcemia, and hyperkalemia) were not found to be initiators nor were direct irritation of the right atrium by central venous catheters or pulmonary artery catheters. However, older age was associated with the development of new-onset SVAs. Similar observations were made among patients who developed atrial arrhythmias after lung resection surgery (18). Sepsis/systemic inflammatory response syndrome greatly increased the odds of developing SVAs. This is consistent with reports that sepsis is closely associated with SVA development, especially in postoperative patients (4,22–25). Further evidence for adrenergic stimulation comes from the greater success with treating new-onset supraventricular tachyarrhythmias in ICU patients with \( \beta \)-adrenergic antagonists than with calcium channel blockers (34).

New-onset SVAs after cardiac and thoracic surgery result in longer ICU and hospital stays. This was also observed in the present study of ICU patients who had not undergone cardiothoracic surgery. New-onset SVAs in ICU patients have been linked to increased mortality secondary to severe acute illness (4). This was confirmed by the present study where indicators of illness severity, such as sepsis, myocardial ischemia, acute renal failure, and APACHE II scores, were associated with the increased mortality. Therefore, new-onset SVAs appear to be associated with the homeostatic dysfunction seen with multiple organ system failure.

In conclusion, SVAs, whether previous or new-onset, are frequently encountered in ICU patients. In this study, 21% of ICU patients were in one of these two categories. This study has a number of limitations; among them are the lack of continuous cardiac monitoring after discharge from the ICU, its performance in a single institution, a sample size that spanned only 1 yr and an observational design. Despite these limitations, this study was clearly able to confirm previous investigations showing that the severity of the underlying critical illness, especially sepsis/systemic inflammatory response syndrome, is associated with the development of new-onset arrhythmias and its greater mortality. Furthermore, it extends previous observations by revealing the extremely high (65%) 1-yr mortality rate in such patients and showing different 4-yr survival patterns of the three groups of patients.
studied. Therefore, new-onset SVAs in critically ill patients are associated with extremely poor prognosis.

REFERENCES