Primary Prevention of Sudden Cardiac Death in Idiopathic Dilated Cardiomyopathy

The Cardiomyopathy Trial (CAT)

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Background—Patients with idiopathic dilated cardiomyopathy (DCM) and impaired left ventricular ejection fraction have an increased risk of dying suddenly.

Methods and Results—Patients with recent onset of DCM (≤9 months) and an ejection fraction ≤30% were randomly assigned to the implantation of an implantable cardioverter-defibrillator (ICD) or control. The primary end point of the trial was all-cause mortality at 1 year of follow-up. The trial was terminated after the inclusion of 104 patients because the all-cause mortality rate at 1 year did not reach the expected 30% in the control group. In August 2000, the vital status of all patients was updated by contacting patients, relatives, or local registration offices. One hundred four patients were enrolled in the trial: Fifty were assigned to ICD therapy and 54 to the control group. Mean follow-up was 22.8±4.3 months, on the basis of investigators' follow-up. After 1 year, 6 patients were dead (4 in the ICD group and 2 in the control group). No sudden death occurred during the first and second years of follow-up. In August 2000, after a mean follow-up of 5.5±2.2 years, 30 deaths had occurred (13 in the ICD group and 17 in the control group). Cumulative survival was not significantly different between the two groups (93% and 80% in the control group versus 92% and 86% in the ICD group after 2 and 4 years, respectively).

Conclusions—This trial did not provide evidence in favor of prophylactic ICD implantation in patients with DCM of recent onset and impaired left ventricular ejection fraction. **(*Circulation*. 2002;105:1453-1458.)**

Key Words: cardiomyopathy ■ defibrillation ■ tachycardia ■ fibrillation ■ death, sudden

Implantable cardioverter-defibrillators (ICD) terminate ventricular tachycardias (VT) and ventricular fibrillation (VF) with high efficacy.[1–3] In secondary prevention trials of sudden cardiac death (SCD), ICD treatment was superior to antiarrhythmic drug therapy.[4–6] In primary prevention of SCD, data on ICD treatment compared with conventional therapy are inconclusive. ICD treatment did not provide survival benefits over bypass graft surgery alone in the Coronary Artery Bypass Graft Patch Trial (CABG-Patch).[7] In contrast, the Multicenter Automatic Defibrillator Implantation Trial (MADIT) revealed a striking survival benefit of ICD treatment compared with drug therapy in patients with spontaneous nonsustained VTs, impaired left ventricular (LV) function, and inducible VTs that could not be suppressed by procainamide.[8] These results are supported by the Multicenter Unsustained Tachycardia Trial (MUSST).[9]

Only limited information is available in patients with dilated cardiomyopathy (DCM). In most heart failure studies, patients with DCM represent a minor subgroup of the overall study population. Mortality rates after 1 year have been reported to range between 14% and 44% in New York Heart Association functional class III to IV; 21% to 51% of deaths are supposedly due to ventricular tachyarrhythmias.[10–19]

In 1991, we started a primary prevention trial in patients with DCM of recent onset (≤9 months) and impaired LV ejection fraction (EF ≤30%) without documented symptomatic ventricular tachyarrhythmias who were randomly assigned to ICD therapy or to a control group.[20–23] The results of the pilot phase are reported in this article.

Methods

Patients

Patients were eligible if they were between 18 and 70 years of age, had symptomatic DCM for ≤9 months and impaired LV function (LVEF ≤30% obtained during LV angiography), and were in NYHA class II or III. Coronary artery disease (coronary stenosis >70%) had to be excluded by angiography. Patients with a history of prior

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myocardial infarction, myocarditis, or excessive alcohol consumption were not included in the trial. Furthermore, patients were excluded if they had a history of symptomatic bradycardia, VT, and VF or if they were listed for heart transplantation at the time of presentation. Patients with significant valvular disease and hypertrophic or restricted cardiomyopathy were also excluded from the trial, as were patients in NYHA class I or IV and patients who were mentally unable to understand the protocol.

Randomization

The trial was conducted at 15 German centers. The protocol was approved by the institutional review board in Freiburg, Germany, and by the review boards at each clinical center. Enrollment began in May 1991 and ended in March 1997.

Patients were enrolled after written informed consent had been obtained. Random assignment was performed centrally. Closed envelopes with the assigned study group were sent to each center. The envelopes were opened when a patient was enrolled.

Baseline Examinations

Detailed information on the study protocol has been published previously.[21–23] In short, all patients underwent the following examinations: physical examination, baseline ECG, exercise test, and echocardiography. A Holter ECG was performed to document bradycardia and tachycardia. Bradycardia was defined as a documented heart rate <40 bpm caused by sinus arrest and atrioventricular block. Wide-complex tachycardias, QRS complex >120 ms, were defined as VT if there was no evidence for supraventricular tachycardia with aberrant conduction. VTs with >3 beats and a duration of <30 seconds were defined as nonsustained; with a duration of >30 seconds, they were defined as sustained. Left heart catheterization was performed to exclude coronary artery disease. Ventricular function and EF were calculated on the basis of LV angiography. Electrophysiological study and right ventricular stimulation were performed with a maximum of 3 extrastimuli at 2 different cycle lengths at the right ventricular apex and outflow tract until VT or VF was induced.

Implantation Procedure

Patients assigned to ICD therapy underwent implantation of a transvenous defibrillator system, under general anesthesia. ICD testing was performed according to current guidelines for ICD implantation.[24] A defibrillation threshold of <20 J was mandatory. Guidant/CPI provided transvenous electrode systems (Endotak, Cardiac Pacemakers, Inc [CPI]) and pulse generators capable of delivering biphasic shocks (Ventak P2, P3, PrX II, CPI). All devices were capable of storing episode data and electrograms. A VT zone with a detection rate of 200 bpm was programmed in all patients. All shocks were programmed to a maximum output of 30 J. The pacemaker rate was programmed to 40 bpm.

End Points

The primary end point of the trial was all-cause mortality at 1 year. Secondary end points were heart transplantation, cardiac mortality (sudden and nonsudden cardiac death), sustained VT (adequate ICD therapy), and symptomatic ventricular tachyarrhythmias requiring antiarrhythmic treatment (Table 1). Adequate ICD therapy was assumed if documented tachycardia electrograms had a morphology different from sinus rhythm and if tachycardias started suddenly with no cycle length variation, indicative of atrial fibrillation. Furthermore, complications caused by ICD therapy, such as revision procedures, infection, and so forth, were documented.

Follow-Up

Patients were scheduled for follow-up visits every 3 months and were encouraged to schedule additional visits if the first shock, cluster of shocks, or syncope had occurred.

The central data registry was at the University Hospital of Eppendorf, Department of Mathematics and Data Processing in Medicine. For quality control and completeness of data forms, data monitoring was performed.

TABLE 1. Primary and Secondary End Points

End Point	Incidence at 1 y
All-cause mortality	
ICD	4 (8.0)
Control	2 (3.7)
Sudden death	
ICD	0
Control	0
Cardiac death	
ICD	4 (8.0)
Control	1 (1.9)
Heart transplantation	
ICD	2 (4.0)
Control	1 (1.9)

Values are numbers of patients (%). Total number of patients in the ICD group was 50; total number of patients in the control group was 54.

Study Design and Statistical Considerations

On the basis of the literature in the late 1980s, all-cause mortality rate was assumed to be 30% in the first year, with 40% of deaths being sudden.[10–16] On this assumption, 1348 patients had to be included to show a 1-year survival benefit of 6% for ICD treatment, with a power of 80% and a probability value of 0.05.

Because all-cause mortality rate varied considerably between different heart failure studies and included mostly patients with heart failure caused by coronary artery disease, it was decided to perform a blinded interim analysis after the inclusion of 100 patients at 1 year of follow-up (pilot phase).

In June 1997, a survival analysis of all patients with at least 1 year of follow-up was performed. The interim analysis showed that the overall 1-year mortality rate for all patients was only 5.6% and markedly below the assumed value of 30%. The difference between the survival rates of the two groups was only 2.6%. According to the protocol, the randomization was stopped, and all randomly assigned patients completed the scheduled follow-up period of 2 years.

In August 2000, at the time of final analysis, the vital status of all patients was assessed to determine long-term mortality rate. The survival rates for the groups were presented as Kaplan-Meier curves and compared by log-rank statistics.[25] Despite having an interim analysis, there was no need to perform an α-adjustment on significance for the primary end point because no differences were detected between the groups.

To estimate the prognostic relevance of patient characteristics, Cox proportional regression models were calculated. Data were described by the arithmetic mean±SD if normally distributed or otherwise by median with 25% to 75% percentiles. Quantitative comparisons between groups were performed by 2-sided analysis, with the use of the Mann-Whitney exact test; qualitative characteristics were compared by means of the exact Fisher χ^2 test. Statistical analysis was performed with SPSS for Windows, release 9.0.1.

Results

Study Sample and Comparison of Groups

A total of 104 patients were enrolled between July 1991 and March 1997. Fifty patients were randomly assigned to ICD treatment and 54 to the control group. Baseline characteristics did not differ between groups except for bradycardias caused by sinus arrest and atrioventricular block I and II (Wenckebach),

TABLE 2. Baseline Characteristics of Patients

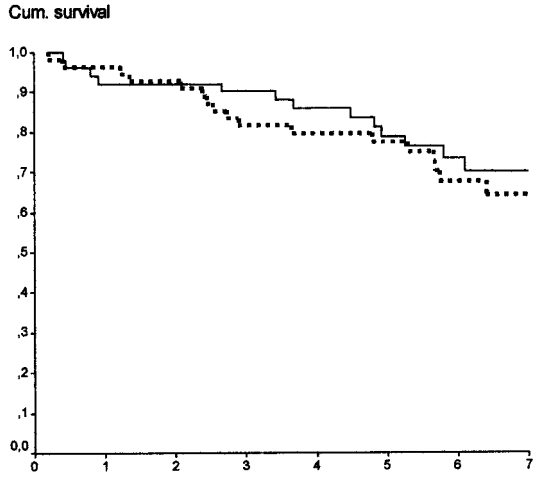
Patients	All	ICD Treatment	Control Group	P
Randomized	104	50	54	
Sex, male/female	83/21	43/7	40/14	NS
Follow-up, mo (per protocol)	22.8±4.3	22.7±4.5	22.9±4.2	NS
Follow-up, y (per August 2000)	5.5±2.2	5.7±2.2	5.2±2.1	NS
Age, y	52±11	52±12	52±10	NS
Duration of symptoms, mo (median)	3.0	3.0	2.5	NS
Baseline violators	19	8	11	NS
Class of heart failure				
NYHA II	65.3%	66.7%	64.1%	NS
NYHA III	34.6%	33.3%	35.8%	
Orthopnoe	3.0%	4.1%	2.0%	NS
Edema	8.9%	8.3%	9.4%	NS
Ejection fraction, %	24±7	24±6	25±8	NS
LVED pressure, mm Hg	17±10	16±9	18±10	NS
Echocardiography				
LV diastolic, mm	69±8	69±7	69±8	NS
LV systolic, mm	58±9	58±9	59±10	NS
Baseline ECG				
Rhythm				
Sinus rhythm	83.3%	79.6%	86.8%	NS
Atrial fibrillation/flutter†	15.7%	20.4%	11.3%	
Paced	1%	0%	1.9%	
QRS morphology				
Normal	63.9%	72.9%	55.1%	NS (0.091)
Not normal	36.1%	27.1%	44.9%	
LBBB	82.9%	84.6%	81.8%	NS
RBBB	2.9%	7.7%	0%	
Other or undefined BB	14.3%	7.7%	18.2%	
QRS width,‡ ms	108±29	102±29	114±29	NS
QT duration,‡ ms	358±58	347±69	369±43	NS
Holter ECG				
Patients with nsVTs	52.9%	53.1%	58.0%	NS
Median duration of nsVT, s (25%/75%)	4.0 (3.0/6.0)	5 (3.0/6.5)	3.5 (2.3/6.0)	NS
Rate of nsVTs, bpm	165±32	175±39	157±23	NS
Bradycardias, % of patients	10.4%	2.1%	18.8%	0.015
SA block	2.1%	0%	4.2%	
AV block	8.3%	2.1%	14.6%	NS
Baseline EPS				
AH, ms	91±27	86±22	97±31	NS
HV, ms	53±14	51±15	55±14	NS
Inducible VT	2.9%	6.1%	0%	NS
Inducible VF	9.6%	16.0%	3.7%	NS
Medication				
β-Blocker	3.8%	4.0%	3.7%	NS
Ca antagonist	11.5%	16.0%	7.4%	NS
Digitalis	80.8%	86.0%	75.9%	NS
Diuretics	86.5%	88.0%	85.2%	NS
Nitrates	28.8%	32.0%	25.9%	NS
ACE inhibitor	96.2%	94.0%	98.1%	NS
Warfarin	29.8%	24.0%	35.2%	NS

AA indicates antiarrhythmic agent; ACE, angiotensin-converting enzyme; LBBB, left bundle-brunch block; LVEDD, left ventricular end-diastolic diameter; mVT, monomorphic ventricular tachycardia; PVS, programmed ventricular stimulation; pVT, polymorphic ventricular tachycardia; RBBB, right bundle brunch block; VTC, ventricular tachycardia cluster; EPS, electrophysiological study; AH, AH interval; and HV, HV interval.

*Echocardiographic M-mode data available for 70 patients only.

†Chronic or intermittent.

‡Patients with pacemakers not included.



Number remaining at risk

ICD _____	50	46	46	45	41	32	24	15
Control	54	52	50	44	40	35	25	13

	Logrank statistic	df	Significance (not adjusted)
1 year	0.81	1	0.3672
2 years	0.01	1	0.9098
7 years	0.35	1	0.5539

Figure 1. Survival in the ICD group vs the control group.

which were noted more frequently during Holter monitoring in the control group (18.8% versus 2.1%, $P=0.015$). Furthermore, mean QRS duration was longer in the control group than in the ICD group (114 versus 102 ms) as the result of a higher incidence of left bundle-branch block (36.7% versus 22.9%). This difference did not reach statistical significance. Furthermore, 3 patients (6.1%) had monomorphic VTs induced during electrophysiological study. All were randomly assigned to ICD therapy. In 10 patients, VF was inducible. Eight patients were randomly assigned to ICD treatment and 2 patients were randomly assigned to the control group. This difference was not statistically significant.

ACE inhibitors were used in 96% of patients without any difference between the groups. Four percent of patients received β-blocker therapy in both study arms at baseline (Table 2). No changes in the medication of ACE inhibitors, digitalis, and diuretics between baseline and 24-month follow-up were documented.

Mortality

All-cause mortality rates were neither different between ICD treatment and control group after 1 year (primary end point) nor during long-term follow-up: Four patients the ICD group and 2 patients in the control group died during the first year of follow-up. No sudden death occurred in either group during this time. All four deaths were cardiac in the ICD group, whereas both patients died of noncardiac causes in the control group.

After a mean follow-up of 5.5±2.2 years, 13 patients in the ICD group and 17 in the control group died. Cumulative survival was 92%, 86%, and 73% in the ICD treatment group versus 93%, 80%, and 68% in the control group after 2, 4, and 6 years, respectively (log rank $P=0.554$, Figure 1).

Complications Caused by ICD Therapy

No deaths occurred as a result of the implantation procedure, that is, within 30 days of ICD implantation. Two revisions caused by device dislocation and bleeding had to be performed; two electrodes dislocated and had to be revised during surgery. During the following 24 months, 10 complications occurred in 7 patients: 7 incidences of electrode dislocation and sensing/isolation defects, 2 incidences of infection with total device replacement, and 1 perforation.

Predictors of Death

The only predictor of total mortality was impaired LVEF. Compared with patients with EF ≥28%, the odds ratio was 4.1 (95% CI, 1.5 to 11.3, $P=0.006$) for patients with EF ≤21% and 2.1 (95% CI, 0.7 to 6.2, $P=0.19$) for patients with EF 22% to 27%.

Survival in patients with nonsustained (ns)VTs during baseline Holter monitoring was 87%, 72%, and 63%, as opposed to 98%, 93%, and 77% in patients without nsVTs after 2, 4, and 6 years, respectively (NS). Survival of patients with nsVTs during baseline Holter monitoring was not improved by ICD therapy, whereas 85%, 77%, and 72% of patients with nsVTs survived in the ICD group and 90%, 67%, and 55% survived in the control group after 2, 4, and 6 years, respectively (NS).

All baseline variables presented in Table 2, such as age, sex, and so forth, were tested accordingly but did not show any statistically significant impact on survival.

Shocks and Syncope in Patients With ICD

Eleven patients received adequate therapies for VTs >200 bpm, and 6 patients had syncope during VTs in the ICD treatment group. Survival free of VTs and adequate therapies in the ICD group was 90%, 87%, and 82% after 2, 4, and 6 years, respectively. All-cause mortality rates were different in patients with and without adequate therapies in the ICD group. Whereas 92%, 90%, and 83% of patients survived, if no VT was stored in the ICD (n=39), only 91%, 73%, and 44% survived 2, 4, and 6 years if VTs were stored and adequately terminated by the ICD (n=11, $P=0.024$).

Discussion

This study revealed that short- and long-term overall mortality rates in patients with DCM and significantly impaired LV function were surprisingly low. Therefore, ICD therapy did not provide any survival benefit in these patients.

Only 4 patients in the ICD group and 2 in the control group died during the first year of follow-up. Long-term survival was 92%, 86%, and 73% in the ICD group versus 93%, 80%, and 68% in the control group after 2, 4, and 6 years, respectively (Figure 1). This is in strong contrast to most series of patients with DCM or heart failure published before this trial was started: One-year mortality rate had been reported to be between 14% and 44% in NYHA functional class III-IV, with 30% to 50% of deaths being be sudden.[10–14,18,19,26] Patients in NYHA class II show a lower 1-year-mortality rate of 6% to 14%.[10,13] However, most of these data had been collected before the ACE inhibitor era. ACE inhibitors have significantly improved survival in pa-

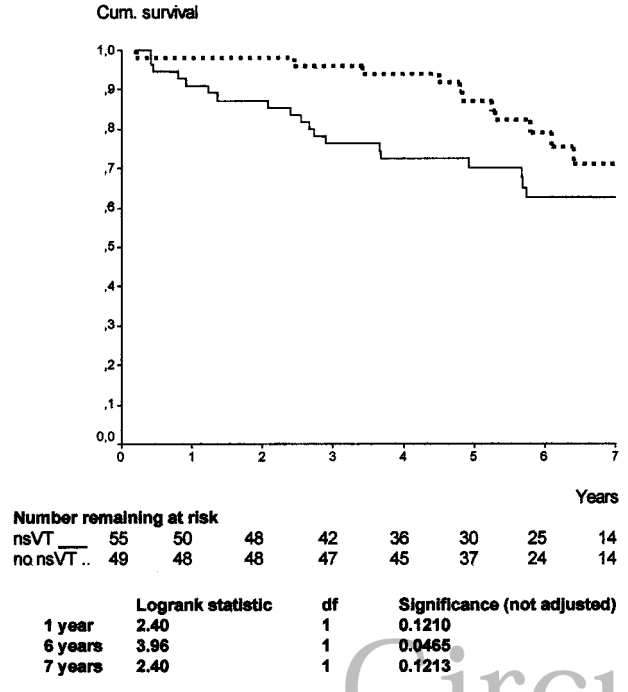


Figure 2. Survival of patients with and those without nsVTs during baseline Holter ECG.

tients with impaired LV function[11–13,16–19]; 65.4% of patients were in NYHA class II in our trial and 96.2% of patients received ACE inhibitors. This may explain the rather low mortality rate, even though only a minority of patients was treated with β-blockers.

The lack of any survival benefit of ICD therapy is most likely due to the overall low event rate in our cohort. Even if the study had been continued to include 1348 patients, the power to show the expected difference of 6% would have been <50%. Thus, the trial was stopped for futility after 1 year.

Even in patients with an increased mortality rate, such as patients with a lower EF or nsVTs during Holter monitoring (Figure 2), ICD therapy did not reveal any significant survival benefit in the Cardiomyopathy Trial (CAT). This is in contrast to other studies, which revealed a strong association between nsVTs and the risk of sudden death.[10–16,26–30] However, it is in concordance with the results of the Amiodarone Versus Implantable Defibrillators trial in patients with nonischemic cardiomyopathy and nonsustained ventricular tachycardias (AMIOVIRT), which did not reveal any survival benefit of ICD treatment over the treatment with amiodarone. The survival rates were 88% and 85% in the amiodarone-treated group versus 89% and 79% in the ICD treatment group after 2 and 4 years, respectively.[30]

In contrast to CAT and AMIOVIRT, which did not show any survival benefit of ICD therapy, a subgroup analysis of patients with DCM in the secondary prevention trial AVID demonstrated a survival benefit despite similar low mortality rates in the ICD arm (11% after 2 years compared with 8% in CAT). However, because the control arm in AVID had a higher mortality rate (18% after 2 years) compared with CAT (7% after 2 years), mortality rate was significantly reduced by ICD treatment.

Interestingly, survival in patients with an ICD with VTs stored in the ICD was significantly impaired compared with patients without VTs (Figure 3). Only 44% of patients with VTs survived 6 years compared with 83% if no VT had occurred ($P=0.024$). One explanation may be that the occur-

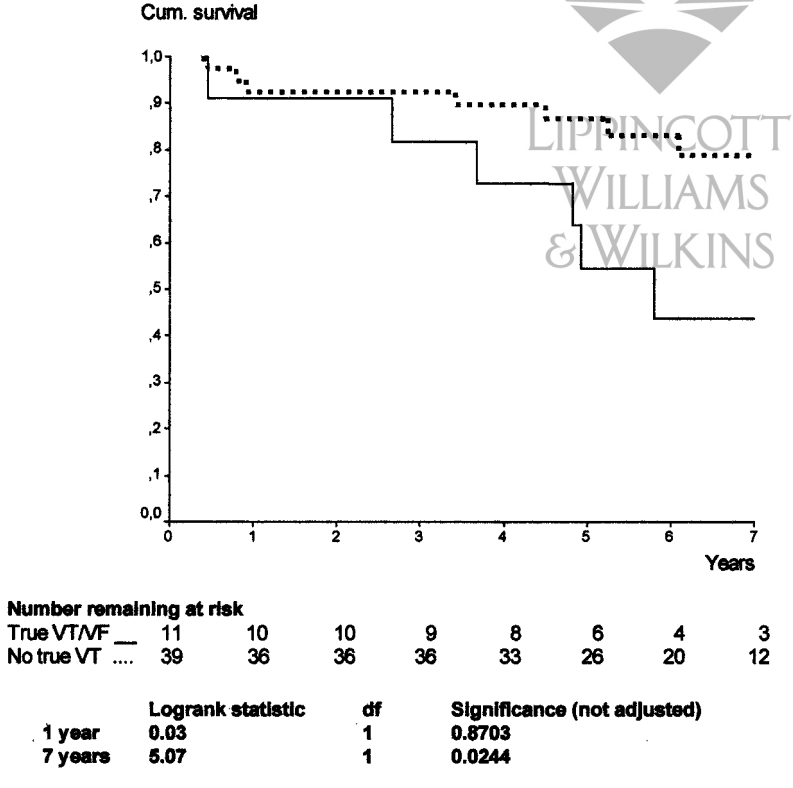


Figure 3. Survival of patients with and those without VTs in the ICD group.

rence of VTs may be closely associated with a progression of heart failure, such that VTs are a sign of cardiac deterioration rather than an independent risk factor of SCD. One study in patients with DCM and clusters of VTs supports this hypothesis. In this study, only 16% of patients survived and were not given transplantation 4 years after the first cluster of VTs, as opposed to 80% of patients without VTs.[31]

SCD and Defibrillator Use

VTs have been made responsible for 30% to 51% of sudden deaths in DCM.[13,15,16,26] In CAT, 11 patients received adequate therapies for VTs >200 bpm and 6 patients had syncope during VTs. However, this did not lead to a survival benefit. It has been suggested that the occurrence of fast VTs (>240 bpm) or VTs associated with syncope be used as a surrogate end point for SCD.[32] This concept is called into question by the findings of the present study.

Limitations

Because the overall mortality rate was too low, the study was stopped for futility after the pilot phase. Even if 1348 patients had been included, as initially planned, the trial would have been underpowered.

Conclusions

ICD therapy did not reveal any survival benefit in the setting of DCM of recent onset and impaired LV function (EF ≤30%). This was most likely due to the low overall mortality rate in the control group. However, even in patients with a significantly increased mortality rate caused by a lower EF and nonsustained VTs, ICD therapy did not reveal any survival benefit. Therefore, the results of CAT do not favor prophylactic ICD implantation in patients with DCM of recent onset and impaired LVEF without any further risk stratification.

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