Low-energy Synchronous Cardioversion of Ventricular Tachycardia Using a Catheter Electrode in a Canine Model of Subacute Myocardial Infarction

WARREN M. JACKMAN, M.D., AND DOUGLAS P. ZIPES, M.D.

SUMMARY The purpose of this study was to determine the feasibility and safety of terminating sustained ventricular tachycardia by low-energy, synchronized shocks delivered through transvenous, intracardiac catheter electrodes. Adult mongrel dogs underwent 2-hour occlusion-release of the left anterior descending coronary artery. Programmed electrical stimulation 3–8 days later in 14 of 24 surviving dogs induced 627 episodes of sustained ventricular tachycardia that had 35 morphologically distinct contours. Truncated exponential shocks, timed from the biphasic R wave recorded in the right ventricular apex, were delivered between the right ventricular apex (cathode) and superior vena cava (anode). Shocks of 0.008–1.0 J (median 0.5 J) reproducibly terminated 25 of 30 (83%) sustained ventricular tachycardias that had a cycle length (CL) ≥ 200 msec. One of five sustained ventricular tachycardias with CLs < 200 msec was terminated by ≤ 1.0 J. During ventricular tachycardias with a CL ≥ 200 msec, only 12 of 748 (1.6%) shocks of 0.008–1.0 J applied within 3% of the QRS produced repetitive ventricular responses, and none accelerated the ventricular tachycardia or produced ventricular fibrillation. Shocks ≥ 0.008 J in the T wave (nine of 85) or ≥ 0.5 J in the QRS of ventricular tachycardias with CLs < 200 msec (two of 17) produced ventricular fibrillation. Atrial flutter or atrial fibrillation, usually terminating within 3 seconds, occurred in 9% of shocks ≥ 0.5 J. The energy required to terminate sustained ventricular tachycardia was decreased 20–250-fold using an epicardial apex cone electrode for the cathode and the superior vena cava electrode for the anode, but was not significantly altered when the shock was delivered between electrodes in the right ventricular apex (cathode) and coronary sinus (anode). We conclude that transvenous, intracardiac, R-wave-synchronous shocks ≤ 1.0 J safely terminate sustained ventricular tachycardia with CLs ≥ 200 msec in dogs.

PERMANENT patient-activated or automatic ventricular pacemakers delivering single stimuli1–3 or rapid bursts4–7 have been used successfully to treat drug-resistant recurrent sustained ventricular tachycardia. However, single ventricular stimuli often fail to terminate ventricular tachycardia (primarily in tachycardias faster than 170 beats/min),8–11 and burst ventricular pacing may result in acceleration of ventricular tachycardia or ventricular fibrillation in as many as 43% of patients.3, 6, 7, 9, 11

Based on the safety and success of synchronized transthoracic direct-current cardioversion12–15 and the feasibility of administering intracardiac shocks,16–25 we reasoned that ventricular tachycardia might be safely and effectively terminated by synchronized shocks of very low energy delivered through intracardiac electrodes. The purpose of this study was to test this hypothesis using a canine model in which programmed electrical stimulation induced sustained ventricular tachycardia 3–8 days after coronary occlusion-reperfusion.26 We used a specially designed catheter electrode because if the technique were feasible, implantation by a transvenous route would have obvious advantages. We also tested the effectiveness of an epicardial apex cone electrode that would have to be implanted by a surgical procedure.21–25

Methods

Experimental Preparation

Forty-seven mongrel dogs that weighed 13–34 kg were anesthetized with secobarbital sodium (25 mg/kg, i.v.), intubated and mechanically ventilated with room air. Using an aseptic technique, a left thoracotomy was performed in the fourth intercostal space, the pericardium reflected, and the heart exposed. A 1-cm segment of the left anterior descending coronary artery (LAD) proximal to the main diagonal branches supplying the apical region was dissected free from surrounding tissue. All visible epicardial branches from the left circumflex and posterior descending coronary arteries in the apical region of the left ventricle were ligated. The isolated segment of the LAD was occluded using a Harris two-stage procedure.27 Two hours after complete occlusion, arterial flow was reestablished. Lidocaine (3 mg/kg, i.v.) was administered 3 minutes before reperfusion.26 The chest was closed in layers, and the dog was allowed to recover. Postoperative care was supervised by a licensed veterinarian.

Three to 8 days after myocardial infarction, 24 surviving dogs were anesthetized with α chloralose (80 mg/kg, i.v.), intubated and mechanically ventilated, and a median sternotomy was performed. Body temperature and arterial pH, PCO₂, and PO₂ were monitored and maintained within the normal physiologic range throughout the experiment.

A #10F electrode catheter, specially designed for cardioversion (Medtronic model 6880), was inserted...
through the right external jugular vein and advanced to
the right ventricular apex. The catheter consisted of
four stainless steel electrodes; two electrodes at the tip
separated by 5 mm and two similarly spaced electrodes
13 cm from the tip. Each electrode had a surface area
of 1.25 cm. The distal pair of electrodes located in the
right ventricular apex was used for bipolar R-wave
sensing to time cardioversion shocks. When a shock
was delivered, the distal pair of electrodes was coupled
together to form the cathode and the proximal pair of
electrodes (located in the superior vena cava) was
coupled together for the anode. In five dogs, a second
6880 electrode catheter was inserted into the left exteri-
or jugular vein and advanced into the distal coronary
sinus. The distal pair of electrodes on this catheter was
used as the anode, with the cathode being the two
electrodes of the first catheter located in the right ven-
tricular apex. Finally, in three dogs, a 5-cm diameter
cone electrode,25 composed of two stainless steel wire
mesh sheets separated by a 5-mm Silastic band, was
sutured onto the apex of the heart. The apex cone
electrode was the cathode and the superior vena cava
electrode formed the anode. Shocks were timed from the
bipolar R wave recorded between the two wire
mesh sheets.

A bipolar His bundle electrogram was recorded using a #6F electrode catheter inserted through the
right carotid artery and advanced retrogradely into the
right coronary sinus of the aortic valve. Bipolar stain-
less steel wire electrodes 1 mm apart, insulated with
Teflon except at the tips, were inserted using a 21-
gauge needle into the epicardium of the right ventricu-
lar outflow tract and the right ventricular apex over-
ying the tip of the 6880 electrode catheter. Five
additional bipolar electrodes were inserted circumfer-
entially into the epicardium around the border of the
anteroapical left ventricular infarction. The above
electrograms, a bipolar atrial electrogram, and scalar
ECG lead II or III were amplified, filtered (40-500 Hz
for electrograms and 0.08-50 Hz for scalar ECG),
displayed on an oscilloscope and recorded on light-
sensitive paper at 50 and 100 mm/sec. Programmed
electrical stimulation (1.8-msec rectangular stimuli at
twice late diastolic current threshold) of the epicardial
sites near the infarction border zone was used to induce
sustained ventricular tachycardia.26,28,29 Pancuronium
bromide (Pavulon), 4 mg i.v., was administered to
prevent skeletal muscle stimulation by cardioversion
shocks and the resultant electrogram interference.

**Cardioversion Protocol**

The cardioverter (Medtronic model 2316) delivered
a truncated exponential wave form,30 6 msec in dura-
tion, at nine energy levels: 0.0005, 0.001, 0.008,
0.025, 0.09, 0.5, 1.0, 2.0 and 2.5 J. A programmable
stimulator (Medtronic model 5325) was interfaced
with the pulse generator to provide the sensing func-
tion and timing for the cardioversion shocks.

After induction of sustained ventricular tachycardia,
shocks between the right ventricular apex and superior
vena cava were introduced at a fixed time within the
QRS complex. Beginning with 0.0005 J, shocks of
progressively increasing energy were delivered until
ventricular tachycardia was terminated. Ventricular
tachycardia was reinitiated and the same sequence re-
peated two to five times to determine the minimum
energy required to reproducibly terminate the tachy-
cardia at that time in the tachycardia cycle. Shocks of
greater energy (up to 2.5 J) were applied to determine
the safety of higher-energy pulses.

Similar testing was done at 5–20-msec intervals
throughout the tachycardia cycle. This protocol was
repeated with shocks delivered between the right ven-
tricular apex and coronary sinus in five dogs and be-
tween the epicardial apex cone electrode and superior
vena cava electrodes in three.

**Definitions**

**Sustained ventricular tachycardia** — ventricular
tachycardia that had a constant QRS contour, ventricu-
lar activation sequence and cycle length, and a dura-
tion of \( \geq 30 \) seconds.

During ventricular tachycardia: **QRS interval** — in-
terval between earliest and last recorded ventricular
activation in any of the intracardiac or surface electro-
grams; **ST-T interval** — interval between last recorded
ventricular activation and last recorded portion of the T
wave in any of the electrograms; **TQ interval** — in-
terval between end of the T wave and onset of ventricular
activation of the next tachycardia cycle; **VS interval** —
interval between earliest recorded ventricular activa-
tion and time at which the cardioversion shock was
applied.

**RVA(-)** — cathode composed of endocardial cath-
eter electrodes in the right ventricular apex.

**Apex cone (−)** — cathode composed of the epicardial
apex cone electrode.

**SVC (+)** — anode composed of catheter electrodes
in the superior vena cava.

**CS (+)** — anode composed of catheter electrodes in
the distal coronary sinus.

**Minimum termination energy** — the minimum
energy that terminated a single episode of ventricular tachy-
cardia at a specific time in the tachycardia cycle. If
more than one episode of ventricular tachycardia was
tested at the same time in the tachycardia cycle, the
largest value was used.

**Maximum required termination energy** — the largest
value of the minimum termination energies for a 25% por-
tion of the QRS interval.

**Repetitive ventricular response** — one or more sponta-
neous premature ventricular depolarizations after a
cardioversion shock, including acceleration of ventricu-
lar tachycardia and development of ventricular fibril-
lation.

**Repetitive atrial response** — one or more sponta-
neous premature atrial depolarizations after a cardiover-
sion shock, including atrial flutter and atrial fibril-
lation.

**Data Analysis**

Linear regression analysis was used to assess the
relationship between ventricular tachycardia cycle length and maximum required termination energy. Significant differences in maximum required termination energy between modes of stimulation (RVA[−] SVC[+] vs RVA[−] CS[+] and RVA[−] SVC[+] vs apex cone[−] SVC[+]) were determined by the Wilcoxon signed-rank test. A chi-square analysis for contingency tables was used to determine the relationship between the incidence of repetitive atrial responses, cardioversion energy levels and modes of stimulation (RVA[−] SVC[+] vs RVA[−] CS[+]).

Results

Energy Required for Termination of Ventricular Tachycardia

All dogs had sinus rhythm at the onset of study. Programmed electrical stimulation induced sustained ventricular tachycardia with constant cycle length, ventricular activation sequence and QRS morphology (fig. 1) in 14 of the 24 dogs (58%): three of nine dogs (33%) studied 3 days after infarction, five of eight dogs (63%) studied 4 days after infarction, and in six of seven dogs (86%) studied 5–8 days after infarction. Two or more morphologically different forms of sustained ventricular tachycardia, each with a distinct cycle length and ventricular activation sequence, were induced in 12 of the 14 dogs, for a total of 35 different ventricular tachycardias. A mean of 17.9 ± 29.7 (± SD) episodes of each tachycardia was induced, for a total of 627 episodes of sustained ventricular tachycardia tested by intracardiac DC shock. The mean ventricular tachycardia cycle length was 257 ± 63 msec.

Figure 2 shows the analog record of a successful termination of ventricular tachycardia by a shock delivered between the right ventricular apex and superior vena cava. For this tachycardia, the minimum termination energy is plotted as a function of time within the tachycardia cycle (VS interval) in figure 3. Between 20 and 40 msec from the onset of ventricular activation, shocks of 0.09 J reproducibly terminated the ventricular tachycardia. Six shocks of 1.0 J (not plotted) also terminated the tachycardia, and did not produce repetitive ventricular responses. Late in the QRS interval and within the first half of the ST-T interval, lower-

FIGURE 1. Programmed electrical stimulation induced sustained ventricular tachycardia 4 days after 2-hour occlusion-reperfusion of the left anterior descending coronary artery. ECG lead III, left atrial (LA) and left ventricular (LV) electrograms are shown. Sinus rhythm (cycle length 550 msec) is present at the left. After a train of eight left ventricular paced complexes (S₃) at a cycle length of 300 msec, two premature ventricular stimuli (S₂ and S₁) at coupling intervals of 210 msec initiated sustained ventricular tachycardia. After five cycles, the cycle length of the ventricular tachycardia became regular at 250 msec.

FIGURE 2. Termination of sustained ventricular tachycardia (cycle length of 245 msec) by a shock of 0.09 J delivered between the right ventricular apex (RVₐ) and superior vena cava. ECG lead III, right atrial (RA), His bundle (HBE) and epicardial electrograms were recorded by the RV outflow tract (RVOT), RVₐ, and the left ventricle (LV) around the border of the anterolateral infarction (LV₁-LV₃). LV₃ is the left ventricular electrogram recorded closest to the catheter electrode in the RVₐ in this and subsequent figures. The shock was introduced 25 msec after the onset of ventricular activation (VS 25), which is recorded earliest in lead III. The shock restored sinus rhythm, evidenced by the narrow QRS complexes preceding His bundle potentials (H) at a normal HV interval, and fixed association with atrial depolarization.
energy shocks produced repetitive ventricular responses, including acceleration of ventricular tachycardia to ventricular flutter. Shocks of 0.0005–0.025 J delivered late in the ST-T interval and within the TQ interval also terminated the tachycardia. These very low energy shocks did not depolarize simultaneously the ventricular myocardium at all recorded epicardial sites; rather, they resulted in a conducted ventricular depolarization similar to that produced by competitive ventricular pacing.

Enough determinations were made throughout the QRS interval in 12 ventricular tachycardias to examine the relationship between minimum termination energy and time the shock was applied for shocks delivered between the right ventricular apex and superior vena cava. In one of the 12 tachycardias, the minimum termination energy was constant (0.5 J) throughout the QRS interval. In eight tachycardias, the minimum termination energy was lowest in the first or second quarter of the QRS interval, while in three tachycardias it was lowest in the third quarter of the QRS interval.

The maximum required termination energy (shocks between the right ventricular apex and superior vena cava) for each of the 35 ventricular tachycardias is plotted as a function of tachycardia cycle length in figure 4. Of the 30 sustained ventricular tachycardias with a cycle length ≥ 200 msec (mean 275 ± 46 msec), 25 (83%) were reproducibly terminated by shocks of ≤ 1.0 J and 20 (67%) by shocks of ≤ 0.5 J. Only one of the five tachycardias with a cycle length < 200 msec (mean 145 ± 25 msec) was terminated reproducibly by shocks of ≤ 1.0 J. There was no significant linear or log-linear relationship between the maximum required termination energy and the tachycardia cycle length.

There was also no direct relationship between the
maximum required termination energy and the proximity of the earliest recorded epicardial activation during ventricular tachycardia to the site at which the shock was delivered. In fact, in two of the 10 tachycardias with cycle lengths $\geq 200$ msec and maximum required termination energy $\geq 1.0$ J, epicardial activation was recorded earliest in the right ventricular apex electrogram and was within 5 msec of the onset of ventricular activation recorded in the His bundle or surface electrograms.

The maximum required termination energy for shocks delivered between the right ventricular apex (cathode) and coronary sinus (anode) was not significantly different from that of shocks between the right ventricular apex (cathode) and superior vena cava (anode) for the seven tachycardias in the five dogs tested. For shocks delivered between the epicardial apex cone electrode (cathode) and the superior vena cava electrodes (anode), the maximum required termination energy was decreased by a factor of 20–250 for all six tachycardias in the three dogs tested (fig. 5).

Safety

Ventricular Response

The ventricular response to shocks between the right ventricular apex and superior vena cava, applied during ventricular tachycardia, is shown in figure 6.

Of the 748 shocks of 0.008–1.0 J introduced within the first 80% of the QRS interval, only 12 (1.6%) produced repetitive ventricular responses and none accelerated the ventricular tachycardia or precipitated ventricular fibrillation. For shocks of 2.0 and 2.5 J, there was an 11% incidence of repetitive ventricular responses of four or more complexes. However, these repetitive responses began more than 400 msec after the shock, had a cycle length longer than that of the original tachycardia, slowed progressively and terminated spontaneously (fig. 7). A similar response occurred frequently with conventional defibrillation using 10 J or more delivered across paddles placed on the heart.

Shocks introduced during the last 20% of the QRS interval and within the ST-T interval resulted in a different ventricular response (fig. 6). In this zone, shocks as low as 0.001 J produced a repetitive ventricular response. As the energy was increased progressively, multiple-beat repetitive ventricular responses or ventricular fibrillation occurred.

In the ventricular tachycardias with cycle lengths < 200 msec, the safety of shocks introduced during the QRS interval was inconsistent. Two of 17 shocks $\geq 0.5$ J placed within the first 80% of the QRS interval resulted in ventricular fibrillation (fig. 8).

Shocks delivered through the other two electrode configurations also did not produce repetitive ventricular responses when introduced within the first 80% of the QRS interval in tachycardias with a cycle length $\geq 200$ msec. Only one of 177 shocks of 0.008–1.0 J delivered between the right ventricular apex and coronary sinus and none of 181 between the epicardial apex cone electrode and superior vena cava resulted in repetitive ventricular response.

Atrial Response

Atrioventricular dissociation occurred frequently during ventricular tachycardia. Therefore, shocks synchronized to the QRS interval fell randomly in the atrial cycle. Shocks introduced during the second quarter of the atrial cycle occasionally resulted in repetitive atrial responses, including atrial flutter or fibrillation (fig. 9). When induced, atrial flutter or fibrillation usually terminated spontaneously within 2–3 seconds after onset.

The occurrence of a repetitive atrial response was directly related to the energy level of the shock ($p < 0.001$). Figure 10 shows the relative incidence of repetitive atrial responses for shocks at each energy level delivered between the right ventricular apex and superior vena cava. For shocks $\leq 0.09$ J, the incidence of atrial flutter or fibrillation was less than 2%. For shocks $\geq 0.5$ J, the incidence of atrial flutter or fibrillation was 5–11% (9% overall). Placing the anode in the coronary sinus did not significantly change the incidence of repetitive atrial responses or atrial flutter and fibrillation. For shocks delivered between the epicardial apex cone electrode and superior vena cava, atrial flutter or fibrillation occurred in two of 213 shocks (1%) $\leq 0.09$ J and in none of 10 shocks $\geq 0.5$ J.

Discussion

Shocks of 1.0 J or less, delivered through transvenous intracardiac catheter electrodes and synchronized to the QRS complex, reproducibly terminated 83% of the sustained ventricular tachycardias with cycle lengths $\geq 200$ msec that were induced in dogs 3–8 days after myocardial infarction. For shocks introduced within the first 80% of the QRS interval, repeti-
FIGURE 6. Incidence of repetitive ventricular response (RVR) produced by shocks delivered between the right ventricular apex and superior vena cava, plotted as a function of energy level and time within the tachycardia cycle at which shocks were applied. Data are cumulative for all tachycardias with cycle lengths \( \geq 200 \) msec. Time is normalized to percent of the QRS, ST-T and TQ intervals. Numbers inside boxes indicate the number of shocks tested. The percent of each box that is unfilled represents the percent of shocks that produced no RVR. The percent of each box that is coarsest stippled, finest stippled, or solid black represents the percent of shocks that produced repetitive ventricular responses of one to three complexes, four or more complexes (including acceleration of ventricular tachycardia and ventricular flutter), or ventricular fibrillation (VF), respectively.

FIGURE 7. A shock of 2.0 J, delivered between the right ventricular apex (RV) and superior vena cava (SVC) 60 msec after onset of ventricular activation, terminated the ventricular tachycardia (cycle length 230 msec) and produced a repetitive ventricular response. The first ventricular complex after the shock had an activation sequence slightly different from the subsequent seven beats. Four hundred sixty milliseconds after this complex, and 800 msec after the shock, a slower ventricular tachycardia with a different ventricular activation sequence and QRS morphology began. This repetitive ventricular response had an irregular cycle length and terminated spontaneously after seven complexes. The last three ventricular complexes are conducted sinus impulses, as evidenced by the preceding small His bundle potential (H). HBE = His bundle electrogram; RA = right atrium; RV = right ventricular outflow tract; LV = left ventricle.
Duration

VT

RVA

FIGURE 8. Ventricular fibrillation produced by a shock of 0.5 J (between right ventricular apex [RVA] and superior vena cava [SVC]) introduced 70 msec after onset of ventricular activation during ventricular tachycardia with a cycle length of 150 msec. Duration of ventricular activation during ventricular tachycardia is 90 msec. RA = right atrium; HBE = His bundle electrogram; RVA = RV outflow tract; LV = left ventricle.

itive ventricular responses were rare and acceleration of the ventricular tachycardia or degeneration to ventricular fibrillation never occurred. For shocks introduced during the ST-T interval, corresponding to the vulnerable period of the ventricular cycle,31 energies as low as 0.008 J accelerated the ventricular tachycardia or produced ventricular fibrillation. Since shocks were not synchronized to the atrial cycle due to the presence of atrioventricular dissociation, atrial flutter or fibrillation was precipitated in 9% of shocks \( \geq 0.5 \) J, but usually terminated spontaneously within 3 seconds.

Only one of five ventricular tachycardias with cycle length \(< 200 \) msec (\( \geq 300 \) beats/min) was terminated by shocks \( \leq 2.5 \) J. The reason for this is not clear. The electrophysiologic mechanism responsible for the more rapid ventricular tachycardias may differ from that of the slower ventricular tachycardias. More likely, if the area involved in the genesis of the ventricular tachycardia is relatively small and protected, possibly by an intervening region of partially excitable myocardium,8, 28, 32 rapid rates may increase the degree of protection by allowing the intervening tissue less time to recover excitability. Greater energies may be required to penetrate partially excitable tissue. The fact that a shock of 0.09 J terminated one of these tachycardias may be related to the timing of the shock in that particular tachycardia.

For ventricular tachycardias with cycle lengths \(< 200 \) msec, shocks \( \geq 0.5 \) J introduced within the QRS interval occasionally produced ventricular fibrillation. Since ventricular activation extended through most of the cycle length in these tachycardias, repolarization of some part of the ventricular myocardium (and therefore the vulnerable period) probably extended into the next QRS interval. There may exist no discrete period in the QRS interval during which a low-energy shock can safely terminate these very rapid tachycardias.

Placing the anode in the coronary sinus did not significantly decrease the energy requirement to terminate ventricular tachycardia or decrease the incidence of atrial fibrillation, and therefore appeared to offer no advantage over the single catheter electrode configuration. This result may be explained by a "short-circuiting" effect of the blood pool. Since the resistivity of blood is approximately one-third that of the total biologic media (blood, interstitial fluid and muscle),33 much of the current was probably lost to the blood pool between the right ventricular apex and coronary sinus. In contrast, the large epicardial apex cone electrode reduced dramatically (20–250-fold) the maximum required termination energy, although this finding is limited by the fact that only six ventricular tachycar-
Clinical Implications

The goal of this study was to determine whether our approach provided a safe and effective method to terminate ventricular tachycardia and, if so, to extend the testing to man. We have accomplished our goal in the dog, and preliminary data indicate that such an approach can be used successfully in man as well. 24

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Considerations of the Model

The ventricular tachycardia induced in this model is similar to that in man. 26, 28, 29 However, testing was done only after occlusion-release of the LAD. Occlusion-release of the left circumflex coronary artery could produce different sites of origin of ventricular tachycardia, and this could alter the energy requirements for a catheter electrode in the right ventricular apex. Finally, the catheter electrode we used was designed for man. The proximal two electrodes were situated high in the superior vena cava, generally 2–3 cm above the right atrial junction. This location may influence the incidence of induction of atrial fibrillation as well as the energy requirement to terminate ventricular tachycardia. Several times, shocks of 1.0 J restored sinus rhythm by terminating the atrial flutter or fibrillation previously induced.

Figure 10. Incidence of repetitive atrial responses (RAR) for shocks at each energy level delivered between the right ventricular apex (RVA) and superior vena cava (SVC) and between the RVA and coronary sinus (CS). The number of shocks tested is indicated above the bars. The percent of each bar that is un-filled represents the percent of shocks that produced no RAR. The percent of each bar that is cross-hatched, coarsely stippled, finely stippled, or solid black represents the percent of shocks that produced RARs of one complex, two or three complexes, four or more complexes, or atrial flutter or fibrillation.

Dias in three dogs were tested. The apparent decrease in energy requirement may relate in part to the pathway traveled by the current, so that shocks of less energy depolarized a region critical to the maintenance of tachycardia. More likely, the large surface area of the cone electrode reduced electrical resistance and the epicardial location minimized loss of current to the blood pool.
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W M Jackman and D P Zipes

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