Diphenylhydantoin in the Prevention of Recurring Ventricular Tachycardia

By Neil Stone, M.D., Michael D. Klein, M.D., and Bernard Lown, M.D.

SUMMARY
The efficacy of diphenylhydantoin in preventing recurrent ventricular tachycardia was studied in ten patients, nine of whom had documented coronary heart disease. Prolonged continuous ECG monitoring of up to 72 hr was performed in each case. Oral and intravenous loading doses of 1000 to 1500 mg were given on the first day of therapy to insure rapid attainment of therapeutic blood levels. Diphenylhydantoin was uniformly ineffective in preventing recurrence of ventricular tachycardia despite the presence of what is regarded as adequate therapeutic plasma concentrations (greater than 19.0 μg/ml in five patients, 10.8–18.0 μg/ml in the remaining five) shortly after onset of ventricular tachycardia. Nystagmus was noted in eight patients, but plasma diphenylhydantoin levels varied widely (8.6–35.0 μg/ml) when this sign of neurotoxicity was first detected.

Additional Indexing Words:
Coronary heart disease Myocardial infarction Nystagmus Sudden cardiac death

The lowering of hospital death rate from myocardial infarction (MI) achieved by coronary care units (CCU) has not substantially affected mortality from coronary heart disease (CHD).1 No significant inroad into coronary mortality is likely until the toll of sudden death occurring within the community is reduced.2 This is necessarily so, since about 65% of all coronary deaths transpire before the patient reaches a hospital.3 Both coronary care unit experience4 and several community based studies5, 6 have suggested that sudden death is due to ventricular arrhythmia. Many of these deaths occur within less than 1 hr, and are unwitnessed. It is, therefore, unlikely that any system of care will be completely effective unless the potential victim can be identified and treated prophylactically. Since there is no current method for predicting the individual likely to be afflicted with fatal arrhythmia, large groups presumed to be at risk will have to be subjected to prophylactic antiarrhythmic measures. Any drug or drugs to be employed in such a program would not only have to be effective in preventing ventricular tachycardia (VT) and ventricular fibrillation (VF), but also have to be free from serious and frequent adverse reactions.

Diphenylhydantoin (DPH), widely used as an antiepileptic drug, has recently gained popularity as an antiarrhythmic agent. It is a drug with but few serious side effects. When given in doses sufficient to produce a blood level of 10 to 20 μg/ml, it is reported to be consistently effective in suppressing ventricular ectopic activity.7 Therefore, administration of DPH might be utilized as a preventive measure against sudden cardiac death. Before it is to be employed in a mass prophylactic program, it is necessary for its efficacy in preventing VT and VF in the patient with ischemic heart disease to be established. While it is difficult to establish whether a drug protects against VF, effectiveness against VT

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is more readily ascertainable. The present study is, therefore, focused on this latter problem. Specifically, the question examined is whether, in patients with coronary heart disease having frequently recurring episodes of ventricular tachycardia, DPH is an effective prophylactic drug.

**Materials and Methods**

Ten patients with coronary heart disease, nine males and one female, experiencing frequent episodes of sustained VT were studied. Clinical information on these patients is listed in table 1. Nine had a documented history of MI. One patient (J.B.) was reported to have sustained a diaphragmatic myocardial infarction, although the electrocardiogram (ECG) at the time of development of VT was nondiagnostic, and coronary arteriograms revealed no occlusive lesions. In these ten patients the arrhythmia first occurred within 72 hr to 15 months after the episode of MI. In four patients the interval was less than 3 months, and in the remaining group the interval was longer. One patient had recurrent VT for more than 5 years.

VT was diagnosed in the presence of a tachycardia with a rate exceeding 120 beats/min, a bizarre QRS morphology, and A-V dissociation. Independent P-wave activity, if not discernable in conventional leads, was verified with atrial electrograms, recorded from a transvenously placed right electrode wire. In all patients, onset of the tachycardia was recorded on a number of occasions. In each instance the initial complex was premature, and demonstrated the same abnormal morphology that characterized the ensuing tachycardia. All patients exhibited frequent ventricular premature beats which resembled in configuration the ventricular complex of the underlying arrhythmia. Fusion and capture beats were consistently noted. If the arrhythmia was sustained for more than 30 to 60 min, evidence of hemodynamic deterioration developed, manifesting in pulmonary congestion, hypotension, or both. This was true even when the rate was less than 150 beats/min.

All patients were monitored continuously for periods ranging up to 72 hr while receiving antiarrhythmic drug therapy with DPH. Monitoring was accomplished by one or more of three methods: (1) surveillance on a CCU slave oscilloscope and memory tape system; (2) tele-metering the ECG signal through special telephone lines linked to a special monitoring console on the CCU; and (3) recording the ECG on a modified Holter portable tape recorder with playback at 60 times real time on an Avionics audiovisual analyzer. Two patients underwent treadmill exercise testing* to verify the efficacy of DPH in preventing VT induced by stress.

DPH was given in loading dose administered over 24 to 48 hr, followed by a maintenance dose. While loading dose varied, each patient received from 1000 to 1500 mg on the first day, followed by 400 to 1500 mg on the second day, and a maintenance dose of 400 to 600 mg thereafter (table 2). If DPH was given during a bout of VT, an intravenous or intramuscular route was employed. When administered intravenously, the drug was dissolved in commercial diluent (40% propylene glycol and 10% ethanol in water, pH 12). Injection rates did not exceed 50 mg/min, and were performed under continuous electrocardiographic monitoring with frequent blood pressure checks. Blood samples for DPH analysis were withdrawn from a venous site separate from that used for drug infusion.

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<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Location of infarction</th>
<th>Duration of VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.G.</td>
<td>61</td>
<td>M</td>
<td>Diaphragmatic and lateral</td>
<td>14 months</td>
</tr>
<tr>
<td>R.B.</td>
<td>62</td>
<td>M</td>
<td>Diaphragmatic</td>
<td>3 months</td>
</tr>
<tr>
<td>R.P.</td>
<td>53</td>
<td>M</td>
<td>Anterior</td>
<td>2 months</td>
</tr>
<tr>
<td>T.D.</td>
<td>44</td>
<td>M</td>
<td>Anteroseptal and posterior</td>
<td>1 day</td>
</tr>
<tr>
<td>G.Fl.</td>
<td>49</td>
<td>M</td>
<td>Anteroseptal</td>
<td>4 years</td>
</tr>
<tr>
<td>G.Fo.</td>
<td>65</td>
<td>F</td>
<td>Diaphragmatic</td>
<td>6 months</td>
</tr>
<tr>
<td>H.H.</td>
<td>63</td>
<td>M</td>
<td>Diaphragmatic</td>
<td>9 months</td>
</tr>
<tr>
<td>J.B.</td>
<td>40</td>
<td>M</td>
<td>Diaphragmatic*</td>
<td>5 years</td>
</tr>
<tr>
<td>D.P.</td>
<td>54</td>
<td>M</td>
<td>Diaphragmatic intramural†</td>
<td>1 month</td>
</tr>
<tr>
<td>J.H.</td>
<td>61</td>
<td>M</td>
<td>Anteroseptal</td>
<td>1 day</td>
</tr>
</tbody>
</table>

*No evidence on electrocardiogram of infarction, coronary angiogram normal.
†Patient experiencing VT in association with Prinzmetal's variant form of angina pectoris.
### Table 2

**Dose of DPH and Blood Levels in 10 Patients with VT**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Weight (kg)</th>
<th>Loading dose (mg)</th>
<th>Dose on day 2 (mg)</th>
<th>Daily maintenance (mg)</th>
<th>Blood levels (µg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>W.G.</td>
<td>94</td>
<td>1000</td>
<td>1600</td>
<td>500</td>
<td>8.6</td>
</tr>
<tr>
<td>R.B.</td>
<td>67</td>
<td>1000</td>
<td>1000</td>
<td>400 i.v.</td>
<td>20.0</td>
</tr>
<tr>
<td>R.P.</td>
<td>77</td>
<td>1500</td>
<td>500</td>
<td>500</td>
<td>11.0</td>
</tr>
<tr>
<td>T.D.</td>
<td>88</td>
<td>1000</td>
<td>400</td>
<td>400</td>
<td>13.8</td>
</tr>
<tr>
<td>G.Fi.</td>
<td>77</td>
<td>1000</td>
<td>800 i.v.</td>
<td>600</td>
<td>16.8</td>
</tr>
<tr>
<td>G.Fo.</td>
<td>70</td>
<td>1500</td>
<td>400</td>
<td>400</td>
<td>18.2</td>
</tr>
<tr>
<td>H.H.</td>
<td>50</td>
<td>500 i.m.</td>
<td>500</td>
<td>500</td>
<td>14.0</td>
</tr>
<tr>
<td>J.B.</td>
<td>70</td>
<td>150 i.m.</td>
<td>600</td>
<td>600</td>
<td>8.6</td>
</tr>
<tr>
<td>D.P.</td>
<td>83</td>
<td>1500</td>
<td>1000</td>
<td>500</td>
<td>12.6</td>
</tr>
<tr>
<td>J.H.</td>
<td>82</td>
<td>1600</td>
<td>100</td>
<td>500</td>
<td>5.0</td>
</tr>
</tbody>
</table>

Plasma DPH concentration was determined by the colorimetric method of Dill et al. Duplicate samples agreed within ± 0.3 µg/ml with standard solutions, all prepared from powdered lot no. 593611.* Drug levels were measured on the first day of therapy if VT occurred or if signs of neurotoxicity appeared, such as nystagmus, slurred speech, blurred vision, or ataxic gait. On subsequent days, blood samples were drawn within 1 hr of the next scheduled maintenance dose of DPH or during recrudescence of VT. In eight patients absence of lateral gaze nystagmus was confirmed prior to administration of DPH. During drug treatment nystagmus was searched for on repeated examinations. When nystagmus was first detected, blood specimens were taken for DPH determinations.

**Results**

DPH failed to prevent recurrence of VT in each of the ten patients (table 3). In three patients inspection of monitoring data obtained over several hours suggested a reduction in the incidence of VT and ventricular ectopic frequency. However, more detailed analysis of monitoring records obtained over

*Supplied by Dr. A. Glazko, Parke-Davis, Detroit, Michigan.

### Table 3

**Result of DPH and Ultimate Effective Therapy**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Result of DPH</th>
<th>Effective therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.G.</td>
<td>VT unchanged</td>
<td>Cholecystectomy; propranolol (160 mg/day)</td>
</tr>
<tr>
<td>R.B.</td>
<td>Ectopic activity in salvos and brief runs of VT</td>
<td>Propranolol (320 mg/day)</td>
</tr>
<tr>
<td>R.P.</td>
<td>VT unchanged</td>
<td>Aneurysmectomy; procainamide (2.0 g/day)</td>
</tr>
<tr>
<td>T.D.</td>
<td>Short paroxysm of VT</td>
<td>Quinidine (1.2 g/day); propranolol (80 mg/day)</td>
</tr>
<tr>
<td>G.Fi.</td>
<td>VT precipitated by exercise and recurred on day 5</td>
<td>Procainamide (6.0 g/day); propranolol (30 mg/day)</td>
</tr>
<tr>
<td>G.Fo.</td>
<td>VT unchanged</td>
<td>No effective treatment; death during aneurysmectomy</td>
</tr>
<tr>
<td>H.H.</td>
<td>VT unchanged</td>
<td>Procainamide (4.0 g/day); isosorbide dinitrate in large dose</td>
</tr>
<tr>
<td>J.B.</td>
<td>VT precipitated by exercise</td>
<td>Procainamide (6.0 g/day)</td>
</tr>
<tr>
<td>D.P.</td>
<td>Short paroxysms and sustained VT on day 5</td>
<td>Propranolol (160 mg/day); isosorbide dinitrate in large dose</td>
</tr>
<tr>
<td>J.H.</td>
<td>VT unchanged</td>
<td>Quinidine (2.4 g/day)</td>
</tr>
</tbody>
</table>

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48 to 96 hr showed no modification in frequency, duration, or type of ventricular arrhythmia. Salvos of ventricular premature beats (VPB's) and brief paroxysms of VT persisted, and were not influenced by the time elapsed from the previous dose of DPH. In two patients who had less frequent episodes of VT after DPH was administered to the point of nystagmus, the arrhythmia was precipitated by means of modest exercise on a motorized treadmill.

Bigger and coworkers7 have emphasized the need for a large loading dose of the drug in order to achieve expeditiously an effective blood level. Nine of the ten patients received such loading doses of 1000 to 1500 mg during the first day of therapy. Four of these subjects received an additional 1000 mg or more on the second day (table 2). Levels were determined promptly after recurrence of VT. Five of the ten patients had plasma concentrations greater than 19.0 μg/ml. The other five patients had levels ranging between 10.8 and 18.0 μg/ml. Nystagmus was detected in all eight patients in whom it was looked for. Plasma DPH levels exceeded 8.0 μg/ml in all these cases, and ranged from 8.6 to 35.0 μg/ml at the time when neurotoxicity was first detected.

The following patient is illustrative of this experience: G.Fi., (Hospital Number 10-34-33), a 49-year-old male, had sustained an anterior wall myocardial infarction 11 years earlier. Thereafter ECG suggested ventricular aneurysm (fig. 1). VT first developed 4 years after infarction, and was readily terminated

Figure 1

Electrocardiographic changes consistent with anterior transmural infarction of uncertain duration. The elevated ST segments in precordial leads suggest the possibility of ventricular aneurysm.

Figure 2

Right bundle-branch block, A-V dissociation, and ventricular rate of 125 beats/min suggest mechanism to be that of ventricular tachycardia.
by means of cardioversion. VT recurred, however, with ever shorter intervals between successive attacks. The ECG pattern of the arrhythmia was that of right bundle-branch block (fig. 2). The rate generally did not exceed 135 beats/min, and the mechanism was unaffected by carotid sinus pressure. A loading dose of 1000 mg of DPH was given orally the first day, followed by 300 mg the next morning, at which time the blood level was 23.6 μg/ml. Later that same day VT recurred; DPH was thereupon administered intravenously in a dose of 100 mg every 10 min. Nystagmus first appeared after a dose of 400 mg, which resulted in a blood level of 27.8 μg/ml. After an additional 500 mg of DPH intravenously, the patient complained of dizziness and diplopia; he became dysarthric, confused, and agitated. A blood level of 39.7 μg/ml was recorded. VT persisted (fig. 3), and was finally terminated with a cardioversion discharge of 1 w-sec. While DPH failed to abolish VT, it remained uncertain whether it might prove effective in preventing recurrences. DPH was therefore continued in a dose of 800 mg daily. Blood levels persisted in the 30.0 μg/ml range (table 2). On the fifth day VT redeveloped, requiring cardioversion. On the sixth day the arrhythmia was precipitated by treadmill exercise; at this time the blood level was 38.0 μg/ml. A successful antiarrhythmic program was eventually evolved, including procainamide and propranolol in respective daily doses of 6.0 g and 30.0 mg.

Whereas DPH uniformly failed to prevent VT, other antiarrhythmic measures were successful in eight of the ten patients, and are listed in table 3. In two cases multiple drug regimens, including large doses of bretylium tosylate, propranolol, procainamide, and quinidine, were tried without avail. Overdrive ventricular pacing at rates of 80 to 120 beats/min was similarly unsuccessful. Both patients had angiographically demonstrable ventricular aneurysms. Patient G.Fo. died on the operating table during attempted aneurysmectomy. Resection of the aneurysm in patient R.P. abolished disabling daily attacks of VT, though ventricular ectopic beats continued and required procainamide to reduce their frequency.

**Discussion**

Experimental evidence for the antiarrhythmic action of DPH first appeared in 1950 with the work of Harris and Kokernot. Using large doses intravenously (20–50 mg/kg), they abolished VT that followed coronary artery ligation in dogs. They postulated that DPH suppressed discharge between injured and normal myocardium analogous to its action in subduing cortical foci in epilepsy. In 1954 Mosey and Tyler demonstrated that DPH abolished ouabain-induced ventricular arrhythmias. Scherf et al. found the drug effective in VT when the arrhythmia followed focal application of the chemical irritants, aconitine and delphinine. Covino and coworkers noted that DPH decreased the tendency to ventricular fibrillation resulting from immersion hypothermia in dogs. White et al. reported that large doses of DPH abolished epinephrine-cyclopropane induced arrhythmias in the experimental animal. Helfant and coworkers found that DPH protected against cardioversion-provoked VT in the digitalized animal. These findings indicate that in the experimental animal, DPH is effective against
ventricular tachyarrhythmias of diverse pathogenesis.

Demonstration of DPH antiarrhythmic action in man had a less auspicious beginning. In 1951 Nadas et al. used DPH to control recurrent VT in a 51-year-old girl who had sustained a basal skull fracture. The drug was given in a divided dose of 300 mg daily for a week. Although the electroencephalogram improved, VT was not prevented. The antiarrhythmic efficacy of DPH in man was first proved by Leonard 7 years later in a patient with VT after myocardial infarction. Though the arrhythmia was refractory to quinidine and procainamide, intravenous DPH in a dose of 250 mg abolished the disorder. Subsequent doses given over longer intervals were required to maintain sinus rhythm. Since this initial success, a number of reports have offered testimony of the antiarrhythmic properties of DPH in a variety of ventricular and atrial arrhythmias. These experiences, summarized in several recent reviews, indicate that the drug has little effect in chronic atrial rhythm disorders, such as atrial flutter and fibrillation, but is of value in treating ventricular ectopic mechanisms, especially when these result from digitalis toxicity or complicated anesthesia, or when they are induced by cardiac catheterization or by surgical operation.

The effect of DPH on ventricular arrhythmias in patients with coronary artery disease is analyzed in the extensive study by Mercer and Osborne. They treated a total of 774 arrhythmic episodes with intravenous DPH. Of this number, 676 were ventricular and 218 occurred in patients with ischemic heart disease. Drug was given in increments of 125 mg, not exceeding 500 mg/hr. Complete correction of the arrhythmia, though at times transient, was achieved in 27.5% of the patients with CHD. A nearly similar success rate was noted in those with angina pectoris (23%) as in those who had suffered prior MI (33%). By contrast, in the 226 episodes of ventricular arrhythmias in postcardiac surgical patients, the rate of reversion was 61%. In the 184 episodes of ventricular arrhythmia following anesthesia, cardioversion, and cardiac catheterization, DPH was an effective antiarrhythmic agent in 80%.

Mercer and Osborne cite 17 patients with VT reported in the literature, including eight of their own, who responded to DPH. Bigger et al., in their careful studies, noted that DPH reverted all their eight cases of VT to sinus rhythm. It is uncertain in how many of the above cited episodes the arrhythmia was the result of ischemic heart disease, however. Eddy and Singh restored sinus rhythm with 250 mg of DPH administered intravenously over 5 min in four patients with VT after acute MI. With the exception of Helfant et al., who failed to revert each of three patients with VT, DPH, when administered intravenously, has been reported as consistently effective in terminating this arrhythmia.

In the present study DPH was uniformly ineffective in preventing recurrence of VT. Bigger and coworkers have emphasized that effectiveness of DPH as an antiarrhythmic depends largely on achieving a blood level of 10 to 18 \( \mu \text{g/ml} \). It is likely that many treatment failures with DPH are indeed attributable to an inadequate blood concentration, since DPH is commonly prescribed in divided doses of 300 to 400 mg daily without a loading dose. Patients receiving 400 mg DPH orally attain a maximum plasma level of only 2 to 5 \( \mu \text{g/ml} \) in an average of 8 hr. If a 70-kg man is given 100 mg of DPH four times daily, it will require approximately 6 to 9 days to reach a concentration ranging from 10 to 25 \( \mu \text{g/ml} \). The serum DPH level rises linearly with increasing dose at about 3.5 \( \mu \text{g/ml} \) for each milligram of drug per kilogram of body weight. Kutt and McDowell showed that a 1000-mg loading dose given in divided increments over a few hours produced an antiepileptic level of 10 to 20 \( \mu \text{g/ml} \) in 3 to 10 hr. To sustain this concentration it required 500 mg on the second day and 400 mg in divided daily doses thereafter. In the present study, all patients received a large loading dose of DPH. In nine of the ten patients VT was not prevented despite a blood level exceeding 10 \( \mu \text{g/ml} \). In four patients VT persisted or

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recurred, notwithstanding a DPH level equal to or greater than 20 μg/ml. Bigger et al.\textsuperscript{7} have recommended that further DPH administration be abandoned upon development of nystagmus; this neurotoxic manifestation was found in all eight patients in whom this sign was sought.

The reason for the failure of DPH as a prophylactic agent against VT is unclear. The explanation may relate to blood level of the drug. Assuming that DPH is effective in preventing VT due to ischemic heart disease, it may be that peak blood levels have to be continuously maintained to overcome the tendency to tachycardia which is consistently present in these patients. The difference in result between abolishing and preventing VT would then be accounted for. To terminate an episode of VT requires but a momentary achievement of a critically effective therapeutic level. This can be readily accomplished by administering intravenously a small total dose. Even though the blood concentration reached may be associated with neurotoxicity, this is of little clinical consequence, however, since the adverse reaction is brief and unlikely to be detected. Observations of Glazko and coworkers\textsuperscript{29} are relevant in this context. They administered 250 mg intravenously over 5 min to normal human volunteers. Though a plasma level as high as 17.4 μg/ml was reached, this concentration receded to a mean value of 6.3 μg/ml within 20 min. When the drug is used for prophylaxis against paroxysmal VT, the route of administration is oral; peak levels equivalent to those following intravenous injection are not reached nor can they be maintained; tachycardia is, therefore, likely to recur.

The above explanation is difficult to reconcile with findings in the present study. Blood concentrations were checked promptly after redevelopment of arrhythmia and were found consistently to be at what is regarded as an adequate therapeutic range. In five patients the concentration was approaching a potential toxic level, and four exhibited nystagmus. The explanation for DPH failure may rather relate to the etiology of VT. Mercer and Osborne,\textsuperscript{32} in their exhaustive study, demonstrated that when VT is due to ischemic heart disease, intravenous DPH is effective in only 30% of patients. These clinical observations are consistent with the experimental findings of Yenkomshian and coworkers\textsuperscript{30} who noted that DPH did not protect against death from ventricular arrhythmia in animals with coronary artery occlusions. Unanesthetized dogs were subjected to abrupt closure of the left anterior descending artery by inflating a previously placed balloon around this vessel. Though DPH was given in a dose that resulted in high blood levels exceeding 20 μg/ml, VT and VF occurred in 70% of animals, an incidence identical to that of the control group.

The findings to date suggest that DPH is not a consistently effective drug for VT when the arrhythmia is due to coronary artery disease. The results of the present study suggest that DPH is an unsuitable agent for use in prevention of sudden death from ischemic heart disease.

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Correction
Stone N, Klein MD, Lown B: Diphenylhydantoin in the prevention of recurring ventricular tachycardia. Circulation 43: 420, 1971. On page 426, the last sentence should be replaced by the following:
If the consistent failure of DPH to prevent VT in the patients studied is generally applicable to VT in other patients with coronary artery disease, it appears improbable that DPH would be suitably effective in the prevention of sudden death from ischemic heart disease due to VT and VF.