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# Phenol Motor Point Blocks in Children: Plasma Concentrations and Cardiac Dysrhythmias

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The use of dilute phenol (5% in water) for neurolysis at the motor point insertion ("motor point block") has been reported to provide temporary relief of spasticity in children.[1] Topically administered phenol in higher concentrations for chemical face-peels is associated with cardiac dysrhythmias in up to 30% of adults,[2,3] but only a single case report[4] of a phenol-associated cardiac dysrhythmia in a child has been published. The current study was undertaken to determine the incidence of cardiac dysrhythmias in children receiving phenol parenterally while anesthetized with halothane in a day-surgery suite and to ascertain doses that might predispose to such dysrhythmias.

## CASE REPORTS

Twenty-four patients scheduled for motor point block were initially enrolled in the study after approval of the protocol by our Medical Research Review committee had been given and parental informed consent obtained. All data are presented as mean ± standard deviation. The patients' ages were 85.4 ± 52 months, and their weights were 19 ± 10 kg. No preanesthetic medication was given. Preoperatively in the day-surgery suite they were scheduled for 12-lead ECG, echocardiography (two-dimensional, M-mode, ATL MK600 or Ultramark 8, Advanced Technology Laboratories, Bothell, WA), and application of Holter monitor (models 90201/90205, Space Labs, Seattle, WA) to serve as a their own control prior to subsequent administration of halothane and phenol.

In the induction room general anesthesia was induced with halothane (maximum 2%), nitrous oxide (60%), and oxygen (40%) after placement of routine monitoring equipment (ECG, precordial stethoscope, pulse oximetry, and end-tidal carbon dioxide and automatic blood pressure monitoring [Dinamap®]). Halothane concentration was reduced to 1 to 1.5% for the remainder of the procedure, and end-tidal carbon dioxide tension was maintained between 35–45 mmHg. No opioids were administered. An intravenous catheter was inserted into a vein of the upper extremity for intravenous fluid administration and subsequent blood sampling. A control whole blood sample was obtained immediately prior to the administration of phenol intramuscularly at the motor point site.

After alcohol-swabbing of the skin and placement of a grounding lead, a Teflon®-coated 22-G needle was connected to a nerve stimulator (0.1 ms/1–10 mA/0.5 Hz, battery-powered) and syringe (containing 5% phenol in water). Upon eliciting twitching of the involved muscles, with negative aspiration of blood to avoid intravascular needle placement, the phenol was injected slowly until twitching ceased. The total number of muscle sites injected, the amount of phenol given, and the duration required for the complete phenol administration was determined clinically by the physiatrist performing the blocks. The clinical criteria depended on the patient's spastic involvement, on facility in identifying the motor point insertion, and on the cessation of twitching as the end-point of administration of phenol. In all cases the total amount of phenol given was noted. All patients in this study had only lower extremity blocks involving adductor, gastrocnemius, and hamstring muscle groups.

Upon completion of the phenol administration at all sites and simultaneous termination of anesthetic agents, venous blood samples (7 ml each) were obtained *via* the intravenous catheter at 5, 15, 30, 60, and 120 min and frozen at −35 °C pending assay. Two hours after cessation of anesthesia and phenol administration, Holter monitoring was discontinued, and repeat 12-lead ECG and echocardiogram again were obtained.

Assays for plasma phenol concentrations were performed at the NIH Mass Spectrometry Research Resource, the University of Colorado Health Science Center, using a modification of the procedure of Cline *et al.*[5] Each blood sample was spiked with an internal standard (paracresol) to a concentration of 25 μg/ml. The samples were extracted using isooctane:diethyl ether (80:20 vol:vol). The phenols were derivatized with heptafluorobutyric anhydride to form volatile compounds that could be subjected to gas chromatography. Separation on the gas chromatograph was accomplished using a 15-m capillary column (DB-1, J&W Scientific, Cordorba, CA). Under the conditions of analysis, phenol eluted in 1.8 ± 0.1 min and paracresol (4-methyl phenol) at 3.0 ± 0.1 min. To identify each peak further, the gas chromatograph was interfaced to a mass spectrometer (Hewlett-Packard 5970 MSD),

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TABLE 1. Patient Characteristics and Data after Motor Point Block

| Patient Number | Age (months) | Weight (kg) | Phenol Injections | | Blood Phenol Concentration (μg/ml) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Dose (mg/kg) | Duration (min) | Control | 5 | 15 | 30 | 60 | 120 |
| 1 | 46 | 11.3 | 37.6 | 27 | 0.3 | 8.4 | 8.9 | 7.3 | 3.2 | 0.9 |
| 2 | 71 | 12.5 | 16.0 | 13 | * | 5.4 | 5.4 | 4.3 | 2.5 | 0.9 |
| 3 | 68 | 15.0 | 23.3 | 25 | * | 9.3 | 5.1 | 4.3 | 1.5 | 0.4 |
| 4 | 240 | 55.0 | 6.7 | 31 | * | 3.1 | 2.1 | 1.5 | 0.5 | 0.1 |
| 5 | 52 | 15.8 | 25.6 | 37 | 0.3 | 8.7 | 10.2 | 7.1 | 4.9 | 2.3 |
| 6 | 56 | 13.6 | 39.3 | 43 | 0.3 | 9.8 | 14.1 | 10.9 | 7.5 | 1.9 |
| 7 | 156 | 30.0 | 30.2 | 36 | 0.3 | 9.2 | 9.1 | 7.0 | 6.0 | 2.7 |
| 8 | 54 | 13.0 | 15.4 | 28 | 0.3 | 4.0 | 5.0 | 3.5 | 2.1 | — |
| 9 | 121 | 31.3 | 13.1 | 22 | 0.4 | 5.4 | 4.5 | 3.7 | 1.8 | 1.3 |
| 10 | 39 | 14.3 | 26.2 | 33 | 0.4 | 8.0 | 8.3 | 8.6 | 4.5 | 1.7 |
| 11 | 96 | 17.0 | 33.5 | 33 | * | 9.4 | 6.6 | 7.2 | 4.7 | 0.5 |
| 12 | 108 | 28.5 | 24.0 | 33 | * | 7.0 | 8.2 | 6.2 | 3.2 | 0.4 |
| 13 | 92 | 17.0 | 29.4 | 45 | * | 7.2 | 6.2 | 3.2 | — | — |
| 14 | 123 | 31.0 | 12.9 | 42 | * | 2.5 | 2.0 | 0.7 | * | * |
| 15 | 75 | 16.0 | 22.5 | 32 | 0.8 | 6.8 | 9.7 | 7.3 | 4.4 | 1.2 |
| 16 | 192 | 25.0 | 45.0 | 36 | 0.3 | 20.5 | 20.0 | 15.4 | — | — |
| 17 | 38 | 13.1 | 39.7 | 33 | 0.4 | 17.8 | 17.0 | 13.2 | 3.7 | 2.5 |
| 18 | 51 | 10.4 | 57.2 | 40 | * | 13.1 | 18.5 | 19.4 | 11.1 | 3.5 |
| 19 | 86 | 21.3 | 30.0 | 33 | * | 15.5 | — | 11.9 | 3.6 | — |
| 20 | 53 | 12.5 | 44.0 | 25 | * | 8.5 | 8.5 | 8.9 | 4.9 | 2.0 |
| 22 | 50 | 11.0 | 70.0 | 37 | 0.3 | 31.8 | 36.0 | 36.1 | 27.7 | 10.6 |
| 23 | 38 | 14.0 | 48.6 | 35 | * | 25.0 | 32.6 | 31.8 | 24.0 | 10.4 |
| 24 | 59 | 11.8 | 51.3 | 41 | * | 21.1 | 20.3 | 18.2 | 14.1 | — |
| Mean | 85.4 | 19.1 | 32.2 | 32 | 0.4 | 11.2 | 11.7 | 10.3 | 6.8 | 2.5 |
| SD | 52.0 | 10.4 | 15.6 | 8 | 0.1 | 7.4 | 9.1 | 8.9 | 7.3 | 3.1 |

Dash indicates that no specimen was obtained. Patient 21 was excluded because of insufficient data samples.

* Blood concentration was less than the lowest standard used.

and the areas of the molecular ions of the derivatives of phenol (mass-to-charge ratio 290) and paracresol (mass-to-charge ratio 304) were measured. The area ratio of phenol/paracresol was used to measure concentration in each sample. The quantitation technique was validated using normal plasma (shown to be free of peaks at the retention times of phenol and paracresol) that was spiked with phenol over a concentration range of 1–50 μg/ml blood. At the lowest test concentration (1 μg/ml), we had a signal-to-noise ratio of > 100. This allowed us to extrapolate to background levels of 0.1 ± 0.1 μg/ml phenol.

ECG, echocardiogram, and Holter monitor interpretations all were performed by one of the authors (RW) at the end of the study, prior to the assay, and without knowledge of the patients' clinical course. The shortening fraction (*i.e.*, the percentage change from diastolic to systolic dimension during one complete cardiac cycle; normal range 28–45%) was obtained from the echocardiograms performed approximately 1 h before and 2 h after administration of halothane and phenol. Holter monitor interpretation was according to the Lown grading system[6]: 1 = infrequent ventricular premature contractions (VPCs) less than 30 per hour; 2 = frequent VPCs (including bigeminy), more than 30 per hour; 3 = multiform VPCs; and 4 = sequential ectopy (ventricular couplets and/or ventricular tachycardia).

One patient was excluded from the analysis because of insufficient sampling of blood specimens. The remaining 23 patients received 32 ± 16 mg/kg phenol (range 6.7–70) over a period of 33 ± 8 min (range 13–45). Plasma phenol concentrations are presented in table 1. Data are significant to ± 0.1 μg/ml. Figure 1 illustrates the correlation between phenol total dose administered and peak phenol plasma concentrations. (Patient 19 was excluded because of absence of the 15-min sample). Because of nonfunctioning catheters, blood phenol specimens were not obtained from all patients at the scheduled times. Missing values (table 1) indicate that no blood sample was obtained or, if a control sample, that the concentration of phenol was less than the lowest standard used.

Sixteen patients had successful Holter monitoring. Two patients had dysrhythmias *prior* to the administration of phenol. Patient 14 had a grade-1 onset prior to arrival in the operating suite; patient 24 experienced a grade-1 dysrhythmia coincident with the onset of anesthetic induction. Another patient (11), who had the only grade-4 dysrhythmia

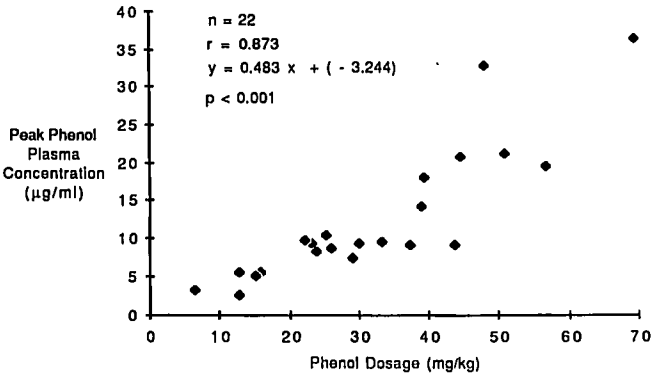


FIG. 1. Relationship between dose of phenol administered (mg/kg) and peak phenol plasma concentration (μg/ml). Patient 19 is excluded because the 15-min sample was not obtained.

recorded, experienced clinical laryngospasm attributed to "light anesthesia" when the Teflon® needle was initially inserted and during administration of approximately 0.5 ml phenol. Plasma phenol concentration from a specimen drawn immediately was 0.85 μg/ml. No other dysrhythmias were noted during subsequent phenol administration.

Three other patients (19%) demonstrated dysrhythmias after phenol administration. Patient 16 was noted to have brief VPCs intraoperatively (a grade-2 dysrhythmia), whereas the other two were observed on Holter analysis only after departure from the operating suite. Patient 23 had a grade-2 dysrhythmia, and patient 2 had a grade-1 dysrhythmia.

Electrocardiograms of all patients were normal both before and 2 h after phenol administration. Likewise echocardiography analysis revealed a mean shortening fraction of 36.7% ± 5% before administration of halothane and phenol and 37.2% ± 6% 2 hours after cessation of anesthetic ($P$ = not significant). Ventricular function was normal before and 2 h after phenol administration in all cases.

## Discussion

Cardiac dysrhythmias in children, associated with administration of phenol (5% in water) during halothane anesthesia occurred in 3 of 16 patients (19%). Three other patients studied also were noted to have dysrhythmias, but those were not attributable to phenol. The most impressive dysrhythmia, a grade 4 on the Lown scale, was attributed to mild laryngospasm intraoperatively and was associated with low plasma concentrations of phenol. This makes intravascular injection of phenol an unlikely source of that dysrhythmia. Whereas two of the patients (16 and 23) with dysrhythmias were noted to have blood phenol concentrations of greater than 20 μg/ml, other patients (18, 22, and 24) had higher measured concentrations and no incidence of dysrhythmias. No correlation could be made between the dosage or blood concentration of phenol and the incidence of dysrhythmias in this study population. The rather long duration (33 ± 8 min) during which the phenol was parenterally administered may have been a factor in the lower-than-expected incidence of dysrhythmias in this pediatric series, in contrast to the experience reported in the plastic surgery literature, in which rapid absorption and possibly greater plasma concentrations might have been contributory factors.[2,3]

Wood[7] has provided a detailed historic review of the use of phenol as a neurolytic agent, including experience with a variety of regional blocks, and Kempthorne and Brown[8] have reported their use of tibial and common peroneal nerve blocks in children after brain injury for brief diagnostic and prognostic purposes. Easton *et al.*[1] described motor point blocks (neurolysis) using 5% phenol in water for temporary relief of spasticity in 42 children with cerebral palsy to improve physical therapy of affected limbs and to help ascertain potential benefit from orthopedic surgery. No complications from the use of ketamine were noted in that series; however, ECG monitoring was not mentioned. While Wood[7] cites 8.5 g as the lethal dose of phenol in humans, the toxic dose of phenol in the pediatric population is not known. Deichmann and Keplinger[9] extensively describe cardiovascular and central nervous system depression and collapse in animals and humans associated with phenol administration, citing death after the ingestion of as little as 1 g in an adult. The only published phenol-related complication in a pediatric patient is that of Warner and Harper[4] reporting the occurrence of multifocal and coupled VPCs in a 10-yr-old boy having removal of a hairy nevus while anesthetized with halothane; this occurrence they attributed to cutaneous phenol administration (total dose 2.4 g, *i.e.*, 67 mg/kg). The dysrhythmias occurred approximately 55 min after phenol administration; however, no blood samples were drawn for assay of blood concentration.

In summary, the use of 5% phenol in water for motor point blocks in pediatric patients with cerebral palsy anesthetized with halothane was not noted to be associated with an increased incidence of cardiac dysrhythmias when administered in the doses and over the duration reported. However, the safe dose, the duration of administration or absorption of phenol, and the threshold blood concentration at which dysrhythmias may become more pronounced have not been established. In the current small series, two patients, with phenol blood concentrations of greater than 20 μg/ml, displayed dysrhythmias attributable to phenol administration, whereas only one patient with a concentration of less than 20 μg/ml had a dysrhythmia. ECG monitoring, particularly with the larger dosages of phenol, may identify a greater incidence of dysrhythmias and is recommended regardless of the anesthetic administered. All dysrhythmias in this study occurred within 30 min of the completion of phenol administration and would have been noticed while the patient was in the operating room or postanesthetic care unit. The procedure appears appropriate to perform in the day-surgery context.

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# Successful Defibrillation with Near-simultaneous Orthogonal Discharges

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Implantation of the automatic cardioverter–defibrillator (AICD) permits patients with life-threatening arrhythmias to continue their normal daily activities. Continuing operative care of these patients includes replacement of pulse generators and electrodes. Each episode of device-testing risks the inability to restore a viable cardiac rhythm.[1]

We encountered a case of ventricular fibrillation during AICD testing that was unresponsive to internal or external countershocks. In this instance, however, near simultaneous orthogonal countershocks using both internal paddles and the defibrillator spring–patch electrodes eventually restored sinus rhythm.

## CASE REPORT

A 58-yr-old man required left thoracotomy for placement of new AICD patches. Recurrent symptomatic ventricular tachycardia unresponsive to anti-arrhythmic therapy had prompted insertion 2 yr earlier of an INTEC® spring–epicardial-patch-configured AICD. Since implantation, that AICD had discharged successfully four times. Magnet testing of the device at a subsequent routine office visit disclosed a magnet charge of time of 30.3 s, indicating battery depletion of the pulse generator. Follow-up electrophysiologic study revealed normal resistance of the single epicardial patch, as well as normal pacing threshold, R-wave amplitude, and pulse duration. However, shocks of 20 and 30 J from an external generator, applied *via* the indwelling spring wire and patch, could not convert an induced ventricular tachycardia, and 40 J rescue cardioversion was required, similarly applied. Based on these findings, the patient's cardiologists planned replacement of the spring–patch-configured AICD with large patch defibrillation plates.

The patient had undergone previous coronary artery bypass graft surgery and had had previous myocardial infarctions, and suffered from hypertension and chronic obstructive pulmonary disease. Repeat cardiac catheterization just prior to AICD replacement revealed an ejection fraction of 13%, totally occluded left anterior descending and right coronary arteries, normal left main and left circumflex coronary arteries, and patent vein grafts to the left anterior descending and right coronary arteries. Medical therapy included oral amiodarone 200 mg twice daily, 1 g sustained release procainamide four times daily, diltiazem 60 mg three times daily, digoxin 0.25 mg daily, and furosemide 40 mg daily.

He received preanesthetic medication with intramuscular morphine sulfate and scopolamine. Monitors included surface electrocardiogram (leads II and V5, continuously), pulse oximetry, a radial arterial catheter, and an oximetry pulmonary artery catheter.‡ Etomidate 20 mg, lidocaine 140 mg, and sufentanil 100 μg provided anesthesia, and vecuronium 8 mg provided muscle relaxation for intubation. One episode of spontaneous ventricular tachycardia converted to sinus rhythm with 20 J from an external generator applied *via* the spring wire and patch.

New large patch–patch electrode placement proceeded. A CPI® external cardioverter defibrillator tested the defibrillation threshold of the new patches. During testing, ventricular fibrillation induced with alternating current by epicardial leads could not be successfully converted despite incremental increases in defibrillation energy (15, 20, and then 40 J). Internal cardiac massage began. External defibrillation at 360 J *via* R2 rescue pads failed five times. More than ten additional attempts to defibrillate *via* the old spring and patch, internal paddles, and external R2 pads also were unsuccessful. Defibrillation attempts continued during and after administration of each of the following drugs: lidocaine 100 mg intravenous (iv) bolus twice and infusion at 2 $mg \cdot min^{-1}$; epinephrine 6 μg iv bolus and infusion of 4 $\mu g \cdot min^{-1}$; sodium bicarbonate 44 mEq iv; bretylium 350 mg iv and then 700 mg iv with 2 $mg \cdot min^{-1}$ infusion; and isoproterenol infusion at 2 $\mu g \cdot min^{-1}$.

After 15 min of cardiopulmonary resuscitation, extracorporeal circulation (ECC) was instituted *via* femoral artery and vein. During 52 min of ECC, 28 attempts to defibrillate with the spring–patch, epicardial patches, internal paddles, and external R2 pads at maximal energies proved unsuccessful. Finally, near-simultaneous (50 ms apart as recorded on a rapid-speed ECG recorder) discharge of internal paddles (50 J) placed perpendicular (fig. 1) to the spring-wire–epicardial-patch plane (40 J) achieved successful defibrillation. With restoration of sinus

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