Transcatheter Thoracic Epidural Neurolysis Using Ethyl Alcohol

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Sporadic reports of pain relief following the injection of ethyl alcohol into the epidural space have appeared in the literature since 1930. Early reports described the injection of large volumes of local anesthetic and alcohol into the caudal epidural space for relief of intractable pelvic and perineal pain due to rectal or prostatic cancers.1-3

Despite mention in scattered case reports, no study of the efficacy and safety of epidural neurolysis using ethyl alcohol has been reported.4-7 Of concern are the inaccuracies of the technique, the possibility of injection pain, the chance that "alcohol neuritis" might develop, and the danger that accidental unrecognized dural puncture might lead to disastrous consequences in the face of the relatively large volume of alcohol that is used. After an initial success using transcatheter thoracic epidural neurolysis with ethyl alcohol to treat a patient with pancreatic cancer pain who did not respond to celiac plexus block, this study was undertaken to document the safety and efficacy of this technique in the management of intractable pain of malignant and benign origins.

MATERIALS AND METHODS

Following approval of the institutional review board, informed consent was obtained from 36 consecutive in-
Patients with intractable somatic or visceral pain of benign and malignant origin referable to cervical or thoracic nerve roots. Patients were followed for up to 18 months to determine immediate and long-term relief of pain. They were asked to rate their percent relief of the original pain subjectively. This was corroborated by the patient's ability to decrease narcotic medication dose. Successful treatment was defined as a report of 70% or more pain relief and a reduction in narcotic dose of at least 25%. Patients were followed until their death or for 18 months to determine the duration of successful epidural neuralysis.

The site for placement of the epidural catheter was selected based on the underlying pathophysiology, as well as on each patient's description of the dermatomal pain patterns (table 1). A 20-gauge nylon catheter was placed 3-5 cm within the epidural space using an 18-gauge Hustead needle and loss-of-resistance technique. Correct catheter placement was confirmed by the patient's report of complete pain relief using 5-7 ml of local anesthetic (0.25% bupivacaine) given in 2-ml increments. Subarachnoid or intravascular placement of the catheter was excluded by negative aspiration for blood or cerebrospinal fluid and by the presence of segmental hypesthesia after the above test doses. The patient was positioned 30° head up and supine for visceral or bilateral somatic pain or with the affected side upward in a lateral position for unilateral somatic pain. The patients were positioned primarily for their comfort, with secondary consideration given to the slight hypobaricity of alcohol in tissue fluids. Alcohol injection was begun 20 min later.

Over 20-30 min, 3-5 ml of ethyl alcohol were slowly injected. The first six patients received 86% ethyl alcohol mixed with 0.25% bupivacaine; the remainder of the patients received absolute alcohol. No more than 0.2 ml was ever given as a bolus. The rate of injection was slowed for any report of pain. The injection was stopped immediately in one patient for reports of tingling and numbness in the fifth fingers and anterior surfaces of the thighs. At completion of the alcohol injection, the catheter was flushed with 0.25 ml of 0.25% bupivacaine, and capped.

Patients were returned to their hospital rooms 30 min after completing the injection with the epidural catheter taped securely in place. They were followed for pain relief as subjectively reported from 0 to 100% and as corroborated by the amount of narcotic required on an "only when necessary" basis. Patients were specifically asked about complications, including catheter site pain, weakness, sensory loss, bowel or bladder dysfunction of new onset, or neuritis.

Second and third alcohol injections were made on a daily basis unless a patient experienced 100% pain relief persistent over 24 h and a decrease in narcotic use of at least 25%. On the second and third days, 3-5 ml of ethyl alcohol were slowly injected 10 min after the catheter was tested using 2 ml of 0.25% bupivacaine and aspirated to verify absence of cerebrospinal fluid or blood. Patient position was the same as on the first day and excessive spread of alcohol was again avoided by continuous questioning of the patient regarding tingling or numbness of the fifth fingers or anterior skin of the thigh. Intravenous hydration on the second and third days of treatment was only instituted in cases where patients experienced anorexia, nausea, vomiting, or diarrhea over the preceding 24 h. Heart rate and arterial blood pressure monitoring was utilized during each injection. Catheters were removed at least 1 h following the final injection of alcohol and a flushing dose of 0.25% bupivacaine.

Any complications of the treatment were recorded and the duration of each was noted. Patients were asked specifically regarding any treatment-related pain, any new sensory or motor deficit, any change in appetite, bowel or bladder function, and any skin problems at the catheter insertion site.

**RESULTS**

Thirty-six patients completed the study. Eighty-nine percent of the patients were judged to have been successfully treated because they reported 70% or greater pain relief, which was supported by a decrease in narcotic dose of 25% or more. All patients experiencing cancer pain of visceral or mixed somatic and visceral origin had immediate pain relief reported during the first week compared to 55% of patients with chronic benign pain (chronic pancreatitis, post-herpetic neuralgia, and diffuse abdominal pain) (table 2).

Of the patients judged to have been successfully treated, the overall duration of relief was from 2 weeks to 7.25 months. The mean overall was 3.3 months; these results reflect the large number of patients who died of their underlying illness during the study period. Twelve patients with cancer were dead within 4 months after treatment. Twenty patients who died during the study reported continued relief of pain until death. Twelve pa-
tients survived to report a return in their pain. Of these patients, duration of pain relief extended from 3 weeks to 7.25 months, with a mean duration of relief of 4.4 months (table 3).

Adverse effects of the procedure for the first 7 days following institution of treatment are reported in table 4. There were no cases of dural puncture, orthostasis, new sensory or motor loss, or “alcohol neuritis.” Ten patients complained of catheter site pain by the second day of treatment. Seven patients required treatment with naloxone once thoracic epidural analgesia was instituted. Four patients required treatment for initial hypotension.

The second patient in the study developed complete sensory and motor loss involving both upper and lower extremities of 2 min duration during the alcohol injection. There was complete resolution of symptoms with cessation of the injection. He experienced no loss of consciousness or apparent diminution in ventilation. Two subsequent injections of ethyl alcohol were completed without difficulty. In addition, he survived to return at 7.5 months for a second series of injections, which was performed without difficulty. He reported continued pain relief until he died of his pancreatic cancer 3.5 months after the second treatment.

Beyond the first week of treatment, five patients complained of back pain at the catheter insertion site, which persisted up to 3 weeks. There were no cases of catheter site infection or skin breakdown. A total of 94 injections of alcohol into the thoracic epidural space were performed and no other adverse effects were noted.

### DISCUSSION

In 1940, Odom reported the injection of 10–15 ml of 95% alcohol into the epidural space for treatment of intractable pain secondary to generalized carcinomatosis. He was impressed with the lack of motor complications in his patients. The instillation of ethyl alcohol into the thoracic epidural space for the purpose of neurolysis was first described in the literature by Groenendijk in 1954. He used rapid peridural injection of 33% alcohol through a needle to treat 17 patients with cancer pain. Each injection provided 1–3 weeks of pain relief, and the technique was repeated as necessary (up to eight times) to achieve long-term relief of pain. He also found no motor complications following epidural neurolysis; there was transient back pain for 20 s following alcohol injection, and pain relief was not always complete. Two patients required intravenous hydration for transient light-headedness following the injection.

In 1967, Bronage described the successful treatment of intractable pain caused by Pancoast’s syndrome using a single injection of 5 ml absolute alcohol into the epidural space at T2. Although he reported no adverse consequences of this treatment, he concluded his report by warning that thoracic epidural neurolysis had potential complications, and implying that it be reserved for intractable pain due to malignancy.

Afferent impulses of somatic and visceral origins can be blocked as the nerve roots traverse the epidural space. Alcohol is a nonspecific, irritating, hypobaric neurolytic agent that spreads along tissue planes easily. It has local anesthetic properties that appear 10–20 min after injection, except in the subarachnoid space where the injection of alcohol is associated with a burning dermal pain within 5–10 min of injection. Previous descriptions of epidural alcohol neurolysis have cited incomplete long-term analgesia as a problem, with repeated injections resulting in optimal results. To avoid injection pain and untoward spread and to permit repeated injections on a daily basis as necessary, a transcatheter approach to tho-
racic epidural neurolysis using ethyl alcohol has been developed.

A transcatheter approach to phenol epidural neurolysis has been described with reported success rates of 50–86% and a total duration ranging from 2 weeks to 3 months. Alcohol and phenol have not been systematically compared as neurolytic agents, although Bromage compared ten cases of thoracic epidural neurolysis using ethyl alcohol with seven cases using 6% aqueous phenol. He performed single injections with each agent, and gave the alcohol as a bolus with the patients lightly anesthetized. Although he noted better pain relief using alcohol, he also described one case of transient motor weakness. Accidental injection of alcohol into the subarachnoid space will result in an immediate burning dermatomal pain, which should provide adequate warning to stop the injection and assess the patient. Subarachnoid injection of phenol is painless. Phenol has a greater affinity for vascular structures, a property that has resulted in severe neurologic sequelae. Both alcohol and phenol can result in a 10% incidence of painful paresthesias or neuritis after injection onto peripheral nerves. Although Katz is often cited for his postulation that a leak of alcohol into the epidural space during subarachnoid alcohol injection may have resulted in one case of neuritis, the only two documented cases of neuritis following epidural neurolysis occurred following 7% phenol injection.

The second patient studied developed complete motor and sensory block of all four extremities during the third ml of the 5-ml alcohol injection. His recovery was complete within 2 min following cessation of the injection, which suggested a too-rapid rate of injection. The importance of the small bolus of local anesthetic preceding each daily aliquot of alcohol injected was emphasized by this case, since the sensory and motor block were most likely caused by the spread of local anesthetic away from the catheter tip. The remainder of the alcohol was injected more slowly and without further incident.

Seven patients required treatment with naltrexone once epidural analgesia was achieved. These were patients with cancer pain who required treatment with intravenous morphine in a dosage range of 10–70 mg/h. The pain relief achieved with thoracic epidural analgesia was substantial, and resulted in a relative narcotic overdose requiring naltrexone and a decrease in the infusion rate of morphine.

All patients with cancer pain of visceral or mixed somatic and visceral origin reported immediate pain relief with this technique. The results were less promising in patients with chronic benign pain syndromes, including chronic pancreatitis, post-herpetic neuralgia, and diffuse abdominal pain. Similar mixed success has been reported for the use of celiac plexus blocks and phenol epidural neurolysis in the treatment of chronic benign pain syndromes. These results may be explained by the multifactorial nature of chronic benign pain syndromes, and for this reason we now reserve epidural neurolytic procedures for patients with cancer pain.

Thoracic epidural neurolysis using serial transcatheter injections of ethyl alcohol can contribute greatly to an increased quality of life in cancer patients whose pain is referable to thoracic nerve roots. Cancer pain often results from a mixture of somatic and visceral afferent stimuli. The epidural space is rarely displaced by intra-abdominal or by intra-thoracic tumors, and represents the least central location for achieving afferent blockade. There has been no study comparing the relative efficacy of celiac plexus neurolysis with thoracic epidural neurolysis in the treatment of cancer pain. The potential risks of celiac plexus neurolysis include hypotension, diarrhea, spillover onto lumbar nerve roots, failure of ejaculation, hematuria, paraplegia, etc.

In the 36 patients studied with intractable pain referable to thoracic nerve roots, there were no serious adverse effects associated with the treatment. The safety of the technique in this study is attributable to: 1) transcatheter instillation of alcohol in bolus amounts no greater than 0.2 ml; 2) daily verification of pain relief and catheter position using local anesthetic prior to beginning any alcohol injection; and 3) daily dosing with total volumes of ethyl alcohol never exceeding 5 ml.

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Anesthetic Management of Hemodynamic Changes during Vein of Galen Aneurysm Clipping

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The Vein of Galen malformation is a midline intracranial arteriovenous fistula with aneurysmal dilatation of the Vein of Galen. This abnormality comprises less than 1% of all arteriovenous malformations,1,2 the highest incidence being in the infant and young child. The lesion is responsible for a 91% cumulative mortality in neonates (both surgically treated and medically managed), with mortality dropping to 53% in surgically treated infants aged 1–12 months, and to 25% in children who present for surgical repair after 1 yr of age.1-3 Mortality is usually due to cardiac complications or uncontrollable hemorrhage at operation. In survivors, long-term central nervous system morbidity in the form of seizures, hemiparesis, blindness, and intellectual impairment remains high. Factors complicating anesthetic management include massive intraoperative hemorrhage, acute congestive heart failure from increased systemic vascular resistance when the aneurysm is clipped, and acute coronary insufficiency due to low diastolic arterial pressure.2,4,5 We report successful outcome in a 9-week-old infant undergoing Vein of Galen aneurysm clipping, under high-dose narcotic anesthesia.

Therapeutic intervention was guided by hemodynamic data obtained from a pulmonary artery catheter.

CASE REPORT

A 9-week-old, 6-kg male infant presented with seizures and cardiomegaly. Diagnosis of Vein of Galen malformation was made by CAT Scan and cerebral angiogram (fig. 1). Preoperative echocardiogram demonstrated increased left atrial and left ventricular dimensions with good contractility of both right and left ventricles. On physical examination, heart rate ranged from 160 to 190 bpm and arterial blood pressure from 110/40 to 90/35 mmHg with bounding peripheral pulses, no gallop, cranial bruit, or hepatomegaly. Neurological examination was only remarkable for mild hypertonicity of the lower extremities. Blood volume was estimated by Cr51 labeled red blood cell study to be 780 ml, 150% of the predicted value. Twelve hours preoperatively, ketamine 1 mg/kg was given iv to facilitate placement of a right femoral pulmonary artery catheter and ulnar arterial line. Initial cardiac index (CI) was 7.0 L/min·m², central venous pressure (CVP) was zero, pulmonary artery diastolic (PAD) pressure was 4 mmHg; pulmonary wedge pressure was 3 mmHg; and systemic vascular resistance index (SVRI) was 680 dynes·s⁻¹·cm⁵. Pulmonary artery diastolic filling pressures correlated with the wedge pressure and were followed as a guide to left ventricular filling, due to the concern that repeated balloon inflation might obstruct right heart output. Due to low filling pressures and preoperative anemia (Hct 24%), the infant was transfused to a hematocrit of 35% the evening prior to surgery. The normocytic hypochromic anemia was attributed to chronic disease and hemodilution with no evidence of hemolysis. Maintenance fluids were given iv to prevent a preoperative fluid deficit.

Intraoperative monitoring consisted of a V̇s ECG lead, precordial stethoscope, precordial doppler, automatic oscillometric BP cuff, continuous ECG monitor (PSA-I), mass spectrometer for measurement of end-tidal CO2 and inspired oxygen concentrations, and pulse oximeter. A V̇s lead was used for detection of myocardial ischemia. Anesthesia was induced with iv fentanyl, 10 µg/kg, and neuromuscular blockade was provided by pancuronium bromide, 0.1 mg/kg iv. A nasal route was chosen for tracheal intubation to provide for postoperative stability of the tracheal tube.

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