plications,[3] and dysphagia, dysphonia, and dyspnea[9] have been found following subcutaneous emphysema caused by high-speed air-driven turbine handpieces.

The treatment of iatrogenic subcutaneous emphysema varies from close observation to emergency airway management. When this complication can be detected intraoperatively, administration of nitrous oxide should be discontinued to avoid enlargement of the air mass. Roentgenograms of the chest should be obtained to determine the extent of the emphysema. The use of clear plastic drapes is helpful in permitting observation of the patient intraoperatively. Prophylactic antibiotic therapy, although controversial, is probably justified, because the oral flora are potentially pathogenic, and the possibility of cellulitis from organisms carried interstitially with the air is always present.[10,11]

We report this case to stress that the use of ultrahigh-speed instruments with increased air pressures and the use of compressed-air syringes can cause subcutaneous emphysema, mediastinal emphysema, and possibly air embolism during dental procedures with anesthesia.

References

1. Thawley SE: Air in the neck. Laryngoscope 84:1445–1453, 1974
2. Cardo VA, Mooney JW, Stratigos GT: Iatrogenic dental-air emphysema: Report of a case. J Am Dent Assoc 85:144–146, 1972
3. Quisling RW, Kangen TT, Jahrsdoefer RA: Otologic complications following use of a high-speed air-turbine handpiece. J Am Dent Assoc 94:895–897, 1977
4. Duncan JM, Ferrillo PJ: Interstitial emphysema after a restoration. J Am Dent Assoc 74:407–409, 1967
5. Trummer MJ, Fasburg RG: Mediastinal emphysema following the use of a high-speed air-turbine dental drill. Ann Thorac Surg 9:378–381, 1970
6. Hunt RB, Sahler OD: Mediastinal emphysema produced by an air-turbine dental drill. JAMA 205:241–242, 1968
7. Rickles NH, Joshi BA: Death from air embolism during root canal therapy: A possible case in a human and an investigation in dogs. J Am Dent Assoc 67:397–404, 1963
8. Lloyd RE: Surgical emphysema is a complication in endodontics. Br Dent J 138:393–397, 1975
9. McGrannahan WW: Tissue space emphysema from an air-turbine handpiece. J Am Dent Assoc 71:884–886, 1965
10. Rhymes R: Postextraction subcutaneous emphysema. Oral Surg 17:271–273, 1964
11. Kleinman HZ: Subcutaneous and mediastinal emphysema after oral surgery: Report of a case. J. Oral Surg 19:527–529, 1961

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Age, Height, and Speed of Injection as Factors Determining Caudal Anesthetic Level, and Occurrence of Severe Hypertension

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Bromage documented that the level of anesthesia achieved with lumbar epidural block was influenced by age and height.[1] However, factors influencing the level of caudal epidural block have not been well investigated in adult patients. Therefore, we decided to study the effects of age, height, and speed of injection on the level of caudal anesthesia in adult patients. During this study unexpected severe hypertension developed in several patients with caudal injection of local anesthetic.

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Methods

Subjects of the study were 135 consenting adult male patients given caudal anesthesia for various operations. Premedication consisted of diazepam, 5–10 mg, and morphine sulfate, 5–10 mg im, given approximately an hour prior to operation. Patients were placed in the jackknife position and conventional caudal blocks performed by use of a #20, caudal needle 3.75 cm long. A 30-ml volume 1.5 per cent lidocaine with epinephrine (1:200,000) was injected over 1 min in Group I (85 patients) and over 2 min in Group II (50 patients). On completion of the injection, patients were either turned to the supine position or kept in the jackknife position maintaining the spine horizontal.

TABLE 1. Operations Performed with Caudal Anesthesia

	Number of Patients
Cystoscopy, transurethral resection of the prostate, circumcision, orchiectomy	32
Sigmoidoscopy, hemorrhoidectomy, fissurectomy, excision of anal fistula	30
Inguinal herniorrhaphy	18
Toe amputation, bunionectomy	17
Leg amputation (above or below knee)	8
Meniscectomy of knee	6
Others	24

Blood pressure and pulse were checked during caudal injection of the anesthetic, every 2 min for the first 10 min and thereafter every 5 min. Response of the anal sphincter reflex and level of sensory anesthesia were tested by pinprick, and motor block by toe movement, every minute for the first 10 min and then every 10 min for an hour. Time to recovery of the anal sphincter reflex was also recorded. Only patients in whom abolition of the anal sphincter reflex was accomplished were included in the study. Statistical analysis of results was by use of correlation coefficients and the Student *t* test.

RESULTS

Operations performed with caudal anesthesia are summarized in table 1. The mean level of anesthesia achieved with 30 ml 1.5 per cent lidocaine with epinephrine (1:200,000) in Group I was T7, higher than the T11 level achieved in Group II (table 2). Anesthesia in the thoracolumbar area was often poor and regressed rapidly, with recovery often occurring within an hour after the block. Patients' ages and heights correlated poorly with sensory levels of caudal anesthesia in both groups (fig. 1).

Of 85 patients in Group I, seven experienced acute severe hypertension (blood pressure more than 200/100 torr) and tachycardia either during or within 2 min following caudal injection of local anesthetic. The hypertension was transient, lasting less than 10 min. It was associated with mental confusion in three patients and incomplete anesthesia in three other patients (table 3). None of the 50 patients in Group II had acute hypertension.

DISCUSSION

Many factors can affect the level of caudal anesthesia: 1) the size of the caudal epidural space; 2) the amount of areolar and vesicular tissues in the epidural space; 3) the patency of sacral canals and intervertebral foramina; 4) the various septa of the epidural space; 5) the permeability of the neural and dural sheaths to the drug; 6) capillary action exerted on the local anesthetic; 7) amount of local anesthetic injected; 8) gravity effect with positional change; 9) speed of injection.[2,3] With all of these variables unknown or uncontrolled except 7, 8, and 9 in our study, differences in either age or height did not produce any significant effect on anesthesia level in either group. These uncontrollable variables may explain the difficulty that clinicians have in controlling the level of caudal anesthesia. Although age bears no relationship to the anesthetic level achieved in the adult patient, age may exert a far different effect in children.[4]

The anesthetic level was assessed by pinprick in all patients, and the relationship between speed of injection and anesthetic level achieved was apparent (table 2). Even though there was a difference in age between the two groups, we do not believe that this contributed to our finding of a higher sensory level achieved with a more rapid injection because there was no relationship between age and anesthetic level achieved (fig. 1). Anesthesia in the thoracolumbar area achieved by rapid caudal injection was, however, often inadequate in intensity and duration. It is therefore suggested that thoracolumbar anesthesia be managed by another technique, such as lumbar epidural anesthesia.

The development of acute transient hypertension

TABLE 2. Onset, Level and Duration of Caudal Anesthesia* (Mean ± SEM, Range)

	Age (Years)	Height (cm)	Time to Abolition of Anal Sphincter Reflex (Min)	Highest Sensory Block Level Achieved (Dermatome)	Time to Recovery of Anal Sphincter Reflex (Min)
Group I (85 patients)	53.2 ± 1.5 (21–83)	175.0 ± 1.0 (152–198)	3.9 ± 0.3 (2–15)	T6.7 ± 0.3 (L3–T1)	202.9 ± 5.3 (125–346)
Group II (50 patients)	44.6 ± 2.7 (23–70)	177.0 ± 1.8 (161.5–193)	3.6 ± 0.5 (2–12)	T11.0 ± 0.9 (S2–T2)	191.8 ± 11.1 (110–267)
Significance of difference	$P < 0.002$	NS	NS	$P < 0.001$	NS

* Local anesthetic used: 1.5 per cent lidocaine with epinephrine (1:200,000), 30 ml. Anesthetic was injected taking 1 min in group I and 2 min in Group II.

during or soon after caudal anesthetic injection is interesting. The mechanism involved in this phenomenon is not clear. We can postulate two possibilities for the hypertension. One is the creation of high epidural pressure during rapid caudal injection of local anesthetic and subsequent compression of the lower segment of the spinal cord, resulting in activation of sympathetic preganglionic cells in the spinal cord and severe transient hypertension. This occurrence of hypertension resulting from direct compression of the spinal cord has been well demonstrated in several animal studies.[5-7]

When rapid caudal injection of 40–55 ml saline solution was used for treatment of atypical sciatica, convulsions occurred in several patients.[2] When these pressure caudal or pressure lumbar epidural procedures were performed during general anesthesia retinal hemorrhage was found in some patients.[2,8] It was postulated that these phenomena were due to movement of the spinal cord and increase of intracranial pressure by rapid injection of saline solution. Even though nothing was mentioned about alteration of the blood pressure of these patients, it is conceivable that some of the retinal hemorrhage could have been attributed to severe hypertension.

The second possible cause of hypertension is inadvertent iv injection of local anesthetic and/or epinephrine. Local anesthetic toxicity often results in various neurologic signs such as restlessness, tremor, convulsions, and unconsciousness, and the hypertension is often followed by hypotension.[9] In our study, however, none of the patients subsequently had hypotension.

Three of our seven patients in whom hypertension developed had mental confusion, one with tonic seizure activity. Mental confusion lasted less than a minute. Both local anesthetic toxicity and increase in epidural pressure are known to produce mental confusion and convulsions.[2,9] Three patients had incomplete or patchy sensory blocks. This also could have been caused by either wide spread of the local anesthetic during injection with high pressure, resulting in a low concentration in the epidural space, or inadvertent iv injection of local anesthetic, leaving less anesthetic in the epidural space.

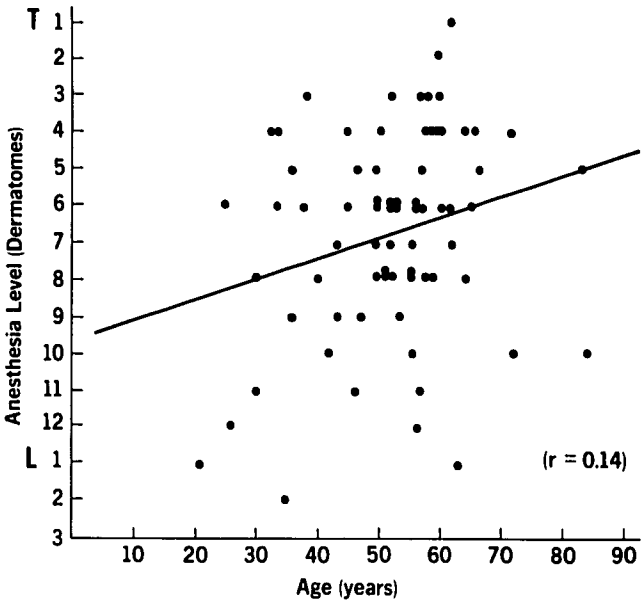

FIG. 1. Relationship between patient's age and level of caudal anesthesia (dermatome) in Group I. T = thoracic; L = lumbar; r = correlation coefficient.

When caudal anesthesia was administered over 2 min in Group II, there was no hypertension or mental confusion. This leads us to believe that pressure and compression of the spinal cord, resulting in sympathetic activation, were a more likely cause of severe hypertension in some patients in Group I.

We recommend, therefore, that the selection of patients for caudal anesthesia be limited to those having surgical procedures involving the sacroperineal area, and that the local anesthetic be injected slowly (>2 min), while blood pressure is carefully monitored.

REFERENCES

1. Bromage PR: Ageing and epidural dose requirement. Br J Anaesth 41:1016–1022, 1969
2. Moore DC: Regional Block. Fourth edition. Springfield, Ill., Charles C Thomas, 1976, pp 439–451

TABLE 3. Patients in Whom Hypertension Developed during Caudal Anesthesia

	Highest Blood Pressure (Control) (torr)	Anesthetic Level	Remarks
Patient 1	220/130 (135/90)	Incomplete	Confusion
Patient 2	205/135 (130/85)	T6	Slight confusion with diplopia
Patient 3	200/110 (110/70)	Incomplete	Shakes
Patient 4	260/105 (140/90)	T4	History of hypertension
Patient 5	240/115 (160/100)	T7	History of hypertension
Patient 6	270/140 (130/90)	Incomplete	Confusion and tonic convulsion—history of hypertension
Patient 7	250/140 (130/90)	L1 (Patchy)	Stiff neck

3. Erdemir HA, Sopper LE, Sweet RB: Studies of factors affecting peridural anesthesia. Anesth Analg (Cleve) 44:400–404, 1965

4. Schulte-Steinberg O, Rahlfs VW: Spread of extradural analgesia following caudal injection in children. A statistical study. Br J Anaesth 49:1027–1034, 1977

5. Eidelberg EE: Cardiovascular response to experimental spinal cord compression. J Neurosurg 38:326–331, 1973

6. Greenhoot JH, Shiel FO, Mauck HP: Experimental spinal cord injury. Arch Neurol 26:524–529, 1972

7. Alexander S, Kerr FWL: Blood pressure response in acute compression of the spinal cord. J Neurosurg 21:485–491, 1964

8. Clark CJ, Whitwell J: Intraocular hemorrhage after epidural injection. Br Med J 2:1612–1613, 1961

9. Moore DC: Regional Block. Fourth edition. Springfield, Ill., Charles C Thomas, 1976, pp 22–23

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Chronic Arsenic Poisoning, A Problem in Anesthetic Management

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Anesthesiologists are occasionally confronted by patients who have cutaneous disorders that affect anesthetic management. Epidermolysis bullosa has frequently been a problem.[1,2] Other dermatologic problems of concern to the anesthesiologist include pemphigus, scleroderma, neurofibromatosis, congenital anhidrotic ectodermal defect, and mastocytosis. These have been well described by Zackheim *et al.*[3]

The medical literature appears to contain no reference to chronic arsenism, with its associated cutaneous disorders, as an anesthetic problem. This report presents the anesthetic considerations in a case of a patient who had chronic arsenism. A further anesthetic problem in this case was previous tracheal resection for carcinoma due to chronic arsenism.

REPORT OF A CASE

A well-nourished, 50-year-old white woman was admitted for excision of a large ulcerating mass on the right posterior chest wall.

Past history revealed that psoriasis had developed when the patient was 20 years old. Her dermatologist had treated her with a combination of x-ray and Fowler's solution (1 per cent potassium arsenite solution). The psoriasis had abated and the patient had terminated her medical supervision but continued to take daily doses of Fowler's solution for the next ten years.

In 1964, a cutaneous lesion developed at the base of the neck. It was excised and diagnosed as a granuloma secondary to chronic arsenism. In 1970, the patient was admitted to another hospital with a four-week history of dyspnea and increasing stridor following an upper respiratory infection. Bronchoscopy revealed a mass below the vocal cords, about 4 cm above the carina. Biopsy demonstrated a squamous-cell carcinoma extending into the paratracheal region. Subsequently, the entire tumor was removed through a right anterior thoracotomy. Reconstruction of the tracheal wall necessitated the use of a small plastic implant. In the immediate postoperative period, it was clear that paralysis of the right vocal cord

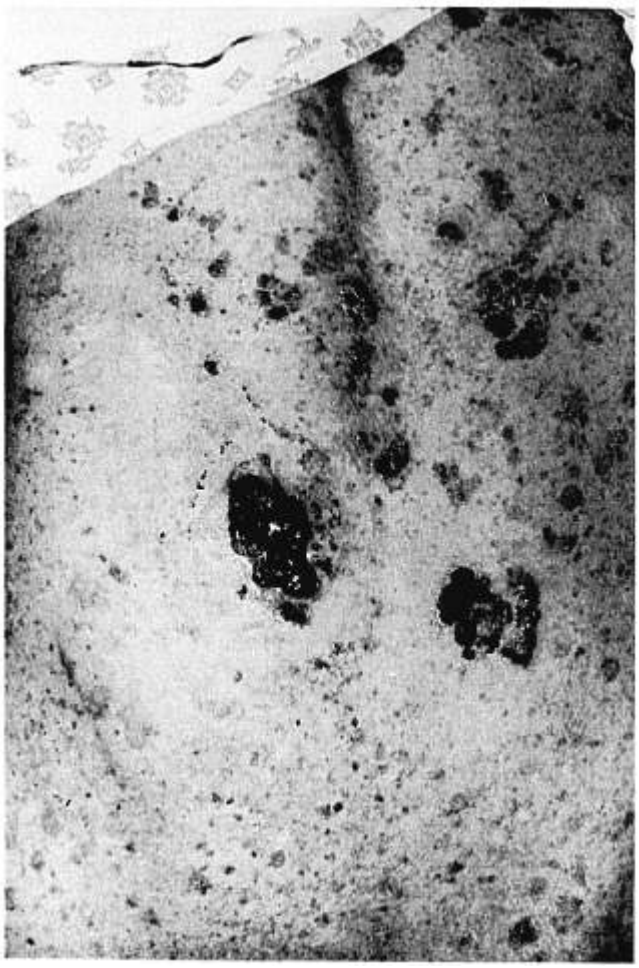

FIG. 1. Posterior chest wall, showing hyperpigmentation, multiple keratoses, and ulcerating squamous-cell carcinoma, secondary to chronic arsenism.

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