plex the P wave immediately followed the R wave, the second was idionodal and the third occurred normally as a sinus complex (fig. 2, second tracing from top). This rhythm continued for one minute after which there were two episodes of pacemaker arrest (fig. 2, third tracing from top). During the next minute pacemaker failure occurred once after every four normal cycles (fig. 2, fourth tracing from top) and after three normal cycles (fig. 2, fifth tracing). Thirty seconds later there was a normal electrocardiogram except for two more episodes of pacemaker failure as illustrated in the bottom tracing. The pH was 7.37 (table 2), the heart rate was 100 beats per minute, the CVP 11 cm. of water and the blood pressure 160/80 mm. of mercury. Frequent postoperative examinations confirmed that the patient had an uneventful recovery without any further episodes of hypotension or arrhythmias.

COMMENT

A case is reported of idionodal rhythm. The arrhythmia was associated with persistent bradycardia and hypotension following controlled hypotensive anesthesia which included the possibility of lignocaine toxicity. The idionodal rhythm, persistent bradycardia and associated hypotension unresponsive to atropine, metaraminol, propranolol, isoproterenol, ephedrine and aminophylline were apparently corrected with 44.6 milli-equivalents of sodium bicarbonate in the presence of an arterial pH of 7.415 prior to the bicarbonate administration. Arterial pH rose to 7.57 five minutes after the bicarbonate was injected. This situation in this case is worthy of note because Reid et al. found no electrocardiographic alterations which they could ascribe to changes in pH in the induced range of 7.30 to 7.64. While electrocardiographic abnormalities may not be experimentally induced by minor variations in pH, the corollary, however, does not seem to hold true. In our experience we have frequently been able to correct arrhythmias, particularly those found in association with halothane and cyclopropane, with a small dose of sodium bicarbonate. A slight variation in pH toward alkalinity clearly corrected the arrhythmia and associated hemodynamic disturbance in the reported case.

REFERENCES


Exaggerated Spread of Epidural Block

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A 69 year old man (5 feet 11 inches and 199 pounds) was to undergo Knowles pin insertion for a five day old right femoral neck fracture. Abnormal preoperative findings included blood pressure 160/75 mm. of mercury, pulse 100/minute, temperature 100° F., rales in both pulmonary bases and blood urea nitrogen 40 mg./100 ml. He had also been treated for severe thoraco-lumbar degenerative arthropathy, paralysis agitans, obstructive pulmonary emphysema, essential hy-

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1 per cent with 1:200,000 epinephrine and 0.1 per cent tetracaine was injected. Dural penetration was ruled out (motility of legs, small amount of cold outflowing fluid, narrow segment of hypalgesia). After another aspiration, 20 ml. lidocaine 1 per cent with 1:200,000 epinephrine and 0.1 per cent tetracaine was slowly injected through the needle carefully immobilized by the anesthetist’s left hand.

During the next 10 minutes, which were clinically uneventful, the upper analgesic level reached T 4 (pin prick). The patient’s pressure then fell suddenly from 160/80 to 120/60 mm. of mercury, and he became markedly dyspneic, breathing with small, jerky, abdominal movements. The block was now felt to be at T 1–2 level. Within two minutes, respiratory and cardiovascular signs were unobtainable. Intubation, ventilation with oxygen, external cardiac massage and intravenous injection of 2 mg. phenylephrine were immediately carried out. Regular sinus rhythm, and preoperative pulse and pressure values resumed in 2–3 minutes. Surgery was postponed. Since the patient was making no voluntary respiratory efforts, he was placed on a respirator. Shortly after, the pressure had to be maintained with a 0.004 per cent phenylephrine drip. The pupils were extremely miotic.

Adequate spontaneous respirations resumed after 3½ hours. Two hours later, the endotracheal tube was removed and the phenylephrine drip discontinued. At that time, the patient was conscious and articulate, but did not recall the incident. He, unfortunately, died 108 hours later, after a course suggesting bronchopneumonia. Autopsy revealed: lobar pneumonia, bilateral pleural effusion, generalized pulmonary edema and emphysema, generalized arteriosclerosis, cardiomegaly, nephrosclerosis and an apparently primary carcinoma of the liver.

The present case exhibits several similarities with some or all of the cases previously reported: (1) large dose of lidocaine, (2) identical clinical picture including a delayed onset, and (3) elderly patient, with arteriosclerosis, hypertension, and arthritis. The last three factors, of course, frequently co-exist in the geriatric patient. Bromage claims that arteriosclerosis is a critical causative factor, but this inference, essentially, only suggests that an elderly patient group, in which arteriosclerosis, among other possible degenerative processes, is common, developed higher epidural levels. Definite conclusions as to specific etiology clearly require more critical methods of study. If degenerative obstruction of the intervertebral foramina influences the epidural spread more than a decreased permeability of arteriosclerotic spinal vessels, arthrospondylitis may also be the principal cause of this complication. Still other factors might have enhanced the spread in our patient; preoperative dehydration (with decreased epidural content) or liver and lung pathology with increased vertebral vein pressure. The dose injected here (250 mg. lidocaine and 25 mg. tetracaine) is admittedly very large for a poor risk patient; however, we commonly use it in elderly arteriosclerotics receiving an epidural block for abdominal surgery. Analgesia, in our experience, rarely trespasses T 4.

REFERENCES