

then reducing the dose of succinylcholine would provide the information while giving added protection against harmful side effects, as shown by the original work of Tolmie, Joyce, and Mitchell.<sup>2</sup>

The time course of this patient's sensitivity correlates closely with the three to eight week "vulnerable period" described by Cooperman.<sup>3</sup>

While it is known that succinylcholine-induced hyperkalemia can occur in association with both upper and lower motor-neuron diseases,<sup>4,5</sup> it is probably not correct to lump all neuromuscular diseases in these categories. First, it is not clear what makes these patients hyperkalemic. Second, there is no common denominator for the cases thus far reported. Certainly the lesions of cerebral palsy, encephalitis, Parkinson's disease, multiple sclerosis, muscular dystrophy, and now cerebral hematoma are different.

Some have suggested that disuse atrophy from prolonged bed rest is a common denominator, but Gronert and Theye demonstrated that this had only a minor effect, at least in dogs.<sup>6</sup> Nutrition may play a role, but definitive studies have not been done. This patient was receiving 2,400 kcal per day, with additional multivitamins, through a nasogastric tube. His weight was stable, without gross edema or obvious muscle wasting.

Although it has been shown that pretreatment with

a nondepolarizing muscle relaxant prior to succinylcholine administration will attenuate the hyperkalemia in susceptible individuals,<sup>7</sup> this case illustrates that this therapy is not entirely protective. In summary, closed head injuries should be added to the growing list of disorders that have the potential for serious hyperkalemia following intravenous administration of succinylcholine.

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Anesthesiology  
51:90-92, 1979

## Preoperative Clonidine Withdrawal Syndrome

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Clonidine is an antihypertensive drug available only in oral form. Rebound hypertension following abrupt cessation of clonidine therapy was described in 1973,<sup>1,2</sup> and since then, more reports have ap-

peared.<sup>3-8</sup> Brodsky and Bravo described the case of a patient in whom clonidine withdrawal syndrome manifested postoperatively in the recovery room,<sup>9</sup> but there has been no published report of this problem occurring preoperatively.

## REPORT OF TWO CASES

**Patient 1.** A 54-year-old man who had an ischemic leg was scheduled for a femoral-popliteal bypass operation, to begin about noon. He had been receiving clonidine, digoxin, hydralazine, propranolol, furosemide and nitroglycerin. Since midnight, all drugs except nitroglycerin had been discontinued. Premedicated with meperidine, 25 mg, and atropine, 0.6 mg, the patient arrived in the operating room shortly after noon. He had just taken nitroglycerin for moderate chest pain, with resultant relief of this discomfort. Radial and pulmonary arterial lines were inserted promptly and provided pressure readings of 280 torr systolic and 30 torr in the occluded (PAOP, "wedge") position, respectively. The abnormal PAOP indicated cardiac failure and the

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Received from the Anesthesiology Service, Veterans Administration Medical Center, 5901 East Seventh Street, Long Beach, California 90822. Accepted for publication December 7, 1978.

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high systemic pressure suggested this was secondary to a marked increase in afterload. The reason for this condition was not clear. Chart review revealed that the patient had not received his scheduled dose of clonidine on the previous evening, in addition to its being withheld after midnight. Thus, he had abruptly changed from a schedule of 0.2 mg clonidine four times daily to none for about 18 hours. In addition, he had received no propranolol for about 14 hours. Since it is known that some patients have elevated plasma catecholamine concentrations after clonidine withdrawal, it seemed that this might explain the status of this patient. Residual beta-adrenergic blockade of the heart would have compromised its capacity to respond to endogenous catecholamines, and thereby to overcome a marked afterload elevation following alpha-adrenergic agonism. An alpha-adrenergic-blocking drug, phentolamine, was given in two 5-mg injections, iv, spaced 5 min apart. This caused radial arterial pressure to fall to 160/100 torr and PAOP to change from 30 to 15 torr. The surgeons felt that immediate operation was necessary to save the ischemic leg. Anesthesia was induced with thiopental and maintained with halothane-N<sub>2</sub>O-O<sub>2</sub>, and blood pressure was controlled with sodium nitroprusside (SNP). The operation lasted 3 hours, 10 min and was uneventful. The SNP was continued postoperatively and hydralazine was also given, im. As blood pressure responded gradually to the hydralazine, the SNP infusion rate was tapered until it was stopped approximately 24 hours postoperatively. The operation was successful, and recovery was uneventful.

**Patient 2.** A 52-year-old man was scheduled for gastrectomy about a month after the episode described above. This patient was also receiving clonidine, at the same dosage of 0.2 mg four times daily, as well as hydralazine and furosemide. He was taking propranolol in addition, but this had been tapered slowly and stopped three days previously in anticipation of discontinuation of clonidine therapy. Because we did not wish to repeat the experience with clonidine withdrawal syndrome, care was taken to order all oral medications given as usual on the morning of the operation, scheduled for about 11 AM. When he arrived in the operating room, the patient stated that he had received his "pills" that morning at 6 AM and he felt well. The first sign of any problem was seen after the arterial line was inserted. Systolic pressure was more than 300 torr. Nothing more was done while the chart was reviewed. The medication sheet showed that he had received all his usual medications *except* clonidine that morning. Infusion of SNP was begun to control blood pressure, the operation was cancelled, and the patient was sent to the Surgical Intensive Care Unit. Since operation was necessary as soon as possible, clonidine was not given, but treatment with hydralazine, im, was begun in much the same manner as done for the previous patient postoperatively. Over the next 24 hours, blood pressure came under control with hydralazine, and SNP was slowly tapered and stopped. The following day, the gastrectomy was done without incident with enflurane-N<sub>2</sub>O-O<sub>2</sub> anesthesia. Recovery was uneventful.

## DISCUSSION

Clonidine is an antihypertensive drug that is thought to cause vasodilation secondary to reduction of sympathetic outflow from the brain.<sup>10,11</sup> This central site of action sets it apart from other antihypertensive vasodilators such as hydralazine and prazosin, which act peripherally on the vessels themselves.

A recent review gave a plan for treatment of hypertension that included clonidine.<sup>12</sup> The plan began with a diuretic, then added propranolol, methyldopa,

or reserpine, then in the third step added hydralazine or prazosin or clonidine. This program of staged therapy is apparently in common use today. Since clonidine is added after propranolol has failed to control adequately the blood pressure, it is evident that patients receiving clonidine according to this plan will have fairly severe hypertension and will usually also be receiving a beta-adrenergic blocking drug. Such patients differ from the subjects of a recent controlled investigation of clonidine withdrawal.<sup>13</sup> Rebound hypertension was not seen in that study. The subjects had been untreated for four weeks before they began to take clonidine as their only antihypertensive drug. They obviously were neither severely hypertensive nor at risk for problems associated with multiple medications.

Arterial blood catecholamine concentrations can double during clonidine withdrawal.<sup>3</sup> To the only moderately hypertensive patient who has good myocardial reserve function, this fact may be of no importance. For the severely hypertensive patient with demonstrated peripheral vascular constriction and poor myocardial reserve, a dangerous increase in afterload may result. The presence of beta-adrenergic blockade will aggravate this situation both by blocking any peripheral vascular dilation caused by catecholamines and by blocking some or all of the cardiotoxic action of these transmitters. The result will be a predominantly alpha-adrenergic agonism by these catecholamines in the propranolol-treated patient. This is not peculiar to propranolol, since the same phenomenon has been observed in a patient receiving timolol, another beta-adrenergic blocking drug not yet available commercially in the United States.<sup>4</sup>

Based on these considerations and in light of our experiences described in the case reports, guidelines are suggested for patients receiving antihypertensive medications and scheduled for operation. A generally desirable tactic is to continue treatment throughout the perioperative period, including usual medication by mouth on the morning of operation. When propranolol is part of the antihypertensive treatment program and clonidine is not, the propranolol should be continued. When clonidine is the only drug being taken, it should be continued.

When both propranolol and clonidine are being taken, both should be withdrawn. First, the propranolol should be tapered to a low dose from preoperative day 7 to preoperative day 3, when it should be stopped. From preoperative days 3 to 1, residual beta-adrenergic blockade should dissipate; during this time clonidine should be continued. On the morning of preoperative day 1, clonidine should be stopped and oral administration of hydralazine either

started, if it was not already being given, or increased if it was. During that final preoperative day the patient should be monitored closely to ensure adequate blood pressure control with the hydralazine. This should avoid any hypertensive crisis, but if it does not, SNP may be used temporarily to regain control until effective hydralazine therapy is established.

Enflurane would be the current anesthetic of choice, since catecholamine-induced cardiac arrhythmias are less likely to occur with this agent than with others. Postoperatively, oral medications may be resumed fully as soon as the patient can manage to swallow and retain them. Obviously, those who have had gastrointestinal operations and are receiving gastric suction therapy will need maintenance therapy with parenteral drugs. Of the three vasodilators, hydralazine, prazosin and clonidine, only hydralazine is available for injection.

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Anesthesiology  
51:92-94, 1979

## Post-spinal Headache or Intracranial Tumor after Obstetric Anesthesia

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Headache and sixth-cranial-nerve palsy are well-known complications of spinal anesthesia.<sup>1-6</sup> Subarachnoid blocks have also reportedly exacerbated symptoms of pre-existing neurologic disease.<sup>7-10</sup> We are unaware of any report of a case of an intracerebral lesion that initially manifests as headache and abducens-nerve palsy following spinal anesthesia. We recently encountered such a case, in which symptoms

developed following a saddle block for a normal vaginal delivery.

#### REPORT OF A CASE

An 18-year-old Mexican-American primigravida was admitted to another hospital in labor after an uncomplicated term pregnancy. A low forceps vaginal delivery was facilitated by the use of a subarachnoid block administered by the obstetrician. A 25-gauge needle was introduced at the L<sub>4-5</sub> interspace atraumatically, and 40 mg (5 per cent sol.) lidocaine were injected. Paresthesias were not elicited. The patient was discharged from the hospital the following day.

Two weeks later she consulted a physician in Tijuana, Mexico, complaining of the recent onset of a retro-orbital headache, partially relieved by recumbency. There was no previous history of headache occurring during the pregnancy. She was unsuccessfully treated with ergotamine tartrate-caffeine tablets (Cafergot) and aspirin. Five weeks post partum she returned to her obstetrician with persistent headache, which he ascribed to cerebrospinal fluid (CSF) leakage. Bed rest, increased oral hydration, but no

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Received from the University of California, San Diego, and Veterans Administration Hospital, La Jolla, California. Accepted for publication December 7, 1978.

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