

impossible to inflate and the patient became grossly cyanotic. The nebulizer was interposed between the slip joint of the endotracheal tube and the Y-piece of the circle carbon dioxide absorption system (fig. 2). The cartridge was squeezed ten times and within 60 seconds, gas was able to enter the lungs and breath sounds were heard. Five minutes after the initiation of nebulization of isoproterenol, the cyanosis disappeared.

CONCLUSION

The clinical experience with this convenient, effective and accurate nebulizer during the past year has been gratifying.

CASE REPORTS

Management of Hemorrhage Following Induced Hypotension

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Reactionary hemorrhage is one of the frequently reported complications following deliberate hypotension. Hampton and Little, in an extensive review, found an incidence of 1.16 per cent in 6,805 cases of controlled hypotension performed in the United States.¹⁻² Tough (1960) reported that there was no evidence that the risk of reactionary hemorrhage was increased owing to hypotension during anesthesia.^{3,4} Reviewing data published between 1958 and 1963, Larson found that the overall incidence of reactionary hemorrhage was 0.27 per cent in 13,264 patients subjected to deliberate hypotension.⁵ By achieving adequate hemostasis, using pressure dressings, and allowing a slow return of arterial blood pressure to preoperative levels, McLaughlin reported only one instance of reactionary hemorrhage in one thousand consecutive cases of deliberate hypotension.⁵

During 1965-67, we used controlled hypotension in 92 cases for extensive surgery of the

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head and neck and thorax. Following the precautionary measures outlined by McLaughlin, reactionary hemorrhage occurred in only one patient. In this instance, hemorrhage was precipitated by straining in the early post-operative period while changing a tracheostomy tube and after the arterial blood pressure had returned to the preoperative level. The hemorrhage was successfully managed by re-instituting hypotension for a period of three hours.

CASE REPORT

A 54 year old woman, weighing 80 kg., was admitted for treatment of a malignant mixed tumor of the right parotid gland, of 40 years duration. The tumor has been slowly but relentlessly destroying the right side of the face, which presented an open ulcerated area. Her past history included a radical excision of the tumor at the age of 16, complicated by right facial paralysis; total abdominal hysterectomy for a fibroid uterus; right radical neck dissection; first and second stages of chest flaps; and extensive irradiation to the right neck and

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parotid region. Recent skull roentgenograms revealed invasion of the right mandible, zygoma, external auditory canal, mastoid and temporal bones. No other significant medical conditions existed prior to the surgical procedure. Chest roentgenograms and ECG were essentially normal and the hematocrit was 43.5 per cent.

Tracheostomy was performed under local anesthesia and a cuffed, rubber tracheostomy tube was inserted. Anesthesia was induced with thiopental (Pentothal) sodium and maintained with 60 per cent nitrous oxide, 0.2 to 0.3 per cent halothane, intermittent injections of *d*-tubocurarine to a total of 66 mg., and artificial ventilation to maintain an end-expired $P_{T_{1/2}}$ between 30 and 35 mm. of mercury. After a steady state of anesthesia was obtained, hypotension was deliberately induced, using intermittent doses of pentolinium (Ansolylen) to a total of 23.5 mg. and gradual tilting of the patient from the horizontal to the head-up position, until the systolic pressure had fallen from 140 to 80 mm. of mercury. At this level of hypotension, the operation was begun and a relatively bloodless field was observed. An extensive operative procedure, lasting seven hours, was performed by three surgical teams including those specializing in ear, nose and throat surgery and neurosurgeons and plastic surgeons. The procedure involved radical resection of a recurrent malignant mixed parotid tumor with removal of the right hemi-mandible, soft tissues overlying the carotid arteries, right ear, mastoid air cell system, right temporal bone and involved dura. A fascia lata graft was used to close the gap in the dura, the chest full thickness pedicle was rotated to close the defect and the chest wound was covered by skin grafts taken from the right thigh. Near the termination of the procedure, the blood pressure was allowed to return gradually to a systolic pressure of 130 mm. of mercury. Two units of blood, 2,000 ml. of lactated Ringers solution and 500 ml. of 5 per cent dextrose in 0.2 per cent saline were given during the procedure. After a pressure dressing was applied, the anesthetics were discontinued and 1.2 mg. of atropine and 3 mg. of neostigmine were administered.

The patient was awake, responsive and

breathing adequately. Replacement of the rubber tracheostomy tube by a metal one was accompanied by severe coughing and straining which was rapidly followed by profuse bleeding from the wound. Two more units of blood were given and the patient was re-curarized to facilitate positive pressure artificial ventilation. Hypotension was re-instituted with 4 mg. of pentolinium and change of position, until the systolic pressure reached 80 mm. of mercury. Shortly after the reduction in arterial pressure, there was a significant decrease in bleeding. Hemorrhage stopped completely after 2 hours of hypotension. Subsequently, the hematoma beneath the skin graft was evacuated under local anesthesia; no bleeding points were encountered. Thereafter, the postoperative course was uneventful except for development of a mild infection in the upper portion of the graft. The graft survived and the patient was discharged from the hospital in good condition 18 days after operation.

COMMENT

This case demonstrates the occurrence of reactionary hemorrhage following deliberate hypotension. Postoperative bleeding was related to coughing and straining, which occurred when the tracheostomy tube was changed. Because of the hazard of straining in the early postoperative period we have modified our technique by maintaining these patients on artificial ventilation and delaying reversal of *d*-tubocurarine paralysis for several hours after termination of anesthesia. By preventing coughing and straining, abrupt changes in venous and arterial pressures, which may dislodge clots, can be avoided.

We suggest that the incidence of reactionary hemorrhage can be minimized by following these procedures: establish adequate hemostasis before closure of the surgical wound; allow blood pressure to return to normal gradually, avoid the use of vasopressors; application of pressure dressings whenever feasible; and prevent coughing and straining for several hours postoperatively.

Ganglion blocking drugs have been used to abolish vasoconstriction associated with hemorrhage during operation,⁶ and their beneficial

effect has been confirmed.⁷ This case illustrates that re-induction of hypotension may be of value to control reactionary hemorrhage if it occurs following deliberate hypotension.

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An Unexpected Complication (Hyperthermia) While Using the Emerson Postoperative Ventilator

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The introduction of the Emerson Postoperative Ventilator in 1965 has contributed substantially to the effective management of patients with acute or chronic respiratory insufficiency. This piston type, volume limited, pressure variable respirator has many advantages, including great flexibility in volume, rate and pressure settings; a built-in "sighing device" to provide increased inflation at regular intervals; excellent humidification provided by a reflux type, heated vaporizer; independent controls for inspiratory and expiratory rate and the ability to administer oxygen at concentrations from 20 to approximately 90 per cent. The disadvantages of this respirator are its inability to function as an assistor and the great bulk of the equipment which makes its use cumbersome in a crowded patient area. We have successfully used this respirator at the University of Michigan Medical Center in many types of patients ranging from 1 week to 70 years in age and from 5 to 300 pounds in weight. This report concerns a hazard inherent in the use of the Emerson Postopera-

tive Ventilator which may lead to serious complications in some patients.

CASE REPORT

The patient was an 18 month old white male child, admitted from another hospital with the diagnosis of Guillain-Barre syndrome. On admission the child was found to be acutely ill showing evidence of generalized profound muscle weakness and absent deep tendon reflexes.

Shortly after admission a tracheostomy was performed under local anesthesia and the child's lungs were ventilated with a pressure-limited ventilator. During the next 30 days, the patient had repeated bouts of atelectasis and bronchopneumonia. During this entire period, artificial respiration was provided with the pressure-limited respirator, with the exception of one long and one brief period of time when an attempt was made to provide adequate ventilation with a Drinker-type, tank respirator. These attempts were unsuccessful and the patient developed increasingly severe respiratory distress. He eventually developed pneumonia and was quite obviously in serious difficulties. At this time it was decided to switch from the pressure-limited device to the Emerson Postoperative Ventilator. Within 30

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