When proper position of the tube and integrity of the balloon had been verified, subsequent bronchoscopy revealed the fistula. An awareness of the conditions predisposing to the formation of tracheoesophageal fistula which existed in this case prompted immediate bronchoscopy and rapid identification of the problem. These conditions were: prolonged tracheal intubation with a cuffed tube,[1, 2, 3] simultaneous use of a nasogastric tube,[2] tracheobronchial inhalation injury with superimposed infection, systemic sepsis,[4] and intermittent episodes of moderately severe hypotension secondary to gastrointestinal hemorrhage.[4]

The use of endobronchial tubes in the management of a comparable problem has been described by Butlin, *et al.*[5] This technique permits adequate ventilation if care is taken to prevent obstruction of the right upper lobe bronchus and protects against soilage of the lower airway. Because mediastinitis, a complication of tracheoesophageal fistula formation, invariably occurs, normal esophageal function is impaired. Therefore, esophageal diversion and gastrostomy may be useful in protecting the airway if adequate drainage can be achieved. The combined usage of endobronchial tubes for airway maintenance and protection and esophageal diversion for drainage may be helpful for management of problems such as that described here until the patient's condition will permit corrective surgery.

References

1. Flege JB Jr: Tracheo-esophageal fistula caused by cuffed tracheostomy tube. Ann Surg 166: 153–156, 1967
2. Hedden M, Ersoz CJ, Safar P: Tracheoesophageal fistulas following prolonged artificial ventilation via cuffed tracheostomy tubes. Anesthesiology 31:281–289, 1969
3. Martinez LR, Kalter RD: Extensive tracheal necrosis associated with a prestretched tracheostomy tube cuff. Anesthesiology 34: 488–489, 1971
4. Andrews MJ, Pearson FG: Incidence and pathogenesis of tracheal injury following cuffed tracheostomy with assisted ventilation: Analysis of a two-year prospective study. Ann Surg 173:249–263, 1971
5. Butlin GR, Sellery GR, Spoevel WE: An unusual airway problem. Can Anaesth Soc J 17:78–82, 1970

Circulatory Changes Following Implantation of Methylmethacrylate Bone Cement

F. T. Schuh, M.D.,* Sigrid Moelling Schuh, M.D.,† M. G. Viguera, M.D.,‡ R. N. Terry, M.D.§

Methylmethacrylate has been extensively used as a dental prosthetic and as a cranial-bone substitute for over two decades. In the early 1960's, Charnley introduced methylmethacrylate into hip-joint replacement surgery for fixation of prostheses.[1] Recently the Food and Drug Administration approved its use in the United States for that purpose. However, episodes of acute hypotension and cases of cardiovascular collapse at the time of intraosseus implantation of the cement have been reported.[2, 3]

The present investigation was undertaken to determine the frequency and magnitude of circulatory changes following methylmethacrylate implantation.

Materials and Method

Studies were made in 32 patients undergoing total hip replacement (Mueller–Charnley

* Resident in Anesthesiology, Buffalo General Hospital; Clinical Assistant Instructor in Anesthesiology, State University of New York (SUNY) at Buffalo; on leave of absence from Christian Albrechts University, Kiel, W. Germany.
† Resident in Anesthesiology, Buffalo General Hospital; on leave of absence from Christian Albrechts University, Kiel, W. Germany.
‡ Clinical Associate in Anesthesiology, Buffalo General Hospital; Clinical Assistant Professor of Anesthesiology, SUNY at Buffalo.
§ Chief, Department of Anesthesiology, Buffalo General Hospital; Clinical Professor of Anesthesiology, SUNY at Buffalo.
Received from the Department of Anesthesiology Buffalo General Hospital. SUNY at Buffalo, 100 High Street, Buffalo, New York 14203. Accepted for publication February 20, 1973. A preliminary abstract was presented at the Fifth International Congress on Pharmacology, San Francisco, 1972.

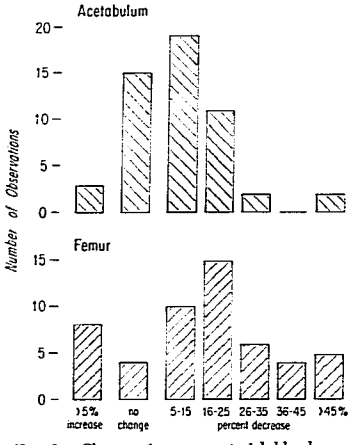

FIG. 1. Changes in mean arterial blood pressure following implantation of methylmethacrylate bone cement into the acetabulum (*top*) and into the femur (*bottom*) during 52 total-hip-joint replacements.

prostheses). The patients averaged 63 years in age and 72 kg in weight. ASA classifications were Physical Status I–III.

The bone cement used for fixation of prostheses was prepared by mixing one unit, 20 ml, of liquid methylmethacrylate solvent with 40 mg of polymethyl methacrylate powder.

Anesthesia. Premedication consisted of atropine sulfate, 0.4 mg, and morphine, 7.5–10 mg, or meperidine, 50–100 mg, one hour before operation.

Anesthesia was induced with thiamylal sodium (125–300 mg, iv) and maintained with a 6-ml/min total flow of 65 per cent N_2O in oxygen in a circle filter system, supplemented as needed by intravenous meperidine or morphine. The trachea was intubated following succinylcholine, 1 mg/kg body weight. After return of spontaneous breathing, appropriate muscle relaxation was maintained by *d*-tubocurarine, avoiding injection during the period of implantation because of its potential hypotensive effect.[4]

Respiration was controlled mechanically throughout the procedure employing a Ventimeter ventilator at a respiratory rate of 10–12/min at an initial minute volume of 85–90 ml/kg body weight. Ventilation was thereafter adjusted as necessary to maintain an arterial *p*H of 7.36–7.44.

During the first hour of anesthesia, Ringer's lactate solution was infused at 15 ml/kg, and 10 ml/kg each additional hour. At the moment of hip implantation total fluid infused averaged 1297 ± 46 ml. Whole blood was transfused as indicated beginning before insertion of the prosthesis.

Systemic arterial blood pressure was measured directly via a percutaneous radial-artery cannula using a Statham P23De transducer and was recorded throughout the operation on a 1508 Honeywell Visicorder oscillograph. Control blood pressure was established for each individual as that recorded immediately prior to insertion of the cement. Variations of ±5 per cent from control were considered within normal limits. The electrocardiogram was visually monitored throughout on a Tektronix P40 oscilloscope.

RESULTS

Of the 52 patients who had hip replacements, two (3 per cent) showed no cardiovascular changes following methylmethacrylate insertion. A decrease in blood pressure immediately following cement implacement into the acetabular bed, and a second hypotensive episode coincident with placement of methylmethacrylate into the marrow cavity of the femur, occurred in 29 patients (55 per cent). One patient responded by an increase in blood pressure following each of the two-site placements. In the remaining 20 patients (39 per cent) combinations of increase, decrease, or no change of pressure followed cement placement.

Figure 1 summarizes the blood pressure data as a frequency histogram.

In all the aforementioned hypotensive responses the decrease in blood pressure occurred less than a minute after cement insertion. Hypotension was maximal at 1.5 to 2 minutes. When an increase in blood pressure followed implantation it appeared more rapidly, within half a minute, returning to control within three minutes.

The magnitude of blood pressure decreases following implantation was found to be directly related to patient age (fig. 2).

An increase in heart rate invariably accompanied a hypertensive response, whereas no consistent changes in heart rate occurred during hypotensive episodes. Arrythmias were observed in only one patient of this series. Ventricular extrasystoles developed during one of the more profound hypotensive responses (a 66 per cent decrease of mean arterial pressure), persisted until the blood pressure returned to control, and did not subsequently recur.

Discussion

In our patients intraosseus application of methylmethacrylate was followed in almost every instance by alteration of arterial pressure or heart rate. These data are in agreement with previous reports.

Charnley and others postulated that the transient circulatory changes resulted from absorption of methylmethacrylate monomer into the vascular compartment. The prompt onset and brief duration of the cardiovascular alterations support this assumption. This pattern closely resembles the hypotension following intravenous injection of the liquid monomer in dogs.[6]

Inasmuch as the quantity of monomer actually entering the circulation at hip replacement is unknown, it is not possible to establish a dose–response relationship. The maximum amount of monomer that can be absorbed, however, should be a function of the mixing time (during which vaporization of monomer into the atmosphere occurs) and of the surface area exposed to the implanted cement.

The surface area of contact between the cement and femoral medullary canal generally exceeds that of the acetabulum, which may account for the greater hypotensive response following femoral versus acetabular implantation.

The mechanism of action underlying the cardiovascular side-effects has not been identified. These results suggest, however, as do responses in experimental animals,[5, 6] that methylmethacrylate monomer exerts its effect through a vasodilatory action. The occasional increase in heart rate together with an increase in blood pressure might be explained as a compensatory mechanism.

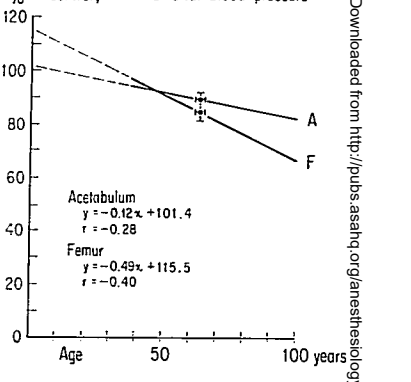

Fig. 2. Relationship between age of the patient and alteration in blood pressure following insertion of methylmethacrylate bone cement into the acetabulum (*A*) and femur (*F*) during 52 total-hip-joint replacements.

Hypotensive episodes and cardiovascular collapse are inherent hazards in the course of operations employing methylmethacrylate bone cement. Inasmuch as the hypotensive mechanism appears to result from sudden vasodilatation, appropriate prophylaxis would seem to be adequate replacement of fluid and blood, with secondary reliance on vasoconstrictor agents.

References

1. Charnley J: Acrylic Cement in Orthopedic Surgery. Edinburgh and London, Livingstone, 1970
2. Powell JN, McGraft PJ, Lahiri SK, et al: Cardiac arrest associated with bone cement. Br Med J 3:326, 1970
3. Newens AF, Volz RG: Severe hypotension during prosthetic hip surgery with acrylic bone cement. Anesthesiology 36:298–300, 1972
4. Stoelting RK: Blood pressure responses to *d*-tubocurarine and its preservatives in anesthetized patients. Anesthesiology 35:315–317, 1971
5. Schuh FT, Terry RN, Viguera MG: Cardiovascular side effects of methyl methacrylate in canine (unpublished data)
6. Pebles DJ, Ellis RH, Stride SDK, et al: Cardiovascular effects of methyl methacrylate cement. Br Med J 1:349–351, 1972