of nitrous oxide in laminated, wire-reinforced endotracheal tubes. A wide variety of foreign bodies also have been reported to cause endotracheal tube obstruction, including nasal turbinates avulsed during nasotracheal intubation, surgical tape, dried lubricant, a broken endotracheal tube stylet sheath and retained plastic debris from the manufacturing of the Murphy eye.

Another common complication of endotracheal tube use is right mainstem bronchus intubation which has been reported, in one series, to occur in 9.6 percent of all cases of intubation. Alveolar hyperinflation, atelectasis and right-sided tension pneumothorax can result from right mainstem bronchus intubation. We are unaware, however, of any previously reported cases of tension pneumothorax resulting from valv-alle endotracheal tube obstruction.

As demonstrated by the removed specimen (Fig 1 and 2) obstruction of an endotracheal tube by such a ball-valve mechanism requires both endotracheal tube ports to be involved in the occlusion. In this case, a mass of dried secretions and clotted blood was adherent inside the tube, completely occluding the Murphy eye. The remainder of the dried secretions and clot extended beyond the tip of the endotracheal tube. The large diameter and mobility of this mass allowed it to open and close over the distal endotracheal tube port. Inspiratory pressure would move the mass away from the distal port allowing inspiratory flow to occur; expiratory pressure distal to the endotracheal tube would push the mass into the distal port, occluding expiratory flow. The repeated ball-valve action of this mass resulted in pulmonary hyperinflation and consequent barotrauma, as manifested by a tension pneumothorax and subcutaneous emphysema.

Whenever airway obstruction is suspected in an intubated patient, prompt, thoughtful intervention is necessary to locate and relieve the obstruction. By utilizing physical examination findings, manual ventilation, a suction catheter, and by deflating the endotracheal cuff the problem should be localizable to either the patient, the endotracheal tube, the cuff or the ventilator circuit. In this patient, difficult passage of the suction catheter suggested a problem related to the endotracheal tube. Urgent extubation and reintubation revealed the nature of the endotracheal tube obstruction, while the physical findings allowed prompt recognition and treatment of the tension pneumothorax.

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Deterioration of Ventricular Tachycardia to Ventricular Fibrillation after Rapid Intravenous Administration of Magnesium Sulfate*

Sam Vaskin, M.D.; Bernard Belhassen, M.D.; and Shlomo Laniado, M.D.

We report the case of a patient who had development of ventricular fibrillation following rapid intravenous administration of magnesium sulfate (2 g over 5 s) for treatment of sustained monomorphic ventricular tachycardia. Initial slowing of the tachycardia was followed by gradual widening of QRS complexes and electrical alternans, leading to ventricular fibrillation within 3 min of magnesium administration.

(Chest 1992; 101:1445-47)

VT = ventricular tachycardia; VF = ventricular fibrillation; LVEF = left ventricular ejection fraction; RBBB = right bundle branch block

Intravenous magnesium sulfate is a well-established treatment of polymorphic ventricular tachycardia (VT) associated with QT prolongation. Recently, the use of magnesium was proposed as an alternative therapy for sustained monomorphic VT. Reservations concerning rapid administration of magnesium sulfate have been expressed, mainly relating to possible cardiac arrest; however, electrocardiographic documentation of the sequence of events leading to cardiac arrest is not available.

We report the case of a patient who had development of ventricular fibrillation (VF) following rapid administration of magnesium sulfate for treatment of sustained monomorphic VT.

CASE REPORT

A 65-year-old man was hospitalized with an extensive anterior myocardial infarction complicated by severe left ventricular dysfunction (left ventricular ejection fraction [LVEF]=21 percent, right bundle branch block [RBBB], and left anterior hemiblock). Two weeks later, he developed multiple episodes of sustained VT. The tachycardia was of monomorphic configuration, with rates ranging from 180 to 190 beats/min. VT resulted in hemodynamic deterioration and invariably required electrical cardioversion for termination. Spontaneous deterioration of VT to VF, however, was never observed. Successive treatment with intravenous lidocaine, procainamide, bretylium tosylate, amiodarone, and magnesium sulfate (6 g over 24 h) failed to terminate or to prevent the episodes of sustained VT. Nonoliguric renal failure occurred (with gradual increase of serum creatinine levels up to 4.5 mg/dl) and led to discontinuation of magnesium sulfate infusion. The last serum magnesium level, obtained shortly before discontinuation of treatment, was 3.7 mg/dl.

Three days later, during a new episode of sustained monomorphic VT, which occurred while the patient was receiving a continuous infusion of procainamide and amiodarone (2 mg/min and 0.5 mg/ min, respectively), a bolus of 2 g of magnesium sulfate (as a 10 percent solution) was administered over a period of 5 s (Fig 1). Progressive slowing of the VT, from 180 to 150 beats/min, was

*From the Department of Cardiology, Ichilov Hospital, Tel Aviv Medical Center and Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel.
observed within the first minute following magnesium administration; however, gradual widening of QRS complexes and electrical alternans appeared afterwards and led to frankly disorganized ventricular activity culminating in VF within 3 min. The patient was successfully resuscitated with a single 300-J direct current shock, and remained in sinus rhythm for several hours without further changes in QRS-T morphology or QT interval. Additional episodes of VT were terminated by rapid ventricular pacing; nevertheless, the patient died of cardiogenic shock two days later.

DISCUSSION

Slow infusion of magnesium sulfate has been repeatedly found to be safe when administered to patients in sinus rhythm, as well as during supraventricular or ventricular tachyarrhythmias. Rapid intravenous bolus of magnesium sulfate (2 g/5 s), administered to patients with reentrant supraventricular tachyarrhythmias has recently raised controversy regarding the safety of such dosing. Our patient experienced deterioration of sustained VT to VF after receiving magnesium sulfate at this rapid infusion rate. Although spontaneous arrhythmia degeneration cannot be excluded, a direct causal effect of magnesium in the induction of VF is suggested by the following: (1) absence of such deterioration during many similar episodes of VT documented both before and after the administration of the rapid bolus of magnesium sulfate; (2) the sequence of events, culminating in VF within 3 min of the magnesium bolus; and (3) slowing rather than acceleration of VT preceding the degeneration to VF. Of note, the patient had received higher doses of magnesium sulfate at slower infusion rates several days before. This was accompanied by a slight elevation of serum magnesium levels (3.4 mg/dl) but had no detrimental consequences. It was only three days after discontinuation of magnesium infusion that the 2-g bolus in question was administered. Thus, although serum magnesium levels were not obtained following resuscitation from VF, and notwithstanding the deterioration of renal function, we believe that the rate of magnesium administration, rather than the total amount administered, played an important role in deterioration of VT to VF. Also, the fact that magnesium was administered while the patient was under the influence of other antiarrhythmic agents argues against a "pure" magnesium-related side effect. However, such a setting is likely the one that will be encountered in clinical practice since magnesium sulfate is not considered a first-line drug for sustained monomorphic VT.

The mechanism of deterioration of VT to VF in our patient is unclear. Marked widening of the QRS complex and slowing of the ventricular rate before VF induction reflect a strong negative dromotropic effect, similar to that of class 3 antiarrhythmic drugs. Such effect could be due to the rate of drug administration. Magnesium sulfate has not been shown to affect intraventricular conduction and refractoriness significantly when infused slowly (infusion rates of 1 g/min). On the other hand, blockade of conduction over accessory bypass tracts may occur with faster infusion rates (2 g of magnesium sulfate over 15 s or less). Also, decreased intraventricular conduction following magnesium administration has been observed during rapid ventricular pacing. Regardless of the mechanism involved, and as interest in the clinical use of magnesium sulfate increases, a word of caution against its rapid intravenous administration, even during resuscitation for ventricular arrhythmias, is in order.
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Primary Pulmonary Disease Due to Mycobacterium Avium-Intracellulare*

Jerome M. Reich, M.D.

*From the Division of Pulmonary Medicine, Bess Kaiser Medical Center, and Center for Health Research, Kaiser Permanente, Northwest Region, Portland, Oregon.
Reprint requests: Dr. Reich, 3414 North Kaiser Center Drive, Portland, Oregon 97227

Primary pulmonary disease due to Mycobacterium avium-intracellulare (MAI) presented in an immunologically intact child exposed to pet birds. (Chest 1992; 101:1447-48)

MAI = Mycobacterium avium-intracellulare complex; MB = mycobacteria; NTM = nontuberculous mycobacteria; PRIMAC: primary pulmonary disease due to MAI

The rarity with which MAI leads to primary pulmonary disease in humans is one of the more puzzling aspects of its peculiar epidemiology; only three cases, all involving children, have been reported.1 In a comprehensive review of the subject of NTM,2 this pattern was not cited, and in a review of the roentgenographic features of NTM,3 absence of the primary pattern was listed (along with absence of pleural effusion) as one of the two roentgenographic attributes of MAI which differentiated it from tuberculosis.

CASE REPORT

A three-year-old girl, previously in good health, presented in 1980 with complaints of a nonproductive cough and fever of one month's duration. A CR showed atelectasis of the right lower and middle lobe and left hilar adenopathy; the right hilum was obscured by the atelectatic shadows (Fig 1). Complete blood count results were normal; the absolute lymphocyte count was 4,000/µL. Primary tuberculosis was suspected on clinical and roentgenographic grounds. A Mantoux test showed no induration to 5 TU at 48 h. The hilar adenopathy diminished over time.

Bronchoscopy revealed extrinsic compression of the bronchus intermedius from the medial aspect and overlying friable erythematous mucosa. Bronchial washings were negative on smear and culture for MB. At thoracotomy, the two atelectatic lobes were removed along with specimens of enlarged right hilar lymph nodes. The lung and lymph nodes exhibited numerous confluent caseating granulomata. Stains to demonstrate MB and fungi were negative.

Figure 1. Atelectasis of middle and lower lobe and contralateral hilar adenopathy are evident.