acute elevation in ST segments. Severe bradycardia and hypotension followed. Fluids, atropine, and ephedrine did not improve the situation. Closed chest massage and epi-
epinephrine 100 µg restored blood pressure and pulse rate. The ST segments reverted to isoelectric.

I believe this incident represents coronary spasm precipitated by the administration of propranolol. The patient had tolerated a resting pulse of 110–120 beats/min for several days before her surgery. Her pulse was 110–120 beats/min until the epinephrine was applied. Blood pressure was maintained at 90–100/50–60 mmHg throughout the two previous hours of surgery. The central venous pressure was maintained at 8–10 mmHg. A hematocrit drawn following the episode of ischemia was 30% and the potassium was 4 mEq/l.

Sudden ST segment elevation associated with hypertension and bradycardia is consistent with coronary spasm. In the anesthesia literature, 13 of 14 patients reported to have suffered coronary spasm were receiving beta blockers.1–3 This may represent more than just happenstance. Recently, Nussmeier and Slogoff4 reported a case in which they treated intraoperative tachycardia with propranolol. Acute ST elevation followed. The ischemia did not respond to nitroglycerin but was reversed with verapamil.

The cardiology literature documents exacerbation of spasm by beta blockers. Further, spasm has been associated with CaCl infusions and other interventions that promote smooth muscle contraction.

I believe that coronary artery spasm should be considered as a possible consequence of beta blockade therapy for epinephrine-induced tachycardia or for tachycardia in patients with coronary disease.

TERRY STEPHEN VITEZ, M.D.
5700 Hickam Street
Las Vegas, Nevada

References

(Accepted for publication June 21, 1985.)

More on Mass Spectrometers and Aerosol Propellants

To the Editor.—In our recent report,1 we described a misleading mass spectrometer reading caused by an aerosol propellant. Chlorofluorohydrocarbon propellants are interpreted by the Perkin-Elmer® (fixed collector) mass spectrometer as isoflurane. In that report, the effect of these nonrespiratory, nonanesthetic gases was presumed due to mass spectral overlap, for which the spectral overlap erasing algorithm was not designed. With the installation of the new Perkin-Elmer® Advantage and Chemetron Sara II® systems at our institution, we performed additional in vitro testing with interesting results.

Metered nebulizers—iso-proterenol HCl, (Norisodrine, Aerohalor®, Abbott Laboratories), metaproterenol sulfate (Alupent®, Boehringer Ingelheim, Ltd.), and albuterol (Ventolin®, Glaxo, Inc.)—were introduced into both systems by using 100% O2 and then 100% N2 as carrier gases to preclude masking spectral overlap with those gases. The findings demonstrated no differences between the carrier gases. In the Perkin-Elmer® system, clinical doses from the nebulizers (1–2 metered aerosols) were displayed uniformly as isoflurane in concentrations up to 10%. In larger doses (20–30 metered aerosols), the gases were interpreted as up to 10% isoflurane and up to 20% CO2. Presumably, it takes more propellant to stimulate the CO2 collector plate. Chemetron’s Sara II® system was tested identically and interpreted clinical doses of drug/propellant as up to 5% enfuvane. In larger doses, the gases were interpreted as up to 5% enfuvane and up to 20% CO2.

Halogenated hydrocarbon propellants ionized by a research mass spectrometer (Finnigan MAT 4515 GS/MS®, San Jose, California) under controlled conditions yielded ions of similar mass spectra as those of all commonly employed halogenated inhalation anesthetics and CO2. Without a specific algorithm to erase this spectral overlap, these similarities caused the erroneous interpretations. With both the Perkin-Elmer® and the Chemetron® systems, propellant at one sampling station did not affect subsequent measurements from that or other stations. Hence, these errors, lasting 4–5 s, were transient and not propagated.

This problem with propellants is not serious and certainly does not mandate that the fixed collector mass spectrometers be redesigned because hand-held inhalers
are only used infrequently and the effects are transient. The misleading readings for isoflurane, enfurane, and CO₂ partial pressures with these types of propellants are easily obviated by not dispensing medication with such a propellant while the mass spectrometer is sampling. When sampling is continuous, removing the sample port briefly, 10–15 s, from the circuit during administration of medication would prevent misreadings. Readings from a clinical, fixed collector mass spectrometer system must be interpreted cautiously when gases that the system was not designed for are being used.

GARY J. THEISEN, M.D.
Resident
Department of Anesthesiology

NIKOLAUS GRAVENSTEIN, M.D.
Assistant Professor
Department of Anesthesiology

REFERENCE
(Accepted for publication June 21, 1985.)

Using a Priming Dose of Relaxant Is Not New

To the Editor:—The use of a priming dose of relaxant as a means of improving conditions for tracheal intubation is a novel but not a new idea. The use of a “test” dose of tubocurarine (usually 5 mg) given 2–3 min before the main dose was taught as a routine procedure in Liverpool when I was a resident in the late 1940s, early 1950s. It was also accepted practice to give the full “relaxing” dose of tubocurarine (usually 20–40 mg) immediately before the thiopentone. I have personally used this technique as a routine for intubation in thousands of patients with all the available competitive relaxants and, with the exception of laudexium, all produced good conditions for passage of the tube. It is particularly valuable with vecuronium, after which intubation can be as easy as with suxamethonium.

The reasons for this technique, as originally described by Gray and Halton, were twofold. The early administration of tubocurarine was to identify patients with latent myasthenia gravis where the period of apnoea would be very prolonged. Sensitivity would be suspected if the response to the test dose was other than mild ptosis. Giving the full relaxant dose before the thiopentone aims at synchronizing the maximum effect of two drugs with different onset times. This technique demands an “open vein”—a practice not generally accepted at the time—and with this precaution I have never encountered a patient who complained of breathlessness at induction.

The detailed monitoring used in the recent papers of Schwarz et al. and Mehta et al. was not available when Gray popularized his technique (which has appeared in standard British textbooks). While the rationale behind the use of relaxants in divided doses may be different in recent studies, nevertheless the use of the technique by earlier pioneers should be acknowledged.

REFERENCES