EDITORIALS

medicine) and just in case there had been a miracle, we discontinued the PEEP, the patient rapidly deteriorated and began to fight the respirator, became intensely cyanotic, hypotensive, and urine flow decreased. The same improvement was once again achieved with reinstition of PEEP.4,6

These bedside observations led us to believe that PEEP in the adult respiratory distress syndrome (ARDS)7 (in this case produced by acute hemorrhagic pancreatitis) was beneficial in improving systemic oxygen transport.

It has required considerable experience with hundreds of additional patients with the ARDS, an excellent experimental model of hemorrhagic pneumonitis developed by one of our associates, Ashbaugh and his colleagues,7 and the weight of additional reports from the literature8–10 to establish this technique in the adult respiratory distress syndrome.

Nonetheless, the “hard data” on reducing shunt and improving oxygen transport in the clinical setting of desperately sick patients in the intensive care unit have required careful documentation.

The article by Nicotra and associates (see page 10) in this issue of Chest clearly shows that intrapulmonary shunt can be reduced, arterial oxygenation improved and in selected patients tissue oxygenation enhanced as reflected by measurements of mixed venous oxygen tension and saturation. Also included in this article is the important point that PEEP does not help all forms of respiratory insufficiency. Although the requirements of a controlled randomized study with careful manipulation of a single variable have not been met in this clinical study, the data reported are at least convincing to the clinician that PEEP improves pulmonary function and enhances oxygen transport in patients with ARDS.

References


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Yes, Virginia, there is a Vagovagal Reflex!

To paraphrase a famous reply made by editorial writer Francis D. Church in the New York Sun 75 years ago to a little friend’s inquiry about Santa Claus—Yes, Virginia, there is a vagovagal reflex! The significance of the vagus nerve in the causation of cardiac arrest has been poorly understood. Suggested evidence of significant vagovagal effect surfaced as the cause of almost 25 percent of the first 1,200 cardiac arrest cases we reported from the Cardiac Arrest Registry 20 years ago.1 Even so, vagal nerve reflex action is often omitted in a discussion of the etiologic mechanisms of asystole or ventricular fibrillation.

Collating some of the varied clinical relationships may more nearly define and further emphasize the action of the vagi in the neuromuscular control of the heart. Significantly, it is increasingly apparent that preventive measures are currently available to avoid these untoward actions.

Obstructive jaundice associated with the clinical picture of vagotonia, including sinus bradycardia, is well known. In fact, obstructive jaundice was the most frequent clinical entity associated with operative cardiac arrest in our first 1,200 cases. For this reason, administration of atropine sulphate is especially indicated in patients operated on for obstructive jaundice.

Stokes-Adams syncope may be completely relieved after a diseased gallbladder has been removed. Stokes-Adams syncope unexpectedly “cured” by vagotomy for peptic ulcer disease is reported. In fact, intentional right vagotomy has eliminated episodes of Stokes-Adams disease.2

After the strain of the Valsalva maneuver, an “overshoot” in blood pressure is accompanied by marked vagal stimulation and slowing of the heart. Extraordinary expiratory effort during an asthmatic
attack may induce severe bradycardia and cardiac arrest, which is ascribed to excessive vagal tone.\textsuperscript{3} There is no paucity of reports of asystole after swallowing a large bolus of food. Manipulation of the vagus nerve during repair of tracheoesophageal fistulae may cause adhesions between the nerve and the esophagus. Stretching of the esophagus, with subsequent traction on the fifth vagus nerve, regularly caused disappearance of P-waves and a marked slowing of the QRS in one child four years after repair of the fistula.\textsuperscript{4} Atropine therapy and later vagotomy relieved this reflex action. Esophageal distention by carbon dioxide from carbonated beverages may result in bradycardia and atrioventricular dissociation by stimulation of the afferent limb of a vagovagal reflex.

Subarachnoid hemorrhage may produce S-T segment depression on the ECG when there is involvement of area 13 of the brain containing the chief cortical representation of the vagus nerve. Atropine, administered intravenously, may eliminate this change.\textsuperscript{5} Almost invariably, an adequate dose of atropine prevents the frequent arrhythmia and occasional asystole occurring under electroshock therapy.

Wolf\textsuperscript{6} has repeatedly called attention to a striking bradycardia during situations provoking extreme dejection or sudden fright. He compares the pathophysiologic influences produced to the profound bradycardia produced in man and animals by diving.

A disproportionately large number of cardiac arrests during eye operations, especially in children, was seen early in the study of the Registry cases. The afferent arc of the oculocardiac reflex is the trigeminal nerve. The efferent limb of the reflex arc consists of the vagus nerve. The oculocardiac reflex is seldom obtained after 40 years of age. As a matter of fact, if one excludes cardiac arrest in patients with coronary artery disease, cardiac arrest occurs most commonly in the first decade of life. Of the first 1,700 cases studied in our Registry, 24.3 percent were encountered in the first decade.\textsuperscript{7} Strictly speaking, the oculocardiac reflex is not vagovagal, since the afferent arc is a trigeminal nerve. Similarly, vagal inhibition or arrest which accompanies glossopharyngeal tic douloureux is initiated by ninth nerve stimulation. Both, however, can be relieved by atropine administration.

Reflex changes in the coronary circulation and cardiac tone which are mediated through the vagus nerves and suggesting viscerocardiac reflexes with vagal connection (and blocked by atropine) include bradycardia or asystole occasionally accompanying rectal and sigmoidoscopic examinations, prostatic manipulation, and with postmicturition syncope.

How much atropine should be given? When the atropine dosage is calculated on the basis of weight, children should receive a proportionately larger dose than adults. A representative adult dose is 0.6 or 0.7 mg intramuscularly. Three-fourths of this amount can safely be given as a rapid intravenously injected dose. For a newborn infant, 0.1 mg atropine sulphate can be given intramuscularly. A fully adequate dose of atropine is advisable, since some patients react to a small dose by a slower heart rate and an occasional atrial arrhythmia or a period of A-V dissociation. Atropine should be given to children prior to the use of succinylcholine, particularly when it is combined with halothane anesthesia and before certain procedures, such as orotracheal intubation, bronchoscopy and muscular correction of the eye. The chief of anesthesia\textsuperscript{a} at a large metropolitan otolaryngology institute tells me that at least 80 percent of patients anesthetized during adenoectomy incur arrhythmias unless they are given 0.01 mg/kg body weight of atropine.

The vagomimetic action of anoxia has more clearly come into focus with the realization that many acute myocardial infarctions are associated with marked vagotonia, manifested by nausea, vomiting, tenesmus and sinoatrial bradycardia. Partridge\textsuperscript{9} notes a 44 percent bradyarrhythmia incidence in 1,150 patients managed by a mobile coronary care unit. The danger of the prolonged cardiac cycle with its electrical instability and encouragement of ventricular ectopic beats, its decreased threshold for ventricular fibrillation and lowered blood pressure and cardiac output suggests the need for proper dosage of atropine, provided one recognizes that atropine may be harmful or contraindicated in an occasional instance.

In summary, there continues to be a long evolutionary period toward a clear delineation of the vagal reflex patterns. Admittedly, some of our clinical understanding of the vagovagal reflex is based on circumstantial evidence. Nevertheless, it behooves the physician to be familiar with the potential vagovagal actions on cardiac rhythm. The prevention of these deleterious reflexes during periods of disease or drug sensitization is possible and should receive high priority in the care of the patient.

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