Ways and Means of Conduction

Our traditional notions, that the normal sinus impulse spreads radially like an ever-widening ripple and that the A-V node is activated en bloc by the wave front that reaches it, have long held sway. In recent years, considerable electrophysiologic and some anatomic evidence has accumulated giving us cause to question tradition—a healthy process provided it does not spawn undisciplined rioting. Several independent observations have injected doubt and spoken for the presence of specialized conducting fibers within the atria and for multiple paths within the A-V junction:

1. In potassium intoxication "sino-ventricular" conduction is possible, the atrial muscle being electrically bypassed (i.e., sinus rhythm without P waves).
2. Differing forms of QRS complex can be produced by stimulating different areas of the A-V junction.
3. Changes in intraatrial conduction may be associated with alteration of the ventricular complex.
4. Abnormal A-V conduction can alter the amplitude of the ventricular complex.
5. In the W-P-W syndrome, the change to the pre-excitation ventricular complex sometimes results from change in the atrial pacemaker.

Such observations invite an attack on tradition and Sherf and James (page 127) accept the challenge and assail the status quo with a vengeance, a theory and no little ingenuity.

Bundles that Wenckebach, Thorel and Bachmann suspected or partially described half a century ago have been more clearly defined as three tracts connecting S-A to A-V node—anterior, middle and posterior internodal tracts. The anterior and middle tracts enter the crest of the A-V node, whereas the posterior tract bypasses the bulk of the node to enter its lower reaches. Sherf and James formulate the hypothesis that conduction from start to finish is a coordinated affair—each segment of conduction being intimately influenced by what went before. Conceivably, aberrant ventricular conduction could stem from abnormal conduction or impulse formation within sinus node, atrial myocardium, A-V junction or the bundle branches.

In approaching the WPW syndrome, the old tag "tot homines . . ." is apt; there is always room for another opinion and here is an elegant one. In previous theories of WPW mechanisms, several characteristics of the syndrome are usually unaccounted for: 1) increased supraventricular ectopic activity; 2) the tendency to change QRS pattern when the supraventricular pacemaker changes; and 3) the correlation between the length of the P-R interval in normal intraventricular conduction and during pre-excitation in the same patient. Sherf and James believe that all of the features of the WPW syndrome can be explained by their postulated "synchronized sino-ventricular conduction system," assuming that the impulse arises in the posterior internodal tract and so bypasses most of the A-V junction. This hypothesis, however, seems to leave the well known "concertina effect" unexplained, unless one assumes that sequential impulses arise successively lower and lower in the posterior internodal tract.

Extending their theory to embrace the acquired WPW syndrome, they reason that its pathogenesis may be blockade of the anterior and middle internodal tracts which forces the sinus impulse to use the posterior tract.

Their arguments are attractive and are based on considerable experimental evidence; they are also based on an equally impressive foundation of theory. Some of the clinical tracings presented in support of their hypotheses are not as convincing as one would like to see and the presence of the inter-
nodal tracts has not been universally accepted—voices of no inconceivable weight and volume have failed to hail their existence. For the moment, therefore, Sherf and James' hypotheses, though they compel us to reexamine our conventional concepts of conduction, must be regarded as tentative and sub judice; their acceptance must await the verdict of further ingenious anatomic and electrophysiologic study.

Henry J. L. Marriott, M.D.*

*From The Rogers Heart Foundation and St. Anthony's Hospital, St. Petersburg, Florida.

The Potential Hazards of Immoderate Hemodilution In Cardiac Surgery

"Allzu viel ist nicht gesund" (Too much is not healthy).
—Old German Proverb—

Homologous blood priming of heart-lung machines is a putative cause of many physiochemical derangements after extracorporeal circulation. The curtailment of these disorders in recent years may be credited to: a) refinements in pump-oxygenator apparatus; b) better artificial respirators; c) improved anesthetic and other pharmacologic agents; d) avoidance of "aged" bank blood; and, e) substitution of non-hemic media for part or all of the priming volume. The latter technique is aptly named "hemodilution," since, regardless of the composition of the prime, dilution of the patient's blood occurs during bypass.

Hemodilution is claimed to confer numerous benefits. First, whatever adverse mechanical changes may be incurred by blood traversing the extracorporeal circuit are presumably reduced in proportion to the degree of deliberate dilution. Second, some proponents of the method allege that it ameliorates peripheral tissue perfusion and metabolism (although absolute proof of this is lacking). Third, the incidental advantage of decreased demands on the blood bank is gratifying, especially in those institutions where much cardiac surgery is done.

Hemodilution cardiopulmonary bypass is well established nowadays in most medical centers. Originally, only a portion of the pump-oxygenator prime consisted of non-hemic liquids; but scattered reports of successful "bloodless" bypass sessions have stimulated the widespread use of completely blood-free primes. Furthermore, the contemporary resurgent popularity of massive infusions of salt solutions for treating major hemorrhage has encouraged the extension of hemodilution concepts to all phases of cardiac surgical care. The premise is proffered in various quarters that there is merit in withholding blood altogether because transfusions are occasionally attended by hepatitis, disease transmission, hypersensitization, and other reactions.

A few cardiac surgical procedures can doubtless be accomplished efficaciously without administering transfusions at all. However, the majority of operations cannot be done safely without blood. The perverse use under those circumstances of replacement fluids to achieve a "bloodless" surgical tour de force not only distorts hemodilution principles, but also needlessly exposes the patient to a constellation of latent dangers.

If the clinician is to apply hemodilution rationally, he must examine articles on the subject with a critical eye, and be conversant with the physiologic consequences to the organism of massively exchanging blood-free fluids for circulating blood. He will soon discover disparities and hiatuses in the available information. The facts are:—(A) More than 20 different hemodiluent primes (dubbed "white blood" or "colorless blood" in medicales) have been devised to date. Their constituents include: 0.9 per cent saline solution, 5 per cent dextrose in water, 5 per cent dextrose in 0.11 per cent saline solution, 5 per cent dextrose in 0.2 per cent saline solution, Ringer's injection, 5 per cent dextrose in Ringer's solution, lactated Ringer's solution (Hartmann's solution), 10 per cent low (40,000) molecular weight dextran in normal saline solution, 6 per cent high (75,000) molecular weight dextran in normal saline solution, 6 per cent hydroxyethyl starch in normal saline solution, gelatin degradation products or specially prepared poly electrolytic compounds. These are used either alone or in combination, and supplemented with 10 per cent serum albumin solution, mannitol solution, THAM, sodium bicarbonate, and myriad other chemical additives. . . . calcium, magnesium, phosphates, etc. (B) Sundry therapists select varying concentrations of their favorite nostrum for admixture with bank blood, without clearly specifying the clinical indications when particular ratios might prove especially beneficial. One group calculates hemodilution in terms of percentage of diluent in the total priming volume, another on the basis of the patient's predicted blood volume derived from body weight, a third according to the patient's isotopically measured blood volume, a fourth according to the patient's weight, and a fifth by the mixed hematocrit value. What is seldom mentioned is how much an-