Evidence Suggesting Dual A-V Nodal Pathways in Patients without Supraventricular Tachycardias*

Rafael Levites, M.D., and Jacob I. Haft, M.D., F.C.C.P.

Electrophysiologic evidence for dual pathways of conduction through the A-V node is presented in three patients without history of supraventricular tachycardia. In case 1, abrupt spontaneous changes in the PR interval from 0.17 to 0.42 second were seen. His bundle electrophysiologic studies showed two sets of A-H intervals during sinus rhythm and at several atrial pacing rates, although at rates over 100 per minute only the slow pathway conducted. Using the extrastimulus method, different refractory periods for the fast and slow pathways were documented. Cases 2 and 3 underwent His bundle electrophysiology studies to evaluate intraventricular conduction defects. During atrial pacing studies abrupt changes in the A-H interval, from 220 to 470 msec and from 220 to 370 msec, were observed on increasing the pacing rate from 90 to 95 per minute in case 2 and from 120 to 130 per minute in case 3. In these two patients, dual A-V nodal pathways were suggested by the sudden changes in the A-H interval at critical pacing rates. These findings indicate that evidence suggesting dual pathways of conduction through the A-V node may not be an uncommon finding and may be present without the manifestation of recurrent supraventricular tachycardias.

Studies in patients with paroxysmal supraventricular tachycardia have suggested that reentry at the A-V node is responsible for the genesis and maintenance of the arrhythmia in many instances. Two hypotheses have been advanced to explain the mechanism: (1) the presence of dual pathways of A-V nodal conduction and (2) "reflection" from an area of depressed conduction. Recently the presence of dual A-V nodal pathways has been demonstrated by Denes and co-workers in two patients with supraventricular tachycardias. During electrophysiologic studies they found two different refractory periods within the A-V node and evidence for an echo zone in the cardiac cycle.

Similar evidence for dual pathways within the A-V node is presented in three patients who had never experienced tachycardias. This finding suggests that the anatomic or physiologic basis (or both) for reentry in many patients exists even in the absence of overt arrhythmias. One patient manifested two different A-V nodal conduction velocities at each of several rates, during sinus rhythm and while undergoing atrial pacing. Two additional patients showed abrupt large changes in A-V nodal conduction time as the atrial rate was changed only minimally.

**METHODS**

His bundle electrograms were performed by using a modified version of the method described previously by Damato and colleagues. Frequencies below 40 and above 500 Hz were filtered out. All tracings were displayed on the oscilloscope of an Electronics for Medicine Recorder and recorded at paper speeds of 100 mm per second.

All patients were atrially paced to accelerate their rate and records were taken at various rates. Premature atrial beats were delivered by coupling the pacing spike to the patient's sinus P waves. The labels A1 and H1 were used for the atrial and His bundle deflections during the sinus cycle. A2 and H2 were the atrial and His bundle deflections resulting from premature stimuli. A-H, H-V and H1-H2 were measured as previously described by Wit and co-workers. The A-V nodal effective refractory period (ERP) was defined as the longest A1-A2 not propagated to the His bundle. The A-V nodal functional refractory period (FRP) was defined as the shortest H1-H2 interval that was seen during premature stimulation.

**CASE REPORTS**

**CASE 1**

A 63-year-old man was admitted to the hospital with complaints of easy fatigability. He had never experienced episodes of palpitations, dizziness or syncope and was not receiving any medications. During recording of his ECG, his PR interval was noted to intermittently change abruptly from 0.17 to 0.42 second (Fig 1) and was otherwise normal. His bundle studies were performed to further evaluate his A-V conduction.

**Electrophysiologic Studies:** During resting sinus rhythm the rate was 66 beats per minute and A-H intervals were intermittently 130 to 150 and 360 msec, H-V was 40 msec (Fig 2). Prolongation of the A-H interval, a normal response to atrial pacing, was seen to occur in two patterns and suggested two conduction pathways at the A-V node (Fig 2). At 82 beats per minute the A-H interval was either 160 msec or 400 msec. At 90 beats per minute the A-H interval was either 180 msec or 410 msec; the A-H interval changed...
Both patients were studied because of abnormal QRS patterns, left bundle branch block in case 2, and right bundle branch block and left axis deviation in case 1. During His bundle electrographic studies, both patients demonstrated the abrupt changes in PR intervals, left bundle branch block in case 2, and right bundle branch block and left axis deviation in case 1.

These findings suggest that there was a dependence of conduction to the His-Purkinje system on the atrial cycle length. At a resting sinus rate of 600 beats per minute, the A-H interval remained constant at each of the coupling intervals. A-H intervals of 220 to 110 msec as the pacing rate was increased from 500 to 120 beats per minute resulted in longed atrial rates that remained constant at each of the coupling intervals. The A-H intervals remained constant and were plotted against pacing rates to demonstrate the abrupt changes in PR intervals. An evidence suggesting dual A-V nodal pathways was observed.

In these patients, dual A-V nodal pathways of A-V conduction were demonstrated. Both patients were observed during atrial pacing or premature atrial stimulation or both. The atrial rate was increased from 500 to 120 beats per minute. At a resting sinus rate of 600 beats per minute, the A-H interval remained constant at each of the coupling intervals. The A-H intervals remained constant and were plotted against pacing rates to demonstrate the abrupt changes in PR intervals. An evidence suggesting dual A-V nodal pathways was observed. Dual A-V nodal pathways of A-V conduction were also demonstrated. In case 1, atrial pacing at rates of 130, 180, and 220 beats per minute resulted in longed atrial rates that remained constant at each of the coupling intervals. The A-H intervals remained constant and were plotted against pacing rates to demonstrate the abrupt changes in PR intervals. An evidence suggesting dual A-V nodal pathways was observed. Dual A-V nodal pathways of A-V conduction were also demonstrated.
FIGURE 3, Case 1. Effect of premature atrial stimulation (A1-A2) on the A-V conduction of the premature beat. A. An A1-A2 of 650 msec resulted in A2-H2's of 170 or 400 msec. B. At an A1-A2 of 600 msec, A2-H2's of 180 or 400 msec were seen. In the lower panel, also note the rapid return of A-H to the control value. C. Left upper panel. At an A1-A2 of 500 msec a constant A2-H2 of 420 msec was obtained. Lower panel. At an A1-A2 of 400 msec the A2-H2 was constant at 570 msec. Note also that after delivery of a second premature beat with an A1-A2 of 600 msec, the A-H interval promptly returned to the control value. Right upper panel. At an A1-A2 of 390 msec the refractory period of the A-V node (slow pathway) was reached; conduction through the A-V node did not occur.
EVIDENCE SUGGESTING DUAL A-V NODAL PATHWAYS

The type B response was similar to type A, but shortly after the plateau (where further decrease in A1-A2 had no further effect on V1-V2), A-V conduction time was suddenly increased such that the V1-V2 interval at shorter A1-A2 interval was longer and remained at this new prolonged value until the ERP was reached. Type C response showed a gradual decrease in V1-V2 as A1-A2 was decreased until a certain point, the minimum V1-V2 (the FRP), after which further decrease in A1-A2 led to a gradual increase of V1-V2 until the ERP was reached.

Moe and co-workers interpreted these findings to suggest that the type A curve did not deviate from the behavior expected in a simple homogeneous conducting system... between atria and ventricles and hence represented A-V conduction through a single pathway. The type B curve, Moe suggested, "can be... explained by the assumption of two parallel systems, the slower of which appears only when the faster is functionally inoperative," that is, conduction down a "fast" pathway proceeded until its refractory period was reached at which time the abrupt increase in A-V conduction time occurred, signifying that conduction was now going via a second parallel "slow" pathway. The type C curve was interpreted by Moe as "compatible with a synaptic mechanism," and suggesting "several parallel pathways with varying conduction velocities and refractory periods, or two basic systems communicating with each other through branches of different lengths..." Hence he considered this curve (C) to be due to either multiple parallel pathways with shifting conduction from the predominant "faster" pathway to different "slower" pathways or two pathways similar to type B but with various areas of branching.

Rosenbleuth, from data on echo beats, similarly postulated two parallel pathways for A-V conduction.

In 1963 Hoffman and colleagues performed studies similar to those of Moe and co-workers but in addition to recording atrial and ventricular depolarization, these investigators recorded the depolarization of the His bundle and of the distal Purkinje system. They found that type A and C response curves were indeed usually due to delay in conduction at the A-V node, but that type B curves, with the sudden increase in V1-V2 (A-V conduction time) to a new stable level that was the basis for postulation of dual pathways by Moe and co-workers, were due to sudden increase in delay in conduction from the His bundle to the ventricular muscle rather than delay at the A-V node. They also noted that in some instances type A curves were due in part to distal His-Purkinje delay. They concluded that the existence of two or more parallel pathways through the A-V node was not necessary to explain their observations and those of Moe and co-workers.

Wit and associates in 1970, after the development of techniques for recording the His bundle electrogram, performed experiments in humans similar to those of Hoffman and colleagues. Their results concurred with those of Hoffman and colleagues in that type C curves of Moe and coworkers were due to delay mainly at the A-V node; type A curves were due to delay at both the A-V node and in the His-ventricular conduction system; and type B curves were due to gradual delay at the A-V node and abrupt blockage in the conduction system distal to the His bundle deflection. They also concluded that dual A-V nodal transmission was not necessary to explain the observed A-V conduction phenomena.

Although the existence of dual pathways through the A-V node has come into question, a number of investigators have documented that it is reentry through the A-V node that plays an important role in many patients with recurrent supraventricular tachycardias. Goldreyer and Damato have shown that delay at the A-V node is necessary for the
induction of paroxysmal atrial tachycardia. They and others before have implied that if there is sufficient slowing of the conduction through the A-V node, an impulse from the atrium may be so delayed that it can reenter the atrium and result in either an echo beat, or if this occurs repeatedly, a supraventricular tachycardia. They have not felt it necessary to postulate dual pathways through the A-V node, though they do not specifically rule out the possibility.

Direct evidence for the existence of dual A-V nodal conduction pathways in humans has recently been reported by Rosen and co-workers. Using His bundle electrography they studied a patient with PR intervals that changed spontaneously during sinus rhythm from 0.18 to 0.42 second and found the A-H interval to change abruptly from 100 to 290 msec on atrial pacing at 90 per minute. Using the extrastimulus method they found two ERPs through the A-V node, suggesting a slow and a fast
pathway. In two additional patients with history of supraventricular tachycardias they noted similar findings with a sudden increase in the fast pathway and engagement of the slow pathway.

The results of the electrophysiologic studies in the patients reported here confirm that phenomena suggestive of dual pathways through the A-V node occur. In case 1 abrupt spontaneous changes between one pathway (the fast pathway) and the other (the slow pathway) were documented to occur during sinus rhythm and at two paced rates. The ERP of the fast pathway was roughly defined and shown to be different from that of the slow pathway. During premature atrial stimulation, conduction via both the slow and the fast pathways was demonstrated at a number of the same A1-A2 intervals.

In cases 2 and 3 the evidence for two A-V nodal pathways was demonstrated by the abrupt increase in A-H conduction time with a small increase in atrial pacing rate. The usual response to atrial pacing is a gradual increase in the A-H interval without a sudden change as noted by Caracta and coworkers and in our laboratory among 110 patients (unpublished observations). This sudden shift in A-H with a gradual progression of A-H increase before and after the shift strongly suggests two A-V nodal pathways with one taking over after the other fatigue.

Although our findings and those of others are strongly suggestive of dual pathways through the A-V node, the possibility that the abrupt changes in the A-V nodal conduction time seen in our patients might be due to concealed reentry cannot be ruled out in the clinical setting. To accomplish definitive proof of more than one pathway through the A-V node would require the positioning of multiple electrodes directly in the A-V node.

Of interest in our three patients is the finding that although two pathways of A-V nodal conduction were seen, during none of the studies were echo beats or supraventricular tachycardias induced, and in none of the patients was there a history suggestive of tachycardia. These findings suggest that dual pathways may not be an uncommon occurrence, and like Wolff-Parkinson-White or Lown-Ganong-Levine, conduction may be present without the manifestation of recurrent tachycardias.15,24,25

As measurement of the A-V refractory periods using the extrastimulus method becomes a more frequently performed procedure, the incidence of dual A-V nodal pathways may prove to be relatively common.

REFERENCES


CHST, 67: 1, JANUARY, 1975

EVIDENCE SUGGESTING DUAL A-V NODAL PATHWAYS 41
Hypertrophic Osteoarthropathy

Bamberger-Marie disease was observed first over eight decades ago (Wien Klin Wochenschr 2:226, 1889; Rev De Med 10:1, 1890). Since then reports concerning its pathology and pathogenesis have brought forth elucidating observations and theories. Some of the pertinent findings were aptly summarized by Holling, H E et al (JAMA 78:977, 1961). "The primary change in the limbs of patients is an overgrowth of vascular connective tissue investing the structures in the distal parts of the limbs. The newly formed tissue lies over the periosteum and new bone formation takes place under it. Surrounding the joints the newly formed tissue gives the appearance of periartthritis though there are no specific articular changes. Periarthritis rather than arthritis was present in 6 of our 7 patients." The shell-like periosteal new bone formation and widening of the bony cortex are demonstrable radiologically but x-ray reveals no involvement of joint surfaces or joints. It has been shown that increased blood flow in the distal areas of the extremities can be demonstrated by plethysmography. In typical instances there is nonpitting cylindrical swelling of the upper and lower limbs. Limitation in articular motion is due to tenderness. Periosteal proliferation and new bone formation may involve the metacarpals and the proximal intermediate phalanges. The subcutaneous tissues are thickened. The overlying skin is likely to be dusky and warm. Tenderness along the involved areas is attributed to periosteal proliferation. These patients are bound to complain of mild to severe aching pain in the respective bones and joints, including wrists, ankles and knees even though no arthritis is present. Holling, H E et al (JAMA 178:977, 1961) thought that occasional articular effusion resulted from increased vascularity and not from inflammation. This view is supported by the findings of McLaughlin, G E et al (Ann Int Med 67:579, 1967) and of Ropes, M W (Synovial Joint Changes in Joint Disease, Cambridge, Harvard U Press, 1953). There may be thickening and deeply-furrowed coarse appearance of the facial skin (hence the terms pachyderma with pachydermoperiostosis, and pachydermoperiostosis) and also, oiliness of the skin and hyperhidrosis. Gynecomastia may be noted in some instances. Rabson, A S et al (J Nat Ca Inst 50:669, 1973) demonstrated the production of human gonadotropin from a cell culture derived from lung cancer. Prior to this, Ginsburg, J et al (Lancet 2: 1274, 1961) recorded increased urinary estrogen levels in patients with hypertrophic osteoarthropathy due to bronchogenic carcinoma. The latter and other thoracic neoplasms are responsible for the overwhelming majority of hypertrophic osteoarthropathy. Other possible causes include carcinoma of the stomach, and nasopharynx, lung abscess, cyanotic congenital heart disease, patent ductus arteriosus with reversal of blood flow, lymphangio-epithelioma of the nasopharynx. Borden, E C et al (Ann Int Med 71:577, 1969) reported two cases in pregnancy. Hypertrophic osteoarthropathy is more likely to be associated with peripheral than central lung lesions. Familial osteoarthropathy was recorded first by Friedreich, N (Arch Path Anat Klin Med 43:83, 1866). Its association with pronounced thickening of the skin is known as Tournaine-Solente-Golé syndrome (Presse Med 2:1820, 1935). In most cases of hypertrophic osteoarthropathy one finds clubbing of the digits. The latter may precede the former in some instances. In most cases removal of the causal factor is followed by prompt disappearance of swelling of involved areas, cessation of pain, and improved joint function. Beneficial sequel may be noted within 24 hours but flare-up may take place with recurrence of the thoracic lesion. Section of the vagus nerve results in the same benefits, without resection of the respective neoplasm. This observation supports the concept of neural reflex influences which elicit physiopathologic reactions leading to hypertrophic osteoarthropathy. In some instances identical retrogressive changes follow simple thoracotomy without removal of the thoracic tumor and without sectioning the vagus nerve. This seemingly puzzling response is attributable partly to a reflex vagal response in the lung to the incision of the chest wall: Puder’s "dermatopulmonary reaction."

Andrew L. Banyai, M.D.

18 Drury AN: Paroxysmal tachycardia of A-V nodal origin, exhibiting retrograde heart-block and reciprocal rhythm. Heart 11:405-408, 1924
19 Scherf D: An experimental study of reciprocating rhythm.

Arch Intern Med 67:372-382, 1941

42 LEVITES, HAFT

CHEST, 67: 1, JANUARY, 1975