arteriovenous fistulas are so rarely encountered. In our patient, marked deformity of the thorax accompanying her severe scoliosis may have altered anatomic relationships, displacing these vessels laterally and thus favoring passage of the puncture needle through the internal mammary artery before its entry into the innominate vein. Review of her outside medical record, however, disclosed no mention of difficulty encountered during insertion of the CVP catheter.

Since Gianturco coil spring occluders are relatively easy to place and highly effective in occluding vessels of medium to large size, they were selected to close this arteriovenous fistula. Small particulate material such as Gelfoam or Ivalon (polyvinyl alcohol) or injectable liquid polymers (bucrylate) would pass through the fistula with resultant pulmonary embolization and, therefore, were not suitable in this situation. Detachable balloons delivered through a percutaneously inserted catheter would be equally effective as coil spring occluders in closing fistulas of this size; however, because of a significant cost differential, we chose the latter.

Initially, in the early 70's, percutaneous transcatheter vasoocclusive techniques were reserved for those patients considered too ill for conventional surgical therapy, and were used mainly in emergencies. Recently, however, in selected cases, percutaneous vasoocclusion has also been used on an elective basis as an alternative to surgery. In this age of cost-consciousness, use of interventional angiographic vasoocclusion, where feasible, offers advantages over traditional surgical therapy through significant reductions in cost and hospitalization. Furthermore, a percutaneous procedure performed on a conscious patient as opposed to major surgery requiring general anesthesia has obvious benefits.

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Panconductional Defect in Mixed Connective Tissue Disease*

Association with Sjogren's Syndrome

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A patient in whom mixed connective tissue disease in association of Sjogren's syndrome had previously been diagnosed, experienced a syncopal attack. Electrocardiographic monitoring revealed periods of profound sinus bradycardia, sinus arrest with slow junctional escape rhythm, and first degree atrioventricular block during several episodes of dizziness. Complete right bundle branch block was a constant finding in this patient. Sinoatrial conduction time and sinus node recovery time were prolonged. Coronary heart disease was excluded by normal coronary arteriographic findings. This patient represents a rare case of cardiac involvement in mixed connective tissue disease.

Mixed connective tissue disease is a distinct rheumatic syndrome with overlapping features of scleroderma, systemic lupus erythematosus, and polymyositis. Association of Sjogren's syndrome and mixed connective tissue disease is possible, though not frequently encountered. Cardiac manifestations in mixed connective tissue disease are rare. Recently, a case of complete atrioventricular block in this disease was described. This report presents multiple cardiac conductional defects in a patient with mixed connective tissue disease in association with Sjogren's syndrome.

CASE REPORT

A 60-year-old woman had a syncopal episode on the day of admission. She had a history of frequent dizzy spells and increasing dyspnea on exertion. At the age of 50 years she had developed pain involving the wrists and hips. Since then she was repeatedly hospitalized at the rheumatology department, where mixed connective tissue disease was diagnosed on the basis of high titer of antinuclear antibody (usually higher than 1:320), polyarthralgias and polyarthritis of RA type, Raynaud's, hypergammaglobulinemia, positive rheumatoid factor, esophageal hypomotility and sclerodactyly. The patient had also pulmonary fibrosis and facial erythema with teleangiectasia. In the course of the disease, several times we found positive extractable nuclear antigen, detected by the immunofluorescence method. The diagnosis was confirmed by positive anti-RNP antibodies (the double-diffusion technique with nuclear extract of calf thymus). The association with Sjogren's syndrome was confirmed by scintigram and histologic examination of a salivary gland.

On physical examination, loss of facial expression and sclerodactyly was noted. She had slight dyspnea and a regular pulse rate of 88/min. Blood pressure was 135/80

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mm Hg. Examination of the lungs was normal. The apex impulse was in the sixth left interspace just outside the left midclavicular line. The first heart sound was of diminished intensity and the second heart sound was widely split. The chest x-ray film showed linear densities and mottling in the basal parts of the lungs. The cardiac silhouette was moderately enlarged. The blood gas analysis revealed hypoxemia (Po2 46 mm Hg) and hypocapnia (Pco2 22.5 mm Hg).

During hospitalization, electrocardiograms demonstrated different rhythm and conduction disturbances (Fig 1). Usually the patient had sinus rhythm and always complete right bundle branch block. Episodes of first degree atrioventricular block were observed at several instances. During episodes of sinus arrests a slow escape rhythm (21 beats/min) was recorded. The form of the QRS complexes was the same as during the sinus rhythm. Occasionally sinus bradycardia with junctional escape rhythm appeared, producing atrioventricular dissociation. Sometimes ventricular premature beats in the form of bigeminy were noted.

The patient underwent cardiac catheterization and electrophysiologic study after she had given informed consent. Pulmonary arterial pressure was increased to 75/20 mm Hg (mean 38 mm Hg), whereas pulmonary capillary wedge pressure and left ventricular end-diastolic pressure were normal. Left ventricular angiography showed a normal left ventricular cavity and normal ejection fraction. Coronary arteriography revealed a normal coronary artery tree (including sinus node artery and atrioventricular node artery). Both sinoatrial conduction time and corrected sinus node recovery time were prolonged; the former was 320 msec (bidirectional; the upper limit of normal is considered to be 215 msec) and the latter 1630 msec (re-

Figure 1. Electrocardiographic tracings recorded at different times during the hospitalization of the patient (monitor leads at 25 mm/sec). A: sinus rhythm with normal P-R interval. B: first degree atrioventricular block with P-R interval of 0.88 sec. C: marked sinus bradycardia and pause with atrioventricular dissociation and junctional escape beats. D: sinus rhythm 60 beats/min with sinus pauses. Terminated by junctional escape beats. E: junctional escape rhythm 34 beats/min because of sinus bradycardia 27 beats/min, resulting in atrioventricular dissociation with occasional antegrade sinus capture. F: escape-capture bigeminy. The fourth P wave is not conducted to the ventricles because of its prematurity (short R-P interval).

Figure 2. Atrial pacing 105 beats/min with 2:1 atrioventricular block. After cessation of pacing, an asystolic pause of 2637 msec is seen. The P-R interval of the first spontaneous beat is 0.28 sec. (Upper tracing: 1 sec marks. Lower tracings: standard electrocardiographic leads 1, 3)

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ported upper limit of normal values is 525 msec\(^{-1}\). Second degree atrioventricular block appeared at atrial stimulation rate of 105 beats/min (Fig 2).

After some weeks the rhythm and conduction disturbances spontaneously disappeared, only complete right bundle branch block persisted.

**DISCUSSION**

Conductional disturbances in progressive systemic sclerosis\(^6\) and systemic lupus erythematosus\(^7\) are well known. They are due to both microvascular and primary collagen abnormalities.\(^5\)\(^-\)\(^7\) In patients with mixed connective tissue disease, serious systemic complications are rare. Until now, only one case of complete heart block in this disease has been reported.\(^8\) A case of congenital complete atrioventricular block associated with maternal mixed connective tissue disease was described recently.\(^9\)

In our patient, multiple conductional defects were noted. The sinus bradycardia, sinus pauses and sinus arrests with junctional escape rhythm are characteristic of the sick sinus syndrome.\(^*\) The sinus node dysfunction was also confirmed by electrophysiologic tests.

The most frequent etiologic factors of the sick sinus syndrome are coronary atherosclerosis, infiltrative disorders, idiopathic fibrosis, collagen-vascular diseases, infectious processes, and pericardial disease.\(^9\)\(^,\)\(^10\) Since coronary heart disease was excluded, the sick sinus syndrome in our patient seems to be a cardiac manifestation of the underlying disease (ie, mixed connective tissue disease). Naturally the idiopathic form of the sinus node disease cannot be ruled out with absolute certainty. In our patient, the rhythm and conduction disturbances were intermittent and at least in part related to clinical changes of the underlying disease; they were most prominent during the exacerbations of other symptoms and signs of the mixed connective tissue disease. Therefore, it is most likely that conduction disturbances in this patient were due to the systemic collagen disease, though clinical expressions of the sick sinus syndrome are frequently periodic regardless of its etiology.\(^9\)

Atrioventricular conduction defects are often associated with the sick sinus syndrome,\(^11\)\(^,\)\(^12\) especially if the etiologic factor can produce both dysfunctions, as collagen diseases do. The right bundle branch block in our patient could be a manifestation of the same disease; the term panconductional defect (introduced by Rasmussen\(^13\)\) is often used to denote such multiple conductional disorders involving the sinus node and perinodal zone, as well as the atrioventricular node and His-Purkinje system. In our case, the right bundle branch block could be related to the pulmonary hypertension with secondary right ventricular stretch, too.

Our experience with this patient suggests that in all collagen diseases, multiple conductional defects in the heart are possible and that mixed connective tissue disease forms no exception to this rule.

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**References**


**Cardiac Surgery for Total Rupture of the Anterolateral Papillary Muscle**

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A 62-year-old man had acute myocardial infarction complicated by total rupture of the anterolateral papillary muscle causing cardiogenic shock and pulmonary edema. Prompt hemodynamic support, medical evaluation and mitral valve replacement resulted in marked improvement and later uneventful recovery.

**Total rupture of a papillary muscle after a myocardial infarction is felt to be incompatible with life. Re-**

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