There has been recent clinical interest in this entity because of possible associations with supraventricular arrhythmias, sudden death and right-sided venous obstruction.

Lipomatous atrial septal hypertrophy is characterized by deposition of excessive adipose tissue in the atrial septum; the result is a globular thickening of the septum. Mature adipose cells mixed with granular or vacuolated fetal fat cells—the hallmark of lipomatous hypertrophy—infiltrate the myocardial fibers. In a series of 32 autopsy specimens reported by McAllister and Fenoglio,[3] the maximal diameter ranged from 1 to 8 cm.

Two-dimensional echocardiography is the technique of choice for the diagnosis of this entity. The subcostal transducer position is considered the best. A bilobed atrial septum with a thickness of at least 15 mm is highly characteristic of lipomatous hypertrophy. In our previous report of 17 patients with this entity, the average thickness of the atrial septum was 21 mm (range 15 to 29).[1]

Because cardiac imaging with magnetic resonance is still in its infancy, few cases of atrial septal hypertrophy have been reported.[4] The intensity of cardiac tissue in magnetic resonance images depends on spin density, relaxation times (T1 and T2) and cardiac motion. The cardiac chambers are clearly differentiated from the myocardium because of the low intensity of flowing blood. This capability, as well as high intrinsic contrast between different soft tissues, allows accurate demonstration of the size and location of cardiac masses.[5,6] Some degree of tissue characterization is possible on the basis of relative intensity alone. Fatty tissue is particularly different from other soft tissues because of its relaxation times and spin density. The T1 relaxation of fat is much shorter than that of other tissue, and the result is the high intensity in partial saturation spin-echo images. In these 2 patients, the magnetic resonance images substantiated the echocardiographic diagnosis of lipomatous hypertrophy of the atrial septum. Because the images were consistent with short spin-lattice relaxation times in the atrial mass, the occurrence of a malignant process was much less likely, since malignancy generally prolongs relaxation times.

1. Fyke FE III, Tajik AJ, Edwards WD, Seward JB. *Diagnosis of lipomatous hypertrophy of the atrial septum by two-dimensional echocardiography. JACC 1983;1:1352-1357.*
2. Prior JT. *Lipomatous hypertrophy of cardiac inter-atrial septum: a lesion resembling hibernoma, lipo-blastomatosis and infiltrating lipoma. Arch Pathol 1964;78:11-15.*
3. McAllister HA Jr, Fenoglio JJ Jr. *Tumors of the cardiovascular system. In: Atlas of Tumor Pathology, Fascicle 15. Washington DC: Armed Forces Institute of Pathology, 1978:40-46.*
4. Levine RA, Weyman AE, Dinsmore RE, Southern J, Rosen BR, Guyer DE, Brady TJ, Okada RD. *Noninvasive tissue characterization: diagnosis of lipomatous hypertrophy of the atrial septum by nuclear magnetic resonance imaging. JACC 1986;7:688-692.*
5. Amparo EG, Higgins CB, Farmer D, Gamsu G, McNamara M. *Gated MRI of cardiac and paracardiac masses: initial experience. AJR 1984;143:1151-1156.*
6. Go RT, O'Donnell JK, Underwood DA, Feiglin DH, Salcedo EE, Pantoja M, MacIntyre WJ, Meaney TF. *Comparison of gated cardiac MRI and 2D echocardiography of intracardiac neoplasms. AJR 1985;145:21-25.*

Paradoxical Response of Plasma Atrial Natriuretic Hormone to Pericardiocentesis in Cardiac Tamponade

PATRICK T. KOLLER, MD
ROGER J. GREKIN, MD
JOHN M. NICKLAS, MD

Vasoactive peptides, termed atrial natriuretic hormone (ANH), have been isolated from animal and human atrial tissue.[1] Serum levels of immunoreactive ANH have been reported to be elevated in pathophysiologic conditions characterized by elevation of atrial pressure.[2-5] Investigations have revealed a direct linear correlation between ANH concentration and the magnitude of atrial pressure in normal persons and in patients with heart failure.[2,3] However, the mechanism by which atrial pressure mediates ANH secretion is unknown. We describe a case of cardiac tamponade in which hemodynamic recordings and ANH measurements were obtained during therapeutic pericardiocentesis. From our observations we suggest that atrial distention produced by changes in the pressure gradient across the atrial wall is the major determinant of ANH release.

A 42-year-old man presented with the sudden onset of dull substernal chest pain. An electrocardiogram revealed an inferior wall acute myocardial infarction. Creatinine kinase levels increased to 4,800 IU/liter with positive MB bands. On the third hospital day the patient had new, sharp, substernal chest pain that increased with inspiration and recumbency, and a new pericardial friction rub was heard on auscultation. The paradoxical pulse pressure was 14 mm Hg. The electrocardiogram did not reveal new ST-segment changes. The patient's symptoms subsided and he was discharged on the 10th hospital day.

The patient returned to the emergency room 3 days later with increased sharp substernal chest pain. The paradoxical pulse pressure had increased to 25 mm Hg. Heart sounds were normal except for an S4 gallop and the friction rub was no longer audible. Jugular venous distention was observed. The chest x-ray showed an enlarged cardiac silhouette relative to previous studies, but the electrocardiogram was unchanged. An echocardiogram showed a large circum-

From the Department of Internal Medicine, University of Michigan Medical Center, Ann Arbor, Michigan 48109-0366. This study was supported by Grant HL 18575 from the National Heart, Lung, and Blood Institute, Bethesda, Maryland, and the Research Service of the Veterans Administration, Washington, D.C. Manuscript received August 18, 1986; revised manuscript received September 12, 1986, accepted September 15, 1986.

TABLE I Hemodynamic and Atrial Natriuretic Hormone Measurements Before and After Pericardiocentesis

	Before	After	
		Immediate	1 Hour
Heart rate (beats/min)	110	109	110
Pressures (mm Hg)			
Systemic artery (s/d)	140/100	152/102	160/100
Right atrial mean	25	19	16
Pulmonary artery (s/d)	44/29	43/25	43/25
Pulmonary artery wedge mean	24	15	17
Intrapericardial pressure	23	19	
ANH (pmol/liter)	13,9	112	68

ANH = atrial natriuretic hormone; s/d = peak systole/end diastole.

ferential pericardial effusion. A pulmonary artery (Swan-Ganz) catheter was placed and revealed elevation and equalization of right atrial, right ventricular end-diastolic and pulmonary artery occlusion wedge pressures (Table I). The right atrial pressure tracing revealed absence of a significant y descent. Blood samples were taken from the right atrial lumen for ANH determinations. ANH concentrations drawn 30 minutes and immediately before pericardiocentesis were 13 and 9 pmol/liter, respectively (normal: mean 18 ± 2 pmol/liter; range 3 to 30; n = 31). Bedside pericardiocentesis was then performed and 540 ml of noncoagulable hemorrhagic fluid was withdrawn. The paradoxical pulse pressure after pericardiocentesis decreased to 14 mm Hg, with similar decreases in right atrial and pulmonary capillary wedge pressures. The right atrial pressure tracing showed appearance of a significant y descent. A subsequent echocardiogram showed a reduced but persistent pericardial effusion. Left atrial dimension increased from 43 mm to 47 mm. Plasma ANH increased to 112 pmol/liter immediately after pericardiocentesis and remained elevated 1 hour afterward, at 68 pmol/liter.

This report describes a case of pericardial tamponade in which paradoxically reduced plasma ANH levels were found despite elevated right atrial and pulmonary capillary wedge pressures. During pericardiocentesis, reduction of these pressures was associated with a significant elevation in ANH concentration. These observations are in contrast to studies showing a direct correlation between plasma ANH concentration and atrial pressure. Clinical conditions characterized by increased atrial pressure, such as congestive heart failure and chronic renal failure with fluid overload, are associated with elevated levels of ANH.[2-5] Rapid infusions of saline solution have produced increases in ANH levels corresponding to atrial pressure elevations.[6] Hemodynamic measurements in normal persons and patients with congestive heart failure have revealed a linear relation between ANH concentration and atrial pressure.[2-4]

The common factor apparently underlying the changes in ANH seen both after pericardiocentesis for cardiac tamponade and during acute or chronic extracellular fluid overload states is atrial distention. The decompression and reexpansion of the atrium during pericardiocentesis may provide the stimulus for ANH release, as suggested by the present case. In saline solution infusion and chronic congestive heart failure, the increase in extracellular fluid volume produces an increase in atrial pressure, leading to atrial dilation. Chronic mitral stenosis and cor pulmonale, conditions marked by enlargement of the left and right atria, respectively, are associated with significant ANH elevations.[3] ANH is also elevated during atrioventricular nodal tachycardia, in which near-simultaneous atrial and ventricular contractions result in elevated atrial pressure as the atrium contracts against a closed atrioventricular valve.[2] Thus, ANH secretion is apparently associated with atrial distention, either from dilation due to elevated intraatrial pressure or from pericardial decompression with an increase in the pressure gradient across the atrial wall. What role atrial stretch receptors have in this response may be a useful area for further investigation.

1. Atlas SA, Kleinert HD, Camargo MJ, et al. *Purification, sequencing and synthesis of natriuretic and vasoactive rate atrial peptides. Nature 1984; 309:717-719.*

2. Nicklas JM, DiCarlo LA, Koller PT, Diltz EA, Shenker Y, Grekin RJ. *Plasma levels of immunoreactive atrial natrouretic factor increase during supraventricular tachycardia. Am Heart J, in press.*

3. Bates ER, Shenker Y, Grekin RJ. *The relationship between plasma levels of immunoreactive atrial natriuretic and hemodynamic function in man. Circulation 1986;73:1155-1161.*

4. Raine AEG, Phil D, Erne P, Burgisser E, Muller FB, Bolli P, Burkart F, Buhler FR. *Atrial natriuretic peptide and atrial pressure in patients with congestive heart failure. N Engl J Med 1986;315:533-537.*

5. Shenker Y, Sider RS, Ostafin EA, Grekin RJ. *Plasma levels of immunoreactive atrial natriuretic factor in healthy subjects and in patients with edema. J Clin Invest 1985;76:1684-1687.*

6. Yamaji T, Ishibashi M, Takaku F. *Atrial natriuretic factor in human blood. J Clin Invest 1985;76:1705-1709.*