



DEPARTMENT OF DEFENSE
ARMED FORCES INSTITUTE OF PATHOLOGY
WASHINGTON, DC 20306-6000

REPLY TO
ATTENTION OF

PATIENT IDENTIFICATION

<u>AFIP ACCESSION NO.</u>	<u>SEQUENCE NO.</u>
2753335	00
ADANALIAR, Linda	SSN:
YR00-45	AB/apb
November 1, 2000	

Venu Gopal, M.D.
Senior Forensic Pathologist
760 West Nielsen Avenue
Fresno, CA 93706

AFIP REPORT:

History: 37 year old woman with chest pain and near syncope, transported to emergency room, died within 1 hour

Heart (gross description per protocol): 280 grams; normal epicardial fat; right ventricle 5 cm in diameter, with dilatation; submitted histologic sections:

Right ventricle (8 sections mounted on 3 slides [in duplicate], slides labeled 5 RVA, 6 RVI, 7 RVA): Physiologic fat infiltration, predominantly in anterior wall, no changes of dysplasia or myocarditis noted

Sinoatrial nodal area (5 sections mounted on 1 slide [in duplicate], slide labeled 9 SN): Focal interstitial hemorrhage, normal artery and node, without dysplasia or inflammation

Atrioventricular nodal area (5 sections mounted on 2 slides [AVN1 in duplicate, AVN2 in triplicate], slides labeled 10 AVN1, 11 AVN2): Compact atrioventricular node, mild fat infiltration, penetrating bundle, mild thickening, artery to atrioventricular node, moderate to focally severe narrowing, with intimal and medial thickening, arterial branches within central fibrous body and ventricular septum; unremarkable proximal right and left bundle branches; mild to moderate increased fibrosis, ventricular myocardium, crest of ventricular septum

Left ventricular free wall (3 sections mounted on 3 slides [slide 8 LVL mid in duplicate], slides labeled 3 LVA, 4 LVL, 8 LVL mid): unremarkable myocardium, focus of contraction band necrosis (marked with ink dots, slide 4), with neutrophilic response consistent with prolonged resuscitation; focus of lymphocytic infiltrate, without myocyte necrosis, slide 8 (marked with ink dots)

Coronary arteries: Ostia not described; minimal atherosclerosis (per protocol)

Diagnosis: Focal arterial thickening, central fibrous body and crest of the ventricular septum, with associated fibrosis

Comment: The significance of the small vessel changes (see attached photograph) is unclear. We see these changes more frequently in cases of sudden unexpected death in young men and women than controls. However, causality is impossible to prove in an individual case, and of

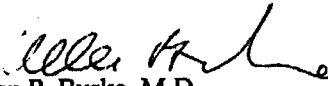
Department of Cardiovascular Pathology
6825 16th St., N.W., Bldg 54, Room 2005, Washington, DC 20306-6000
Telephone: (202) 782-2840 Fax: (202) 782-9021

course noncardiac causes of death must be excluded before ascribing death to intramural coronary arterial thickening. Unfortunately, in about 20% of sudden unexpected deaths due to presumed cardiac arrhythmias, the source of the arrhythmia cannot be ascertained with certainty despite complete cardiac investigation.

The heart has been extensively and appropriately sampled, with excellent identification of conduction system structures, right ventricle, and ventricular septum. Therefore, we do not see the need to study the wet tissue. Of note, the coronary ostia were not described in the protocol provided; as coronary ostial abnormalities are among the more common causes of sudden cardiac death, we would wish to exclude any abnormality at this site.

References:

1. Burke AP, Subramanian R, Smialek J, et al. Nonatherosclerotic narrowing of the atrioventricular node artery and sudden death. *J Am Coll Cardiol* 1993; 21:117-22.
2. Burke AP, Virmani R. Intramural coronary artery dysplasia of the ventricular septum and sudden death. *Hum Pathol* 1998; 29:1124-7.
3. James TN, Marshall TK. De subitaneis mortibus. XVII. Multifocal stenoses due to fibromuscular dysplasia of the sinus node artery. *Circulation* 1976; 53:736-42.
4. James T. Morphologic characteristics and functional significance of focal fibromuscular dysplasia of small coronary arteries. *Am J Cardiol* 1990; 65:12G-22G.


Allen P. Burke, M.D.
Staff Pathologist
Department of Cardiovascular Pathology