Letters to the Editor

Improvement of Left Ventricular Aneurysm After Myocardial Infarction: Report of Three Cases

K. Iwasaki, T. Kita, G. Tanaguchi, S. Kusachi: *Clin Cardiol* 14, 355-360 (1991)

To the Editor:

I read with interest the report of three cases of improvement of left ventricular aneurysm after myocardial infarction by Iwasaki and associates. Their concept that the myocardium presenting as a ventricular aneurysm might be hibernating is provocative. But I seriously question the appropriateness of the use of the term ventricular aneurysm by the authors in their report.

All their patients were studied within one month of acute myocardial infarction. Since it takes at least six weeks for infarct to heal,[1] one is hardly justified in terming any ventriculographic motion abnormality within one month of an acute myocardial infarction a ventricular aneurysm.

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Reference

1. Mallory GK, White PD, Salcedo-Salger J: The speed of healing of myocardial infarction: A study of the pathological anatomy in 72 cases. *Am Heart J* 18, 647 (1939)

Author's reply:

In Case 1, radionuclide ventriculography performed six weeks after the acute myocardial infarction (AMI) revealed dyskinetic anterolateral wall motion. In Case 2, echocardiography done one year after the AMI showed akinetic motion of the anterior and apical wall. In Case 3, dyskinetic apical wall motion was observed in radionuclide ventriculography performed eight weeks after the AMI. From these findings, akinesis/dyskinesis continued at least six weeks in case 1, one year in Case 2, and eight weeks in Case 3. Mallory *et al.*[1] demonstrated that very little healing change occurs after the second month of the infarction. Therefore, it would be unlikely, at least in Cases 2 and 3, that improvement in ventricular abnormal motion was solely dependent on simple healing of the infarct.

Ventricular aneurysm, as defined in our report, used the following criteria: presence of ventricular bulge, and akinesis and/or dyskinesis in wall motion. Not a few of the investigators used these criteria,[2] and considering the clinical setting of our report, such a definition would be reasonable. We could not define ventricular aneurysm histopathologically, i.e., scar aneurysm. Ventriculography was carried out four weeks after the AMI. Therefore, ventricular aneurysm in our report could be viewed as "physiological aneurysm".[2]

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References

1. Mallory GK, White PD, Salcedo-Salger J: The speed of healing of myocardial infarction. *Am Heart J* 6, 647 (1939)
2. Hamer DH, Lindsay J Jr: Redefining true ventricular aneurysm. *Am J Cardiol* 64, 1192 (1989)

Prolonged QT Interval: A Marker of Sudden Infant Death Syndrome?

F. Perticone, R. Ceravolo, P. L. Mattioli: *Clin Cardiol* 14, 417–421 (1991)

To the Editor:

Perticone, Ceravalo and Mattioli ask if prolonged QT interval is a marker of sudden death. Presumably premature or rapid supraventricular impulses would be conducted to the ventricle during the prolonged vulnerable period.

Preston *et al.*[1] described such transmission and the consequent development of ventricular arrhythmias in young goats and pigs but not in puppies, although the effective refractory period (RP) was limited by the ventricular RP in each species. In adult animals transmission delay was limited by the conduction system. Ventricular arrhythmia was not produced by premature atrial excitation.

Thus, in addition to the long QT hypothesis, transmission of early or frequent supraventricular beats to a ventricle in varying degrees of refractoriness might be a factor in development of ventricular arrhythmias and sudden death in suckling humans, even without prolonged QT interval.

In spite of an experimental model, identification of this phenomenon as a cause of SIDS in human infants will be only serendipitous unless a population of parents willing to subject their infants to long-term cardiac monitoring and a wealthy banker willing to provide financing can be identified.

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Calendar of Continuing Medical Education

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The 57th Annual Scientific Assembly of the American College of Chest Physicians

November 4–8—San Francisco, California (USA)

Information: American College of Chest Physicians, Division of Education, 3300 Dundee Road, Northbrook, IL 60062-2348, USA (708) 498-1400

American Heart Association 64th Scientific Sessions

November 11–14—Anaheim, California (USA)

Information: American Heart Association, 7320 Greenville Avenue, Dallas, TX 75231, USA (214) 706-1230

The 6th Congress of Western Pacific Association of Critical Care Medicine

December 1–4—Bangkok, Thailand

Information: Secretariat Office, 6th WPACCM, Dept. of Anesthesiology, Siriraj Hospital, Bangkok 10700, Thailand

1991 Radiological Society of North America Scientific Assembly and Annual Meeting

December 1–6—Chicago, Illinois (USA)

Information: Jodi Skrip, Public Communications Inc., 35 East Wacker Drive, Chicago, IL 60601, USA telephone: (312) 558-1770, fax: (312) 558-5425

1992

Cardiopulmonary Emergencies

January 18–25—Kona, Hawaii (USA)

Information: Edith S. Bookstein/AIPE, P.O. Box 2586, La Jolla, CA 92038, USA; telephone: (619) 454-3212

Sixth San Diego Symposium on Vascular Diagnosis

February 17–20—San Diego, California (USA)

Information: Thomas S. Clark Assoc., 4970 Cleveland Street, Suite 107, Virginia Beach, VA 23462, USA (804) 490-1389, fax: (804) 497-4209

Advanced Clinical Magnetic Resonance Imaging

February 17–21—Waikoloa, Hawaii (USA)

Information: Harvard Med-CME, P.O. Box 825, Boston, MA 02117, USA; telephone: (617) 432-1525

The 36th Annual Convention of the American Institute of Ultrasound in Medicine (AIUM)

March 8–11—San Diego, California (USA)

Information: AIUM Convention Department, 11200 Rockville Pike, Suite 205, Rockville, MD 20852-3139, USA; telephone: (301) 881-2486, fax: (301) 881-7303

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Reference

1. Preston JB, McFadden S, Moe GK: Atrioventricular transmission in young mammals. *Am J Physiol* 197(1), 236–240 (1959)

Author's reply:

I agree with Dr. McFadden's conclusions: the QT hypothesis of SIDS is not exclusive. Many large prospective and pathologic studies are necessary to identify the specific mechanism of SIDS.

Therefore, it would be desirable to perform a new study by 24-hour holter ECG in most newborns to show the ventricular arrhythmias as possible cardiac mechanisms of SIDS. In fact, in one of our newborns (three days old) we recorded by 24-hour holter ECG several episodes of torsade de pointes, the longer of which lasted 13.5 s (data unpublished).

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