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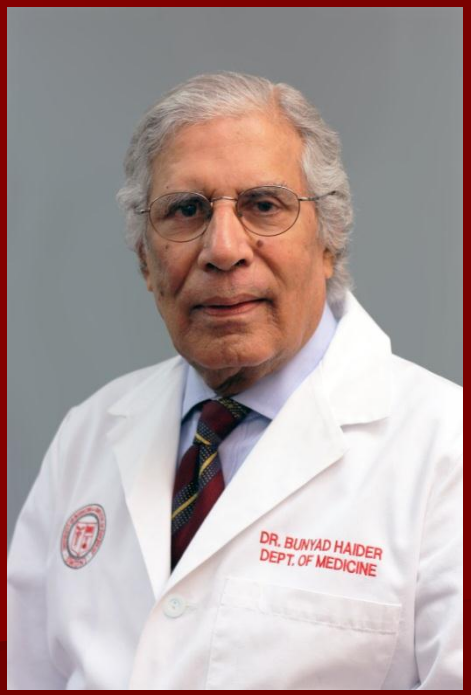

Bunyad Haider, M.D.

Professor of Medicine

Division of Cardiology

Comparative antiarrhythmic effects of intravenously administered lidocaine and procainamide and orally administered quinidine☆

Mortimer L. Schwartz, MD, FACC, N.Conant Webb, MD, Benjamin G. Covino, MD, PhD, Edward M. Finck, MD, Bunyad Haider, MD

From the Department of Medicine, New Jersey College of Medicine and Dentistry, Newark, N. J. USA

Received 12 September 1969; accepted 16 February 1970.

Abstract

A double-blind control study compared the ventricular antiarrhythmic efficacy of a single dose each of intravenously administered lidocaine and procainamide and orally administered quinidine. A statistically significant reduction in ventricular ectopic contractions occurred immediately and was present 30 minutes and 1 hour after the onset of injection of procainamide. Lidocaine produced a statistically significant reduction in ventricular extrasystoles immediately and for 30 minutes thereafter. No decrease in the incidence of ectopic contractions was observed with either orally administered quinidine or placebo therapy alone. The duration of ventricular antiarrhythmic action of a single injection of procainamide was significantly greater than that of lidocaine. A statistically but probably not clinically significant reduction in systolic blood pressure was observed with procainamide, quinidine and placebo therapy. No change in either systolic or diastolic blood pressure was observed with lidocaine in the dose employed.

Ischemic heart failure: Sustained inotropic response to small doses of L-epinephrine without toxicity☆

Bunyad Haider, MD, FACC, Mohammad I. Khan, MD, William M. Burke, MD, FACC, Timothy J. Regan, MD, FACC

From the Department of Medicine, College of Medicine and Dentistry of New Jersey, New Jersey Medical School, Newark, N. J., USA

Accepted 30 October 1974.

Abstract

As a prelude to a study of severe ischemic heart failure, the therapeutic response of the ischemic ventricle to epinephrine and acetylstrophanthidin in nontoxic doses was determined in 24 intact anesthetized dogs undergoing a first episode of acute regional ischemia. A thrombotic obstruction was produced in the left anterior descending coronary artery, effecting moderate left ventricular dysfunction. The elevation of end-diastolic pressure and reduced stroke volume in control dogs were not significantly altered by administration of strophanthidin. Epinephrine (0.05 μg/kg per min) elicited a significant reduction in end-diastolic pressure and increase in stroke volume. The latter was not attended by an increased incidence of ventricular fibrillation, whereas fibrillation occurred in half of the group given strophanthidin. Thus, the catecholamine was selected to study pump failure.

Severe ischemic heart failure was assessed in two groups with scar from previous infarction for up to 4 hours. By 60 minutes of ischemia, the increase in end-diastolic pressure and volume and decrease in stroke volume and ejection fraction were comparable in both groups. Thereafter, alternate animals received small doses of epinephrine (0.05 to 0.15 μg/kg per min) with graded increments at 60 minute intervals to counter tachyphylaxis and findings were compared with those in control dogs. Over the subsequent 3 hours, there was progressive deterioration of left ventricular function in the untreated group with an increase in end-diastolic pressure from 10 ± 1 to 33 ± 2.4 mm Hg. End-diastolic volume increased by 63 percent; stroke volume and ejection fraction decreased by 48 and 66 percent, respectively. The infusion of epinephrine was attended by a significantly lower end-diastolic pressure of 20 ± 2.5 mm Hg, whereas end-diastolic volume, stroke volume and ejection fraction were restored to control levels after 4 hours of ischemia. Mortality in the untreated group was 62 percent by 4 hours; all seven animals in the treated group survived.

Evidence for Cardiomyopathy in Familial Diabetes Mellitus

TIMOTHY J. REGAN, MICHAEL M. LYONS, S. SULTAN AHMED, GILBERT E. LEVINSON, HENRY A. OLDEWURTEL, MEHMOOD R. AHMAD, and BUNYAD HAIDER, *The Departments of Medicine and Pathology, College of Medicine and Dentistry of New Jersey-New Jersey Medical School, and the Martland Hospital Unit, Newark, New Jersey 07103*

ABSTRACT Recent epidemiologic studies have suggested that cardiac disease is common in diabetics and may often have a noncoronary basis. To examine the status of the left ventricle, 17 adult-onset diabetics of familial type without hypertension or obesity underwent hemodynamic study and were compared to 9 controls of similar age.

Of the 17, 12 subjects had no significant occlusive lesions by coronary angiography. From this group eight without heart failure had a modest, but significant, elevation of left ventricular end-diastolic pressure. End-diastolic and stroke volumes were reduced, but ejection fraction and mean rate of fiber shortening were within normal limits. The left ventricular end-diastolic pressure/volume ratio was significantly higher than controls. Afterload increments effected a significant increase of filling pressure compared to normals without a stroke volume response, consistent with a preclinical cardiomyopathy. Four patients with prior heart failure had similar but more extensive abnormalities. None had local dyskinesia by angiography, and lactate production was not observed during pacing-induced tachycardia. Left ventricular biopsy in two patients without ventricular decompensation showed interstitial collagen deposition with relatively normal muscle cells. These findings suggest a myopathic process without ischemia.

Postmortem studies were performed in 11 uncomplicated diabetics. Nine were without significant obstructive disease of the proximal coronary arteries, and the majority succumbed with cardiac failure. On left ventricular sections, none had evident luminal narrowing of the intramural vessels. All nine exhibited periodic acid-Schiff–positive material in the interstitium. Collagen accumulation was present in perivascular loci, between myofibers, or as replacement fibrosis. Multiple samples of left ventricle and septum revealed enhanced triglyceride and cholesterol concentrations, as compared to controls. Thus, a diffuse extravascular abnormality may be a basis for cardiomyopathic features in diabetes.

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INTRODUCTION

A relatively high incidence of cardiac deaths in patients with diabetes mellitus has been attributed to coronary atherosclerosis and its complications (1). However, two recent studies of vessel pathology in diabetics failed to support the assumption that all cardiac deaths were related to this process. In a quantitative study of the extent of surface involvement in the coronary vessels, only a modest increase in the quantity of atherosclerotic disease was present in diabetics compared to age- and sex-matched controls (2). In addition, Ledet observed that intramural vessels commonly exhibited accumulation of glycoprotein but did not show luminal narrowing (3). Thus, the question is raised as to whether a portion of the cardiac diabetic population may have primary myocardial abnormalities as a basis for their symptomatology, independent of ischemia, hypertension, or obesity.

As an initial approach to this problem in a canine model with chronic diabetes, ventricular function and compliance were observed to be abnormal (4), associated with accumulation of periodic acid-Schiff–(PAS) positive glycoprotein in the myocardium as well as increments of triglyceride. In humans, a functional abnormality was also found in noncardiac adult diabetics by the systolic time-interval method (5), similar to the preclinical abnormality of ethanolism (6, 7).

This report is concerned with an examination of left ventricular function and coronary arteriograms after the development of cardiac symptoms in uncomplicated adult diabetics with a familial history, to determine if the development of symptomatology is de-

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Myocardial Function and Coronary Blood Flow Response to Acute Ischemia in Chronic Canine Diabetes

BUNYAD HAIDER, S. SULTAN AHMED, CHRISTOS B. MOSCHOS, HENRY A. OLDEWURTEL, AND TIMOTHY J. REGAN

SUMMARY To examine the influence of preexistent diabetes mellitus on left ventricular performance and coronary blood flow responses to acute ischemia, mild normoglycemic diabetes was induced in nine mongrel dogs after three doses of alloxan, (20 mg/kg, iv), at monthly intervals. Hemodynamic measurements and coronary blood flow (^{85}Kr clearance) were obtained before and after the onset of ischemia. This was produced by occlusion of the proximal left anterior descending coronary artery via a balloon-type catheter in nine intact anesthetized diabetic dogs and 10 nondiabetic dogs. During the 1st hour of ischemia in the diabetic group, the end-diastolic pressure rose from 7 ± 1.1 (mean ± SE) mm Hg to 23.8 ± 2.3 without a significant increase of end-diastolic volume. In controls end-diastolic pressure rose from 8.6 ± 1.1 mm Hg to 15.3 ± 1.4, and end-diastolic volume was significantly increased, so that the ratio of end-diastolic pressure and volume was significantly higher in the diabetic group ($P < 0.005$). Although indices of contractility did not differ, stroke volume and work reductions were significantly greater in diabetics, despite the fact that coronary blood flow was reduced to a similar extent. Size of the ischemic areas appeared comparable as judged by distribution of dye injected distal to the occlusion. Since potassium loss and sodium gain in the inner and outer layers of ischemic tissue did not differ between the two groups, the intensity of ischemia seemed similar. Glycogenolysis was unimpaired in the diabetic ischemic muscle but triglyceride levels remained elevated. Morphologically the diabetic myocardium was characterized by a diffuse accumulation of periodic acid-Schiff-positive glycoprotein in the interstitium, which was thought to limit diastolic filling of the ischemic ventricle and to contribute to the substantial reduction of ventricular performance.

ALTHOUGH the influence of acute regional ischemia on left ventricular function has been well defined in the previously normal animal,[1, 2] the response of the ventricle affected by a chronic metabolic or structural abnormality has not been described. Acute myocardial infarction has been reportedly associated with a greater incidence of pump failure and higher mortality in diabetes mellitus.[3] Although the increased mortality from cardiac disease complicating diabetes mellitus has been traditionally attributed to accelerated atherosclerosis of the coronary arteries,[4] this is a disputed issue since recent evidence in studies using more quantitative methods and age-matched controls has shown that the complicated lesions of atherosclerosis may occur to only a slightly greater extent in diabetics.[5]

From the Department of Medicine, College of Medicine and Dentistry of New Jersey—New Jersey Medical School, Newark, New Jersey. Supported in part by Research Grant HL 09914 and Postgraduate Training Grant HL 05510 from the National Heart and Lung Institute.
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Address for reprints: Bunyad Haider, M.D., CMDNJ-New Jersey Medical School, Department of Medicine, 100 Bergen Street, Newark, New Jersey 07103.

In a previous study from this laboratory,[6] we observed altered myocardial function in chronic diabetes mellitus in dogs, associated with accumulation of periodic acid-Schiff (PAS)-positive glycoprotein in the myocardial interstitium without coronary obstructive lesions; this morphological abnormality also has been observed in man.[7, 8] To examine the response of the diabetic myocardium during acute regional ischemia as compared to normal controls the following study was undertaken.

Methods

Two groups of healthy male mongrel dogs 2–4 years old and weighing 21–28 kg were studied. The dogs had no clinical evidence of disease for 6–8 weeks before admission to the study groups. Hematocrit and serum albumin were initially normal and both groups received the same diet consisting of 8% fat, 22% protein, 58% carbohydrate, 9% ash, and 3% crude fiber. One group (n = 10) served as controls with normal glucose tolerance by intravenous testing. The other group (n = 9) was made diabetic with low doses of alloxan at monthly intervals. To produce mild normoglycemic diabetes, alloxan monohy-

Trans Assoc Am Physicians. 1978;91:197-203.

Altered myocardial function and collagen in diabetic rhesus monkeys on atherogenic diet.

Haider B, Yeh CK, Thomas G, Oldewurtel HA, Lyons MM, Regan TJ.

Abstract

We have demonstrated that in rhesus monkeys, 18 months of diabetes alters the end-diastolic pressure, end-diastolic volume relations without hypertrophy. Accumulation of collagen in the myocardial interstitium was the apparent basis for abnormal left ventricular performance. Neither collagen concentration nor left ventricular performance were signigicantly affected by dietary lipid composition. These myocardial abnormalities occurred at a stage when coronary atherosclerosis was limited. However, the relative influence of coronary atherosclerosis and myocardial alterations during more prolonged lipid feeding remains to be determined.

PMID: 112754 [PubMed - indexed for MEDLINE]

I think an intracardiac banana **would** be indicated at this point!

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ST-Segment Analysis Using Wireless Technology in Acute Myocardial Infarction (STAT-MI) Trial

Vivek N. Dhruva, DO,* Samir I. Abdelhadi, DO,* Ather Anis, MD,* William Gluckman, DO, FAEP,† David Horn, MS,‡ William Dougan, MICP,† Edo Kaluski, MD, FACC,* Bunyad Haider, MD, FACC,* Marc Klapholz, MD, FACC*

Newark, New Jersey

Objectives Our goal was to examine the effects of implementing a fully automated wireless network to reduce door-to-intervention times (D2I) in ST-segment elevation myocardial infarction (STEMI).

Background Wireless technologies used to transmit prehospital electrocardiograms (ECGs) have helped to decrease D2I times but have unrealized potential.

Methods A fully automated wireless network that facilitates simultaneous 12-lead ECG transmission from emergency medical services (EMS) personnel in the field to the emergency department (ED) and offsite cardiologists via smartphones was developed. The system is composed of preconfigured Bluetooth devices, preprogrammed receiving/transmitting stations, dedicated e-mail servers, and smartphones. The network facilitates direct communication between offsite cardiologists and EMS personnel, allowing for patient triage directly to the cardiac catheterization laboratory from the field. Demographic, laboratory, and time interval data were prospectively collected and compared with calendar year 2005 data.

Results From June to December 2006, 80 ECGs with suspected STEMI were transmitted via the network. Twenty patients with ECGs consistent with STEMI were triaged to the catheterization laboratory. Improvement was seen in mean door-to-cardiologist notification (−14.6 vs. 61.4 min, $p < 0.001$), door-to-arterial access (47.6 vs. 108.1 min, $p < 0.001$), time-to-first angiographic injection (52.8 vs. 119.2 min, $p < 0.001$), and D2I times (80.1 vs. 145.6 min, $p < 0.001$) compared with 2005 data.

Conclusions A fully automated wireless network that transmits ECGs simultaneously to the ED and offsite cardiologists for the early evaluation and triage of patients with suspected STEMI can decrease D2I times to <90 min and has the potential to be broadly applied in clinical practice. (J Am Coll Cardiol 2007;50:509–13) © 2007 by the American College of Cardiology Foundation

The benefit of decreasing door-to-balloon times (D2B) in acute ST-segment elevation myocardial infarction (STEMI) has previously been widely reported (1–4). Strategies to decrease D2B times are often limited by multiple time-intensive steps. Implementation of wireless electrocardiogram (ECG) transmissions from the field to the emergency department (ED) have recently been shown to significantly decrease D2B times but still require personnel in the ED to forward the ECG to the cardiologist (5). The STAT-MI project (ST-Segment Analysis Using Wireless Technology in Acute Myocardial Infarction) was devised to demonstrate that newer wireless technologies and software linked in a network can facilitate simultaneous transmission of high-resolution ECGs to both the ED and offsite cardiologists and further improve door-to-intervention (D2I) times. We report on the initial results of implementing this completely automatic notification and response system for the evaluation and management of STEMI and its use in reducing D2I times.

Methods

Network. The STAT-MI network was devised by a collaborative task force at the University of Medicine and Dentistry, New Jersey—University Hospital led by cardiology staff and including members of emergency medical services (EMS), ED, hospital administration, hospital information and technology, telecommunications, medical informatics, and physician staff. Representatives from Medtronic Corporation (LIFEPAK 12, LifeNet Receiving

From the *Division of Cardiology, Department of Medicine, †Division of Emergency Medicine, Department of Surgery, and ‡Department of Medicine, University of Medicine and Dentistry, New Jersey—New Jersey Medical School, Newark, New Jersey. Grants for this study were received from the Verizon Foundation (Basking Ridge, New Jersey) and Medtronic Corporation (Minneapolis, Minnesota).

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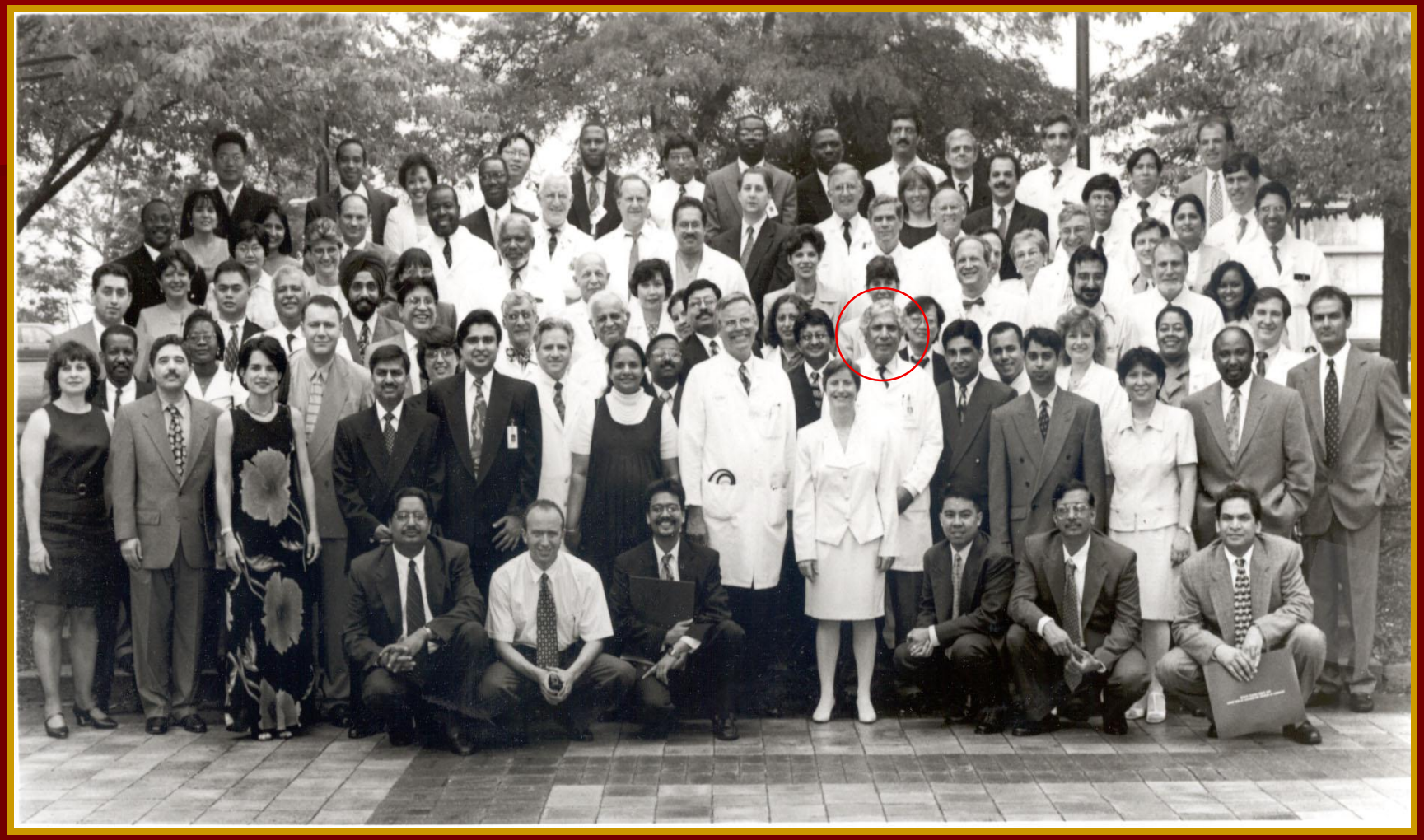

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American Journal of Cardiology
Volume 26, Issue 5 , Pages 520-523, November 1970

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Abstract | Abstract + References | PDF | References

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Volume 35, Issue 4 , Pages 504-513, April 1975

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Circulation

ARTICLES

Acute myocardial infarction in toxic cardiomyopathy without coronary obstruction

TJ Regan, CF Wu, AB Weisse, CB Moschos, SS Ahmed, and MM Lyons

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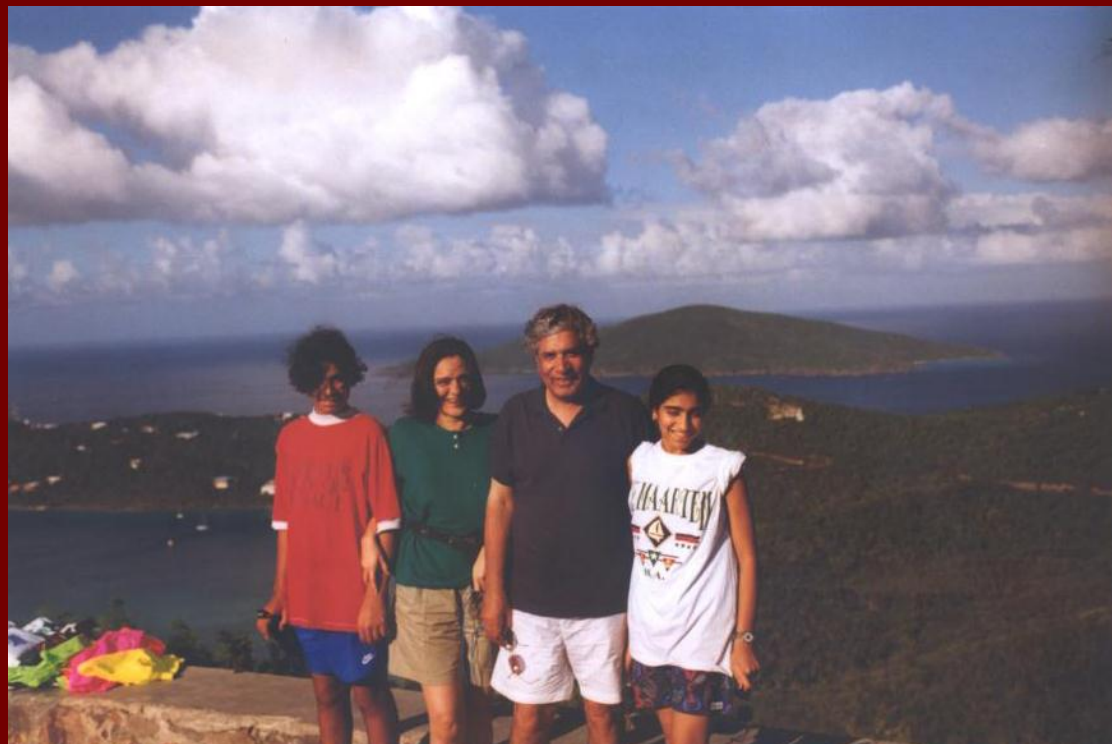
Abstract | PDF



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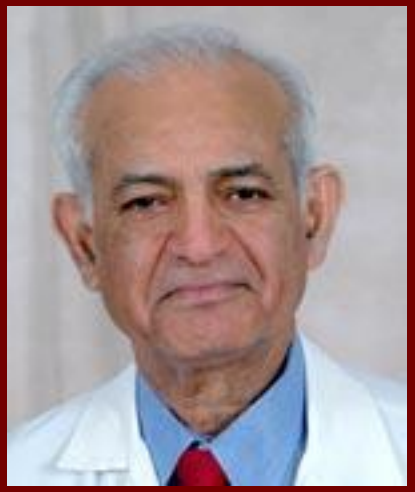

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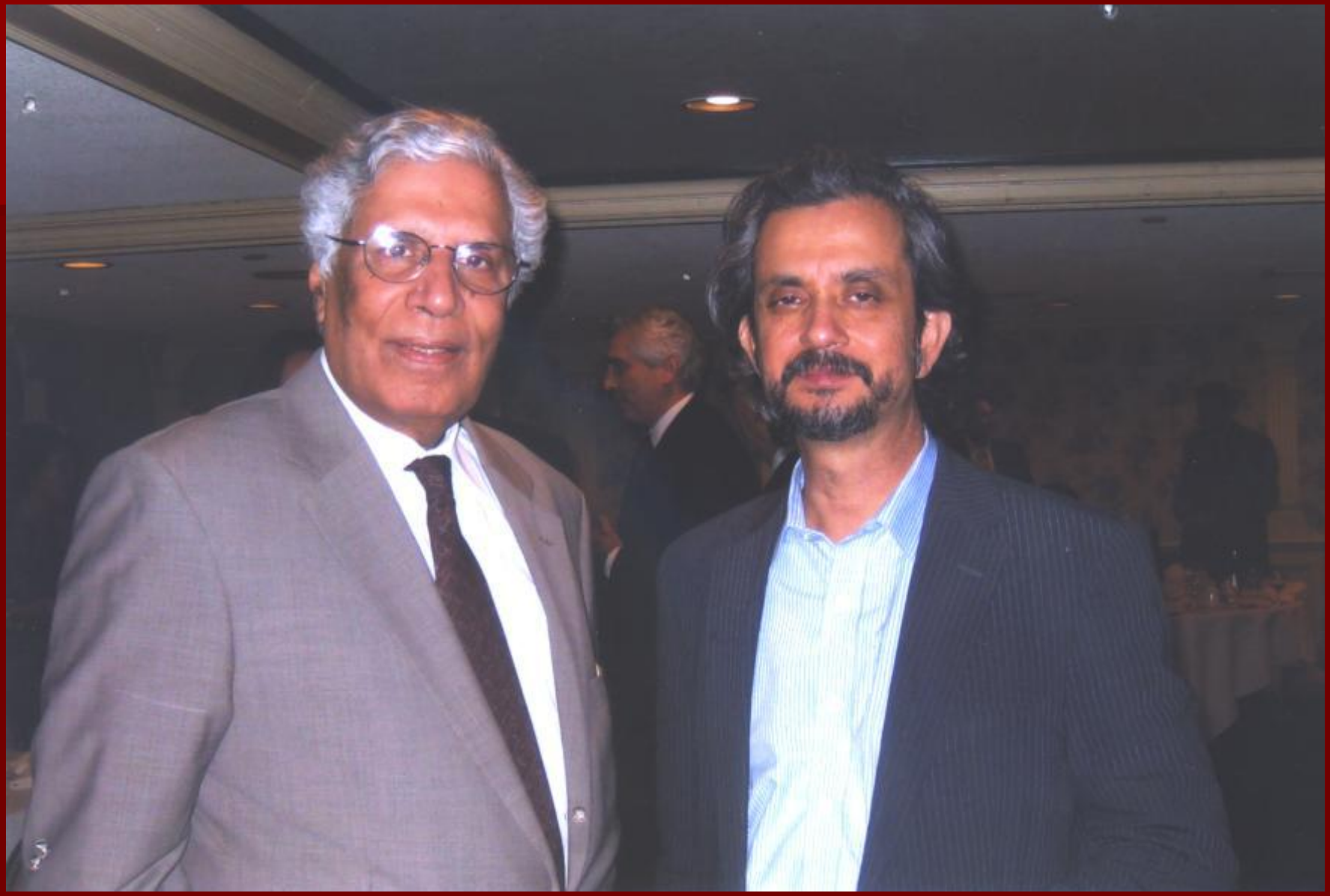


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