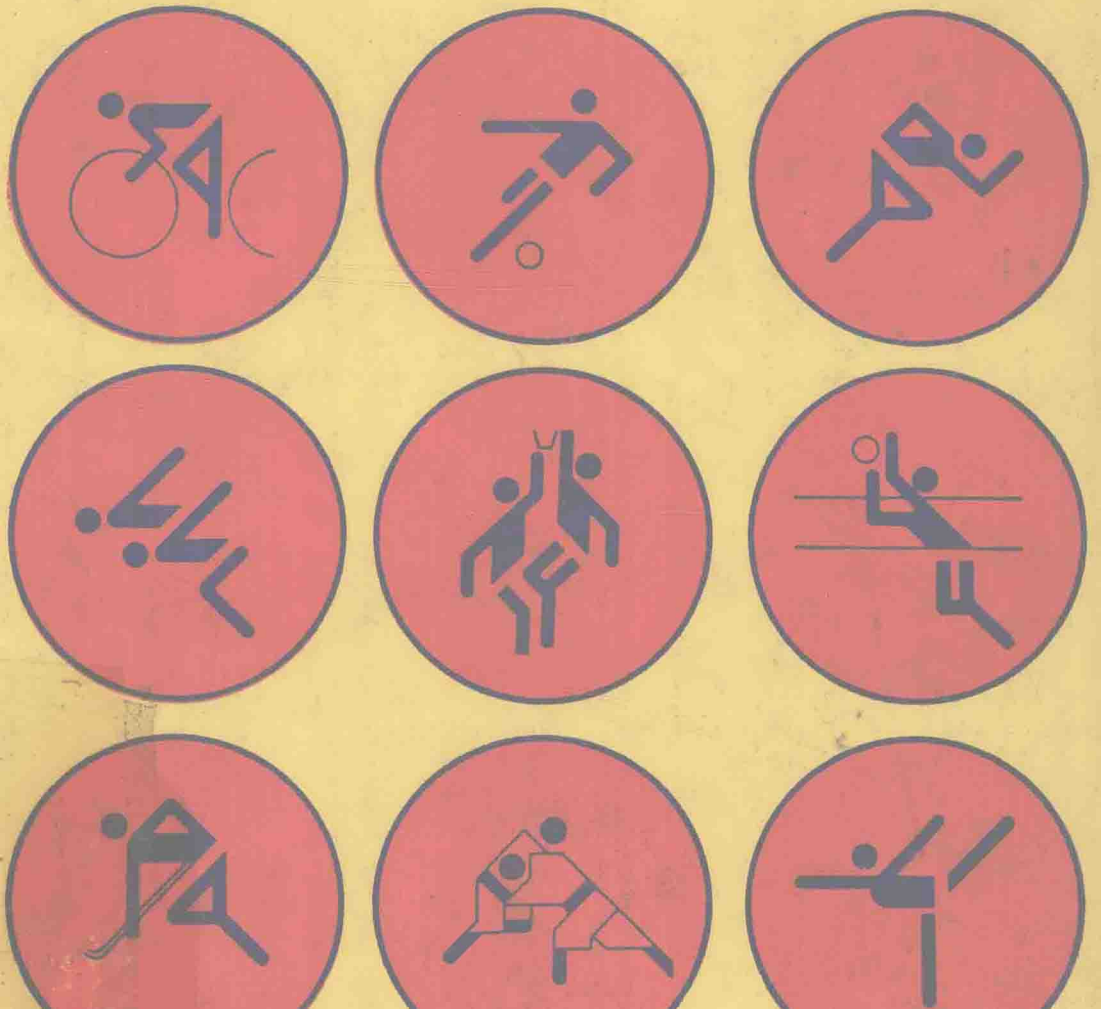
1983 Year Book of SPORTS MEDICINE

J. L. ANDERSON • F. GEORGE
L. J. KRAKAUER • R. J. SHEPHARD
J. S. TORG



The YEAR BOOK of

Sports Medicine

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YEAR BOOK MEDICAL PUBLISHERS, INC.
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THOMAS B. QUIGLEY, M.D.

Tribute to Thomas B. Quigley

American surgery has lost one of its most colorful personages with the death of Thomas B. Quigley, a former editor of this YEAR BOOK.

Born in 1908 in North Platte, Nebraska, Quigley later recalled himself as a child sitting on the knee of Buffalo Bill Cody, a family friend. He went to Harvard College and Harvard Medical School, graduating in 1933 during the bottom of the Depression. A talented amateur actor, Quigley spent vacations working with stock companies on Cape Cod and remained friends with his acting colleagues, who included Henry Fonda. Each had a daughter named Jane—Jane Fonda and Jane Alexander are still friends and mutual admirers.

Quigley spent 1933 as a resident in pathology in New York City, and then became a surgical intern at Peter Bent Brigham Hospital in Boston, staying on as a resident until 1938, when he took a chief residency in New York City. In 1939, he returned to Boston as surgeon to the University Health Service and Athletic Department at Harvard.

As a volunteer in the armed forces, Quigley went overseas in March 1942 with the Harvard Surgical Unit for over three years, first in Ireland and, after D-Day, on the continent, where he was a surgeon and Chief of Surgical Service for the 22nd General Hospital on the Normandy beachhead and on into Germany. He returned to Boston after the war to rejoin his wife, Ruth, and pick up the threads of his practice. He became Attending Surgeon at Peter Bent Brigham Hospital and eventually Clinical Professor of Surgery at Harvard and Lecturer in Orthopedic Surgery at Boston University and Tufts Medical School. Originally trained as a general surgeon, Quigley had acquired broad experience in trauma surgery during the war. Gradually, he devoted more and more of his time to treatment of athletic injuries and bone and joint surgery.

He took his duties as team physician at Harvard seriously, attended games and practices faithfully, and pioneered the training and treatment techniques that have become the gold standard of care for athletic teams. As his fame spread, he became involved in the care of many athletes, professional and amateur, and numbered some of the all-time greats among his patients.

He always had a marvelous flair for teaching, a quick wit, and a wondrous way with the English language. He enlivened conferences and training programs with astute and clever comments. His publications numbered nearly 200 over 45 years and contained many gems of wit and wisdom.

He suffered the depredations of a stroke that made his last years

an ordeal—an ordeal that he surmounted with dignity. His wife, Ruth, died several years ago, and he leaves a son and two daughters.

It is hard to think of anyone who has left a bigger impact on sports medicine than Bart Quigley. Courageous, chivalrous, humorous, dedicated, dramatic—he is sorely missed.

CRAIG B. LEMAN, M.D.

Dr. Leman was a former staff member and close friend of Dr. Quigley at the Peter Bent Brigham Hospital in Boston.

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The material covered in this volume represents literature reviewed up to February, 1983.

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American Heart Journal
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American Journal of Clinical Nutrition
American Journal of Obstetrics and Gynecology
American Journal of Ophthalmology
American Journal of Physiology
American Journal of Sports Medicine
American Review of Respiratory Disease
Annales Chirurgiae et Gynaecologiae
Annals of Allergy
Annals of Internal Medicine
Annals of Neurology
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Archives of Internal Medicine
Archives of Otolaryngology
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Australian Paediatric Journal
British Heart Journal
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European Journal of Clinical Pharmacology
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Journal of the American Medical Association
Journal of the American Osteopathic Association
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Science Digest
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South African Medical Journal
Sports Illustrated
Thorax

1. Exercise Physiology

Cardiopulmonary Physiology

1–1 **Frequency of Intensive, Prolonged Exercise as a Determinant of Relative Coronary Circumference Index.** Although functional cardiac changes in association with regular physical activity have been well documented, structural changes leading to lower susceptibility to coronary occlusive disease are not well defined. R. W. Haslam and R. B. Cobb (Syracuse Univ., N. Y.) examined the effects of exercise training at varying frequencies on the relative coronary circumference index (RCCI), which has been shown to be related to the prevalence of myocardial infarction. Rats participated in 10 weeks of intensive aerobic swimming exercise 1, 5, or 10 times a week, swimming for 30–51 minutes on each occasion while carrying 1.0% to 2.25% of their individual body weight. The load was increased progressively during the 10-week training period. The RCCI was calculated as the ratio of ventricular weight to the sum of the circumferences of the right and left main coronary arteries.

The incremental effectiveness of weighted swimming in causing differential energy expenditures was confirmed by changes in body weight. The mean wet-weight RCCI decreased significantly as exercise frequency increased beyond once a week (table). The mean value for rats trained 10 times a week was 8.7% less than that in sedentary rats. Ventricular wet weight showed a significant inverse linear trend with respect to frequency of exercise; absolute dry ventricular weight showed no significant trend. A hypertrophic response to exercise was evident when ventricular dry weight was adjusted for body weight differences.

Circumference Variable Means For Various Exercise Frequencies*

	Control (sedentary)	Low freq. (1x/wk)	Moderate freq. (5x/wk)	High freq. (10x/wk)	Trend analysis
No. subjects	20	19	11	19	
RCCI (using wet wt.) ($mg \cdot pu^{-1}$)	98.8 ±3.1	99.2 ±3.4	91.0 ±3.5	90.2 ±3.0	Linear $P < 0.01$
RCCI (using dry wt.) ($mg \cdot pu^{-1}$)	18.5 ±0.5	18.7 ±0.6	18.0 ±0.5	16.9 ±0.7	Linear $P < 0.01$
Left coronary circumference (pu)	11.64 ±0.36	11.74 ±0.32	11.60 ±0.48	12.59 ±0.43	Linear $P < 0.10$
Right coronary circumference (pu)	7.88 ±0.28	8.28 ±0.40	8.24 ±0.56	8.14 ±0.42	NS
Summed coronary circumference (pu)	14.11 ±0.35	14.40 ±0.42	14.29 ±0.60	15.07 ±0.49	NS

*Values are means ± SE; pu = planimeter units measured by polar planimeter; NS = not significant.

(1–1) Int. J. Sports Med. 3:118–121, May 1982.

Intensive swimming exercise leads to a reduction in the relative coronary circumference index in rats, which indicates a greater circumference per unit mass of heart. The findings suggest the distinct possibility that the RCCI can be reduced in human beings through regular exercise, and also indicate that the reduction will be greater as the frequency of exercise increases. The RCCI provides a link between exercise-induced structural coronary vascular change and myocardial infarction.

▶ [There has been much discussion as to the extent to which regular exercise can increase the blood supply of the myocardium. Despite early enthusiasm, it is now generally agreed that exercise alone has little influence on the dimensions of the collateral vessels, even in experimental animals (see article 1–2). Several researchers have demonstrated an increased capacity of the coronary arterial tree (as seen in plastic casts), and an increased circumference or cross-section of the main coronary vessels after a subject has undergone vigorous training. However, the potential benefit of such changes could have been outweighed by concomitant cardiac hypertrophy.

The present article overcomes this objection by expressing the circumferences of the two main coronary arteries as a ratio to ventricular mass, an index that is apparently related to the prevalence of myocardial infarction and myocardial failure in humans. The studies of Haslam and Cobb show a supposedly linear trend toward increased myocardial vascularity, as exercise is increased from control to ten 30–51-minute sessions per week. Nevertheless, the main increment of the relative coronary circumference index is seen on moving from 1 to 5 sessions of exercise per week, and it is possible that studies on a larger sample of animals might have revealed some nonlinearity of the exercise dose-response curve.—R.J.S.] ◀

1–2 **Influence of Physical Exercise on Coronary Collateral Blood Flow in Chronic Experimental Two-vessel Occlusion.** Wolfgang Schaper (W. Germany) reports that in 45 purebred German shepherd dogs, approximately 1 year old, the left circumflex and right coronary arteries were chronically occluded by implantation of slowly swelling ameroid constrictors that occluded the artery within 2½ weeks. Before operation, 27 dogs were trained on a treadmill until they could run 8 mph on a 22% incline for 1 hour, 5 days per week. Preoperative exercise training lasted 1–3 months.

Two weeks after operation, exercise was gradually resumed and continued for 12 weeks, when preoperative performance level had been regained and maintained for one month. After the dogs had trained with two chronically occluded coronary arteries, collateral and coronary blood flows were measured 100 ± 22 days after operation with tracer microspheres at maximal coronary vasodilation (adenosine infusion) in an isolated, blood-perfused Langendorff preparation at perfusion pressures of 40, 60, 80, 100, 120, and 140 mm Hg. Eighteen nonexercising dogs with 2-vessel coronary occlusion served as controls. Nine controls and 9 exercising dogs were paired littermates.

Two-vessel occlusion was associated with a 24% mortality rate. All dogs died instantaneously. Exercise had no influence on mortality. Autopsies of these dogs revealed absence of a developed collateral circulation associated with premature arterial closure due to a constric-

(1–2) Circulation 65:905–912, May 1982.

tor fitted too tightly at operation or thrombus formation inside the stenosed artery.

Exercise of relatively high intensity (heart rates greater than 200 beats/minute) before and after occlusion had no effect on coronary collaterals as compared with collateral blood flow achieved spontaneously in nontrained sedentary controls. Collateral conductance in trained and untrained dogs reached only slightly less than 40% of that of the replaced coronary artery. Radiomicrosphere distributions indicated a flow deficit, especially subendocardial, at loads requiring a high blood flow.

Results agreed well with earlier observations in nonexercising dogs with chronic 2-vessel occlusion studied in an identical way.

▶ [Since the classic experiments of Eckstein (*Circ. Res.* 5:230, 1959), opinions have been deeply divided on the possibility of producing collateral vessels by vigorous exercise. The present report is a careful reexamination of this question. In keeping with clinical experience in man, Schaper concludes that intense physical activity (that which raises the heart rate to more than 200 beats per min) does not stimulate collateral development in dogs, relative to the response of sedentary control animals with similar chronic occlusion of two coronary vessels.

Schaper notes a number of interesting differences between dogs and humans: in particular, the collaterals of the dog are restricted to the subepicardial tissue and, although exercise was intense enough to induce subendocardial ischemia, it was not sufficient to induce subepicardial ischemia (and thus it is unlikely that there was any added stimulus to collateral formation in the animals that had exercised). He stresses that some earlier authors assessed collateral function under the rather artificial conditions of retrograde perfusion. From a functional point of view, the key issue is how much blood flow can be delivered through the collateral vessels while the heart is contracting against a heavy afterload.

In man, interest has begun to shift away from the collateral vessels, as it has become recognized that a number of other factors can improve the relative oxygen supply of the myocardium (including not only a reduction of exercise heart rate and systolic pressure, but also an increase in the duration of diastole, a redistribution of blood flow between superficial and deeper parts of the heart wall, and changes of cardiac dimensions).—R.J.S.] ◀

1–3 **Comparison of Platelet Function During Exercise in Normal Subjects and Coronary Artery Disease Patients: Potential Role of Platelet Activation in Myocardial Ischemia.** Jawahar Mehta and Paulette Mehta (Univ. of Florida) studied platelet function measurements in 22 consecutive patients with coronary artery disease (CAD) and in 13 normal subjects. All CAD patients had evidence on selective angiography of significant stenosis in one or more coronary arteries.

In exercise stress tests, 8 CAD patients had normal results and 14 had abnormal results. Platelet counts during exercise increased similarly in normal subjects and in CAD patients. Platelet aggregation response to adenosine diphosphate was unaffected by exercise in normal subjects or in CAD patients (platelet counts were adjusted). Platelets from 7 of the 14 CAD patients with abnormal stress test results had increased sensitivity to endoperoxide analog (U-46619)/ (that is, a U-46619 concentration of less than 200 ng/ml was necessary for 50% platelet aggregation).

(1–3) Am. Heart J. 103:49–53, January 1982.

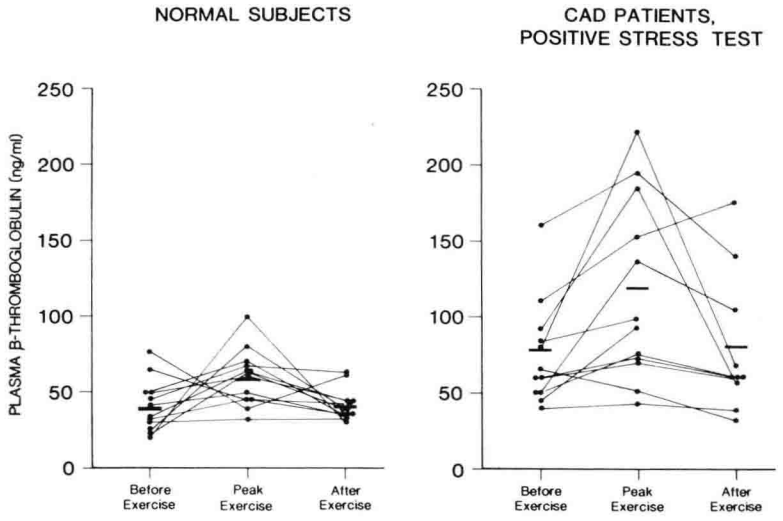

Fig 1–1.—Plasma β-thromboglobulin levels immediately before exercise, at peak exercise, and 15 to 30 minutes after exercise. Preexercise levels were significantly higher in CAD patients with positive exercise stress tests compared with levels in normal subjects. During exercise, plasma β-thromboglobulin levels increased to much higher levels than those seen in normal subjects. After exercise, plasma β-thromboglobulin levels quickly declined to preexercise levels. (Courtesy of Mehta, J., and Mehta, P.: Am. Heart J. 103:49–53, January 1982.)

Resting plasma β-thromboglobulin (B-TG) levels, which are an index of in vivo platelet activation, were significantly higher in CAD patients than in normal subjects (74 ± 7 and 41 ± 5 ng/ml, respectively). During exercise, plasma B-TG levels increased in normal subjects to 60 ± 5 ng/ml. In CAD patients, B-TG concentrations increased to 102 ± 14 ng/ml during exercise but declined to preexercise levels soon after exercise. Of the 12 CAD patients with abnormal exercise stress test results, 11 had increases in plasma B-TG levels during exercise (Fig 1–1), whereas only 3 of the 8 CAD patients with normal stress test results had any increase. The mean increase in plasma B-TG levels in CAD patients with abnormal stress test results was significantly greater than that in CAD patients with normal results (51% vs. 19%).

The results indicate that certain measurements of platelet function at rest are altered in CAD patients. Exercise stress is associated with further platelet function abnormalities in certain CAD patients. Exercise-induced platelet activation may be related to myocardial ischemia. It is not clear whether platelet activation is a primary factor in the genesis of myocardial ischemia or occurs secondary to exercise-induced stress.

► [This article presents interesting evidence that, in some coronary patients, exercise may induce myocardial ischemia through an unusual degree of platelet activation. A potent vasoconstrictor, thromboxane A_2, is released in the early stages of activation, and adherence of platelets to sites of damage in the coronary vascular tree could give rise to further narrowing of the injured vessels, to the point that oxygen delivery is inadequate to meet the demands of exercise. There is some possibility that this