The major fuels for the normal human heart are free fatty acids in the fasted state and glucose in the fed state. During hypoxia, glucose metabolism is accelerated with the production of lactate (anaerobic glycolysis). When lactate accumulates or when the intracellular pH falls, glycolysis may be limited. During hypoxia, products of lipid metabolism, such as triglycerides, accumulate and these might have harmful effects. The state of oxygenation therefore profoundly affects myocardial metabolism. The SCi® indicates that this paper has been cited over 220 publications since 1968.

My interest in myocardial metabolism was awakened by Sir John McMichael, previous director of the department of medicine at the Hamersmith Royal Postgraduate Medical School, in 1959. He pointed out: "When the heart fails, the anatomy looks the same, but something has gone wrong with the chemistry." In a flash of enthusiasm I realized that not only congestive heart failure, but drug action and many clinical aspects of heart function were ultimately going to be explained by myocardial metabolism. I avidly began to read about the subject but could find no suitable reference material except for two: Olson and Schwartz reviewed basic science in Medicine in 1951, and Richard Bing described human heart metabolism in Circulation in 1955. The field was clearly developing, as shown by Bing's Harvardian Oration to the New York Academy of Sciences in 1946, so it seemed to me before any extensive research work should be undertaken that existing knowledge required gathering and analysis. That decision was made in 1960 when I was a research fellow at Harvard Medical School.