Acronymic one-upmanship

I read with amusement the article on the EMPIRE study[1]. Although the authors did not define the acronym EMPIRE — used only in the title but not in the text, I guess that it was referring to the first part of the title ‘Economics of Myocardial Perfusion Imaging in Europe’. But where did the R in EMPIRE come from unless it came from the r that follows the E in Europe?

According to the Webster’s Third International Dictionary, an acronym is a word from the initial letters of each of the successive parts of a compound term. But this rule has been violated recently, e.g. COPERNICUS for Carvedilol Prospective Randomized Cumulative Survival trial, SYMPHYTON for Sibrafiban versus aspirin to Yield Maximum Protection from ischemic Heart Events post acute coronary syndrome, VIGOUR for Virtual coordinating center for Global collaborative cardiovascular Research[2]. But, to reverse the sequence of letters to create a catchy acronym is rather stretching the limit. Could it be that these British authors were still dreaming about their lost empire when they conjured up this acronym?

Inventors of acronyms for cardio- logical trials like to play the game of one-upmanship nowadays. Acronymophilia[3] or acronymmania[4] is getting out of hand. From 1992 to 1998 there had been a tenfold increase in the number of acronyms of cardiologic trials[5]. This exponential growth[3] is bound to continue as long as investigators play the game of one-upmanship. My Plea is to Let Each Acronym be Spelled out Every time (PLEASE)[6].

T. O. CHENG
Professor of Medicine
The George Washington University
Medical Center,
Washington D.C., U.S.A.

References


Neurobiology of respiratory-pattern training in congestive heart failure

Bernard[1] finds that respiratory-pattern training in chronic heart failure results, at least, in better coordination of the respiratory muscles and of the diaphragm, and a greater facility to slow down the breathing rate, regardless of whether or not it permanently modifies the spontaneous breathing habit. This new approach is supported by profound effects on angina by consciously focusing attention on breathing and the intervening pauses, adaptation to stress manifested by slower, deeper breathing (contributing to a 6-fold reduction in mortality), and the role of a slower rate of living which contributes to a 5-fold increase in lifespans[2]. It is also supported by the following: the rate of speech hesitation pauses of 1 s or more, 4·79 ± 2·48 per min, 1·50 ± 0·33 s (Mean ± SD) correlates with the state of the circulation (angina/hypertension) and a sixfold incidence of clinical coronary artery disease in two groups of normal coronary-prone men followed prospectively for 10 years, P<0·05; the reduction of blood pressure is associated with longer, less recurrent pauses of about 2 s[3,4] which correlates with the feeling of rhythmicity; the microvascular response to the onset of neural activity is consistently delayed by several seconds; and short-term laboratory experience demonstrates that adult female speech production is sufficient to influence infant’s speech production occurring in the silent intervals between the adult’s vocalizations (average, 3·37 s). Even brief (1–5 s) spontaneous pauses in ongoing patterned behaviors are accompanied by an immediate reduction in serotonin neuronal activity to, or below, baseline levels, coordinating autonomic, motor, and sensory functions, and modulating dopamine which optimizes response organization and working memory, and regulates the microvasculature[5,6] implicated in slow coronary flow as a cause for anginal[5].

These findings suggest that respiratory-pattern training[1] may promote a primary rhythm in the central nervous system that entrains heart rate, blood pressure, and respiratory rate, maintains cognitive unity through a callosal window between prefrontal cortices[7,8], and orchestrates bimanual coordination through a callosally interconnected and distributed network of frontal and parietal cortical areas of which the supplementary motor area is a part[9]. Therefore, simple training to improve breathing in chronic heart failure[1] may enhance physical and psychological well-being, whether or not it increases survival[7].

Neurobiological features of this training in chronic heart failure, a multisystem syndrome[1], are demonstrated by the association of brain stem cardiovascular control, cardiovascular reactivity in challenging tasks, cortical silent periods, mood, subclinical impairment of lung airways, and vasospasm with dopaminergic activity promoted by plasmogen and endothelin which is activated by mental stress[2,3,8,9]. These findings prompt studying long-term efficacy.
and applicability of this training[1] by longitudinal investigation of decreased prolactin reactivity predictive of ambulatory ischemia[10], and electroencephalographic characterization of brain dopaminergic stimulation[11]. The fact that delay-dependent speeding of reaction time, indicating motor readiness, is abolished by depletion of dopamine[12], suggests evaluating the influence of awareness of training goals on spontaneous attitudes[1] by monitoring temporal features of expressive activity in spontaneous dialogues may be necessary[12].

E. H. FRIEDMAN
Cleveland, OH, U.S.A.

References