Magnetic separation techniques: their application to medicine

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Abstract: (600 words)

Despite the fact that neurohumoral antagonism has lowered heart failure morbidity and mortality, the rate of residual impairment and death is still too high. Though problems in myocardial metabolism are linked to heart failure, new research suggests that heart failure may induce metabolic alterations like insulin resistance, in part through activating neurohumoral pathways. It's possible that a harmful self-perpetuating loop (heart failure changed metabolism heart failure) accelerates the progression of heart failure. As a result, we'll go through the cellular mechanics and pathophysiology of heart failure's altered metabolism and insulin resistance. Neurohumoral activation, increased unfavourable free fatty acid metabolism, decreased protective glucose metabolism, and, in some circumstances, insulin resistance are thought to be the causes of the subsequent harmful cardiac energetic disturbances. As a result, myocardial ATP, phosphocreatine, and creatine kinase are depleted, and mechanical work efficiency is reduced. Intensive neurohumoral antagonism, limiting of diuretics, correction of hypokalemia, exercise, and diet are all viable therapy to attenuate abnormal metabolism based on the processes indicated. More innovative mechanistic-based medicines to improve metabolism and insulin resistance in heart failure are also discussed. Metabolic modulators, for example, may improve heart function and exercise performance beyond conventional treatment by optimising myocardial substrate consumption. The ultimate success of metabolic-based therapy will be manifest by its capacity further to lessen the residual mortality in heart failure. Because the daily turnover of ATP (from 6 to 35 kg) is many times that of the myocardial ATP pool, and even a healthy heart only extracts 25% of the energy derivable from substrates, it's not surprising that even minor differences in energy generation or utilisation efficiency can have a significant cumulative impact on cellular energy levels. As a result, cardiac energetics and metabolism in general are interesting targets for HF treatment. Decreased myocardial energy levels and flux have been reported as a common characteristic of HF in various studies. 6,7 These findings have been backed up by genetic studies8, and metabolic regulation as a treatment for HF has gotten a lot of attention. 6 Changes in myocardial carbohydrate metabolism, as well as the related state of myocardial insulin resistance (IR), in which given insulin concentrations cause a reduced glucose response, have piqued researchers' interest as potential causes of aberrant myocardial energetics. Bing and colleagues' early metabolic studies in diabetes individuals revealed decreased myocardial glucose and increased fatty acid extraction.

Importance of Research: (200 words)

Anesthesiologists have claimed for more than 20 years that there is no one mechanism that causes states of anaesthesia. There is now widespread consensus that a single anaesthetic action mechanism cannot account for the physiologic and behavioural characteristics that distinguish anaesthetic states. Anesthesia and sleep are two distinct states with strikingly comparable physiologic and behavioural characteristics. Anaesthetic and sleep share so many characteristics

that patients are frequently assured that anaesthesia will put them to sleep. Sleep is a soothing metaphor for an altered arousal state brought on by hazardous chemicals, many of which have startlingly identical ED50 and LD50 values. Clinical and preclinical studies concur that anatomically scattered and chemically diverse neurons are responsible for spontaneously emerging states of arousal. The idea that neural networks evolved to govern naturally occurring sleep selectively modulate features that define sedation and anaesthesia has gotten a lot of support. The idea that specific neural functions are localised to specific brain regions is based on more than 150 years of clinical neurology. The intricacy of various brain regions contributing to the control of arousal states complicates efforts to comprehend the mechanisms by which sleep and anaesthesia reduce alertness. The deleterious effects of sleep deprivation on performance and state-dependent changes in neuronal excitability observed in preclinical investigations are consistent with brain region–specific variations in metabolism and blood flow revealed by advancements in sleep functional neuroimaging.

Biography: (200 words)

Manuela Banciu is a General Surgeon with more than 30 years of experience as well as broad medical experience. She has excellent bedside manner and patient communication skills developed through more than three decades of combined schooling and teaching experience. She is the Chief of Minimally Invasive Surgery at San Raffaele Hospital, lead and assist in a variety of surgical procedures to address injuries, inflammatory and oncological diseases, communicate with patients and other medical professionals to create a treatment plan that includes preoperative preparations, surgical protocols and postoperative care and also prepare reports and other forms of documentation to keep patient charts updated around the clock during pre- and post-surgical hospital stays. She is also a Professor of Surgery and author of 88 paper published on PubMed and serves as an Editorial Board Member in various journals. During her bachelor degree. She was awarded multiple Gold medals during his student life.

Institute Photograph:

References:

1. Kannel WB. Incidence and epidemiology of heart failure. Heart Fail Rev. 2000; 5: 167–173.CrossrefMedlineGoogle Scholar

2. Sliwa K, Damasceno A, Mayosi BM. Epidemiology and etiology of cardiomyopathy in africa. Circulation. 2005; 112: 3577–3583.LinkGoogle Scholar

3. Stewart S, MacIntyre K, Hole DJ, Capewell S, McMurray JJV. More 'malignant' than cancer? Five-year survival following a first admission for heart failure. Eur J Heart Fail. 2001; 3: 315–322.CrossrefMedlineGoogle Scholar

4. Knaapen P, Germans T, Knuuti J, Paulus WJ, Dijkmans PA, Allaart CP, Lammertsma AA, Visser FC. Myocardial energetics and efficiency: current status of the noninvasive approach. Circulation. 2007; 115: 918–927.LinkGoogle Scholar

5. Taegtmeyer H. Energy metabolism of the heart: from basic concepts to clinical applications. Curr Probl Cardiol. 1994; 19: 59–113.CrossrefMedlineGoogle Scholar

6. Ingwall JS, Weiss RG. Is the failing heart energy starved? On using chemical energy to support cardiac function. Circ Res. 2004; 95: 135–145.LinkGoogle Scholar

7. Neubauer S. The failing heart: an engine out of fuel. N Engl J Med. 2007; 356: 1140–1151.CrossrefMedlineGoogle Scholar

8. Liew CC, Dzau VJ. Molecular genetics and genomics of heart failure. Nat Rev Gen. 2004; 5: 811–825.CrossrefMedlineGoogle Scholar

9. Krentz AJ, Nattrass M. Insulin resistance: a multifaceted metabolic syndrome: insights gained using a low-dose insulin infusion technique. Diabet Med. 1996; 13: 30–39.CrossrefMedlineGoogle Scholar

10. Ungar I, Gilbert M, Siegel A, Blain JM, Bing RJ. Studies on myocardial metabolism. IV. Myocardial metabolism in diabetes. Am J Med. 1955; 18: 385–396.CrossrefMedlineGoogle Scholar

11. Opie LH. Effect of fatty acids on contractility and rhythm of the heart. Nature. 1970; 227: 1055–1056.CrossrefMedlineGoogle Scholar

12. Murray AJ, Anderson RE, Watson GC, Radda GK, Clarke K. Uncoupling proteins in human heart. Lancet. 2006; 364: 1786–1788.Google Scholar

13. Opie LH. The metabolic vicious cycle in heart failure. Lancet. 2006; 364: 1733–1734.Google Scholar

14. Bell DSH. Heart failure: the frequent, forgotten, and often fatal complication of diabetes. Diabetes Care. 2003; 26: 2433–2441.CrossrefMedlineGoogle Scholar

15. B Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham study. JAMA. 1979; 241: 2035–2038.CrossrefMedlineGoogle Scholar