## **EDITORIAL**

## Is Enhanced External Counterpulsation (EECP) A- 'Last Resort' Treatment of Refractory Angina?

EECP is not a new invention. In fact, the basic theory & techniques of counterpulsation developed more than 50 years ago by researchers at Harvard University & Massachusetts General Hospital USA. In 1953, Kantrowitz and Kantrowitz1 described diastolic augmentation as a means of improving coronary blood flow. Birtwell et al did pioneering work toward the development of this technique and were first to apply this concept by developing the initial arterial counterpulsator in the U.S. Zheng et al were the first to report the benefits of external counterpulsation in the 1980s by using the first pneumatic counterpulsation device. Lawson et al at the State University of New York, Stony Brook, undertook a number of open label studies with the enhanced system, EECP, between 1989 and 1998 using both objective and subjective end points. 2-5,7 In 1999, Arora et al 6 reported results of the first double-blind randomized placebo-controlled multicenter trial (MUST-EECP). Since then, EECP therapy has emerged as a safe, highly beneficial, low cost, non-invasive treatment of refractory angina patients & now for heart failure patients as well.

Enhanced external counter pulsation, or EECP, has been used as a treatment for angina in China for two decades. The technique of EECP therapy consists of electrocardiogram-gated rapid sequential compression of the lower extremities taking place during diastole, followed by simultaneous decompression during systole. A typical treatment course consists of 35 outpatient treatments administered as 1 h per day over 7 weeks. 7 After that therapy can be continued 1-2 days per week as a maintenance dosing. The overall hemodynamic effect is to provide diastolic augmentation & thus increase coronary perfusion pressure; to unload systolic cardiac workload & therefore decrease myocardial oxygen demand; & to increase venous return & subsequently cardiac output. <sup>8,10</sup> This exciting new therapy is presently being explored as a treatment for refractory angina.

Recent evidence suggests that, EECP therapy may improve symptoms & decrease long-term morbidity via more than one mechanism including improvement in endothelial function, promotion of collateralization, enhancement of ventricular function, improvement in oxygen consumption (VO2), regression of atherosclerosis & peripheral training effects similar to exercise. Numerous clinical trials in the last 2 decades have shown EECP therapy to be safe & effective for patients with refractory angina with a clinical response rate averaging 70% to 80%, which is sustained up to 5 years. It is not only safe in patients with co-existing heart failure but also shown to improve quality of life & exercise capacity & to improve left ventricular function long-term. Interestingly EECP have been studied for various potential uses other than heart disease such as restless leg syndrome, sudden deafness, hepatorenal syndrome, erectile dysfunction.<sup>7,9</sup>

Throughout the world, EECP therapy has been studied for various potential uses other than heart disease. It's role in improving endothelial function might be beneficial in the treatment of patients with Cardiac Syndrome X with severely symptomatic coronary endothelial dysfunction in the absence of CAD with standard 35-h course of EECP therapy. However, it is important to realize that EECP therapy is an option for patients with angina refractory to medical treatment who are not candidates for interventional or surgical revascularization. The American Heart Association recommends it as a Class IIb (Level of Evidence:B) intervention for treatment of refractory angina pectoris (RAP), among other non-phrmacological approaches such as neurostimulation (Class IIb, Level of Evidence :B) & transmyocardial laser revascularization (Class IIa, Level of Evidence:A). The European Society of Cardiology views EECP therapy as an interesting modality available for treatment of RAP with more clinical trials needed to define its role in treating RAP. Enhanced external counterpulsation therapy is a valuable out-patient procedure providing acute & long-term relief of angina symptoms & improved quality of life among a group of patients with symptomatic ischemic heart disease with or without congestive HF. 7

## Prof. Md. Abu Siddique PhD

Professor of Cardiology Bangabandhu Sheikh Mujib Medical University Shahbag, Dhaka. E-mail: <u>drabusiddique@yahoo.com</u>

## **Referances:**

- Kantrowitz A, Kantrowitz A; Experimental augmentation of coronary flow by retardation of coronary artery pressure pulse. Surgery 1953; 34: 678-87.
- Lowson WE, Hui JC, Zheng ZS, et al; Improved exercise tolerance following enhanced external couterpulsation: cardiac or peripheral effect? Cardiology 1996; 87: 271-5
- Lowson WE, Hui JCK, Sorrof HS, et al; Efficacy of enhanced external counterpulsation in the treatment of angina pectoris. Am J Cardiol. 1992; 70: 859-62.
- Lowson WE, Hui JCK, Zheng ZS, et al;Can angiographic findings predict which coronary patients will benefit from enhanced external counterpulsation? Am J Cardiol. 1996; 77: 1107-9
- Lowson WE, Hui JCK, Guo T, et al; Prior revascularization increases the effectiveness of enhanced external counterpulsation. Clin Cardiol. 1998; 21: 841-4.

- Arora RR, Chou TM, Jain D, et al; The multicenter study of enhanced external counterpulsation (MUST-EECP): effect of EECP on exercise-induced myocardial ischemia and angina episodes. J Am Coll Cardiol. 1999; 33: 1833-40.
- Manchanda A, Soran O; Enhanced external counterpulsation and future directions. J Am Coll Cardiol. 2007;50(16) : 1523-31.
- Parmley WW, Chatterjee K; Enhanced external counterpulsation. Discussed during the "Cardiology Update, 1997" October 1997; 16-18.
- Bonetti PO, Barsness GW, Keelan PC et al; Enhanced external counterpulsation improves endothelial function in patients with symptomatic coronary artery disease. J Am Coll Cardiol. 2003; 41: 1761-8.
- Kim M C, Kini A, Sharma S K; Refractory angina pectoris mechanism and therapeutic options. J Am Coll Cardiol. 2002;39(6): 923-34