Pericardial and myocardial localization of antimyosin in a case of acute myocarditis. *J Nucl Med* 1994;35:469–470.
18. Calegaro JUM, de Carvalho AC, Campos ER, Medeiros M, Gomes E. Gallium-67 in rheumatic fever: preliminary report. *Arq Bars Cardiol* 1991;56: 487–492.
19. Kao CH, Hsieh KS, Wang YL, Chen CW, Wang SJ, Yeh SH. Tc99m HMPAO labeled white blood scanning for the detection of carditis in the differentiation of rheumatic fever and inactive rheumatic heart disease in children. *Nucl Med Comm* 1992;13,478–481.
20. Narula J, Chandrashekhar Y, Rahimtoola SH. The diagnosis of active rheumatic carditis: the echoes of change. *Circulation* 1999; in press.

Pneumatic External Counterpulsation: A New Noninvasive Method to Improve Organ Perfusion

Dierk Werner, MD, Manfred Schneider, MD, Mathias Weise, MD, Barbara Nonnast-Daniel, MD, and Werner G. Daniel, MD

Intra-aortic balloon counterpulsation (IABC) is the most commonly used cardiac assistance procedure for temporary support of the failing heart after acute myocardial infarction and cardiac surgery. IABC is proven to increase survival in cardiogenic shock unresponsive to pharmacologic treatment.[1] However, the complication rate ranges between 10% and 15%.[2] External counterpulsation as a noninvasive alternative to IABC was developed in the 1960s.[3] This technique operated by applying electrocardiographic-triggered diastolic pressure via water-filled cylinders to the vascular bed of the lower limbs. Despite initial promising results[4,5] this hydraulic external counterpulsation was less effective in comparison with IABC and could not be established.[6] In 1983, pneumatic external counterpulsation (PECP) was described[7] based on experiences with hydraulic external counterpulsation.[8] PECP applies sequential diastolic pressure to calves, thighs, and buttocks by means of 3 air-filled paired cuffs (Figure 1). This system is widely used in China and in some cardiologic centers in the Western World for treatment of patients with angina pectoris and cerebrovascular disease. At the Third Chinese National Congress on external counterpulsation, a significant reduction of angina pectoris was reported in 3,218 of 6,116 patients (53%), and a functional improvement was observed after PECP therapy in 76 of 100 patients with sudden deafness.[9] The American study MUST-EECP (Multicenter Study of Enhanced External Counterpulsation) demonstrated a decrease in anginal symptoms and nitroglycerin usage as well as a reduction in ischemic ST-segment depression after a treatment course of 35 hours (1 hour/day).[10] Lawson et al[11] demonstrated a long-term effect of this therapy in patients with angina pectoris over a period of 3 years. Despite these encouraging clinical results, measurements of hemodynamic changes induced by PECP are very rare.[12] The objective of this study was the estimation of hemodynamic effects of PECP.

From the Medical Clinic, Friedrich-Alexander-University, Erlangen; and Medical Clinic, University Hospital Carl Gustav Carus, Dresden, Germany. Dr. Daniel's address is: Herzzentrum der Universität, Medizinische Klinik II mit Poliklinik der Friedrich-Alexander Universität, Östliche Stadtmauerstr. 29, 91054 Erlangen, Germany. Manuscript received January 5, 1999; revised manuscript received and accepted May 25, 1999.

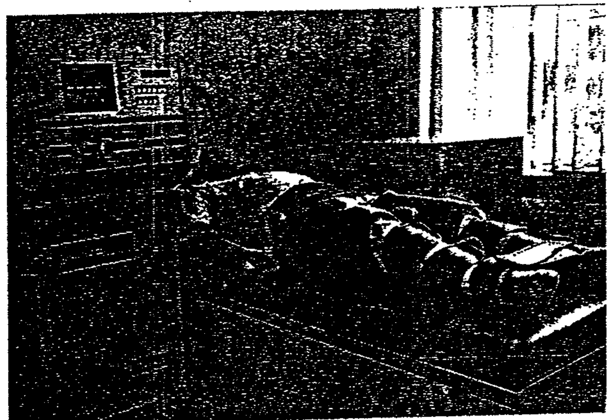

FIGURE 1. Pneumatic external counterpulsation device (EECP, Vasomedical)

TABLE I Duplex Scanning and Echocardiographic Findings During Pneumatic External Counterpulsation*

Artery/Stroke Volume	Increase of Flow Volume/Stroke Volume (%)	p Value
Cuff Pressure: 200 mm Hg, Using Cuffs for Calves and Thighs		
Common carotid artery	19 ± 6	<0.01
Internal carotid artery	19 ± 5	<0.01
Right vertebral artery	12 ± 4	<0.05
Left vertebral artery	11 ± 5	<0.05
Hepatic artery	25 ± 13	<0.01
Right renal artery	21 ± 9	<0.01
Abdominal aorta	88 ± 48	<0.01
Internal iliac artery	144 ± 29	<0.05
Stroke volume	12 ± 9	<0.05
Left coronary main stem	18 ± 6	NS
Cuff Pressure: 300 mm Hg, Using Cuffs for Calves, Thighs, and Buttocks		
Internal carotid artery	26 ± 6	<0.01
Left coronary main stem	42 ± 2	NS

*Sixteen volunteers, transesophageal echo in 3 patients with coronary artery disease.

• • •

For this study, the 2 commercially available PECP devices were used (Vasomedical Inc., Westbury, New York; and Cardiomedics Inc., Irvine, California).

0002-9149/99/$–see front matter
PII S0002-9149(99)00477-4

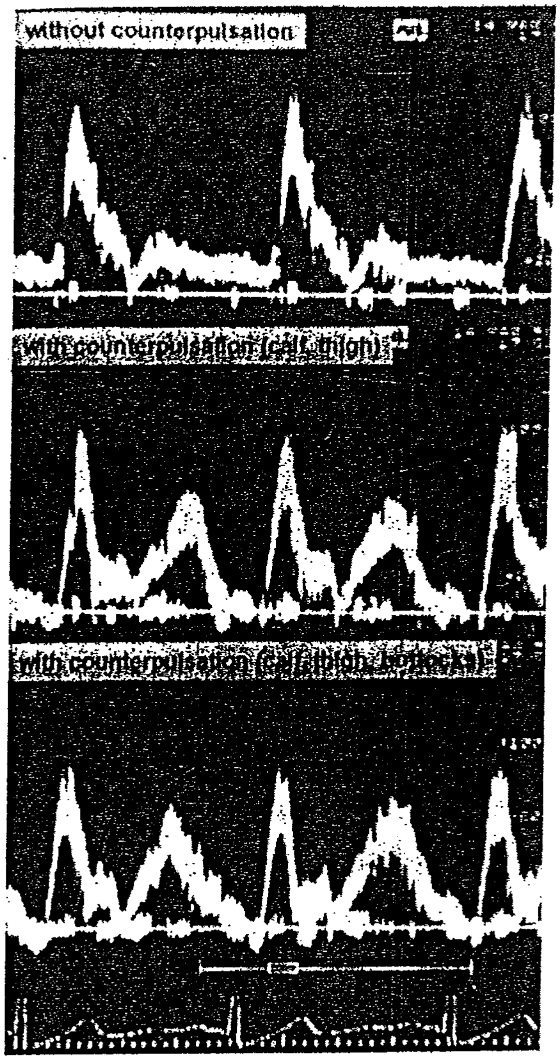

FIGURE 2. Flow pattern in common carotid artery. *(A)* Before PECP. *(B)* PECP using calves and thighs. *(C)* PECP using calves, thighs, and buttocks.

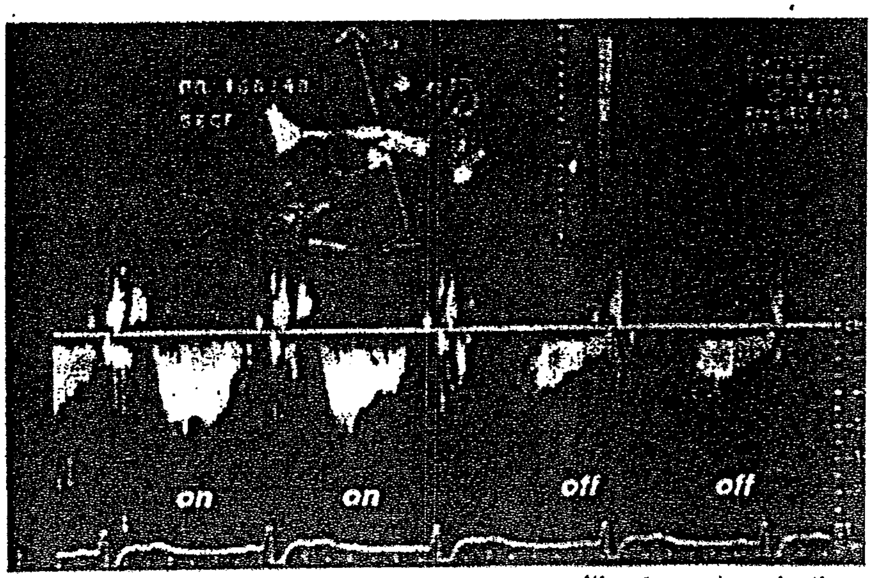

FIGURE 3. Flow pattern in left coronary main stem during and after PECP.

PECP was used in 16 healthy volunteers (ages 28 ± 4 years) using at first a pressure of 200 mm Hg to calves and thighs only, and then a pressure of 300 mm Hg to calves, thighs, and buttocks. Changes in flow volume in carotid, vertebral, hepatic, renal, and internal iliac arteries were measured by duplexsonography and stroke volume by Doppler echocardiography before and at the end of 1 hour of PECP. In 6 patients undergoing transesophageal echocardiography for clinical reasons, flow measurement in the left coronary main stem was performed before and during PECP.

During PECP, using cuffs for thighs and calves and 200 mm Hg pressure flow, all investigated arteries showed a characteristic diastolic augmentation (Figure 2). Flow volume in the common carotid artery increased by 19 ± 6% ($p < 0.01$), in the internal carotid artery by 19 ± 5%, in the right and left vertebral arteries by 12 ± 4% and 11 ± 5% ($p < 0.05$), respectively, in the renal artery by 21 ± 9% ($p < 0.01$), in the hepatic artery by 25 ± 13% ($p < 0.01$), and in the internal iliac artery by 144 ± 29% ($p < 0.05$). Further augmentation was reached when the buttocks belt was used in addition; the cuff pressure of 300 mm Hg flow volume increased by 26 ± 6% in the internal carotid artery. There were no changes in the diameter of the studied arteries during PECP; only the abdominal aorta showed a significant diastolic increase measured by sonography (mean change 1.0 ± 0.8 mm). Stroke volume of the volunteers increased from 67 ± 22 ml before PECP to 75 ± 27 ml (12%; $p < 0.05$) at the end of 1 hour of use. PECP was well tolerated by all volunteers; 3 persons described fatigue of limb muscles on the next day and 1 complained headache immediately after PECP.

In 3 patients, PECP led to an increase of flow volume in the left main coronary stem by 18 ± 6% using cuffs for calves and thighs and a pressure of 200 mm Hg (Figure 3). Using an additional buttocks belt and a cuff pressure of 300 mm Hg, flow volume increased by 42 ± 2% in the 3 other patients.

• • •

Our data demonstrate that PECP leads to a significant increase in perfusion of brain, liver, kidneys, and myocardium, the latter being mainly perfused during diastole. The augmentation of flow volume is accompanied by an increase in mean arterial pressure by 15%[13] and a downregulation of the vasoconstrictive hormones endothelin and renin from 75% to 80% of baseline levels in healthy volunteers and patients with coronary artery disease.[14] These hemodynamic findings help to interpret the reported clinical improvements in patients with coronary artery disease or with cerebrovascular diseases (e.g., transient ischemic attacks, ischemic central retinopathy or sudden deafness). Compared with IABC the advantages of external counterpulsation are simpler application, possibility of repeated use, and a very low risk of complications.

In conclusion, PECP provides an increase in perfusion of all internal organs and could be a new and noninvasive therapeutic option for patients with different diseases caused by disturbed organ perfusion.

1. Waksman R, Weiss AT, Gotsman MS, Hasing, Y. Intra-aortic balloon counterpulsation improves survival in cardiogenic shock complicating acute myocardial infarction. *Eur Heart J* 1993;14:71–74.
2. Eltchanioff H, Alexios PD, Whitlow PL. Complications associated with percutaneous placement and use of intraaortic balloon counterpulsation. *Am J Cardiol* 1993;71:328–333.
3. Ruiz U, Soroff HS, Birtwell WC, Many M, Giron F, Deterling RA. Assisted circulation by external pressure variation. *J Cardiovasc Surg* 1969;15:187–197.
4. Soroff HS, Cloutier TC, Birtwell WC, Begley LA, Messer JV. External counterpulsation—management of cardiogenic shock after myocardial infarction. *JAMA* 1974;229:1441–1450.
5. Amsterdam EA, Banas J, Criley JM, Loeb HS, Mueller H, Willerson JT, Mason DT. Clinical assessment of external pressure circulatory assistance in acute myocardial infarction. *Am J Cardiol* 1980;45:349–355.
6. Kuhn LA. External pressure circulatory assistance: no light on the shadow. *Am J Cardiol* 1980;46:1069–1072.
7. Zheng ZS, Li TM, Kambic H, Chen GH, Yu LQ, Cai SR, Zhan CY, Chen YC, Wo SX, Chen GW, Ma H, Chen PJ, Huang BJ, Nose Y. Sequential external counterpulsation (SECP) in China. *Trans Am Soc Artif Intern Organs* 1983;29:599–603.
8. Soroff HS, Hui J, Giron F. Current status of external counterpulsation. *Critical Care Clincis* 1986;2:277–295.
9. Xu YY, Hu DY, Zheng ZS. Review article: external counterpulsation. *Chin Med J* 1990;103:768–771.
10. Arora RR, Chou TM, Jain D, Nesto RW, Fleishman B, Crawford, L., McKiernan T. Results of the Multicenter Study of Enhanced External Counterpulsation (MUST-EECP): EECP reduces anginal episodes and exercise induced myocardial ischemia (abstr). *Circulation* 1997;95 (suppl. I):466.
11. Lawson WE, Hui JCK, Zheng ZS, Oster Z, Katz JP, Diggs P, Burger L, Cohn CD, Soroff HS, Cohn PF. Three-year sustained benefit from enhanced external counterpulsation in chronic angina pectoris. *Am J Cardiol* 1995;75:840–841.
12. Applebaum RM, Kasliwal R, Tunick PA, Konecky N, Katz ES, Trehan N, Kronzon I. Sequential external counterpulsation increases cerebral and renal blood flow. *Am Heart J* 1997;133:611–615.
13. Werner D, Wonka F, Krinke HE, Klinghammer L, Daniel WG. Blood pressure changes during external counterpulsation (abstr). *Eur Heart J* 1998;19:655.
14. Garlichs CD, Zhang H, Werner D, John A, Traegner P, Daniel WG. Reduction of serum endothelin-1 levels by pneumatic external counterpulsation (abstr). *Can J Cardiol* 1998;14(suppl. F):87.