Vasodilator Therapy in Chronic Congestive Heart Failure

By

ALI MUHAMMAD*, MBBS, FRCP, FCPS, FACC, FCCP.

In the natural history of chronic congestive heart failure, there comes a stage when routine and standard anticongestive treatment does not restore reasonable haemodynamic compensation. Such patients display evidence of inappropriate sympathetic overdrive evidenced by increase in heart rate, elevated peripheral vascular resistance due to arteriolar constriction, and enhanced venoconstriction causing increase in venous return and filling pressure (Fig. 1). Although

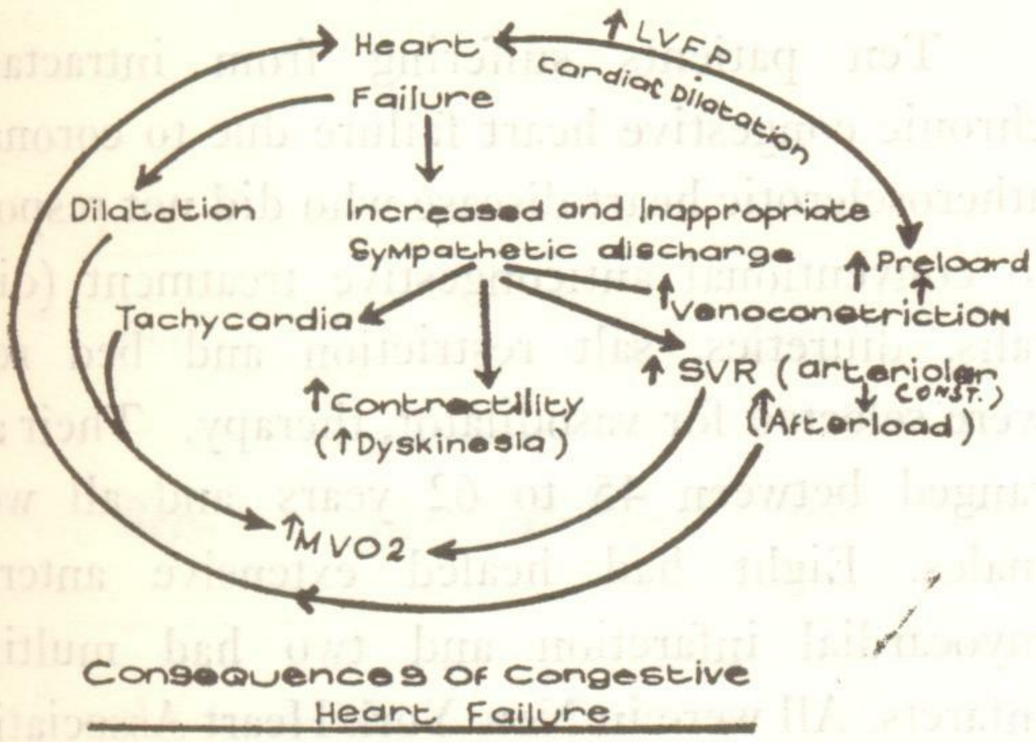


Fig. 1

sympathetic positive inotropism may increase contractility, the latter along with increase in preload and afterload enhances myocardial oxygen demand. Besides, elevated afterload offers greater impedance to left ventricular ejection so that stroke volume and cardiac output are further compromised (Fig. 2). Sympathetic overdrive in congestive heart failure working on the plateau of left ventricular function curve would prove the last straw on the camel's back because it would increase both the preload and the afterload. Recently several studies have reported the salutary effect, of vasodilator therapy in the treatment of both acute and chronic congestive heart failure (Franciosa et al, 1974; Guiha et al, 1974; Chatterjee et al, 1976; Massie and Chatterjee, 1979).

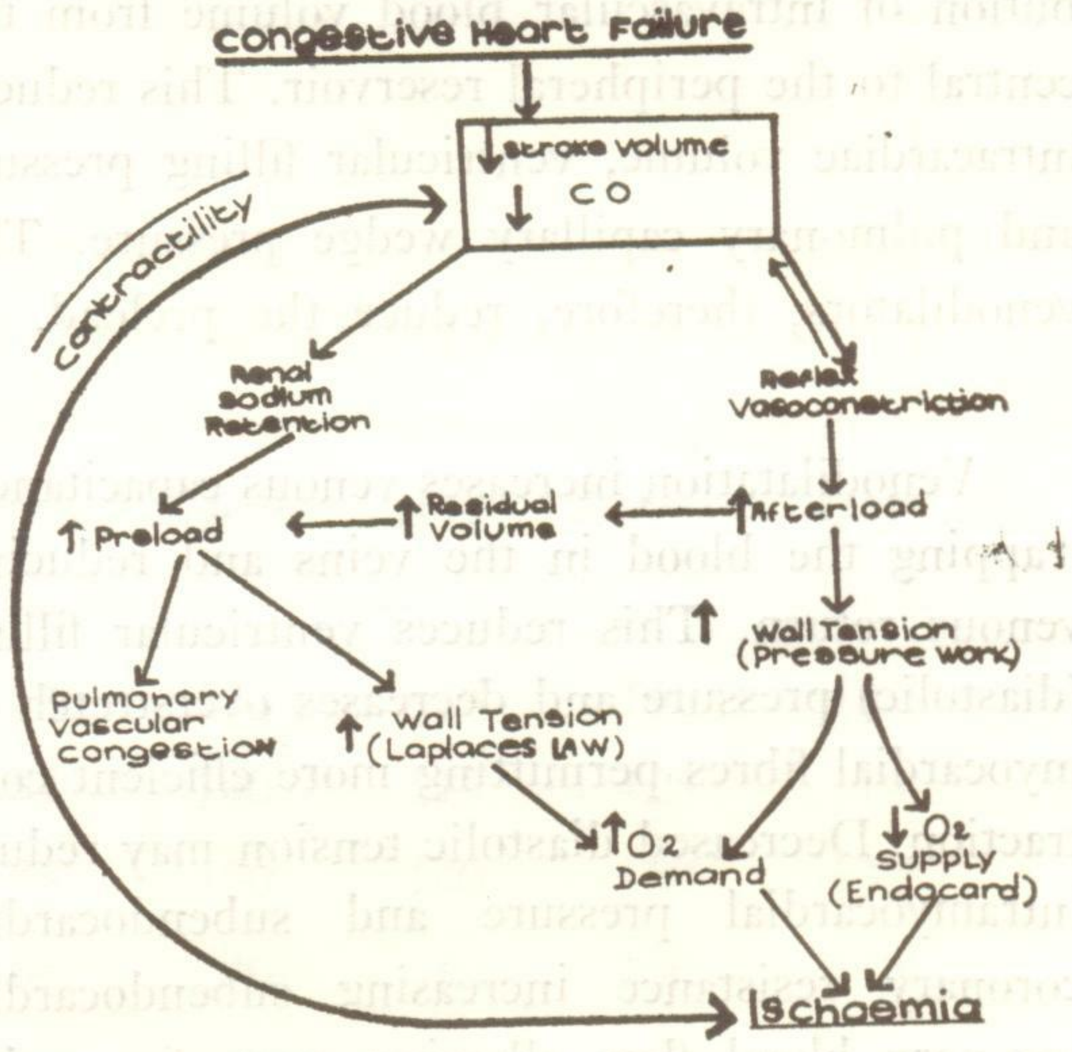


Fig. 2

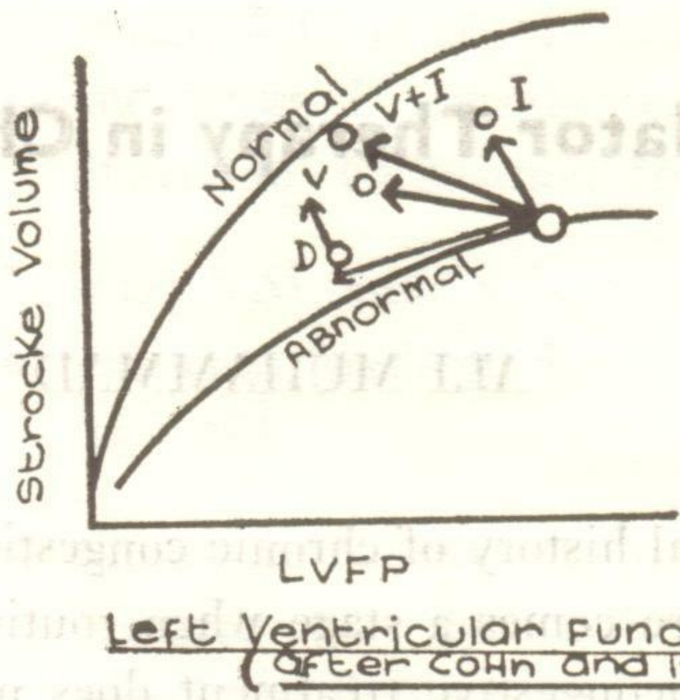
Normally, vasodilators, by reducing peripheral vascular resistance, occasion fall in blood pressure and reflex tachycardia. This response

*Consultant Cardiologist, Hyderabad.

(Based on the paper read at the Fourth Biennial All Pakistan Cardiology Congress held at Karachi in December, 1979).

is blunted in congestive heart failure when reduction in peripheral vascular resistance and transient fall of blood pressure is accompanied by increase in stroke volume without an increase in heart rate. Arterial blood pressure is, therefore, maintained near the control level by increase in stroke volume. Transient fall in arterial blood pressure would reduce afterload (systolic wall tension) allowing enhanced rate and extent of muscle fibre shortening. The reduction in afterload would, therefore result in more effective systolic ejection and increased stroke volume, reduced end-systolic volume and decreased myocardial systolic wall tension. Reduction in systolic wall tension may improve coronary perfusion during systole. Venodilators, by causing increased venous capacitance, occasion redistribution of intravascular blood volume from the central to the peripheral reservoir. This reduces intracardiac volume, ventricular filling pressure and pulmonary capillary wedge pressure. The venodilators, therefore, reduce the preload.

Venodilatation increases venous capacitance, trapping the blood in the veins and reducing venous return. This reduces ventricular filling (diastolic) pressure and decreases overstretch of myocardial fibres permitting more efficient contraction. Decreased diastolic tension may reduce intramyocardial pressure and subendocardial coronary resistance increasing subendocardial coronary blood flow allowing more favourable distribution of coronary flow. Vasodilator therapy, therefore, facilitates increased endocardial blood flow to ischaemic myocardium. The reduction of preload and afterload, therefore, set in motion a series of salutary effects which may improve the efficiency of heart shifting the ventricular function curve up and to the left (Fig. 3).



Left Ventricular Function Curve
(After Cohn and Frankosa)

Fig 3: I:Inotropic therapy
V:Vasodilator therapy
D:Diuretic therapy
V+I:Vasodilator and inotropic therapy

Method:

Ten patients suffering from intractable chronic congestive heart failure due to coronary atherosclerotic heart disease who did not respond to conventional anticongestive treatment (digitalis, diuretics, salt restriction and bed rest) were selected for vasodilator, therapy. Their age ranged between 45 to 62 years and all were males. Eight had healed extensive anterior myocardial infarction and two had multiple infarcts. All were in New York Heart Association Functional Class IV. All were in sinus rhythm and showed signs of biventricular failure (pulmonary congestion, raised jugular venous pressure, hepatomegaly and gravitational oedema). Ventricular gallop rhythm (left) was heard in all the patients; mitral pansystolic murmur grade 3/6 was heard in 7 patients. All showed basal crepitations in the lungs. X-ray chest showed cardiac enlargement and pulmonary congestion.

The following parameters were evaluated daily:

1. Subjective symptoms: dyspnoea, fatigue, weakness, palpitation.
2. Physical sign:pulse, blood pressure, jugular venous pulse and pressure, heart sounds and murmurs, pulmonary congestion, weight and urinary output.
3. X-ray chest and E.C.G. were taken weekly for three weeks and monthly thereafter.

Routine anticongestive treatment was continued. Isosorbide dinitrate was initially given in doses of 10 mg. six hourly by mouth and was later increased to 20 mg. six hourly depending on the response. Prazocin was given initially in doses of 1 mg. six hourly and later increased to 3 to 5 mg. six hourly depending on the response. The patients were followed for six weeks to 3 months.

Results:

Seven patients improved. Three patients showed no response. Those who improved showed decrease in heart rate, fall in jugular venous pressure and increase in diuresis reduction in weight and decrease in pulmonary congestion and heart size. Blood pressure decreased but remained above 100 mm.Hg. systolic in all.

Table 1 summarises the effects of vasodilator therapy.

TABLE I

Functional Class		*JVP CM*		*HR/Min*		*BP (Syst.)*		*WT (lb)*	
Before	*After*	*Before*	*After*	*Before*	*After*	*Before*	*After*	*Before*	*After*
IV	II	6	3	110	84	130	125	145	131
III	II	5	3	100	82	140	115	125	115
IV	II	7	3	104	84	150	120	150	132
IV	II	6	3	110	96	140	110	130	113
IV	IV	8	6	136	138	120	110	160	164
IV	II	7	4	126	94	146	110	120	105
IV	IV	8	8	120	134	140	110	170	180
IV	IV	7	8	132	140	155	115	135	150
IV	III	8	4	130	126	120	120	180	165
IV	II	7	3	112	92	138	124	135	120

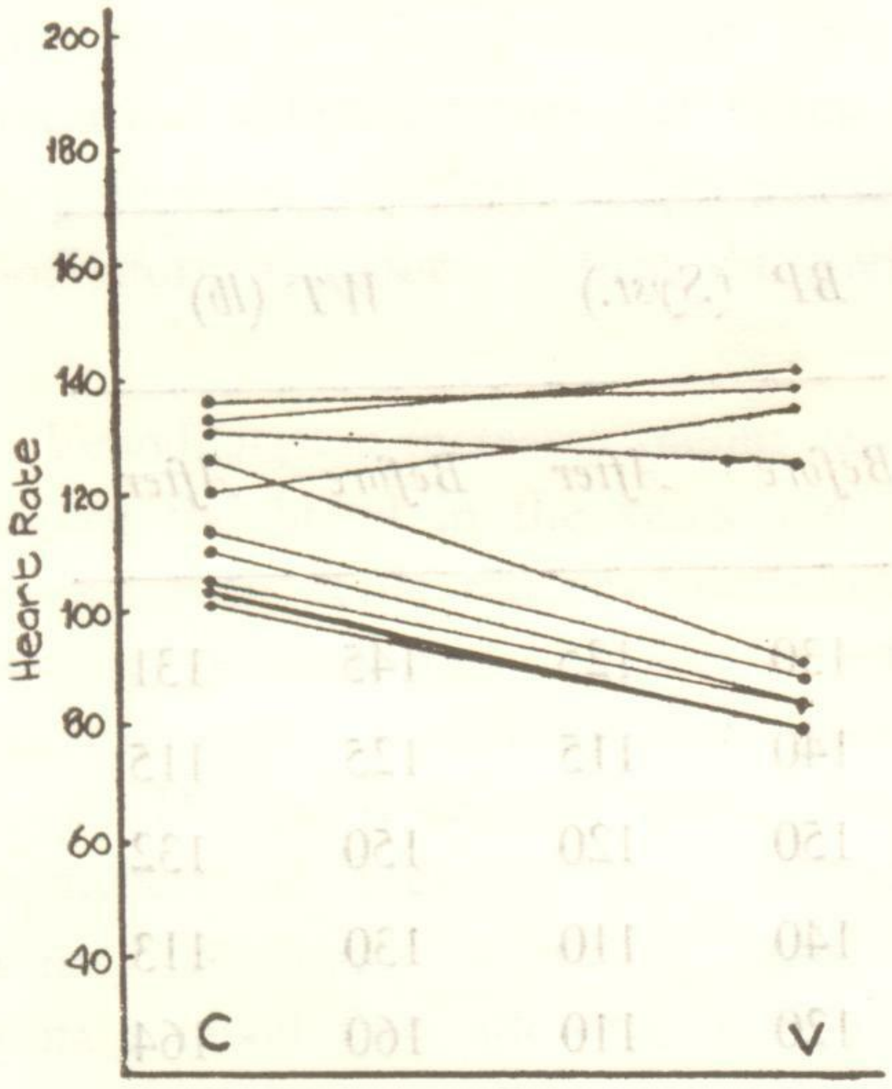
7 patients obtained better functional class.

6 patients improved from functional class IV to Class II and 1 from functional class IV to Class III, 3 patients showed no improvement.

Jugular venous pressure was reduced in all who responded to treatment. In non-responders, it remained unchanged in 1, increased in 1 and slightly decreased in 1.

Heart rate decreased in all who showed response. It increased in the three non-responders (Fig. 4).

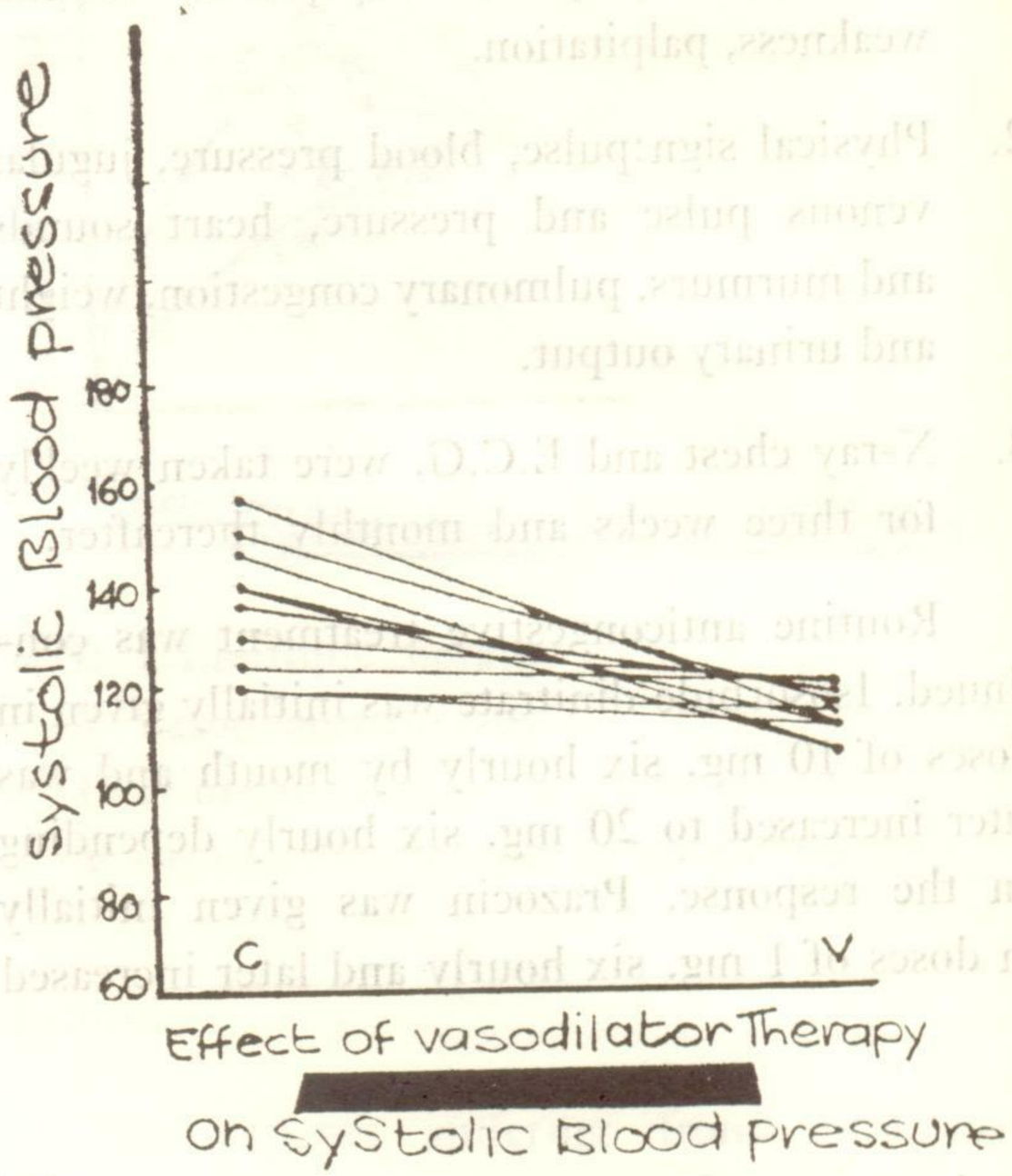
Fig. 4



Effect of vasodilator Therapy on Heart Rate

Blood pressure (systolic) decreased in all (Fig. 5). Weight decreased in all who improved and increased in the three who showed no response.

Fig.5



Effect of vasodilator Therapy on Systolic Blood pressure

Ventricular gallop was heard in all but was muffled in those who responded indicating fall in filling pressure.

Pansystolic murmur decreased in intensity in 4 out of 5 patients who responded and was unchanged in non-responders.

Pulmonary congestion decreased in all who responded.

Heart size decreased in 4 out of 7 patients who responded.

Discussion:

Vasodilator therapy shows salutary effects in chronic congestive heart failure by reducing the afterload and/or preload. Reduction of afterload by dilatation of resistance vessels (arterioles) reduces impedance to left ventricular ejection promoting increase in stroke volume,

elevation of ejection fraction, reduction of left ventricular end-diastolic volume and filling pressure and decrease in left ventricular end-systolic volume. All these parameters reflect enhanced left ventricular performance. Dilatation of venous capacitance vessels reduces preload causing diminution of filling pressure. Isosorbide dinitrate acts by causing venodilatation trapping blood in the venous capacitance vessels so that the venous return and filling pressure is reduced. Prazocin owing to its postsynoptic alpha adrenoreceptor blocking action has a strong effect both on the systemic venous capacitance vessels and arteriolar resistance vessels. The important haemodynamic effects of isosorbide dinitrate and prazocin are summarized in Table 2.

Table II

Effects Of Isosorbide Dinitrate And Prazocin In Heart Failure

Drug	Venous Tone	Arteriolar Resistance	Heart Rate	Arterial B.P.	Cadiac Output	lvfp
Isosorbide Dinitrate	↓↓	↔	↔	↔	↔	↓↓
Prazocin	↓or↔	↓	↔	↔	↑	↓or↔

In this study of ten patients, combined use of isosorbide dinitrate and prazocin was undertaken to potentiate the effect of prazocin on the venous capacitance vessels by isosbide dinitrate. This pharamcologic manipulation of preload and afterload conferred beneficial effect on all the clinically measurable parameters of ventricular performance. It is suggested that vasodilator therapy may be used with benefit in patients suffering from chronic congestive heart failure who fail to respond to conventional anticongestive treatment.

Summary:

Ten patients suffering from chronic congestive heart failure resistant to routine anticongestive therapy were treated with isosorbide dinitrate and prazocin. It is concluded that vasodilator therapy with these drugs has salutary potential.

References:

Chatterjee, K., Parmely, W.W. et al; Oral hydralazine therapy for chronic refractory heart failure: Circulation 54:879, 1976.

Chatterjee, K., Parmley, W.W., et al; The role of Vasodilator therapy in heart failure: Prog. in Cardiov. Dis; 19:301. 1977.

Franciosa, J.A.; Mikulic, E. Cohn, J.N. et al; Haemodynamic effect of orally administered isosorbide dinitrate in patients with congestive heart failure; Circulation; 50:1020, 1974.

Guiha, N.H.; Cohn, J.N. et al; Treatment of refractory heart failure with infusion of nitroprusside; New Eng. J. Med; 291:587, 1974.

Massie, B.M. and Chatterjee, K; Vasodilator therapy of Pump failure; Med. Clin. N. Amer; 63:25, 1979.

—:o:—