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Captopril in pulmonary hypertension

Sir,

In their article in the September 1982 issue, Rich *et al.*[1] report their experience after administration of captopril in four patients with primary pulmonary hypertension. The authors found no significant effect of captopril, either at rest or with exercise, on the cardiac output, pulmonary arterial pressure, or pulmonary vascular resistance. In spite of these results, however, we consider that captopril may be useful in the treatment of some cases of pulmonary hypertension, especially in those with pulmonary hypertension secondary to chronic obstructive pulmonary disease, and our preliminary results seem to indicate this.

We have given captopril (50 mg orally) to three patients with pulmonary hypertension secondary to severe chronic obstructive pulmonary disease and recorded the haemodynamic responses before and one hour after the administration of the drug. Twenty four hours later these same patients received nifedipine (20 mg sublingually) and the haemodynamic response to this drug was also noted. The results obtained are shown in the Table.

In cases 1 and 2, captopril improved cardiac output and decreased the mean pulmonary arterial pressure, as well as the pulmonary and systemic vascular resistance. In the third patient the effects of captopril were deleterious. Nifedipine produced a substantial haemodynamic deterioration in cases 1 and 3 and slightly improved pulmonary vascular resistance, mean pulmonary pressure, and cardiac output in case 2.

Table *Haemodynamic response to captopril and nifedipine*

	HR	*SAP*	*SVR*	*PAP*	$\dot{Q}_T$	*PVR*
Case 1:						
Before captopril	88	90	25·0	40	4·19	9·0
After captopril	95	85	14·5	37	6·75	5·2
Before nifedipine	80	91	20	42	4·57	9·2
After nifedipine	80	85	27·3	38	3·23	11·8
Case 2:						
Before captopril	100	73	32·2	22	2·85	7·0
After captopril	110	70	20·9	16	3·20	3·43
Before nifedipine	100	74	32	22	2·95	6·8
After nifedipine	125	72	22	20	3·08	5·7
Case 3:						
Before captopril	98	76	17·1	39	5·07	7·1
After captopril	95	60	21·8	32	3·3	8·7
Before nifedipine	96	73	13·6	40	5·37	7·44
After nifedipine	94	58	16·3	37	3·56	11·2

HR, heart rate; SAP, mean systemic arterial pressure; SVR, systemic vascular resistance; PAP, mean pulmonary arterial pressure; Q, cardiac output; PVR, pulmonary vascular resistance.

Several vasodilating agents, distinguished by their different mechanism of action, have been used in the treatment of pulmonary hypertension. The response to these drugs has been shown to be variable: pulmonary haemodynamics have improved, have deteriorated, or have remained unchanged after the administration of vasodilating agents.[2-4]

Alternatively, it is known that the factors implicated in the genesis of pulmonary hypertension are multiple; thus, it could be suggested that every individual case may show improvement or deterioration when on vasodilating drugs, and our preliminary results, shown in the Table, indicate this. Pulmonary haemodynamics might be improved by the drug that would act through the concrete pathogenic mechanism responsible in every individual case.

We do not agree with Rich *et al.*[1] that no single drug has been identified which is clearly superior in lowering pulmonary vascular resistance. Effort is still needed before several pathogenic types of pulmonary hypertension are identified and the selective treatment for each case is recognised.

We consider that captopril is a serious candidate in the treatment of pulmonary hypertension. Its known effects[1] on prostaglandins and angiotension II, both of them implicated in the genesis of pulmonary hypertension, as well as clinical experience in the treatment of systemic hypertension and congestive cardiac failure, attest to its possibilities. Further studies, however, are needed in order that the action of captopril in pulmonary hypertension can be clarified.

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