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Static exercise in untreated systemic hypertension

D. J. Ewing, J. B. Irving,[1] F. Kerr, and Brian J. Kirby
From Department of Medicine, University of Edinburgh, Royal Infirmary, Edinburgh

The circulatory responses to sustained handgrip at 30 per cent of a maximum voluntary contraction of the forearm muscles (MVC) were examined in untreated hypertensive patients. Arterial pressure increased in all patients, often attaining very high levels. Two main patterns of circulatory response were observed. In group 1 (8 patients) there was an increase in cardiac index and heart rate, with little or no change in systemic vascular resistance. These patients conformed to the pattern of response previously observed in normal subjects. Group 2 (6 patients) responded by an increase of systemic vascular resistance, a smaller rise in heart rate, and little or no change in cardiac index. All patients in group 2 had either electrocardiographic or radiographic evidence of left ventricular hypertrophy, suggesting that the difference in response was due to impaired left ventricular function. In 4 patients further measurements were made at 10, 20, and 50 per cent maximum voluntary contraction demonstrating an incremental increase in response up to 30 per cent and a more rapid rise of arterial pressure at 50 per cent maximum voluntary contraction.

The very high pressures attained during static exercise are potentially deleterious to patients with an already high blood pressure as they could precipitate cardiac failure or cerebral haemorrhage.

The circulatory responses to static exercise differ considerably from the responses to dynamic exercise. Static, or isometric exercise, in which a muscular contraction is maintained to fatigue, in normal subjects results in a considerable increase in mean arterial pressure with a comparatively modest increase in heart rate. Dynamic exercise, for example, walking, running, and cycling, has the opposite effect, namely little change in mean arterial pressure and a great increase in heart rate (Bruce *et al.*, 1968). There is a much greater rise in mean arterial pressure during dynamic exercise in hypertensive patients when compared to normal subjects (Taylor, Donald, and Bishop, 1957; Sannerstedt, 1966) but relatively little is known of any comparable differences in circulatory response between hypertensive and normal subjects during static exercise (Staunton, 1967; Hoel, Lorentsen, and Lund-Larsen, 1970). Since activities involving static exercise (e.g. handgrip, lifting and supporting weights with arms or legs) are used many times a day an exaggerated pressor response could be a factor in maintaining a high arterial pressure during daily activity in hypertensive patients. We investigated whether the circulatory responses of untreated hypertensive patients differed from normal subjects.

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The circulatory responses are produced in normal subjects when 30 per cent or more of the maximum voluntary contraction is maintained in a muscle or group of muscles (Lind *et al.*, 1964). The response is independent of the muscle mass involved as long as a fatiguing load greater than 20 per cent of a maximum voluntary contraction is maintained. For experimental purposes handgrip at 30 per cent maximum voluntary contraction is convenient as it has been widely investigated in normal subjects and it can be easily reproduced (Clarke, Hellon, and Lind, 1958). Most subjects can maintain 30 per cent maximum voluntary contraction for a few minutes thereby allowing circulatory measurements to be made.

Patients

Fifteen patients (9 men and 6 women) aged between 36 and 59 years (mean age = 45) were investigated (Table 1). Casual diastolic pressure readings by sphygmomanometry were consistently found above 90 mmHg, except in Case 10 who was regarded as a case of 'labile' or 'borderline' hypertension because casual diastolic pressures by sphygmomanometry were recorded above and below 90 mmHg on different occasions. After full investigation it was concluded that 13 had essential hypertension and 2 renal hypertension (Cases 2 and 13). One patient (Case 4) stopped methyldopa 6 weeks before study but the remainder had never received any

TABLE 1 *Clinical details*

Case No.	*Age (yr)*	*Sex*	*Body surface area (m^2)*	*Diagnosis*	*Electro-cardiogram*	*CTR %*	*Remarks*
1	52	F	1·59		N	44	
2	43	F	1·71	Medullary sponge kidney	LV1	51	
3	41	F	1·73		N	51	
4	38	F	1·67		LV1	46	Methyldopa stopped 6 weeks
5	39	F	1·55		LV1	47	
6	48	M	2·14		LV3	45	
7	36	M	2·04		LV2	48	
8	52	M	1·95		N	47	
9	51	M	2·28		N	51	
10	41	M	2·20		N	45	
11	59	F	1·83		LV1	53	
12	42	M	1·77		LV1	43	
13	47	M	1·85	Renal artery stenosis	LV2	43	
14	39	M	1·66		LV1	38	
15	44	M	1·93		LV1	52	

form of hypotensive treatment. There was no clinical evidence of cardiac failure in any patient though Case 7 had a transient episode of atrial fibrillation in the past. Electrocardiographic evidence of left ventricular hypertrophy was found in 10, and in 5 the cardiothoracic ratio measured from a standard 6 ft posteroanterior chest *x*-ray exceeded 50 per cent.

After explaining the nature and purpose of the investigation and obtaining their consent, patients were familiarized with the handgrip dynamometer. Maximum voluntary contraction was determined from the highest of three brief maximum grips.

Methods

Patients were studied in the supine position without sedation. Under local anaesthesia a fine nylon catheter (internal diameter, 1·0 mm; length, 30 cm) was inserted percutaneously by the Seldinger technique approximately 20 cm into the left brachial artery and a second nylon catheter (internal diameter, 1·0 mm; length, 55 cm) into an adjacent antecubital vein and advanced to the subclavian vein. The force of the handgrip was measured with a hand dynamometer in which the original indicating mechanism was replaced by a miniature linear displacement transducer whose electrical output was

TABLE 2 *Individual results at 30 per cent*

Case No.	*Cardiac index (l./min per m^2)*			*Stroke index (ml/m^2)*			*Heart rate (per min)*		
	Control	*Exercise**	*Diff.*	*Control*	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*
1	2·57	5·84	3·27	43	79	36	60	74	14
2	2·56	2·69	0·13	38	35	−3	68	76	8
3	3·44	4·49	1·05	54	59	5	64	76	12
4	3·15	3·62	0·47	41	43	2	76	84	8
5	3·37	4·33	0·96	53	42	−11	64	104	40
6	4·29	4·20	−0·09	56	47	−9	76	90	14
7	1·84	2·67	0·83	33	29	−4	56	92	36
8	2·20	2·68	0·48	34	26	−8	64	104	40
9	2·03	2·22	0·19	34	25	−9	60	88	28
10	3·06	3·05	−0·01	42	35	−7	72	88	16
11	4·36	4·45	0·09	52	46	−6	83	96	13
12	3·03	4·21	1·18	47	53	+6	64	80	16
13	4·12	3·77	−0·35	51	50	−1	80	76	−4
14	2·45	2·87	0·42	32	29	−3	76	100	24
15	—	—	—	—	—	—	72	104	32
Mean	3·03	3·65		44	43		69	89	
SD	0·82	1·00		8·6	14·8		8·1	11·0	
P	<0·02			NS			<0·001		

* Sustained handgrip at 30 per cent maximum voluntary contraction. NS = Not significant.

measured on a voltmeter scale calibrated from 0 to 50 kg. A 50 kg thrust produced a linear displacement of 5 mm. After a period of 15 minutes rest after insertion of the catheters, measurements were made of heart rate, arterial pressure, and cardiac output at approximately 2-minute intervals during (1) a control period, (2) a period of sustained handgrip, and (3) a recovery period. Patients were instructed to maintain 30 per cent of a maximum voluntary contraction for as long as possible usually between 3½ and 4 minutes, though Case 4 could maintain 30 per cent maximum voluntary contraction for no more than 2 minutes. Five patients (Cases 6, 7, 8, 9, and 13) repeated the procedure after periods of rest at 10, 20, and 50 per cent maximum voluntary contraction, respectively. Patients were instructed to count quietly during the handgrip to prevent a Valsalva manoeuvre.

The intra-arterial pressure was measured continuously, except during dye curves, with a Statham P23 DG pressure transducer at a reference level 5 cm below the sternal angle, calibrated to the range of pressures in the patient under study. The frequency response of the catheter manometer system was linear to 14 Hz. Systolic and diastolic pressures were averaged over 10-second periods and mean pressures were obtained by electrical integration. The systolic pressure in Case 14 exceeded 350 mm during 30 per cent grip and was not recorded. The cardiac index was measured by the rapid injection of known amounts of indocyanine green dye into the subclavian venous catheter by a pneumatic injection apparatus with simultaneous sampling of arterial blood through a modified Waters XC 302 cuvette and densitometer by a constant speed Harvard withdrawal pump. Calibration was carried out at the end of the study by drawing samples of 3 known concentrations of dye in the patients' blood through the cuvette. The dye curves in Case 15 were technically unsatisfactory and were therefore discarded. Heart rate was counted from the continuously recorded electrocardiogram. Arterial pressure, electrocardiogram, and indicator dilution curves were recorded onto ultraviolet sensitive paper using a Honeywell recorder (type 1185 mk 2). Systemic resistance was calculated from

$$\frac{\text{mean arterial pressure (mmHg)}_2 \times 1332 \times 60}{\text{cardiac index (ml/min/m}^2\text{)}}$$

dynes sec cm^{-5} m^2.

Standard statistical methods were used to calculate means and standard deviations. Student's paired and unpaired 't' test was used to test statistical significance.

Results

a) **Effect of 30 per cent maximum voluntary contraction** (Table 2)

All 15 patients were studied at rest and immediately before release of 30 per cent maximum voluntary contraction. Resting cardiac index was within normal limits in 11 and low in 3 (Cases 7, 8, and 9), none of whom had clinical signs of failure, though Case 7 had atrial fibrillation in the past. These patients also had a low resting stroke index. Resting heart rate and stroke index in the remainder were within normal limits. Systolic, diastolic, and mean arterial pressure were raised at the time of study in all patients consistent with the diagnosis except in Case 10 who was considered a 'labile' hypertensive.

maximum voluntary contraction

Systolic arterial pressure (mmHg)			*Mean arterial pressure (mmHg)*			*Diastolic arterial pressure (mmHg)*			*Systemic vascular resistance (dynes sec cm^{-5} m^2)*	
Control	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*	*Control*	*Exercise*
165	211	46	124	143	19	95	109	14	3856	1957
243	252	9	161	184	23	111	126	15	5026	5467
200	232	32	139	152	13	91	101	10	3229	2705
190	210	20	136	148	12	96	111	15	3450	3267
256	282	26	172	204	32	125	151	26	4079	3765
215	307	92	137	197	60	97	142	45	2552	3749
221	291	70	167	227	60	130	165	35	7254	6795
190	218	28	133	161	28	104	127	23	4831	4801
205	248	43	134	184	50	101	135	34	5275	6624
116	135	19	87	96	9	73	87	14	2272	2515
224	232	8	161	163	2	112	128	16	2951	2927
228	278	50	179	215	36	153	179	26	4721	4081
213	257	44	160	172	12	111	133	22	3104	3646
258	—	—	177	179	2	126	153	27	5774	4984
208	239	31	169	182	13	127	154	27	—	—
209	242		149	174		110	133		4170	4092
35·8	43·0		24·9	32·4		19·7	24·9		1390	1476
	<0.01			<0.001			<0.001		NS	

His resting intra-arterial diastolic pressure varied between 73 and 114 mmHg on the day of investigation. The systemic vascular resistance was normal or high, with particularly high values in Cases 7 and 9 who had the lowest cardiac indices.

Arterial pressure and heart rate immediately increased when the patient gripped and the response preceded either fatigue or discomfort. The pattern of response is illustrated in Fig. 1. The change in arterial pressure was statistically significant ($P < 0.001$), with the greatest rise in systolic (mean rise, 37 mm) and lesser increases in diastolic (23 mm) and mean arterial pressure (25 mm) (Fig. 2). Concomitantly heart rate increased significantly from 69 ± 8 to 89 ± 11 ($P < 0.001$). Overall there was a significant increase in cardiac index from 3.03 ± 0.82 to 3.65 ± 1.00 l./min per m^2 ($P < 0.02$) but no significant change in stroke index. Systemic vascular resistance did not change significantly during 30 per cent maximum voluntary contraction. There was no relation between the magnitude of response and the resting values for cardiac index, heart rate, systolic, diastolic, or mean pressures.

The interrelations between cardiac index and mean arterial pressure were analysed in greater detail and are illustrated in Fig. 3. Two major patterns of circulatory response emerge. In 8 patients (Cases 1, 3, 4, 5, 7, 8, 12, and 14) designated group 1, the rise in arterial pressure was the result of a rise in cardiac index, with little change or a fall in systemic vascular resistance conforming to the pattern of response observed in normal subjects. The remaining 5 in whom cardiac index was measured (Cases 2, 6, 9, 10, and 13) designated group 2, had an equally conspicuous rise in arterial pressure but little change in cardiac index. In this group the increase in arterial pressure was due to an increase in systemic vascular resistance. There was no statistically significant difference in resting cardiac index, arterial pressure, and systemic vascular resistance between groups 1 and 2 despite the different circulatory responses during handgrip (Table 3). Groups 1 and 2 were

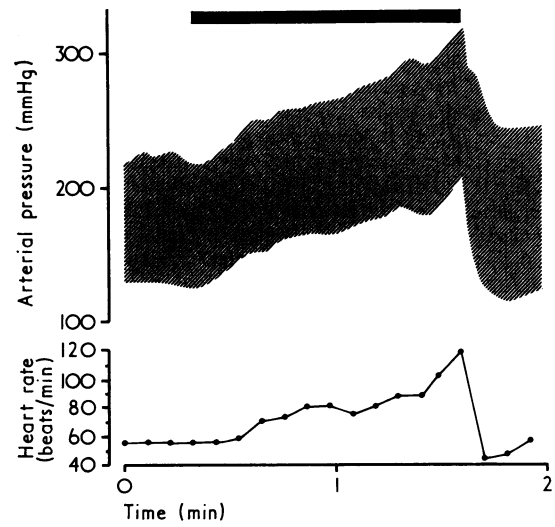

FIG. 1 *Intra-arterial pressure traced from a continuous intra-arterial pressure recording in Case 7 during 50 per cent maximum voluntary contraction. The black bar indicates the duration of handgrip.*

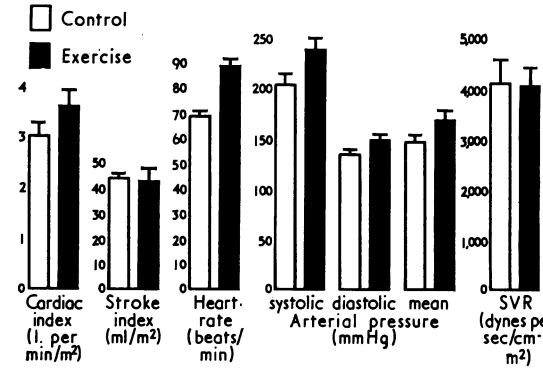

FIG. 2 *The circulatory effect of 30 per cent of maximum voluntary contraction. The vertical bars represent the mean values and the SE of the mean.*

TABLE 3 *Comparison of groups 1 and 2 at rest and*

	Cardiac index (l./min/m²)			*Stroke index (ml/m²)*			*Heart rate (per min)*		
	Group 1	*Group 2*	*P*	*Group 1*	*Group 2*	*P*	*Group 1*	*Group 2*	*P*
Control	M2·76	3·40	NS	42	45	NS	65	73	NS
	SD0·58	0·99		8·8	8·8		7·1	7·7	
Handgrip	M3·84	3·40	NS	45	40	NS	89	88	NS
	SD1·10	0·88		18·1	9·6		12·4	10·1	
P	<0·02	NS		NS	<0·01		<0·01	<0·02	

M = mean; SD, standard deviation.
Note: P values in vertical column relate to differences between groups 1 and 2, and P values in the bottom line to differences between control and handgrip.

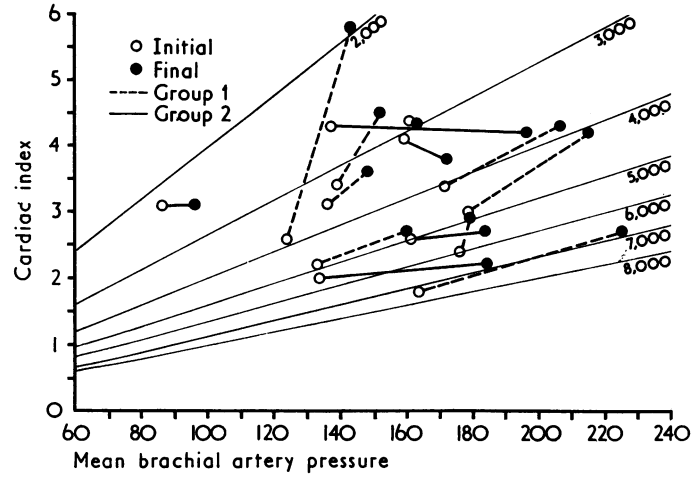

FIG. 3 *The relation between cardiac index and brachial artery pressure at rest and during sustained handgrip at 30 per cent maximum voluntary contraction. The isopleths represent systemic vascular resistance.*

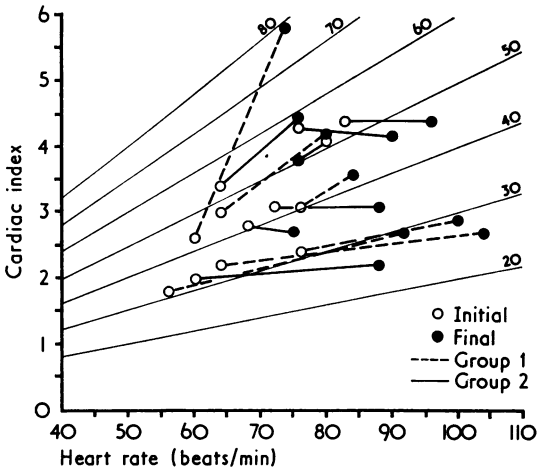

FIG. 4 *The relation between cardiac index and heart rate during sustained handgrip at 30 per cent maximum voluntary contraction. The isopleths represent stroke index.*

further differentiated by a smaller rise in heart rate in group 2 compared with group 1. Paradoxically stroke index was greater in group 1 patients despite the higher heart rate (Fig. 4).

In summary, group 1 patients correspond to the pattern of cardiovascular response observed in normal subjects, namely a heart rate dependent increase in cardiac index, with a consequent rise in arterial pressure. Group 2 patients increased arterial pressure by the same amount by an increase in systemic vascular resistance; they had no change in cardiac index and a smaller change in heart rate. All of the 6 patients in this group 2 had either electrocardiographic evidence of left ventricular hypertrophy or radiographic evidence of cardiac enlargement (Table 1) though none had clinical evidence of cardiac failure.

b) **Comparative effects of 10, 20, 30, and 50 per cent maximum voluntary contraction** (Table 4)

In 5 patients, the effect of handgrip at 10, 20, and 50 per cent maximum voluntary contraction was

during handgrip at 30 per cent maximum voluntary contraction

Systolic pressure (mmHg)			*Mean pressure (mmHg)*			*Diastolic pressure (mmHg)*			*Systemic vascular resistance (dynes sec cm^{-5}m^2)*		
Group 1	*Group 2*	*P*	*Group 1*	*Group 2*	*P*	*Group 1*	*Group 2*	*P*	*Group 1*	*Group 2*	*P*
213	203	NS	153	144	NS	115	105	NS	4649	3530	NS
33·1	40·6		22·5	28·5		21·9	16·9		1336	1291	
246	239		179	168		137	129		4044	4155	
36·2	51·7		32·8	33·6		28·9	20·9		1505	1577	
0·01	0·02		0·01	0·05		0·001	0·01		0·05	0·05	

examined in addition to 30 per cent maximum voluntary contraction. At all levels of handgrip there was a rise in heart rate though arterial pressure did not rise significantly with 10 per cent maximum voluntary contraction. In some patients even the non-fatiguing load of 10 per cent maximum voluntary contraction provoked a pressor response and the response became more obvious with incremental increases in percentage maximum voluntary contraction. Though 50 per cent maximum voluntary contraction could be maintained only for short periods of time, the rate of increase in arterial pressure was so rapid that it often approached or even exceeded the levels attained by several minutes handgrip at 30 per cent maximum voluntary contraction. Arterial pressure and heart rate were still rising when fatigue made the patient release his grip. However, the responses to 10 and 20 per cent maximum voluntary contraction and the immediate response on starting handgrip indicate that fatigue was not the cause of the cardiovascular responses.

Discussion

Both normal and hypertensive subjects respond to sustained handgrip by an increase in arterial pressure and heart rate, but some hypertensive patients differ in the way they achieve the increase in arterial pressure and in the magnitude of the heart rate response. In normal subjects arterial pressure increases due to an increase in cardiac index, with little or no change in systemic vascular resistance (Lind *et al.*, 1964). Eight hypertensive patients in the present study responded in this way but the

TABLE 4 *Effects of increasing percentage*

Handgrip	*Case No.*	*Cardiac index (l./min/m²)*			*Stroke index (ml/m²)*			*Heart rate (per min)*		
		Control	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*
	6	4·00	3·79	−0·21	55	49	−6	72	78	6
	7	2·26	1·86	−0·4	40	31	−9	56	60	4
	8	2·10	2·16	0·06	29	30	1	72	72	0
10 per cent	9	2·32	2·39	0·07	39	40	1	60	60	0
	13	3·16	3·87	0·71	48	54	6	66	72	+6
	Mean	2·77	2·81		42	41		65	68	
	SD	0·08	0·95		9·8	10·7		7·2	8·0	
	P	NS			NS			<0·1		
	6	3·68	4·01	0·33	54	50	−4	68	80	12
	7	1·89	2·53	0·64	29	29	0	64	88	+24
	8	2·30	2·31	0·01	32	29	−3	72	80	8
20 per cent	9	2·14	2·18	0·04	38	30	−8	56	72	16
	13	3·88	4·00	0·12	51	50	−1	76	80	4
	Mean	2·78	3·01		41	38		67	80	
	SD	0·93	0·92		11·2	11·3		7·7	5·7	
	P	NS			<0·1			<0·05		
	6	4·29	4·20	−0·09	56	47	−9	76	90	14
	7	1·84	2·67	0·83	33	29	−4	56	92	36
	8	2·20	2·68	0·48	34	26	−8	64	104	40
30 per cent	9	2·03	2·22	0·19	34	25	−9	60	88	28
	13	4·12	3·77	−0·35	51	50	−1	80	76	−4
	Mean	2·90	3·11		42	35		67	90	
	SD	1·20	0·84		11·0	12·1		10·4	10·0	
	P	NS			<0·02			<0·05		
	6	3·23	3·65	0·42	45	41	−4	72	90	18
	7	2·39	2·30	−0·09	40	22	−18	60	104	44
	8	2·22	2·14	−0·08	34	22	−12	66	96	30
50 per cent	9	2·07	2·75	0·68	34	33	−1	60	84	24
	13	4·00	4·85	0·85	63	55	−8	63	88	25
	Mean	2·78	3·14		43	35		64	92	
	SD	0·82	1·12		12·0	13·9		5·0	7·8	
	P	NS			<0·05			<0·01		

remaining patients increased arterial pressure by an increase in systemic vascular resistance with little change in cardiac index. There was no difference in the magnitude of increase in arterial pressure in the two groups though there was a smaller increment in heart rate in those who responded to handgrip by a rise in systemic vascular resistance. Previous studies by Staunton (1967) and Hoel *et al.* (1970) stated that the circulatory responses to handgrip of hypertensive patients did not differ from normal subjects. However, on detailed examination of their results one of Staunton's (1967) 4 patients and 2 of Hoel *et al.*'s (1970) 9 patients responded to handgrip by an increase in systemic vascular resistance. Furthermore, the hypertension was less obvious in those studies; 4 of the 13 patients had a diastolic pressure below 90 mmHg. Thus, in hypertensive patients arterial pressure increases in response to sustained handgrip, but a proportion differs from normal subjects in that the increase is brought about by an increase in systemic vascular resistance rather than by an increase in cardiac index.

All the patients who responded by an increase in systemic vascular resistance (group 2) had electrocardiographic or radiographic evidence of left ventricular hypertrophy. Recently patients with coronary artery or rheumatic heart disease affecting the left ventricle were shown to respond to static exercise by an increase in arterial pressure due to an increase in systemic vascular resistance with little change in cardiac index (Helfant, deVilla, and Meister, 1971; Kivowitz *et al.*, 1971). Thus, the response in those hypertensive patients with evidence of left ventricular hypertrophy (group 2) probably reflects impair-

maximum voluntary contraction in 5 patients

Systolic arterial pressure (mmHg)			*Mean arterial pressure (mmHg)*			*Diastolic arterial pressure (mmHg)*			*Systemic vascular resistance (dynes sec cm^{-5} m^2)*		
Control	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*	*Control*	*Exercise*	*Diff.*
242	244	2	168	177	9	111	123	12	3356	3752	376
219	249	30	169	182	13	126	146	20	5976	7820	1844
190	179	−11	133	143	10	100	104	4	5062	5291	129
206	222	16	142	153	11	102	114	12	4892	5116	224
202	219	17	145	146	1	110	108	−2	3667	3015	−652
212	223		151	160		110	119		4590	4995	
19·8	27·7		16·2	18·1		10·3	16·7		1073	1844	
NS			<0·02			<0·1			NS		
225	253	28	161	176	15	119	138	19	3496	3508	12
229	279	50	173	207	34	129	165	36	7315	6539	−776
180	197	17	134	145	11	103	110	7	4656	5017	361
202	238	36	144	170	26	106	132	26	5378	6232	854
240	246	6	167	173	6	126	128	2	3440	3456	16
215	243		156	174		117	135		4857	4950	
24·1	29·8		16·3	22·1		11·7	19·9		1598	1456	
<0·05			<0·05			<0·05			NS		
215	307	92	137	197	60	97	142	45	2552	3749	1197
222	292	70	168	227	59	131	166	35	7254	6795	−459
190	218	28	133	161	28	104	127	23	4831	4801	−30
205	248	43	134	184	50	101	135	34	5275	6624	1349
213	257	44	160	172	12	111	133	22	3104	3646	542
209	264		146	188		109	141		4603	5123	
12·2	35·5		16·4	25·5		13·4	15·2		1870	1518	
<0·01			<0·02			<0·01					
236	348	112	177	237	60	115	182	67	4379	5189	810
226	275	49	166	223	57	134	168	34	5551	7749	2198
183	197	14	131	140	9	100	118	18	4716	5228	512
222	226	4	156	166	10	114	134	20	6023	4824	−1199
202	261	59	147	174	27	102	145	43	2937	2867	−70
214	216		155	188		113	149		4721	5172	
21·2	57·2		17·6	40·7		13·6	25·7		1192	1737	
<0·1			<0·05			<0·02					

ment of left ventricular function and corresponds to the less conspicuous increase in cardiac index during dynamic exercise in patients with left ventricular hypertrophy (Taylor *et al.*, 1957).

Although the exact nervous pathways mediating the circulatory responses described are unknown, it is thought that the reflex originates in contracting skeletal muscle (Donald *et al.*, 1967; Freyschuss, 1970). The increase in systemic vascular resistance we observed suggests sympathetically mediated vasoconstriction rather than release of vagal inhibition. Systemic vascular resistance was often high in the control period due to increased sympathetic tone or reduced vascular compliance; therefore even a small increase in cardiac index results in a conspicuous increase in arterial pressure.

It was debated in the past whether the increase in arterial pressure was greater in hypertensive patients than in normal subjects in response to pressor stimuli (Brod, 1960). Neither the pressor responses to handgrip at 30 per cent maximum voluntary contraction, nor the responses at 10 and 20 per cent maximum voluntary contraction were greater than those observed in normal subjects, thus reinforcing the view that hypertensive patients do not have an exaggerated response to pressor stimuli. Both 10 and 20 per cent maximum voluntary contraction elicited a response in arterial pressure and heart rate before the onset of fatigue, indicating that the circulatory responses are not the result of pain or discomfort. Most untrained subjects are unable to maintain 50 per cent maximum voluntary contraction for more than a minute, but the rise in arterial pressure and heart rate during that period often exceeded the values observed after several minutes at lower work loads.

A handgrip of 30 per cent maximum voluntary contraction is roughly equivalent to carrying 10 kg in the hand (Lind and McNicol, 1968), which is a level of work attained transiently many times a day. The percentage change in arterial pressure is no greater in hypertensive patients, but the higher initial pressures result in them attaining very high levels of arterial pressure with only a modest workload. For example Case 6, with a resting arterial pressure of 215/97 mmHg, increased it to 307/142 mmHg at 30 per cent maximum voluntary contraction and 348/182 mmHg at 50 per cent. It is not known whether transient increases of pressure of this magnitude can rupture blood vessels in the brain or disrupt atheromatous plaques in the coronary vessels, but prolonged or repetitive activities involving static exercise will increase the time-averaged arterial pressure for the day, which is known to be related to cerebrovascular disease (Sokolow *et al.*, 1966). Furthermore, in cardiac failure left ventricular end-diastolic pressure increases during static exercise (Helfant *et al.*, 1971; Kivowitz *et al.*, 1971) and hypertensive patients could attain levels high enough to produce pulmonary oedema. Work or recreation involving frequent or prolonged episodes of static exercise is potentially harmful in hypertension, and should be avoided. Clearly, it is impossible for the hypertensive patient to avoid static exercise entirely as even 10 per cent maximum voluntary contraction provokes some response. Further information is needed on the therapeutic modification of the circulatory response to static exercise. Beta-adrenergic blockade does not appear to reduce the increase in arterial pressure (MacDonald *et al.*, 1966). What little is known of the effect of α-adrenergic blockade on this response suggests it too is ineffective (Staunton, 1967; Freyschuss, 1970).

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Requests for reprints to Dr. Brian J. Kirby, Department of Medicine, University of Edinburgh, Royal Infirmary, Edinburgh EH3 9YW.