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CASE REPORT

Cardiovascular and hormonal responses to electroconvulsive therapy

Modification of an exaggerated response in an hypertensive patient by β-receptor blockade

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Summary

A patient suffering from severe psychiatric depression underwent a course of electroconvulsive therapy. A marked rise in systemic arterial pressure, heart rate and plasma catecholamines in response to electroconvulsive therapy was attenuated by β-receptor blockade using propranolol. The significance and mechanism of this attenuation are discussed.

Key words

Blood pressure; hypertension.
Brain; electroconvulsive therapy.

A 74-year-old woman with severe depression was scheduled for a course of electroconvulsive therapy (ECT). The patient had mild to moderate hypertension (arterial blood pressure in the 140/90–170/100 mmHg range), and was taking chlorthalidone 50 mg daily and oral potassium supplements. Although there was no indication of ischaemic heart disease, carotid and femoral artery bruits indicated that some degree of peripheral vascular disease was present. The electrocardiogram demonstrated occasional premature atrial beats, but no ST segment or T-wave changes. The patient was given oxygen for 5 minutes before induction of anaesthesia with methohexitone, 50 mg, intravenously. Muscle relaxation was achieved with suxamethonium 30 mg, intravenously. The electrocardiogram was monitored throughout the procedure. Arterial blood pressure and heart rate were measured after atropine premedication but before induction of anaesthesia (control), 30 seconds after methohexitone administration, 30 seconds after suxamethonium administration, and 15, 30, 60, 90, 120, 180, 300 and 600 seconds after the application of ECT (130 volts for 0·75 seconds) (Table 1). Venous blood was taken for the measurement of plasma catecholamines (adrenaline, noradrenaline and dopamine), and plasma renin activity, before induction of anaesthesia (control) and 60 and 600 seconds after the application of ECT (Table 2). The tables summarise the cardiovascular and hormonal responses, after different ECT, with and without β-adrenergic receptor blockade. β-blockade was achieved with propranolol, in 0·5 mg increments, given intravenously before induction of anaes-

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Table 1. Haemodynamic response with and without β-receptor blockade

	β-Blockade (propranolol 3 mg intravenously)			No β-blockade		
Control	Blood pressure (mmHg) 140/70	Heart rate (beats/min) 98	RPP 13,720	Blood pressure (mmHg) 130/80	Heart rate (beats/min) 88	RPP 11,400
30 Seconds after methohexitone 50 mg	140/70	90	12,600	110/80	90	8,800
30 Seconds after suxamethonium 50 mg	140/70	90	12,600	150/90	96	14,400
Seconds after ECT						
15 seconds	145/75	96	13,920	200/110	120	24,000
30 seconds	150/80	100	15,000	240/130	140	33,600
60 seconds	160/80	100	16,000	250/140	140	35,000
90 seconds	160/80	100	16,000	210/130	130	27,300
120 seconds	150/75	95	14,250	200/110	120	24,000
180 seconds	150/70	95	14,250	180/100	100	18,000
300 seconds	140/70	90	12,600	150/90	96	14,400
600 seconds	140/70	88	9,800	140/90	92	12,600

RPP = Rate Pressure Product (Systolic arterial pressure × heart rate).

thesia, a total of 3 mg reducing the resting heart rate by 10 beats/minute. Cardiovascular and hormonal control values were in the absence of β-receptor blockade.

During ECT, in the absence of β-receptor blockade, the rate pressure product (RPP = systolic arterial pressure × heart rate) reached 35 000 and there was electrocardiographic evidence of myocardial ischaemia, as evidenced by S–T segment depression, which persisted for approximately 180 seconds, the S–T segments returning to the iso-electric line when the rate pressure product (RPP) had returned to less than 20 000. This was associated with an approximately fifteen-fold increase in plasma adrenaline and approximately three-fold increase in plasma noradrenaline. After β-receptor blockade the peak RPP reached 16 000 associated with an increase in plasma adrenaline of approximately 7·5-fold whilst plasma noradrenaline increased only slightly.

Adrenaline, noradrenaline and dopamine levels were determined by the radio-enzymatic method of Peuler & Johnson.[1] Plasma renin activity was determined by radio-immunoassay, using a commercially available kit (RIA-New England Nuclear, Medical Diagnostics Division, North Billerica, Massachusetts 01862, USA).

Discussion

Cardiovascular complications are the main cause of mortality during ECT modified with barbiturates and muscle relaxants (drug modified ECT).[2] Twenty years ago, Griswold[3] demonstrated that ECT stimulated the sympathetic nervous system causing a rise in plasma catecholamines that could be attenuated by intravenous barbiturates. Subsequent experiments in animals[4] demonstrated that high spinal analgesia, in which the sympathetic outflow was blocked, also inhibited the increase in catecholamines as well as the increase in arterial blood pressure and heart rate. In these animal experiments, a dose–response relationship was demonstrated between the duration and strength of the current applied and the autonomic stimulation and cardiovascular response. Another study, in humans, using 120 V lasting 0·6–0·8 seconds, demonstrated a rise in mean arterial pressure of 40 mmHg after drug-modified ECT, associated with an approximately three-fold increase in plasma adrenaline and noradrenaline.[5] However, a rise of 100 mmHg in systolic arterial pressure was observed in one patient in this study, which was associated with a four-fold increase in plasma adrenaline and two-

Table 2. Hormonal response, with and without β-receptor blockade

	Adrenaline (pg/ml)	Noradrenaline (pg/ml)	Dopamine (pg/ml)	Plasma renin activity (ng/ml/hour)	Adrenaline (pg/ml)	Noradrenaline (pg/ml)	Dopamine (pg/ml)	Plasma renin activity (ng/ml/hour)
Normal values	30–100	150–250	0–100	0·6–4·0	30–100	150–250	0–100	0·6–4·0
Control	32·5	563·0	45·0	0·42	24·0	424·0	16·0	0·35
	β-blockade (propranolol 3 mg intravenously)				No β-blockade			
60 seconds after ECT	242·1	845·0	27·0	0·26	350·0	1255·0	26·0	0·17
600 seconds after ECT	21·0	682·0	45·0	0·27	25·0	880·0	13·0	0·29

Normal values for plasma renin activity are in the rested supine subject.

fold increase in plasma noradrenaline. The blood for these catecholamine estimations was drawn 30–60 seconds after ECT. In an early study using unmodified ECT of 5 seconds duration, the increase in plasma adrenaline was less than ten-fold and approximately two-fold for plasma noradrenaline.[3]

In the case described, methohexitone and suxamethonium were used to modify ECT. Blood pressure rose from a pre-induction value of 130/80 to 250/140 mmHg and heart rate increased from 88 to 140 beats/minute. This represents an increase in RPP from 11 400 to 35 000. In common with previous reports[4,5] the maximal increases in arterial blood pressure and pulse rate occurred approximately 60 seconds after ECT and were returning toward control values after 10 minutes. The maximal changes in blood pressure and heart rate corresponded to approximately a fifteen-fold increase in plasma adrenaline and a 3-fold increase in plasma noradrenaline. Although the plasma adrenaline level approached the control value 10 minutes after ECT the noradrenaline level remained approximately twice that of control. These increases are considerably greater than reported in previous studies.[3,5] Current methods of measuring plasma catecholamines, using the radio-immunoassay technique such as that of Peuler & Johnson[1] are more accurate than earlier chemical methods of measurement, such as the method of Anton & Sayre[6] used by Gravenstein *et al.*[3] This may account for the discrepancy in the cardiovascular responses and catecholamine levels reported by the earlier investigators. It must be emphasised, however, that this is a single case report of a patient in whom a particularly striking increase in blood pressure and heart rate occurred in response to ECT. Experiments in man[7] and animals[4] have demonstrated, however, that at least a five-fold increase in circulating catecholamines is required to produce a significant cardiovascular response. The fifteen-fold increase in plasma adrenaline demonstrated in this report would certainly be sufficient to increase blood pressure and heart rate in this patient.

After β-adrenergic receptor blockade, using propranolol, the increases in blood pressure and heart rate were greatly attenuated, the RPP of 16 000 comparing to a RPP of 35 000 without β-receptor blockade. This was associated with an attenuation of catecholamine release. It is difficult to be certain of the precise mechanism for the attenuation of catecholamine release in the case reported, though a number of possibilities exist. It is known that β-receptor agonists increase the release of noradrenaline from adrenergic nerve terminals[8] and that propranolol blocks this effect. Therefore, impairment of the release of noradrenaline following sympathetic stimulation might contribute to the antihypertensive effect. There is also a close relationship between the sympathetic nervous system and the renin–angiotensin system, and stimulation of one system enhances the activity of the other.[9,10] Renin is stored in the granules of the juxtaglomerular cells of the afferent arteriolar wall and its release is inhibited by an increase in blood pressure and propranolol.[11] Inhibition of renin release, after β-receptor blockade, could therefore modify the stimulation of the sympathetic nervous system. Blockade of β-receptors may thus attenuate the cardiovascular response to ECT by a number of interrelated mechanisms. Firstly, block of cardiac β-receptors will modify the increase in blood pressure and heart rate caused by increased circulating catecholamines. Secondly, block of renin release by propranolol may modify the activation of the sympathetic nervous system. Thirdly, propranolol may attenuate the release of noradrenaline from adrenergic nerve terminals following sympathetic stimulation.

We conclude that ECT even when modified by barbiturates and muscle relaxants may cause brief, intense stimulation of the sympathetic nervous system. The cardiovascular and hormonal consequences of this stimulation may be modified by β-adrenergic receptor blockade using propranolol. This is of particular importance in hypertensive patients and, in particular, those with ischaemic heart disease.

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