

SEVERE HYPERTENSION IN PATIENTS WITH HIGH SPINAL CORD LESIONS UNDERGOING ELECTRO-EJACULATION— MANAGEMENT WITH PROSTAGLANDIN E₂

By H. L. FRANKEL, M.B., F.R.C.P. and C. J. MATHIAS, D.Phil., M.R.C.P.
*National Spinal Injuries Centre, Stoke Mandeville Hospital, Aylesbury, Great Britain and
Medical Unit, St Mary's Hospital Medical School, London, Great Britain*

Key words: Electro-ejaculation; Hypertension; Prostaglandin E₂.

Introduction

ELECTRO-EJACULATORY procedures are now increasingly used in patients with spinal cord injuries who are incapable of sexual intercourse, with collection of semen either for fertility studies or for artificial insemination. The cardiovascular effects of such procedures however have not been studied in depth and this is of importance as we have previously observed severe hypertension and bradycardia in a tetraplegic undergoing such a procedure (Frankel *et al.*, 1974). We have therefore studied patients with spinal cord lesions at different levels who were undergoing electro-ejaculation. We report on the cardiovascular changes observed and on a novel therapeutic approach to managing the hypertension in patients with high lesions.

Methods

In each patient blood pressure (BP) was measured by an intra-arterial catheter introduced percutaneously into either the radial artery or the dorsalis pedis artery using previously described techniques (Mathias *et al.*, 1975). Heart rate (HR) was derived from the arterial pressure signal. In all patients the electrocardiogram (ECG) was monitored continuously on an oscilloscope with a facility for recording the trace if necessary. In each patient an intravenous cannula was inserted in a forearm vein and was kept patent with heparinised saline. This cannula was used in three patients for the infusion of Prostaglandin E₂.

All patients enquired about the procedure and requested it of their own accord after details about it and the possible dangers were explained to them. At the onset the rectum was manually evacuated prior to the insertion of an electrode which was placed close to the seminal vesicles. Intermittent stimulation of up to 20 volts was given for a few seconds at a time using the Stoke Mandeville Paraplegic Ejaculation Stimulator. A rectal thermometer was inserted next to the electrode to ensure that a temperature higher than 99°F did not occur so as to prevent local tissue burning.

In the laboratory were facilities for emergency resuscitation. In addition to a series of drugs available in the cardiac arrest trolley were pentolinium, phentolamine and propranolol, each placed in a syringe ready for administration if needed.

In three patients Prostaglandin E₂ (Prostin E₂, Upjohn Limited) was infused intravenously after attempts at electro-ejaculation had resulted in unacceptable levels of hypertension. The drug was diluted in saline and was infused with a Harvard Infusion Pump which had an adjustable infusion rate.

Patients and Results

Patient 1: This 37-year-old man had been tetraplegic, with a complete lesion at C7, for 108 months. He was otherwise in good health. His resting blood pressure varied from 100/55 to 115/68 mmHg with a heart rate between 50 and 54 beats/min. During insertion of the electrode the blood pressure rose sharply to 195/105 mmHg (Table I). The maximum blood pressure recorded during electrical stimulation was 278/130 mmHg with a heart rate of 36 beats/min. Hypertension was accompanied by sweating, flushing of the face and a bilateral, throbbing headache. During stimulation penile erection occurred but not ejaculation.

Prostaglandin E₂ was then intravenously infused. A dose infusion rate of 0.5 µg was initially started which was increased to 1 µg/min after 5 minutes and then on to 3 µg/min. The cardiovascular changes at rest are recorded in Table II. There was a dose dependent fall in BP and a rise in HR. During infusion the patient had a diffuse headache which was similar in nature but less intense than previously, and which was not exacerbated by electrical stimulation. Stimu-

TABLE I

Maximum blood pressure (BP) in mmHg and simultaneous heart rate (HR) in beats/min in Patient 1 during either insertion of the electrode or electrical stimulation before and during the infusion of Prostaglandin E₂. The responses 10 minutes after the infusion of Prostaglandin E₂ are also indicated. The figures in brackets indicate the voltage applied and the number of occasions

Pre-infusion		During Prostaglandin E ₂ infusion		Approx. 10 mins post-infusion	
B.P.	H.R.	B.P.	H.R.	B.P.	H.R.
195	52	138	56		
105		80			
(Electrode insertion)		(Electrode insertion)			
275	40	204	54	246	42
120		105		118	
(10v × 10)		(10v × 5)		(10v × 3)	
276	36	210	52	234	38
126		105		120	
(18v × 3)		(18v × 3)		(18v × 3)	
275	42	220	52		
125		105			
(18v × 5)		(18v × 5)			
278	36	225	53	270	38
130		108		128	
(20v × 6)		(20v × 6)		(18v × 6)	

lation resulted in lower levels of BP elevation but not in ejaculation. The infusion was therefore stopped. Repeat stimulation 10 minutes after stopping the infusion resulted in pressure rises similar to those before the infusion (Fig. 1 and Table I).

Patient 2: This 29-year-old man with a complete C5 lesion had been injured 5 months previously. His resting BP was 135/60 mmHg and HR was 58 beats/min. The responses to electrical stimulation are summarised in Figure 2. Ejaculation did not occur and Prostaglandin E2 was therefore infused. 3.5 µg/min was initially infused for 5 minutes after which stimulation was again performed. With the Prostaglandin E2 infusion stronger stimuli for a longer period were provided. The patient continued to have a flushed face and sweating during the stimulation as was seen previously but did not complain of headache. Ejaculation occurred with the blood pressure rising precipitously to 270/99 mmHg. The semen contained spermatozoa but none were motile. After a short period an 8.5 µg/min infusion was then given and stimulation repeated but no further ejaculation occurred.

TABLE II

Resting systolic and diastolic blood pressure (BP) in mmHg and heart rate (HR) in beats/min in Patients 1, 2 and 3 before and after 5 minutes of infusion of different doses of Prostaglandin E2. (The asterisk in Patient 1 denotes infusion of 3.0 µg/min of PG E2.)

Patient	Pre		Prostaglandin E2 (µg/min)							
	BP	HR	0.5		1.00		3.5		8.5	
1	100/55	50	92/55	52	94/55	55	68/45*	60	—	—
2	135/60	58	—	—	—	—	93/45	77	68/38	86
3	125/80	75	—	—	—	—	144/85	72	100/60	90

Patient 3: This 25-year-old man with a complete T4 lesion had been injured 55 months previously. His resting BP was 110/65 mmHg with a HR of 68 beats/min and this rose to 170/97 mmHg with an HR of 70 beats/min on rectal examination. After insertion of the electrode, the BP rose even higher to 180/105 mmHg with an HR of 74 beats/min. Electrical stimulation resulted in severe hypertension with a BP of 245/135 mmHg recorded with an HR of 55 beats/min during 20 volt stimulation. 3.5 µg/min of PG E2 was then infused. A 20-volt stimulation resulted in the BP rising to 210/120 mmHg with an HR of 66 beats/min. 8.5 mg/min of PG E2 was then infused and 5 minutes later electrical stimulation was again performed. After 11 minutes ejaculation occurred with the BP rising to 224/120 mmHg with an HR of 64 beats/min. The patient felt perfectly well throughout the procedure except for a flushed face and sweating. No further stimulation was accorded and there were no ill effects from the procedure.

Patient 4: This 24-year-old man with an incomplete T7/8 lesion had been injured 6 months previously. His resting BP varied between 120–140/65–70 mmHg with a HR of between 60 and 65 beats/min. During rectal examination his BP rose to 144/72 mmHg and his HR fell to 56 beats/min. Fifteen-volt stimulation raised his BP further to 164/86 mmHg and HR fell to 50 beats/min. The

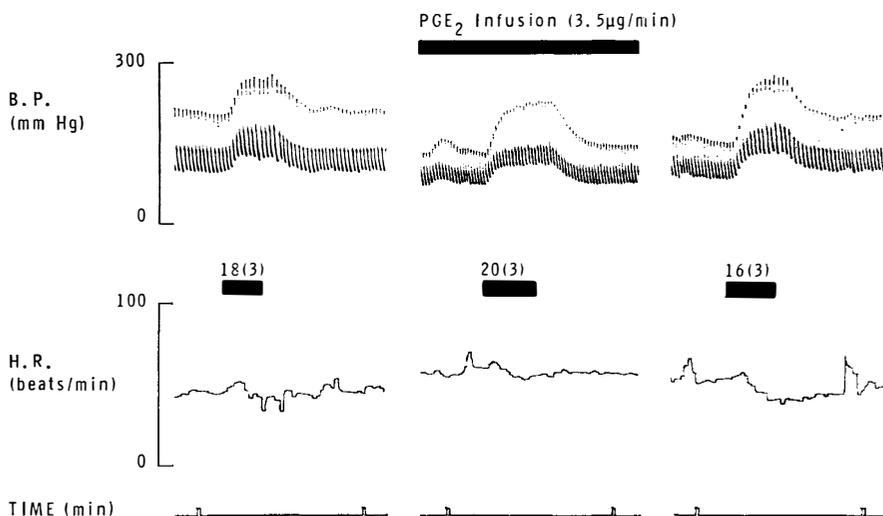


FIG. 1

The effect of electrical stimulation (dark blocks) on blood pressure (BP) and heart rate (HR) of Patient 1 before, during and 10 minutes after Prostaglandin E₂ infusion. The voltage and frequency of electrical stimuli, in brackets, are given above each block.

BP was not raised during repeated stimulation, which caused penile erection but not seminal emission.

Patient 5: This 28-year-old man with a T₁₀ lesion had been injured 12 months previously. His resting BP was 120/60 mmHg with an HR of 70 beats/min. Rectal examination, insertion of the probe and stimulation resulted in minimal cardiovascular changes. Neither erection nor ejaculation occurred during the procedure.

Discussion

In the three patients with cervical or high thoracic lesions severe hypertension and bradycardia occurred during insertion of the electrode and electrical stimulation. These patients felt uncomfortable, one had a headache and all had flushing and sweating over the face and neck. These changes are similar to those previously observed in a tetraplegic undergoing electro-ejaculation (Frankel *et al.*, 1974), and are similar to the changes which occur in tetraplegics during stimulation of the skin and urinary bladder or during skeletal muscle spasms (Guttmann & Whitteridge, 1947; Corbett *et al.*, 1971 A & B). It is likely that the profound cardiovascular changes observed are part of the syndrome of autonomic dysreflexia secondary to visceral stimulation and are the result of uninhibited sympathetic nervous activity via the distal part of the spinal cord. It is not clear whether in man there are inhibitory pathways in the spinal cord that synapse with supra spinal centres; however, it is interesting that in Patient 4 with an incomplete T_{7/8} lesion and in the patient with the T₁₀ lesion there were minimal cardiovascular changes observed. Both these patients with low lesions had normal Valsalva manoeuvres and no postural fall in BP, which suggests that a substantial proportion of their baroreceptor pathways were functioning normally.

Hypertension and tachycardia during coitus have been reported in normal subjects in whom direct arterial blood pressure and heart rate have been recorded

(*Little et al.*, 1974). In their series blood pressures as high as 233/128 and 214/135 mmHg were reported especially during ejaculation. Heart rates as high as 170 beats/min were observed. In our patients with high lesions similar blood pressure changes were recorded, but unlike the normal subjects there was a bradycardia instead of a tachycardia. In the normal subjects, unlike the tetraplegics, muscular exercise accompanied coitus and part of the cardiovascular changes may have been related to those associated with exercise (*Braunwald et al.*, 1967). It may be that in the tetraplegics the absence of either the neural or the humoral components of exercise resulted in a predominance of parasympathetic outflow in the vagus in response to hypertension and this probably overruled the effect of increased sympathetic outflow to the heart.

The morbidity and mortality associated with the paroxysmal elevation in blood pressure in tetraplegics during autonomic dysreflexia has been emphasised in recent reviews (*Frankel and Mathias*, 1976, 1979). Although electro-ejaculation is superior to the previous technique of intrathecal neostigmine (*Guttmann & Walsh*, 1971) in eliciting ejaculations, because it provides a controlled stimulus for a short period of time, our observations suggest that the severe hypertension may be of potential danger to these patients. Furthermore, in our three patients it initially limited the extent of stimulation and therefore reduced the chance of successful ejaculation. In three of our patients, therefore, a novel therapeutic approach to combine both blood pressure control and at the same time not impair the genital smooth muscle responses (which are an integral component of ejaculation) was assessed. Prostaglandin E₂ was therefore infused intravenously during stimulation. In all three patients higher stimuli for a longer period could be given. In the first patient ejaculation was not achieved but this may have been because, being the first patient, an appropriate dose of Prostaglandin was not infused for an adequate period of time. In the other two patients the infusion of PG E₂ was instrumental in achieving ejaculation.

We are not certain by precisely which mechanism PG E₂ reduces both resting blood pressure and the level of blood pressure in response to electrical stimulation. Animal studies indicate that the hypotensive action is largely due to an action on vascular smooth muscle resulting in dilatation of both resistance and capacitance vessels (*Conway and Hatton*, 1975). It has also been suggested that PG E₂ may exert a negative feedback control over sympathetic neural transmission (*Hedqvist*, 1973) and this may have resulted in the diminished responses. It may be argued, however, that this latter action might impair the chance of successful ejaculation. However, in certain animals PG E₂ enhances the contractile responses of the vas deferens and seminal vesicles (*Naimzada*, 1969), and this may have been instrumental in successful ejaculation in two patients.

Our studies indicate that severe hypertension, as part of the syndrome of autonomic dysreflexia, may occur during electro-ejaculatory procedures in patients with high thoracic and cervical spinal cord lesions. There seem to be minimal changes in blood pressure in patients with lower spinal cord lesions. In patients with high lesions infusion of PG E₂ appears to control the blood pressure, without retarding the process of ejaculation.

SUMMARY

Intra-arterial blood pressure (BP) and heart rate (HR) were continuously recorded in five patients with spinal cord injuries at different levels who were undergoing electro-ejaculation. In three patients with lesions at C₇, C₅ and T₄

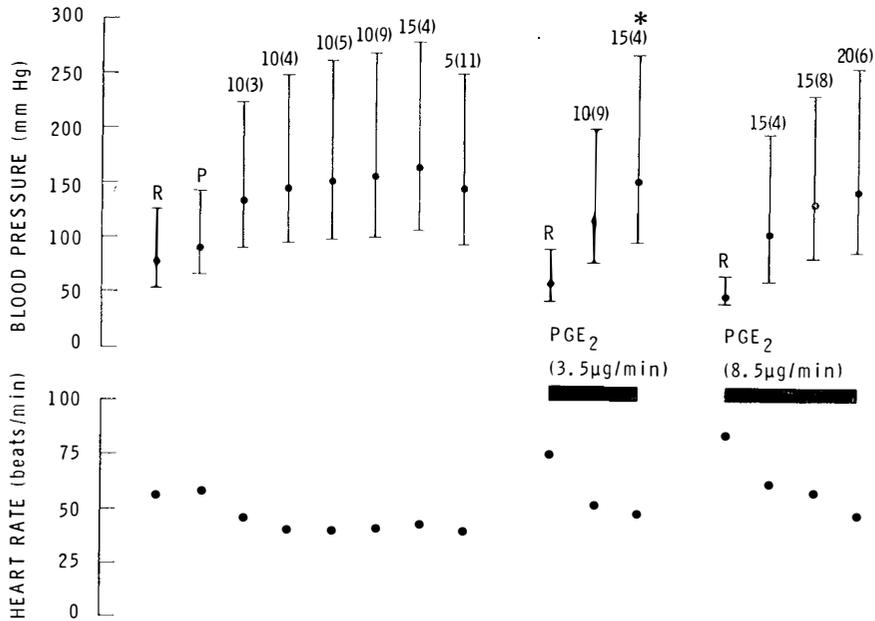


FIG. 2

Systolic and diastolic blood pressure (upper and lower horizontal lines), mean blood pressure (filled circles) and heart rate in Patient 2 at rest (R), during insertion of the rectal probe (P) and during electrical stimulation (voltage is given above each with the number of stimuli in brackets). Measurements have been made before and during infusion of Prostaglandin E₂ (PG E₂). The asterisk denotes when ejaculation occurred.

insertion of the electrode and electrical stimulation caused severe hypertension and bradycardia. In a patient with a T_{7/8} lesion and in another with a T₁₀ lesion there were either moderate or minimal cardiovascular changes. Severe hypertension during electro-ejaculation is a serious problem in patients with high lesions and is probably part of the syndrome of autonomic dysreflexia.

In the three patients with high spinal cord lesions the procedures were repeated during an intravenous infusion of Prostaglandin E₂. Resting BP was lowered and resting HR raised. The level of BP recorded during electrical stimulation was substantially reduced. This enabled larger stimuli to be used for a longer period and resulted in successful ejaculation in two patients.

Acknowledgements. We thank Mrs P. Smith and Sisters M. Brennan and E. Kidd for their valuable assistance, the late Dr. B. L. Copley of Upjohn Ltd. (UK) for his encouragement, and the Lawson Tait Medical and Scientific Research Trust for their support. C. J. M. holds a Wellcome Senior Research Fellowship in Clinical Science.

RÉSUMÉ

La tension intra-artérielle et le débit sanguin furent enregistrés chez 5 malades avec lésions de niveaux différents de la moelle épinière durant l'électroéjaculation.

L'insertion de l'électrode et la stimulation électrique ont causé une hypertension sévère et une bradycardie chez les 3 malades, C₅-C₇ et T₄. Cet effet sur le système cardiovasculaire chez les 2 malades au T_{7/8} et T₁₀ était modéré ou minimal. Les changements cardiovasculaires observés chez les malades de lésions supérieures sont probablement attribués à l'autonomie dysrèflexique.

L'étude était répétée pour les 3 malades aux lésions supérieures C5-C7-T4 durant l'infusion intraveineuse de la prostaglandine E₂; la tension intra-artérielle était substantiellement réduite et l'on a pu obtenir l'éjaculation de 2 malades, en employant de plus fortes stimulations pour une plus longue période de temps.

ZUSAMMENFASSUNG

An 5 Patienten mit Querschnittslähmungen auf verschiedenen Höhen wurden bei elektrisch stimulierter Ejakulation durchgehend der intra-arterielle Blutdruck (BD) und die Pulsfrequenz (PF) gemessen. 3 Patienten mit Verletzungen bei C₇, C₅ und T₄ litten nach Einführung der Elektrode und elektrischem Stimulus an stark überhöhtem Blutdruck und Bradykardie. An zwei Patienten mit Verletzungen bei T_{7/8} bzw. T₁₀ waren entweder mässige oder geringe kardio-vaskuläre Veränderungen zu bemerken. Die kardio-vaskulären Veränderungen bei den Patienten mit hochliegenden Verletzungen sind Teil des Syndroms der autonomen Dysreflexie.

An 3 Patienten mit hochliegenden Rückenmarksverletzungen wurde der Vorgang wiederholt, während ihnen Prostaglandin E₂ intravenös verabreicht wurde. Dabei stieg die PF und sank der BD im Ruhezustand. Während der elektrischen Stimulierung wurde der BD erheblich gesenkt. Dadurch wurde es möglich, stärker und länger zu stimulieren, was dann bei zwei Patienten zur Ejakulation führte.

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