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going elective prostatectomy. All had been starved for at least 12 h before surgery. None of them showed evidence of endocrine renal, hepatic or metabolic disorders. Pre-medication consisted of atropine 0.6 mg and pethidine 100 mg i.m. at 7.05 a.m.

Anaesthesia was induced with thiopentone 5 mg/kg, which was followed by pancuronium 0.1 mg/kg body weight. Anaesthesia was maintained with nitrous oxide 3.5 l/min and oxygen 1.5 l/min administered via an endotracheal tube and a circle absorber system. Supplementary doses of fentanyl 0.05 mg were given i.v. at approximately 30-min intervals and pancuronium 1–2 mg when indicated. Ventilation was controlled with a Cape–Waine Mk 3 ventilator with a minute volume of 10 l/min. Blood loss from each patient was minimal and none required blood transfusion. During surgery each patient received between 100 and 400 ml Ringer lactate solution. The pulse rate and arterial pressure were measured at 10-min intervals using a pulse monitor (Cotel Keating) and a pneumatic cuff. After surgery, the residual effect of pancuronium was antagonized with neostigmine 2.5 mg preceded by atropine 1.2 mg i.v.

Nine samples of venous blood were collected via a 19-gauge butterfly needle in the cephalic vein from each patient:
(1) 07.00 hours before pre-medication;
(2) 08.00 hours before induction of anaesthesia;
(3) After 15 min of anaesthesia;
(4) After 30 min of anaesthesia;
(5) After 45 min of anaesthesia;
(6) After 10 min of surgery;
(7) After 30 min of surgery;
(8) After 60 min of surgery; and
(9) 60 min after the end of anaesthesia and surgery.

Ten millilitres of blood was collected for estimation of plasma cortisol concentration into lithium heparin bottles and 2 ml for blood sugar estimation into fluoride oxalate bottles. All samples were received at the laboratory coded.

Plasma cortisol was estimated directly using the radioimmunoassay (RIA) technique, the kits (Amerlex Cortisol RIA kits) being supplied by Radiochemical Centre, Amersham, U.K. This assay employs a specific antiserum which has a low cross-reactivity with other naturally occurring steroids and which has been immobilized on to the surface of polymer particles of uniform diameter (Amerlex antibody suspension). In the assay method, cortisol is released from transcortin by a chemical blocking agent contained in the ^{125}I-labelled cortisol derivative solution. The total cortisol in the sample is then free to compete with the ^{125}I-labelled cortisol derivative for binding sites on the antibody-coated particles. No separate heat denaturation process is carried out. The blood sugar was estimated by the glucose–oxidase method of Huggett and Nixon (1957) with appropriate quality control (College of American Pathologists quality evaluation programme).

Results

Table 1 shows the details of the patients and the duration of surgery.

TABLE 1. Details of patients and duration of surgery

Patient	Age (years)	Weight (kg)	Duration of surgery (min)
1	70	55.1	73
2	76	68	145
3	60	44	45
4	58	64.4	100
5	65	67.6	115
6	66	63	60
7	70	69.4	60
8	60	43.5	53
9	65	50	50
10	89	83.1	52
Mean	67.3	60.81	75.3
Standard deviation	8.81	12.55	33.33
Standard error	2.79	3.97	10.60

Table 2 and Fig. 1 show the blood sugar levels at the different stages of observation. Pre-medication produced a slight but insignificant elevation of blood sugar. Anaesthesia did not cause any further change in blood sugar. The surgical procedure, however, produced a progressive rise of blood sugar, the rise being significant from 30 min of surgery until after the end of anaesthesia and surgery.

Table 3 and Fig. 2 show the plasma cortisol level. Pre-medication caused a slight fall in plasma cortisol; so did general anaesthesia.

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METABOLIC RESPONSE TO ANAESTHESIA AND LOWER ABDOMINAL SURGERY IN NIGERIANS

CHANGES IN PLASMA CORTISOL AND BLOOD SUGAR

O. O. AKINYEMI AND J. A. O. MAGBAGBEOLA

Department of Anaesthesia, College of Medicine, University College Hospital, Ibadan

Summary

In ten Nigerian patients undergoing prostatectomy, plasma cortisol was slightly decreased by pre-medication with atropine and pethidine and during general anaesthesia without surgery. This decrease was not statistically significant. There was, however, a statistically significant increase in plasma cortisol after 1 h of surgery and also in the post-operative period. The blood sugar was elevated by pre-medication and surgery though the rise was not significant. There was a significant rise of blood sugar after 30 min of surgery and in the post-operative period. These findings in general are similar to those observed in ten Nigerian patients undergoing upper abdominal surgery, although the rise in plasma cortisol was higher following upper abdominal surgery, and the rise in blood sugar higher following lower abdominal surgery.

Résumé

Chez dix malades nigériens subissant une prostatectomie le cortisol de plasma fut légèrement diminué par une prémédication avec de l'atropine et de la pethidine au cours d'une anesthésie générale sans chirurgie. Cette diminution ne fut pas significative du point de vue de la statistique. Mais 1 h après la chirurgie il y eut un accroissement significatif de cortisol de plasma qui se maintenait tout le long de la période postopératoire. Le sucre du sang ne fut pas élevé par prémédication et par chirurgie, bien que le relèvement ne fût pas significatif. Il y eut un relèvement significatif de sucre du sang après 30 min de la chirurgie et au cours de la période postopératoire. D'une manière générale ces résultats sont analogues à ceux remarqués chez dix nigériens qui subissent la chirurgie de l'abdomen supérieur; mais après cette dernière chirurgie, il y a un plus haut relèvement du cortisol et du sucre de plasma.

Correspondence: Dr O. O. Akinyemi, Department of Anaesthesia. University College Hospital, Ibadan, Nigeria.

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Introduction

A previous study in this environment showed that Nigerians exhibit significant hyperglycaemia in response to upper abdominal surgery though this is not more marked than in the European, Japanese, Jamaican and Indian patients (Magbagbeola & Adadevoh, 1974). The same study also showed significant increase in plasma cortisol after upper abdominal operation. The aim of the present study was to determine the metabolic response of Nigerians to lower abdominal surgery and see if this response is any different from their response to upper abdominal surgery.

Methods

Ten Nigerian male patients, of physical status 1 (ASA) and between the ages of 58 and 89 years were studied. All of them were under-

TABLE 3. Plasma cortisol during prostatectomy

Patient	Before pre-med	Before induction	During anaesthesia 15 min	30 min	45 min	During surgery 10 min	30 min	60 min	After operation (60 min)
1	13.5	12.0	9.4	10.4	12.2	10.5	14.5	12.2	16.5
2	7.9	4.9	9.2	5.7	6.4	6.5	9.2	16.0	22.0
3	6.5	6.0	11.5	8.2	6.5	11.0	12.0	17.0	12.0
4	12.5	11.4	7.5	9.3	5.3	5.3	7.0	12.5	18.5
5	7.5	4.4	7.6	9.9	10.3	7.0	13.2	12.6	16.5
6	8.7	6.2	5.0	4.7	3.9	2.9	15.0	14.0	17.5
7	6.8	6.8	8.9	6.5	6.4	6.8	17.5	16.0	10.6
8	6.2	15.5	12.2	13.0	13.5	9.5	13.8	15.8	19.0
9	17.5	14.0	14.5	10.1	12.8	15.5	13.8	17.0	19.0
10	25.0	27.0	27.5	29.0	28.5	25.0	28.5	28.0	25.0
Mean	11.21	10.82	11.33	10.68	10.58	10.0	14.45	16.11	17.66
Standard deviation	6.10	6.93	6.28	6.90	7.16	6.33	5.76	4.57	4.24
Standard error	1.93	2.19 NS	1.99 NS	2.18 NS	2.26 NS	2.00 NS	1.82 NS	1.45 S	1.34 S

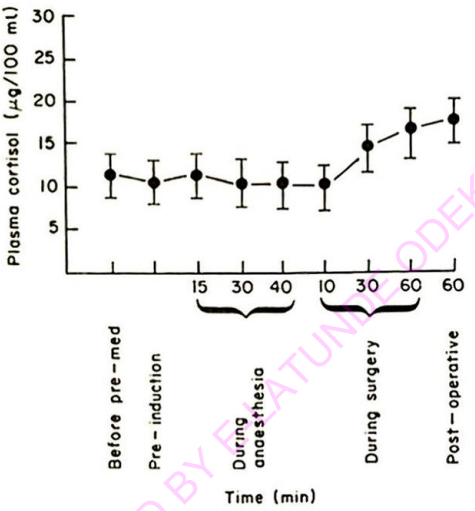

FIG. 2. Plasma cortisol concentration (mean ± s.e. mean) before, during and after surgery and anaesthesia.

of Oyama (1973) that pethidine does not inhibit adreno-cortical stimulation caused by pre-operative emotional stress. The probable reason for the latter was a difference in methodology.

Our finding of insignificant changes in plasma cortisol during anaesthesia without surgery is consistent with that of other workers (Oyama *et al.*, 1969; Clarke, Johnston & Sheridan, 1970).

The marked rise of plasma cortisol after 30 min of surgery and significant rise after 60 min of surgery and post-operatively is also similar to the observations of some other workers (Clarke *et al.*, 1970; Ffoulkes-Crabbe, Abiodun & Johnson, 1971). This rise in plasma cortisol is, however, less than that produced during upper abdominal surgery in the same environment (Table 4).

The observed slight increase in blood sugar following pre-medication and during anaesthesia alone is in accord with the finding of Cullingford (1966) and Clarke (1970, 1973) on European patients who had similar types of anaesthesia. (The significant rise of blood sugar from 30 min of surgery and the post-operative period also tally.) A comparison of the rise in blood sugar during surgery in this study with those of Keating (1958), Cullingford (1966), Clarke (1970) and Oyama, Takiguchi and Kudo (1971) confirms that the significant hyperglycaemia exhibited by Nigerians in response to surgery is not more marked than in the European, Japanese, Jamaican and Indian patients.

These findings in general were similar to those obtained in a previous study on Nigerian patients undergoing upper abdominal surgery (Magbagbeola & Adadevoh, 1974), in spite of the shorter mean duration of surgery (75.3 min) and the older mean age group (67.3 years) as

TABLE 2. Blood sugar values during prostatectomy (mg/100 ml)

Patient	Before pre-med	Before induction	During anaesthesia 15 min	30 min	45 min	During surgery 10 min	30 min	60 min	After operation (60 min)
1	90	81	74	81	74	71	86	85	93
2	60	68	83	66	58	65	81	93	121
3	75	86	68	67	71	66	69	82	102
4	73	68	77	75	73	68	82	100	146
5	74	78	95	117	120	128	135	163	195
6	91	88	84	80	63	70	89	93	194
7	81	93	88	73	81	78	98	88	171
8	68	88	83	83	71	73	95	98	151
9	71	79	81	76	86	110	157	167	224
10	79	91	95	95	89	91	91	105	129
Mean	76.20	82.00	82.80	81.30	78.60	82.00	98.30	107.40	152.60
Standard deviation	9.51	8.87	8.56	15.06	17.38	21.30	26.91	31.15	43.03
Standard error	3.01	2.80 NS	2.71 NS	4.76 NS	5.50 NS	6.74 NS	8.51 S	9.85 S	13.61 S

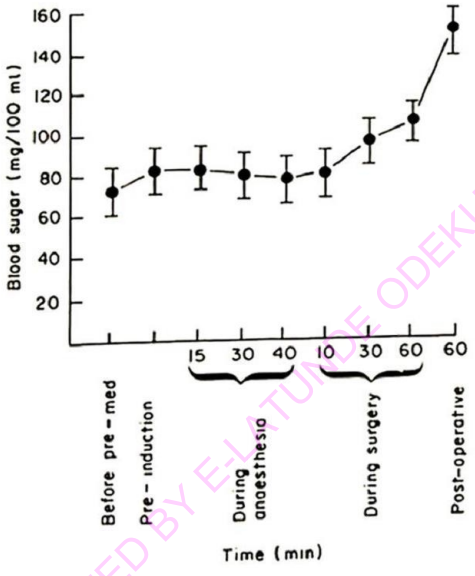

FIG. 1. Blood sugar concentration (mean ± s.e. mean) before, during and after surgery and anaesthesia.

Surgery, however, produced a marked elevation of plasma cortisol, this rise becoming significant from one hour of surgery onwards.

The overall pattern of these results is similar to that of upper abdominal surgery; but the rise of plasma cortisol produced by lower abdominal surgery is less than that caused by upper abdominal surgery. On the other hand, lower abdominal surgery produced a higher rise of blood sugar.

Discussion

The present studies show that anaesthesia with thiopentone-nitrous oxide-pancuronium supplemented with pethidine does not produce significant changes in blood sugar and plasma cortisol in Nigerian patients. During lower abdominal surgery, whilst no significant changes occurred in the first 30 min, there were significant rises of blood sugar and of plasma cortisol from 30 min onwards. Significant increase in blood sugar and plasma cortisol were also recorded after operation.

The range of normal cortisol values in Nigerians estimated at the University College Hospital, Ibadan, have been found to be 6–30 μg/100 ml between 08.00 and 12.00 hours. In this study, all the blood samples were collected before 12.00 hours. Pre-medication with atropine and pethidine given 1 h before induction of general anaesthesia was found in this study to cause a small decrease in plasma cortisol (Table 3). This finding was consistent with that observed in a similar study on Nigerian patients undergoing upper abdominal surgery (Magbagbeola & Adadevoh, 1974), and at variance with the conclusion by Oyama, Takazawa and Kimura (1969) and the opinion

compared with 88.5 min and 43.2 years respectively for upper abdominal surgery.

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TABLE 4. Mean plasma cortisol concentration (μg/100 ml) and blood sugar (mg/100 ml) before, during and after anaesthesia and surgery: values during upper and lower abdominal surgery compared (n = 10)

	Before pre-med	Before induction	During anaesthesia			During surgery			After operation (60 min)
			15 min	30 min	45 min	10 min	30 min	60 min	
Plasma cortisol									
Mean									
Upper abdominal*	20.8	15.1	13.9	15.6	12.3	12.4	23.6	32.2	38.2
Lower abdominal	11.21	10.82	11.33	10.68	10.58	10.0	14.45	16.11	17.66
Mean diff. from initial value									
Upper abdominal*	–	−5.7	−6.9	−5.2	−8.5	−8.4	+2.8	+11.4	+17.4
Lower abdominal	–	−0.39	+1.2	−0.53	−0.63	−1.21	+3.24	+4.9	+6.45
Blood sugar									
Mean									
Upper abdominal*	67.9	73.4	66.7	65.6	69.8	71.6	83.1	97.8	120.1
Lower abdominal	76.20	82.00	82.80	81.30	78.60	82.00	98.30	107.40	152.60
Mean diff. from initial value									
Upper abdominal*	–	+5.5	−1.2	−2.3	+1.9	+3.7	+15.2	+29.9	+52.2
Lower abdominal	–	+5.8	+6.6	+5.1	+2.4	+5.8	+22.1	+31.2	+76.4

*Magbagbeola and Adadevoh (1974) and present study.