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ORIGINAL RESEARCH

Effects of inadvertent preoperative prolonged fasting on Blood Sugar, Electrolytes and Ketogenesis: General vs Regional Anaesthesia

Sajith Damodaran[1], Mahendra Kumar[2], Shikha Agarwal[3], R. S. Rautela[4]

[1]Consultant, Intensive Care, Queen Elizabeth The Queen Mother Hospital, St Peter's Road, Margate, CT9 4AN, United Kingdom
[2]Professor, [3]Assistant Professor, Dept of Anaesthesiology & Critical Care, School of Medical Sciences & Research Sharda University, Greater Noida, G.B. Nagar, U.P., India
[4]Director Professor, Dept of Anaesthesiology & Critical Care, UCMS & GTB Hospital, Dilshad Garden, Delhi, India

Corresponding Author
Shikha Agarwal
Assistant Professor, Dept of Anaesthesiology & Critical Care, School of Medical Sciences & Research Sharda University, Greater Noida, G.B. Nagar, U.P., India
Email: drshikhaagarwal22@gmail.com

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Abstract
Background - Many patients are inadvertently fasted for prolonged periods (≥ 14 hours) preoperatively due to unavoidable reasons. This study was undertaken to asses the effects of inadvertent prolonged preoperative fasting.
Methods - Sixty patients of ASA class I or II scheduled for elective surgery either under general anaesthesia or subarachnoid block, who inadvertently fasted for more than fourteen hours were selected randomly and divided into two groups - Group GA (patients received general anaesthesia) or Group RA (patients received subarachnoid block). Before induction of anaesthesia, all patients were asked for the presence of various symptoms like thirst, dryness of mouth, hunger, concentrated urine etc. Samples of venous blood and urine were collected before induction of anaesthesia and at various points of time peri-operatively for analysis of blood sugar, serum electrolytes, urine for specific gravity and presence of ketone bodies. The data was analyzed using ANOVA, Chi square test and Tukey test.
Results - Preoperative fasting period was statistically comparable in two groups. The most common symptoms were thirst, dry mouth and concentrated urine. Three patients developed hypoglycemia without ketonuria and 6 patients developed ketonuria at pre-induction in both groups. The mean specific gravity of urine was significantly higher during study period than their values from preanaesthetic record. There was statistically significant elevation of blood glucose (although within normal physiological limits) in both groups intraoperatively and postoperatively compared to preinduction value. At 6 hours postoperatively, 60% patients developed ketonuria in both groups.
Conclusions – After prolonged preoperative fasting a significant number of patients developed ketonuria independent to technique of anaesthesia.
Key Words: preoperative fasting, hypoglycemia, ketonuria, ketogenesis

Introduction

Preoperative fasting is a well-established practice to avoid the incidence of gastric regurgitation and pulmonary aspiration in patients undergoing anaesthesia for surgery. General anaesthesia abolishes the protective airway reflexes and thus increases the risk of aspiration of gastric contents after regurgitation. Conventionally, a practice of "nil by mouth for 6 to 8hrs before surgery" is followed. Various national and international anaesthesia societies have proposed patient friendly guidelines that reduce the total fasting period, especially for clear fluids. These guidelines have been decided after adequate studies on the relationship between fasting period and volume of gastric contents.[1] But in a busy hospital setup patients invariably have fasting for more than recommended period due to multiple factors.[2]

During the fasting period patients are allowed to take nothing by mouth – neither water, nor caloric intake. During this period various symptoms like feeling of hunger, thirst, dryness of mouth, concentrated urine, anxiety and discomfort have been reported in literature. The energy stores in form of glucose and glycogen are quickly depleted during fasting and therefore changes

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occur in metabolism of glucose, fats and proteins. Patients for surgery face additional stress in the fasted state over and above the surgical stress. Literature also shows that patients fasted overnight develop intraoperative insulin resistance as compared to patients on carbohydrate diet.[3]

Besides fasting, anaesthesia and surgery also have their effect on blood sugar and metabolic status. Under surgical stress, there is hyperglycemic effect which is more prominent under general anaesthesia.

The effect of fasting and anaesthesia on serum electrolytes, particularly sodium and potassium is not known, though it may be postulated that in normal adults, major deviations from normal levels will not occur. However, the effect of prolonged fluid deprivation and resultant dehydration on serum electrolyte levels in patients undergoing surgery under different techniques of anaesthesia may vary.

The present study was designed to assess the effect of inadvertent preoperative prolonged fasting on blood sugar, serum electrolytes, specific gravity of urine and ketogenesis in patients who underwent elective surgery under general anaesthesia or subarachnoid block.

Materials and Methods

The study was conducted after getting approval by the institutional review board for research. Sixty patients of either gender, aged between 18 and 60 years, belonging to ASA Class I and II scheduled for elective surgery under either general or regional anaesthesia who had been fasting for more than 14 hours inadvertently after their last meal, were selected. Patients with systemic illnesses like diabetes mellitus, systemic hypertension or cardiovascular, respiratory or hepatic diseases were excluded. Informed consent for participation in the study was taken from each patient.

The patients were divided into two groups on that basis of technique of anaesthesia required as per surgical procedure. The patients of GA Group (n=30) received endotracheal general anaesthesia while patients of RA Group (n=30) subarachnoid block.

The last meal time, and symptoms of discomfort due to prolonged fasting like thirst, dryness of mouth, hunger, concentrated urine, giddiness, irritability, anxiety and any other symptoms the patients wished to report were recorded before the procedure.

Patients of group GA were subjected to standard conventional endotracheal general anaesthesia. Monitoring of heart rate (HR), Non invasive blood pressure (NIBP) (systolic, diastolic and mean) and pulse oximetry (SPO_2) was commenced from preinduction time every five minutes throughout the procedure. Fentanyl 1mcg/kg was given as a premedicant and anaesthesia was induced with Inj Thiopentone Sodium 2.5% 4-6 mg/kg, and endotracheal intubation was facilitated by Inj Vecuronium 0.1mg/kg. Intermittent positive pressure ventilation was used to maintain EtCO2 between 35-40mmHg. Anaesthesia was maintained using Nitrous Oxide:Oxygen (66:33) and isoflurane 0.5-1%. Ringer's lactate (RL) solution was infused as per need to maintain systolic blood pressure ±20% of baseline value with adequate depth of anaesthesia. Inj Diclofenac Sodium 1mg/kg IM and Inj Ondansetron 6 mg IV were administered just before reversal. Neuromuscular blockade was reversed using Inj Neostigmine 2.5 mg and Inj Atropine 1.2 mg. Tracheal extubation was done as in routine.

Patients of group RA received subarachnoid block by standard technique under strict aseptic precautions using Quinke's needle 25G. Inj bupivacaine 0.5% heavy 2.5 ml was used. All patients were monitored similar to the group GA. Systolic blood pressure was maintained ±20% of baseline values using intravenous fluid infusions (crystalloids and colloids) and Injection mephentermine (6mg) as per requirement.

Postoperatively, Inj Diclofenac Sodium 1mg/kg was administered I/M when VAS score was 3 or more on complaint of pain.

Blood Sugar and Serum Sodium and Potassium were estimated in all patients before induction of anaesthesia, 90 minutes after induction of anaesthesia and postoperatively at 3 hours and 6 hours. Urine was analyzed just before induction and at 3 hours and 6 hours postoperatively to measure specific gravity and detect ketone bodies in all patients.

All patients received IV infusion of Ringer's Lactate as per need postoperatively. If any patient showed blood sugar levels below 60mg/dl along with signs of hypoglycaemia, infusion of 5% dextrose was administered.

The recorded data was analysed using ANOVA test and chi-square test followed by Tukey's test.

Results

Patients of both groups were statistically comparable for their demographic profile. (Table-1)

Table-1 showing demographic profile of both groups

	Group GA (n=30)	Group RA (n=30)	p value
Age	34 ±10.03	35.7 ±10.68	0.552 (NS)
Body Weight (Kg)	54.2 ±12.36	57.77 ±10.49	0.233 (NS)
Male:Female	10:20	11:19	
Duration of fasting (hrs)	15.70 ± 1.24	16.01 ± 1.22	0.210 (NS)

$p<0.05$ significant, (NS-not significant)

The incidence of the various preoperative symptoms due to prolonged fasting as reported by the patients is shown in Fig 1.

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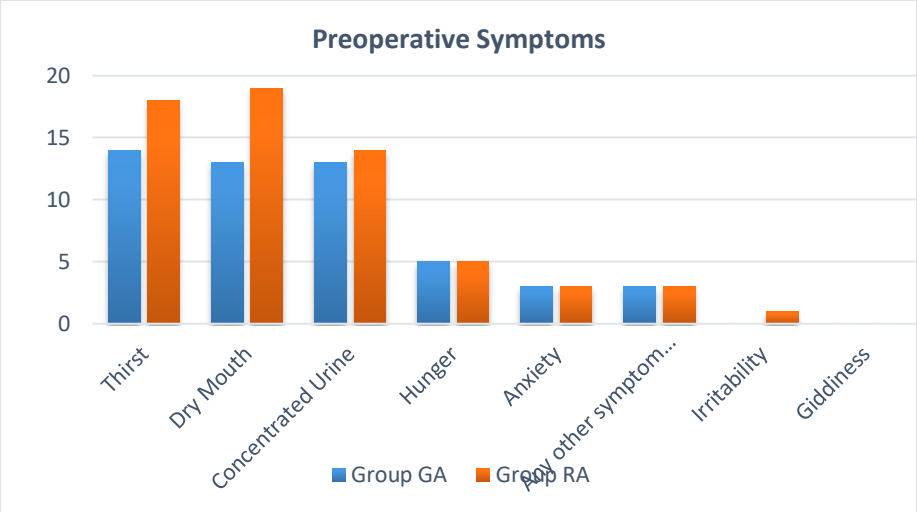

Figure: 1 Incidence of various preoperative symptoms due to prolonged fasting

The most common symptoms were thirst, dry mouth and concentrated urine in both the groups.

Blood sugar levels at each time interval were statistically comparable between two groups. (Table-2). However, in GA group, blood sugar levels were significantly higher at post induction and 3 and 6 hr of postoperative period while in the RA group, it was significantly higher at post induction and 3 hr of postoperative period than preinduction value of respective group. Three patients in the GA groups showed hypoglycaemia (Blood Sugar < 60mg/dL). No patient had perioperative hypoglycaemia.

Table 2. Showing mean of blood sugar levels (mg/dL) with SD at various points of time in both the groups.

Time	Group GA(n=30)	Group RA(n=30)	p (<0.05 sig)
From PAC Record	93.5±11.8	91.07±15.23	0.795(NS)
Pre-Induction	74.33±10.31	80.70±12.09	
Post Induction 90min	94.6±23.18*	87.70±15.33*	
Post operative 3 hrs	88.03±20.40*	88.83±14.61*	
Post operative 6 hrs	88.97±18.64*	86.13±10.65	

*significantly different from their pre-induction value (p = 0.000) (p<0.05 significant), PAC – preanaesthetic check-up, (NS- Not significant)

Statistical analysis revealed that there was no significant difference in the mean of serum sodium and potassium levels between the two groups at any point of time. Intraoperative and postoperative serum sodium and potassium levels in both the groups were comparable (except potassium level at postoperative 6 hours) with preinduction value of respective group. (Table 3 & 4).

Table: 3 showing Mean of Serum Sodium in mEq/L with SD at various time intervals in both the groups.

Time	Group GA (n=30)	Group RA (n=30)	p (<0.05 sig)
From PAC record	140.97±3.88	138.93±3.03	0.394(NS)
Preinduction	145.21±9.66	144.40±8.85	
Post induction 90 min	146±7.21	145.23±7.35	
Post operative 3 hrs	147.55±7.20	144.67±4.16	
Post operative 6 hrs	146.69±7.15	146.87±5.26	

PAC: preanaesthesia check-up, (NS - not significant)

Table: 4 showing mean of serum potassium in mEq/dL with SD at various points of time in both the groups.

Time	Group GA (n=30)	Group RA (n=30)	p (<0.05 sig)
From PAC Record	4.29±0.62	4.22±0.52	0.169
Preinduction	4.03±0.68	4.19±0.75	
Post induction 90 min	3.84±0.61	4.16±0.44	
Post operative 3 hrs	3.97±0.54	4.16±0.42	
Post operative 6 hrs	4.33±0.46*	4.27±0.54	

*significantly different from their preinduction value (p=0.006) (p<0.05 significant) PAC: preanaesthesia checkup (NS- not significant)

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Ketone bodies were detected in 2 patients in the GA group and 4 patients in the RA group even before induction of anaesthesia (Table 5). Postoperatively at 3 hrs, 6 (20%) and 8 (26.7%) patients showed ketonuria in GA and RA group respectively and at 6 hours 60% patients of each groups had ketonuria. Patients who had ketonuria at preanaesthetic time also continued to have it in postoperative period.

The mean of specific gravity of urine was comparable in both the groups at all-time intervals (p>0.05). At preinduction, and postoperative 3 and 6 hrs, it was significantly higher in both the groups as compared to the value recorded in the PAC record of respective group (p<0.000). (Table 6)

Table: 5 showing number of patients with their percentage with positive urine ketone bodies in both the groups.

Interval	Group GA (n=30)	Group RA (n=30)	p Value (< 0.05 significant)
From PAC record	Nil	Nil	1.00 (NS)
Preinduction	2 (6.7%)*	4 (13.3%)*	0.389 (NS)
Post operative 3 hrs	6 (20%)*	8 (26.7%)*	0.542 (NS)
Post operative 6 hrs	18 (60%)*†‡	18 (60%)*†‡	1.00 (NS)

*Significantly different from the PAC value, p < 0.005, †Significantly different from the Preinduction value, p < 0.02, ‡Significantly different from the Postoperative 3 hour value, p < 0.02, PAC: Preanaesthetic check up (NS – Not significant)

Table: 6 showing mean and SD of specific gravity of urine of both the groups

Time interval	Group GA (n=30)	Group RA (n=30)	p value
From PAC record	1.0112 ± 0.0021	1.0108 ± 0.0021	0.057 (NS)
Pre induction	1.0203 ± 0.0070*	1.0208 ± 0.0072*	
Post operative 3 hrs	1.0228 ± 0.0045*	1.0200 ± 0.0054*	
Post operative 6 hrs	1.0247 ± 0.0076*	1.0195 ± 0.0081*	

* significantly different from their PAC value (p= 0.000) (p<0.05 significant) (NS – not significant)

Discussion

Conventionally, preoperative orders of 'nil per orally from midnight' is given to achieve an empty stomach before induction of anaesthesia. Preoperative fasting is practiced to decrease the incidence and severity of pulmonary aspiration due to loss of airway reflexes in patients undergoing general anaesthesia. However, recent guidelines of various international anaesthesia organisations have allowed more liberal intake of fluids and even light meal preoperatively. The consensus at present is that 2 hours of fasting for clear fluids and 6 hours after a light meal is safe enough to reduce the incidence of pulmonary aspiration while at the same time enhancing patient comfort. Despite these guidelines, a large number of patients still end up fasting for prolonged durations due to various reasons.

The present study was conducted to evaluate the effect of prolonged preoperative fasting on blood sugar and serum electrolyte levels and ketogenesis in adult patients undergoing surgery under either general anaesthesia or subarachnoid block who inadvertently fasted for more than 14 hours. The most common symptoms reported by the patients in both the groups were thirst, dry mouth and concentrated urine. Since the symptoms were recorded before induction of anaesthesia, the technique of anaesthesia is not the cause of the difference. Sutherland et al reported an incidence of thirst of 44% in female outpatients undergoing minor gynaecological surgery.[3] This is comparable to the incidence in thirst in the present study (GA group 46.7%, RA group 60%).

Hunger was reported by 16.7% patients in each group. This was in contrast to the study done by Sutherland et al where the mean duration of fasting was 15 ± 3 hours and the incidence of hunger was 50%, which was higher than in the present study.[3] Three patients in the present study had hypoglycaemia (blood sugar < 60mg/dL) preoperatively. All the three patients belonged to the GA group. This included two females and one male patient. The lowest preinduction blood sugar level recorded was 47 mg/dL in the male patient. The duration of fasting in this patient was 14 hours and 15 minutes. The other two patients had fasted for 15 hours and 30 minutes and had blood sugar levels of 54 mg/dL and 57 mg/dL. The mean preinduction blood sugar level was 74.33 ± 10.31mg/dL in the GA group and 80.70 ± 12.09 mg/dL in the RA group with no statistical significant difference (Table 2). Metabolic response to surgical stress manifests as decreased glucose tolerance, increased glucose and cortisol levels intraoperatively. This is primarily mediated by the hypothalamo-pituitary-adrenal axis as explained in an update by Weismann.[4]

It has been shown that the extent of metabolic response was related to the duration of the surgery and its extent.[5] According to Houghton et al intraperitoneal surgery is known to cause the greatest stress response which is in turn due to the stimulation of the splanchnic nerve supply to the pancreas.[6] Extradural local anaesthetics and opioids blunt the surgical stress response by blocking the sensory output from the splanchnic nervous system.[7] Literature search revealed that spinal anaesthesia (Oyama et al[8], Moller et al[9]) and general

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anaesthesia (Oyama et al[10], Geisser et al[11], Baldini et al[12]) did not have a similar effect.

In the present study, blood sugar levels were measured at 90 minutes intraoperatively and at 3 hours and 6 hours postoperatively in both the groups. The mean intraoperative blood sugar was 94.60 ± 23.18 mg/dL in the GA group and 87.70 ± 15.33 mg/dL in the RA groups. The difference was not statistically significant (p = 0.795). However, the intraoperative blood sugar levels in both the groups were significantly higher than the preinduction blood sugar. This was in accordance with other studies concluded that there was no attenuation of surgical stress response to surgery under general[10,11,12] and spinal anaesthesia[8,9].

Oyama et al had demonstrated that there was a significant elevation of blood glucose during surgery under isoflurane anaesthesia.[10] Similarly, Geisser et al had shown that isoflurane cannot prevent the metabolic response to surgery in patients undergoing abdominal hysterectomy.[11] Patients in the present study who underwent general anaesthesia with isoflurane also showed significant elevation in blood sugar intraoperatively. The longer fasting duration in these patients didn't reduce the intraoperative blood sugar.

Oyama et al studied the effect of spinal anaesthesia on intraoperative blood sugar levels in patients undergoing surgery. They concluded that there was no attenuation of metabolic stress response under spinal anaesthesia.[8] This was further confirmed by Moller et al who concluded that spinal anaesthesia with sensory loss up to T4 had only transient effect on the metabolic response to stress. They also postulated that only by maintaining sensory blockade above the level of T4 in spinal anaesthesia could attenuate the elevation of blood sugar in response to surgical stress.[9] This was demonstrated in the study by Webster et al which showed less elevation in blood sugar levels in patients undergoing colonic surgery under continuous spinal anaesthesia up to T4 level than in patients undergoing surgery under general or extradural anaesthesia.[13]

In the present study, the patients in the RA group received subarachnoid block with hyperbaric bupivacaine. The height of sensory block achieved in 83% patients was between T8-T10 and was associated with elevation of intraoperative blood sugar in the RA group. There was no incidence of hypoglycaemia intraoperatively even though these patients had longer fasting times than earlier studies. Bromage et al have shown in their study that the hyperglycemic effect was known to persist up to 48 hours after surgery.[14] In the present study, the mean postoperative blood sugar levels at 3 hours and 6 hours in the GA group remained significantly higher than preinduction values (88.03 ± 20.40 mg/dL and 88.97 ± 18.64 mg/dL respectively). In the RA group the blood sugar at 3 hours postoperative was 88.83 ± 14.61 mg/dL which was significantly more than the preinduction value. At 6 hours, the blood sugar was 86.13 ± 10.65 mg/dL in the RA group, higher but not significantly different statistically from its preinduction value. The metabolisable glucose stores a normal 70 Kg male in the form of liver glycogen is only up to 75 – 100 gm while the central nervous system alone metabolises up to 115 gm/day. The glycogen stored in muscle cells amounts to up to 200 – 250 gm but is unavailable to other tissues.[15] Hence stored glucose alone is inadequate to maintain normal glucose levels in fasting patients. One of the mechanisms maintaining normoglycaemia in fasting patients is increased lipolysis and ketogenesis. This process provides an alternate source of energy for body tissues and to conserve glucose for obligate glucose metabolising tissues like brain and red blood cells. Ketonuria develops only when the level of ketone bodies in blood overwhelms the ketone metabolising pathways, usually at levels more than 12 mmol/L.[16]

In the present study 2 patients in the GA group and 4 patients in the RA group developed preinduction ketonuria. None of these patients had hypoglycaemia. Thus, it can be concluded that ketogenesis was already underway in these patients and this process was contributing to the maintenance of normoglycaemia. However, ketonuria occurs only when the blood level of ketones exceeds 12 mmol/L and doesn't correlate well with ketone levels in the blood. Thus, even in patients who had no evidence of ketone body in urine preoperatively, ketogenesis might have been underway.

Postoperatively, the patients were administered only Lactated Ringer's solution at a rate of 75 mL/hr and urine samples were analysed for ketonuria by dip sticks. The percentage of patients with positive ketones in urine was 20% at 3 hours and 60% at 6 hours in the GA group. In the RA group, 26.7% and 60% patients had positive ketones in urine at 3 hours and 6 hours respectively. These include the patients who had ketonuria at the previous interval. Since ketonuria is only an indirect method of assessing ketogenesis, and there are renal threshold like effects for ketones[16], the difference in the percentage of patients with ketonuria is not of clinical importance. However, these results indicate that in the absence of glucose, post operative patients maintain normoglycaemia by metabolising free fatty acids. One of the ways to prevent ketogenesis is to provide glucose. This would inhibit lipolysis and hence decrease ketogenesis. Metabolic effects of surgical stress persist in the early postoperative period lead to elevated blood sugar levels. Numerous studies (Allison et al[17], Brandt et al[18]) have shown that insulin secretion in response to hyperglycaemia is inhibited during surgery and this effect persists postoperatively.

The present study analysed whether water restriction and consequent dehydration due to prolonged fasting would have any effect on serum electrolyte levels in normal adult patients. Compared to the baseline value obtained from the preanaesthesia record, the preinduction and intraoperative as well as post operative values of serum sodium were higher but within normal range in both the groups. This difference was not statistically significant. Also, there was no significant difference between the groups. Thus dehydration due to prolonged fasting and surgical stress

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has only an insignificant effect on serum Na^+ levels in healthy adults undergoing surgery. In the presence of intact thirst mechanism, even a minor (3-4 mEq/L) increase in serum Na^+ above baseline values cause intense thirst. Though the patients in the present study developed thirst, they were not able to drink water and this explained the increase in their serum Na^+ levels. However, the period of fluid restriction was not long enough to cause clinical hypernatremia.

Serum K^+ levels are influenced by Glucose, Insulin, acid-base status and catecholamine levels.[19,20] Insulin causes shifting of K^+ ions intracellularly. Acidosis has the opposite effect. Catecholamines too shift extracellular K^+ intracellularly. It is well documented that serum levels of cortisol are increased before and during surgery under general anaesthesia[10], [11], [12] and spinal anaesthesia[9],[13]. Increase in serum cortisol levels is due to stress response to surgery. Surgical stress is known to cause a decrease in serum potassium by up to 0.5 meq/L. [19] Studies have shown that there is usually no change in insulin levels during surgery under general[10,11] and spinal anaesthesia[8]. Surgical trauma itself can lead to release of intracellular potassium into the extracellular compartment. The net effect of these processes is difficult to quantify.

The effect of dehydration consequent to prolonged fasting, if any, would be obscured by the effects of other factors. In the present study, mean serum K^+ levels were non-significantly lower than the levels as obtained from the PAC record at preinduction, 90 minutes post induction and 3 hours postoperatively in both the groups. The serum K^+ value at 6 hours postoperatively was higher that value in the PAC record in both groups. The difference was statistically significant only in the GA group. Also, the mean serum K^+ was comparable among both the groups at all points of time.

Specific gravity of urine was analysed at preinduction and at 3 hours and 6 hours post operatively. The mean urine specific gravity at preinduction was comparable in both groups (1.0203 ± 0.007 for GA group vs 1.0208 ± 0.0072 for RA group). The mean urinary specific gravity showed an increase in the postoperative period. On comparison within both the groups there was no significant difference between the two groups. However, the preinduction and postoperative values of specific gravity of urine were significantly higher than the value obtained from the PAC record. Normal urinary specific gravity ranges from 1.002 – 1.030. The specific gravity of glomerular filtrate is 1.007 – 1.010. Hence any value higher than this indicates relative dehydration [21]. The increase in specific gravity was because the patients in the present study had not taken any fluids during the prolonged fasting period.

This study indicated that patients who were inadvertently fasted for prolonged periods developed symptoms and signs in the perioperative period. The most common symptoms were thirst, dry mouth and concentrated urine. Hunger as a major symptom was reported by less than twenty percent of patients. The mean of the specific gravity of urine was on higher side of normal range, indicating relative dehydration.

Though normal adults are capable to maintain normoglycaemia during prolonged fasting. In the present study pre- induction hypoglycaemia was detected in three patients. These patients did not have ketonuria. Therefore it might be possible that lipolysis and ketogenesis had not occurred to a significant level in these patients to maintain normal blood sugar.

Intraoperatively, there was a statistically significant increase in blood sugar levels in patients undergoing surgery under both general anaesthesia and subarachnoid block. Metabolic response to surgical stress induced lipolysis, ketogenesis and gluconeogenesis contributed to maintaining normoglycaemia intraoperatively and postoperatively. Sixty percent of patients in each groups had ketonuria at 6 hours postoperatively.

During preoperative prolonged fasting the effects of anesthesia and surgery increase the levels of glucocorticoids that causes lipid metabolism, which may lead to ketoacidosis.[22]

It is well known that insulin resistance can occur preoperatively due to prolonged fasting, which may lead to development of severe metabolic stress and increase the severity of the underlying trauma.[23]

Recently, Glucose containing fluids have been advocated until 2 hours before surgery to reduce the incidence of thirst and hunger without evidence of increased risk of aspiration[24]. However, insulin resistance due to surgical stress response may lead to postoperative hyperglycaemia.

To avoid all these problems patients should be advised nil per orally as per institutional, national[25] or international guidelines[24]. Clinician should be more liberal in terms of clear fluid intake. Prolonged inadvertent or otherwise preoperative fasting should be avoided while waiting for surgery.

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