The use of Spinal Anaesthesia in Diabetic Patients

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Summary

The modifying effect of low spinal anaesthesia on hyperglycaemic and haemodynamic response to surgery in 22 insulin dependent diabetic patients (IDDM) was studied. No significant alterations from the preoperative valves of blood glucose levels, arterial blood pressure, and ECG pattern were noticed during surgery. These studies indicate the possible advantages of conducting surgery in diabetic patients under regional anaesthesia.

Key Words

Insulin dependent diabetic patients
Stress response to surgery.

Diabetes mellitus is a metabolic disorder caused by many environmental and genetic factors, usually acting mutually.[1] Surgery and trauma induce profound changes in endocrine function, characterized by an increase in the circulating concentrations of the catabolic hormones such as catecholamines, glucagon and cortisol and a concomitant decrease in plasma concentration of the anabolic hormones, insulin and testosterone.[2] The hyperglycaemia of surgery is the result of an increase in glucose production compared with the rate of utilization. This increase in blood glucose concentration is proportional to the severity of the surgical trauma.[3] According to a recent survey (W.H.O. 1981) the prevalence of diabetes mellitus in Malta is 7.7%. It has been concluded that diabetes mellitus is a major health problem in Malta.

It seemed appropriate to investigate and assess the effects of spinal anaesthesia on the perioperative glucose homeostasis in a population with a high prevalence of diabetes mellitus. This was the aim of our clinical study.

Patients and Methods

Twenty two insulin dependent diabetic patients (IDDM) with an ASA physical status classification II and III were given low spinal anaesthesia for lower limb surgery.

Premedication to all patients consisted of 5mg nitrazepam (Mogadon) orally at hour of sleep the night before surgery and 10 mg diazepam (Valium) administrated orally one hour before anaesthesia.

Table 1 summarises the surgical procedures and

TABLE 1. *Summarized kind of surgery, age and sex of the patients.*

No.	Kind of Surgery	Sex	Age
	Amputation of lower limb:		
1.	below knee	F	52
2.	"	M	83
3.	"	F	78
4.	"	F	56
5.	"	F	70
6.	above knee	M	60
7.	"	F	68
8.	"	F	64
9.	"	M	63
10.	"	F	71
11.	"	F	73
12.	"	F	74
13.	"	M	57
14.	"	F	75
15.	"	F	74
16.	"	M	81
17.	"	M	72
	Amputation of great toe and toes		
18.	"	F	84
19.	"	M	65
	Necrectomy of the foot and sole		
20.	"	F	59
21.	"	M	58
22.	"	F	61
Total 22		**Sex ratio F-M 14-8**	**Mean age: 68**

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patients data. Prior to the spinal anaesthesia all patients were infused with 500 ml of 1/2 Ringer lactate in 5% dextrose, with 8 i.u. plain Insulin, followed by 3ml kg^{-1} h^{-1} during surgery and in the first postoperative hour while patients were kept in the recovery room. In the ward patients were left free to take liquids orally.

Spinal anaesthesia: all patients were positioned in the lateral horizontal position. A midline approach was used to insert a spinal needle G 22 in the subarachnoid space and 3 ml 0.5% plain bupivacaine (Marcain) was then injected.

ECG was continuously monitored. All our patients preoperatively showed signs of some of the following: elevation of ST segment, left bundle branch block (LBBB), right bundle branch block (RBBB). Blood was drawn from an ante-cubital vein for determination of blood glucose (1) prior to the induction of spinal anaesthesia, (2) half an hour after commencing of surgery, (3) and one hour after surgery. Blood pressure was measured with an aneroid sphygmomanometer before induction of spinal anaesthesia and every five minutes thereafter during the surgery.

Results

No significant haemodynamic changes were recorded in any of the patients during the induction, surgery, and in the first hour following surgery. The maximum recorded fall in systolic blood pressure was approximately 4kPa (30 mm Hg).

Continuous ECG monitoring showed no significant alterations in the heart rate and the ECG patterns in all cases.

The values of blood glucose taken during the study period correlated well with preoperative levels.

Discussion

Pathophysiology of diabetes mellitus

Diabetes mellitus is a permanent disorder of glucose metabolism in which failure to use glucose properly leads to hyperglycaemia, glucosuria, hyperosmolarity, polyuria, and dehydration. Associated abnormal fat metabolism produces ketonaemia and ketonuria. Disordered protein synthesis and enhanced catabolism, is associated with muscle wasting.[4 5]

Glucose and free fatty acids are the primary and the immediate sources of energy for the body. In diabetes mellitus not only is carbohydrate metabolism seriously deranged, but simultaneously fatty acid synthesis is impaired. The diabetic relies, therefore, for his energy needs, on the metabolism of lipids, (stored or dietary) and so produces an excessive amount of ketone bodies. Ketosis and acidosis result. In the neutralization and excretion of these organic acids, potassium and sodium are lost. Some of the accumulated Acetyl-CoA is diverted to excess synthesis of cholesterol. Thus hypercholesterolaemia is a prominent feature of diabetes mellitus and associated with the grave consequences of accellerated atherosclerosis. Anabolic processes such as synthesis of glycogen, proteins and triglicerides are slowed while proteins and glycogen are catabolized for gluconeogenesis and energy. The impaired carbohydrate, fat and protein metabolism eventually involves all the endocrine glands but principally the anterior pituitary and adrenals. Growth hormone is necessary for metabolism of stored triglycerides but, unfortunately, the liberated fatty acids act as insulin inhibitors. Adrenal steroids are involved in gluconeogenesis. These metabolic alterations lead to pathologic changes in organs and tissues. At post mortem the pancreas in 90% of diabetics, shows hyaline (amyloid) degeneration, fibrosis, atrophy and lymphatytic infiltration of the islets. The liver of long-standing poorly controlled diabetics is often fatty and enlarged. The kidneys are usually the most severely damaged organs in the diabetic. Renal failure, usually due to renal microvascular disease, accounts for many of the diabetic deaths in both juveniles and adults. Any one or any combination of the following lesions may be found:

1) Glomerular involvment with three distinctive patterns: diffuse glomerulosclerosis, nodular glomerulosclerosis (Kimmelstiel-Wilson disease) and exudative lesions.
2) Arteriolosclerosis inducing so-called benign nephrosclerosis.
3) Pyelonephritis, sometimes with necrotizing papillitis.
4) Glycogen accumulation in the tubular cells and
5) Fatty change of the tubular cells.

One of the most threatening aspects of diabetes mellitus is the developoment of blindness as a consequence of retinopathy, cataract formation or glaucoma. Diabetic retinopathy is characterized by microangiopathy, exudates, proliferative changes and vitreous hemorrhages.

Diabetes mellitus is a significant cardiovascular risk factor, with higher levels of blood glucose directly related to the extent of vascular disease. This relationship is true for both the juvenile and adult onset diabetic, and on both groups premature microvascular disease is the major cause of death.[6]

Surgically-induced adverse hormonal and metabolic changes, if not prevented or rapidly treated may seriously harm the patient and prolong his convalescence. Modification of the neuroendocrine response to surgery may be attempted in two ways. This may be achieved either by afferent neuronal blockade with local anaesthetics, for example extradural or spinal anaesthesia, or by inhibition of hypothalamic function with large doses of opiates.[7 8]

Painful stimuli from the site of operation can be partly suppressed by profound anaesthesia but only drugs blocking the spinal cord itself or the afferent or efferent pathways in its neighbourhood can block it completely.[9 10]

In all our patients surgical analgesia was excellent. Haemodynamic stability was another feature during and after surgery in all our patients.

Absence of a significant hyperglycaemic response in our patients, we assume, was due to the afferent neuronal blockade.

Another beneficial effect of spinal anaesthesia in our patients was a prolonged postoperative pain-free period and a decreased requirement for opiate analgesia.

Our results would suggest that lower limb surgery under spinal anaesthesia is associated with mild changes in haemodynamic and metabolic response to surgery and could be a safer method of anaesthesia particularly in diabetic patients.

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