ACTA MEDICA IRANICA
Vol.23. 1981.p. 109-119

PLASMA GLUCOSE RESPONSE AS A MONITOR OF SYMPATHETIC ACTIVITY DURING ANESTHESIA AND SURGERY

Kang Soo Kim, M.D.* Richard N. Terry, M.D.**
Shahpoor Shahgoli, M.D.***

ABSTRACT:

Blood glucose levels measured at half hour intervals in a series of 45 major surgical procedures demonstrated a consistent hyperglycemia during the course of operation. Peaks were associated with periods of cardio-vascular stress.

In contrast, patients in whom autonomic activity was inhibited by ganglionic blockade demonstrated a consistent decrease in blood glucose.

Pharmacologic armamentarium for control of the hypermetabolic state induced by surgical/anesthetic stress is readily available.

* Resident in Anesthesiology
** Clinical Professor
***Visiting Professor
Department of Anesthesiology The Buffalo General Hospital

Open heart patients received 2mg/kg of morphine by drip during induction. Nitrous oxide-oxygen 60/40 was supplemented with d-tubocurarine or pancuronium as necessary to maintain apnea. Ventilation was controlled to maintain PaCo of 38-40 Torr. General surgical procedures were supplemented with meperidine as indicated.Sodium Nitroprusside or enflurane were employed briefly for control of hypertensive episodes on several occasions.

Surgical fluid losses were replaced by lactated Ringer's solution, albumin, fresh-frozen plasma and packed cells. Pump prime was with plasmalyte*. Dextrose 5% in water was used only as a drug-infusion vehicle.

Five patients of the general surgery group given pentolinium ganglionic blockade, 5-15mg. as necessary to maintain a systolic B.P. in the 60-80 Torr range,provided a measure of the relationship autonomic stimulation exerts in blood glucose response to trauma.

RESULTS

Mean blood glucose change in open heart surgical procedures where no dextrose infusion was employed (Figure I) was +31% from induction to by-pass, +129% during pump, followed by a consistent fall in the postbypass period to +39% of control (24 patients).

By contrast, where 5% D/W infusion(200-500cc)was used as drug vehicle(3 pts.)in by-pass proceedures,glucose reach +260 to +290% of control (Figure II). In each instance the hyperglucosemia was quantitatively related to the volume of dextrose infused.

During general surgical procedures (Figure III),in wh-

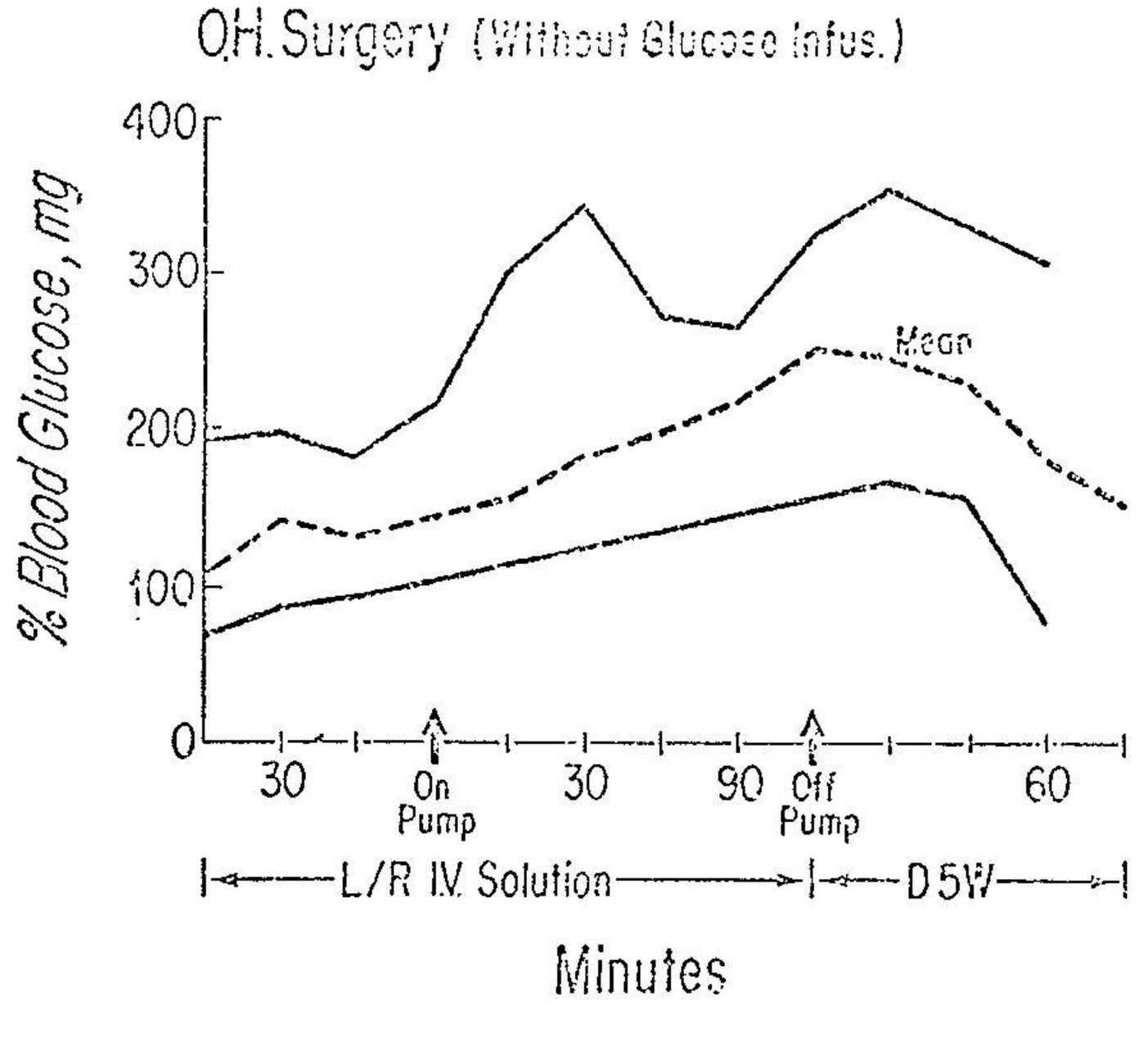

Figure 1

Blood Glucose response during open heart surgery no dextrose infused until after by-pass-A = maximum values, B = mean, C=lowest values for period.

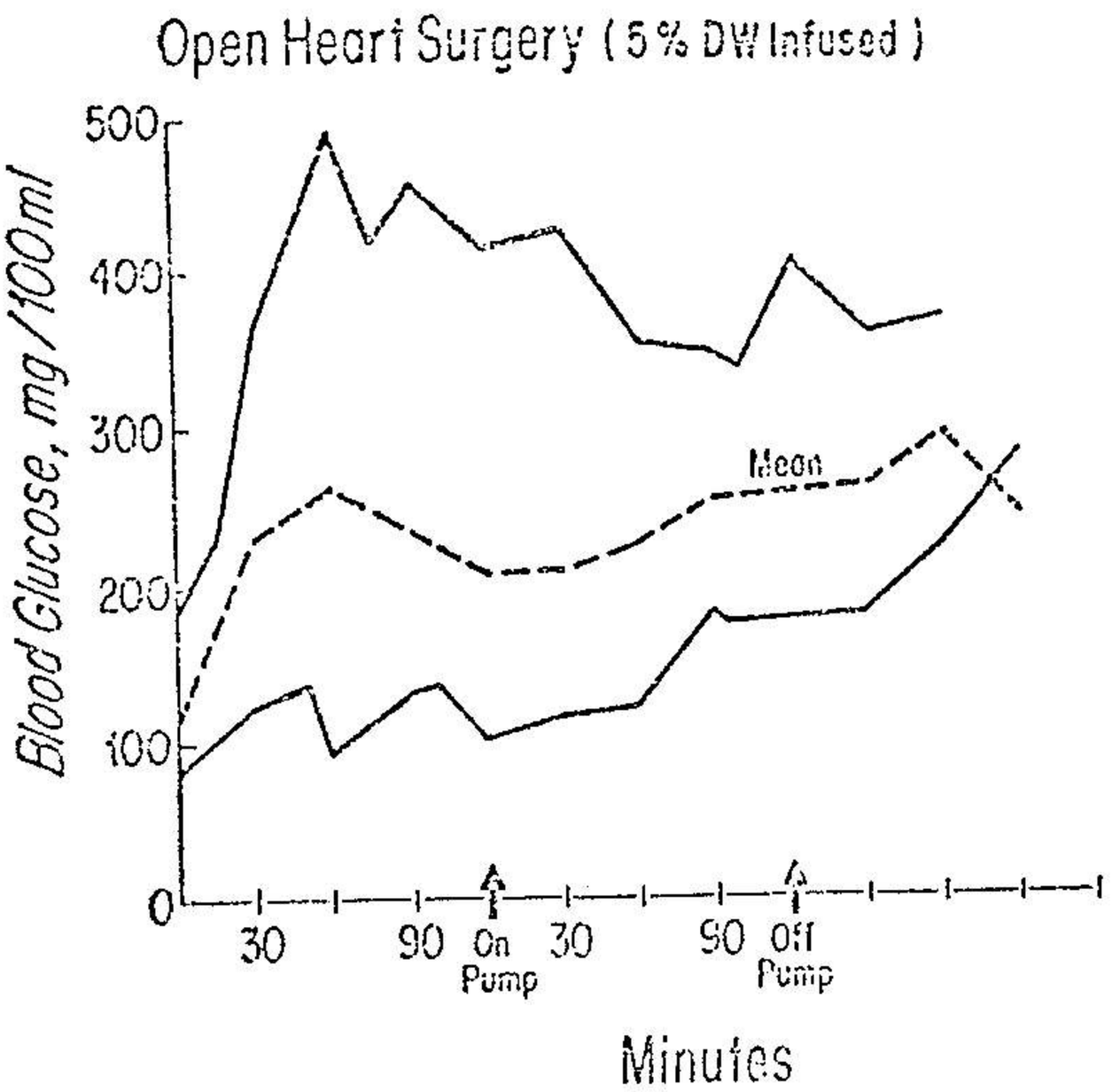

Figure 2

Blood Glucose response during open heart surgery D5W infused pre and post by-pass.

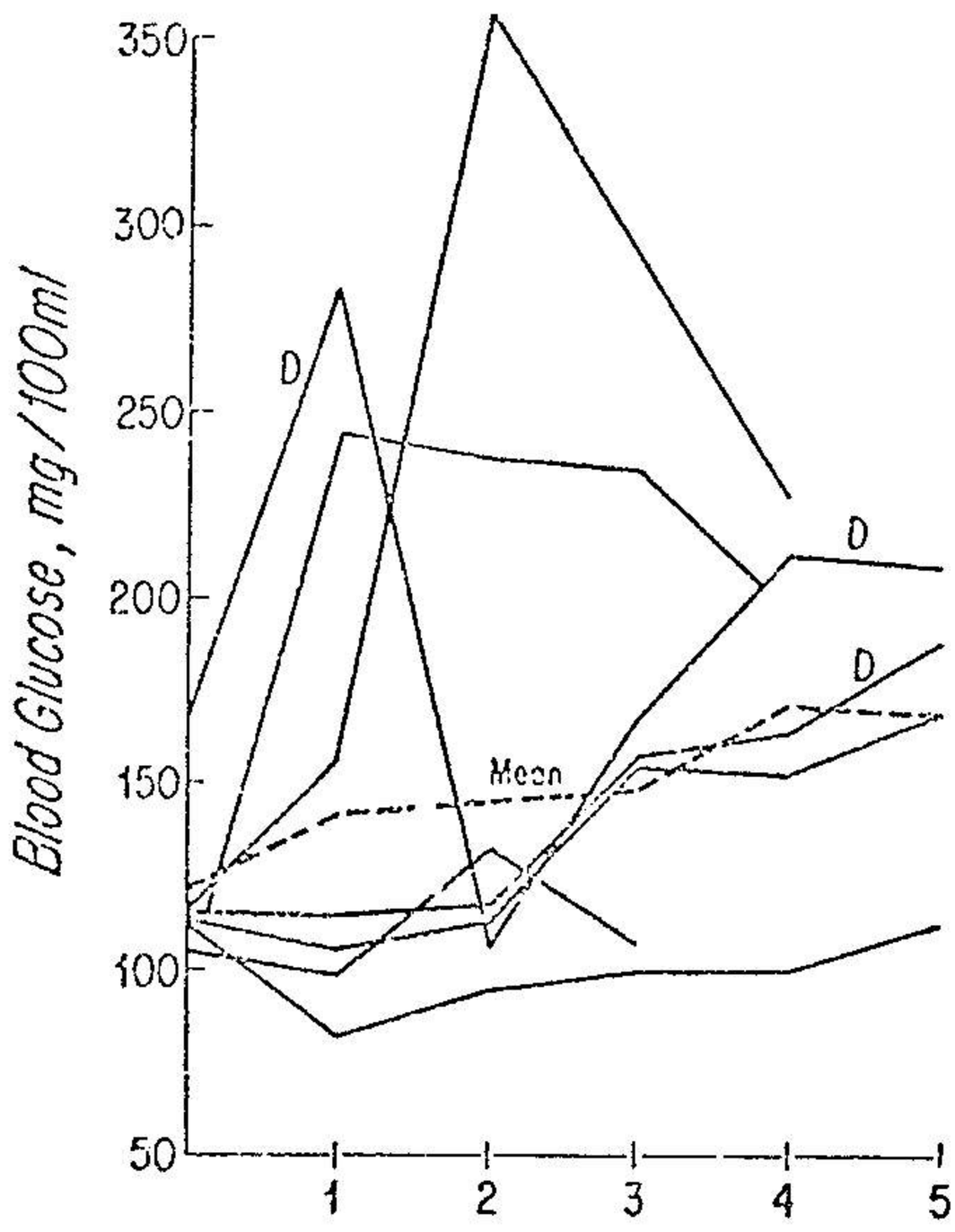

Figure 3

Blood Glucose responses during anesthesia, 13 major general surgical procedures ; no dextrose infused - D = known diabetic patient; C = cachectic patient.

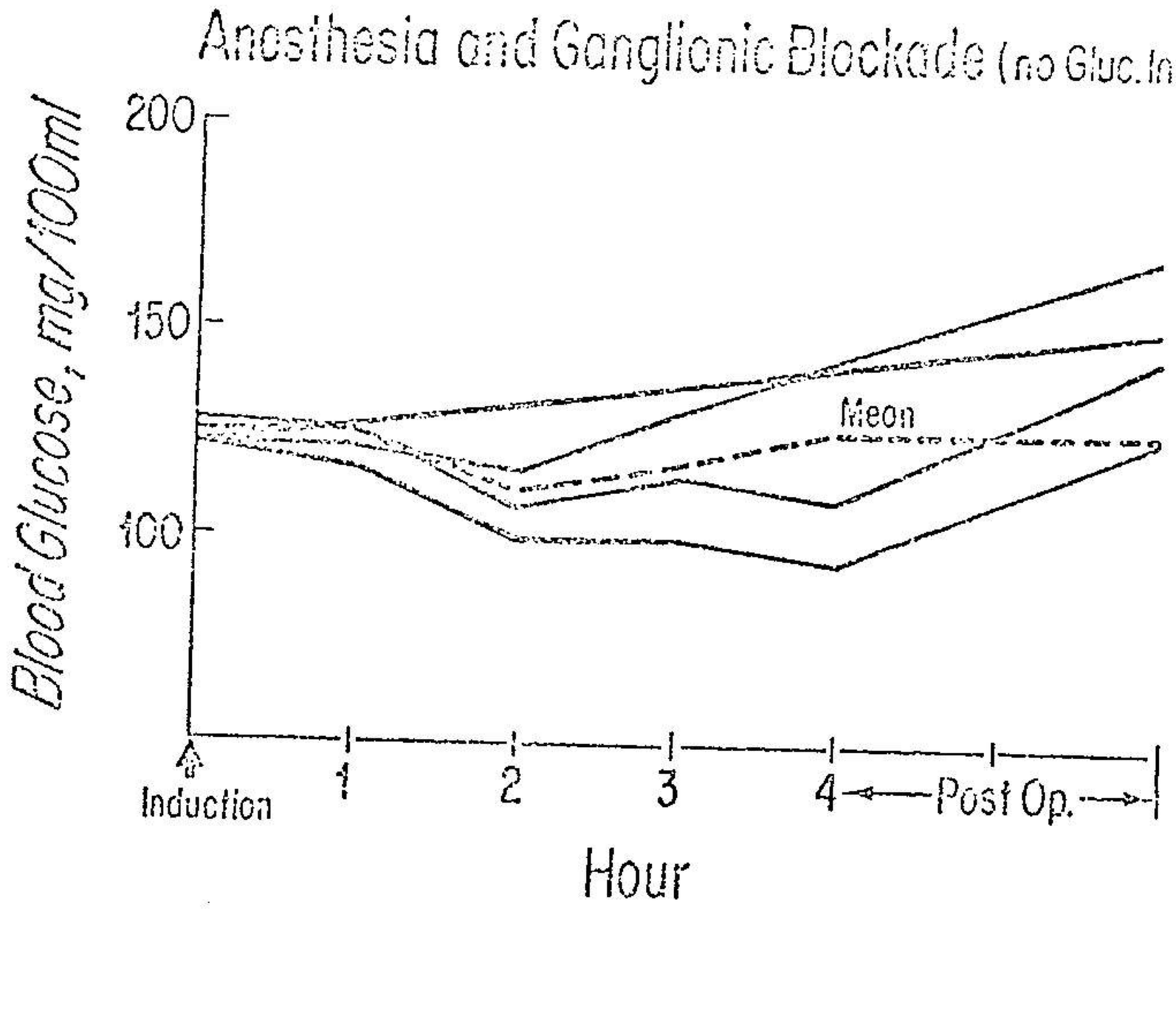

Figure 4

Blood Glucose response during anesthesia and ganglionic blockade for general surgical proceedures.

ich no dextrose was infused, plasma glucose levels rose progressively +17% after 1 hour, +21% after 2 hours,+22% at 3 hours and +41% at 4 hours, +40% during the 5th hour of anesthesia. The single exception in this group was a chronically cachectic cirrhotic male,whose glucose stores may have been so depleted as to be incapable of responding to stress stimulation.

By contrast, in the group of general surgical procedures done under pentolinium ganglionic blockade,blood glucose remained unchanged at 1 hour and consistently decreased overaging -12% from control at 2 hours, -7% at 3 hours, and -11% at four hours.

No consistent relationship was observed between changes in blood glucose, lacticacidemia, or potassium.

DISCUSSION

Since Claude Bernard in 1877 observed that dogs subjected to hemorrhagic shock developed hyperglycemia,a succession of investigators have noted elevated blood sugar in response to the spectrum of trauma.

During stress, cells respond by mobilizing glycogen reserves. Stress induced hyperglucogenemia is accompanied by insulin suppression, and later insensitivity, blocking utilization of sugars,("shock diabetes").Dextrose infusions add to already elevated plasma levels,failing to initiate insulin response.

Elevation of plasma glucose is initiated by the induction of anesthesia, (1)before surgical trauma is added.. The total response is related to the magnitude of surgi-

Plasma glucose response as a monitor of 117

cal stress, the greatest elevation reported during intra-abdominal operations(2).Dehydration,diuretics,sepsis,hypovolemia, and steroids contribute additionally to hyperglycemia.

Anesthetic-induced hyperglycemia is most pronounced with agents evoking catacholamine response (i.e., ether and cyclopropane). It has been observed, however, that anesthetic/stress-induced hyperglycemia may be aborted by epidural or subarachnoid anesthesia presumably through interruption of autonomic innervation of the adrenal (3). The absence of a hyperglycemic response to general anesthesia which we observed when ganglionic block was employed, thus might have been anticipated.

Stress induced hyperglycemia has been most consistently observed following organ ischemia. Such was the case in our series: within minutes of going on by-pass there was a consistent, abrupt, elevation of plasma glucose. That so rapid a response may be related to cerebral ischemia is implied from the work of Braith Waite(4) who noted decreased CBF in more than 50% of patients on initiation of by-pass, As the adverse of this phenomenon, we observed precipitous fall in blood glucose following resumption of pulsatile blood flow.

The consequences of the stress induced hyperglycemic phenomenon during surgery are not well documented.A number of potentially pathophysiologic states, however,must be considered:

1) Hyperosmolar coma, resulting from hyperosmotic hypervolemia, (at the expense of cellular water) inducing a cerebrocellular hypovolemic encephalopathy,prolonging recovery from anesthesia (5).

2) Metabolic acidosis, resulting likewise from cellular hypovolemia.

3) During compensatory restitution, the threat of hypoglycemic shock resulting from "over-shoot" insulin release.

4) The failure of cellular utilization of glucose,(insulin suppression) sets the stage for muscle-wasting protein catabolism(the alternative source of energy)of the post-surgical patient, referred to as "negative nitrogen balance" (6).

CONCLUSION

Blood glucose levels monitored at 30 minute intervals in a series of major surgical procedures showed a consistent hyperglycemia, increasing progressively during the duration of the operation.

By contrast, patients in whom autonomic activity during surgery was inhibited by ganglionic blockade demonstrated a consistent decrease in blood glucose.

The hypermetabolic state induced by surgical/anesthetic stress is seen as an inappropriate sympathetically mediated disruption of homeostasis. Pharmacologic control of excessive autonomic acitivity is readily available in ganglionic blocking agnets.

REFERENCES:

1) Wright, P.D., Henderson, K., Johnson,I.D.A.; Glucose utilization and insulin secretion during surgery in man. survey of Anesth. Vol. 18-No3-19.

2) Clarke, R.S.J.; The hyperglycemic response to different types of surgery and anesthesia. Brit. J. Anes. 42:45-52, 1970. Brit. J. Surg. 61:5-8, 1974.

3) Bevan, D.R.; Modification of the metabolic response to trauma under epidural anesthesia. Anaesthesia 26: 188-196, 1971.

4) Braithwaite, M.A.; Cerebral blood flow and metabolism during open heart surgery. Thorax 29:633-638, 1974.

5) Woods, J.E., Zincke, H., Palumbo, P.J., Johnson,W.J., Anderson, C.F., Fromel , P.P., Service, J.; Hyperosmolar non-ketonic syndrome and steroid diabetes occurrance after renal transplantation. J.A.M.A. 231:1261-1263, 1975.

6) Wilmore, D.M., Long, J.M., Mason, A.D.: Stress in surgical patients as a neurophysiologic reflex response. S.G. &O. 142:257-269. 1976.