ROLE OF SURGERY IN MANAGEMENT OF MORBID OBESITY

ESSAY SUBMITTED FOR PARTIAL FULFILLMENT OF MASTER DEGREE IN GENERAL SURGERY

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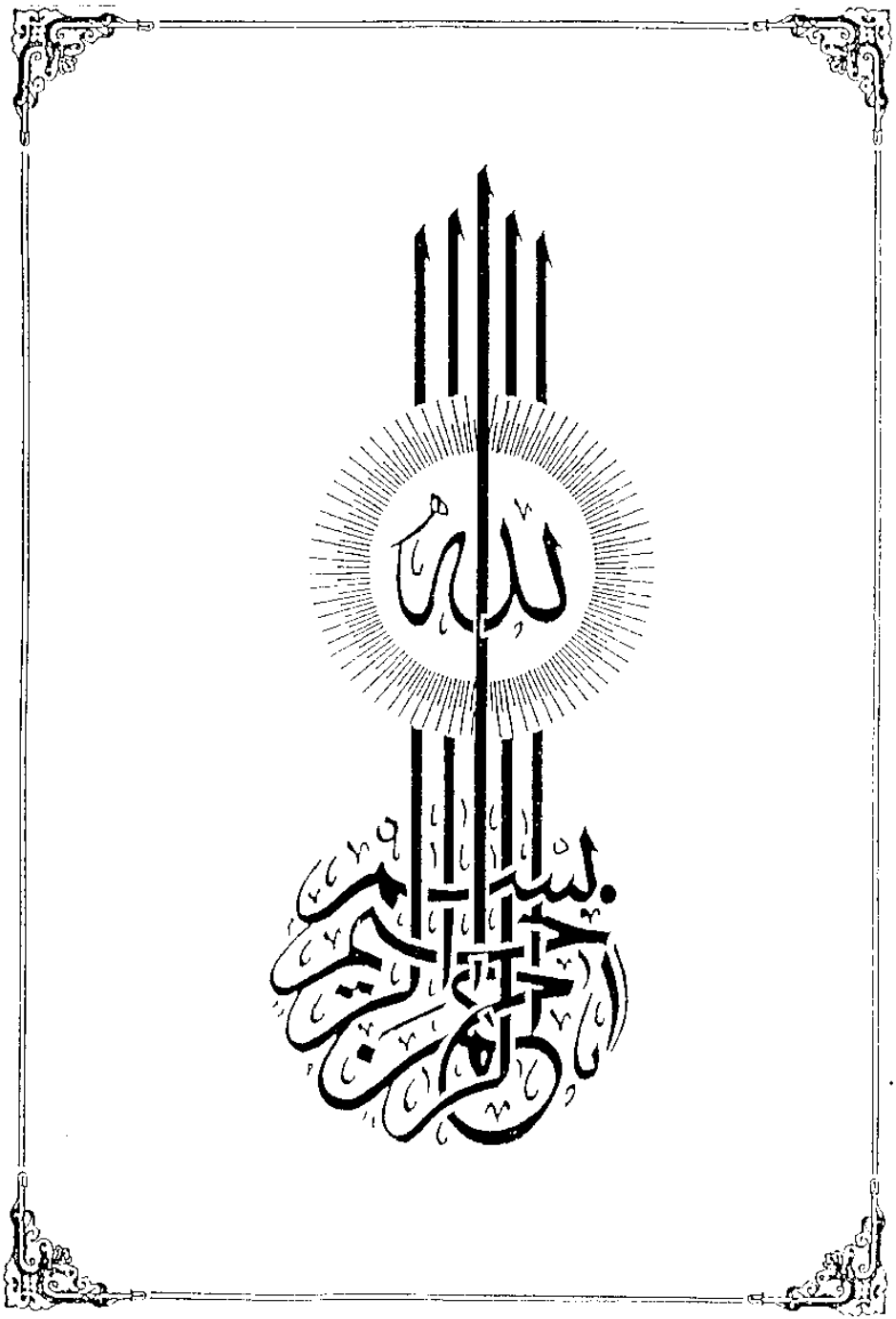

To

my parents

and

my brothers

Content

Physiology of feeding

*Regulation of food intake
*Mechanism of satiety

Physiology of Feeding

* Regulation of food intake:

1- Glucose utilization by the satiety center:

There is a considerable debate about the signals that are sent by the satiety and feeding centers to regulate food intake. The activity of the satiety center is probably governed in part by the level of glucose utilization of cells within the center. These cells have therefore been called glucostates. It has been postulated that when their glucose utilization is low, and consequently when the arterio-venous blood glucose difference across them is low, their activity is decreased. Under these condition the activity of the feeding center is unchecked, and the individual is hungry. When utilization is high, the activity of glucose is increased, the feeding center is inhibited and the individual feels sated.

(Ganong, 1995)

The glucostatic hypothesis of appetite regulation is supported by an appreciable body of experimental data.

Other factors undoubtedly affect appetite, but the glucostatic hypothesis has merit of explaining the increased appetite in diabetes, in which the blood sugar is high but glucose utilization by the cells is low because of the insulin deficiency. The objection has been raised that most neural tissue does not require insulin to metabolise glucose. However, the region of the ventro-medial nucleus has been shown to be different from the rest of the brain in that its rate of glucose utilization does vary with the amount of insulin in the circulation. (Ganong, 1995).

2- The limbic system:

The limbic system is also involved in the neural regulation of appetite. Lesions of the amygloid nuclei produce moderate hyperphagia.

The ascending noradrenergic fibers in the ventral bundle inhibit appetite, and discrete bilateral lesions in the bundle cause hyperphagia. Amphetamine may exert its inhibitory effect on appetite by causing the release of norepinephrine from the terminals of the ascending noradrenergic fibers. (Ganong, 1995)

3- Degree of distention of the stomach:

It has been shown that there are stretch receptors in the stomach whose response is related to the degree of distention, and which are probably situated in the smooth muscles of the stomach. They send afferent impulses through vagus nerve fibers and they are stimulated mechanically by the distention of the stomach.

It is apparent that at mealtimes or after intake of water, large number of these impulses will be exited, their number increase with the course of water or food intake. Maximal stimulation is achieved when the stomach is fully distended. Thereafter, the number of impulses continues to reach the brain at a fairly constant diminished rate but little, since the receptors are very slowly adapting, till the stomach begins to empty. The number of impulses will then gradually decrease until stomach is fully emptied when about a third of receptors are still active discharging spontaneously at about one to ten impulses per second.

It is suggested that gastric receptors are responsible for the immediate feeling of satiation of hunger and thirst, which increases, with progress of a meal or intake of water. When the number of impulses reaches a certain level, feeling of intake of water comes to an end.

(Paintal A, 1954)

4- Other factors:

There is evidence that the size of body fat depots is sensed by either neural or hormonal signals to

the brain, and the appetite is controlled in this fashion (lipostatic hypothesis). A cold climate stimulates and a hot environment depresses appetite. Especially in human, cultural factors, environmental, and past experience relative to sight, smell, and taste of food, also affect food intake. (Ganong 1995)

The sight and smell of food are important signals for initiating food seeking behavior and identifying potential sources of food. Along with the taste and textured of food in the mouth, these sensory signals and quality of food can serve both a positive feedback signals, initiating food ingestion, or negative signals to slow down, terminate, or abort an eating incident. (George, 1989)

Indeed obese subjects usually respond to external signals such as time of day, social setting and smell or taste of food to a greater extent than do persons with normal weight. (Olefsky, 1987)

* Mechanism of satiety:

1- Brain:

Ultimate control of feeding lies in the brain, and the major centers that control eating are in the hypothalamus.

Lateral hypothalamic nuclei (LHN) are the areas that produce active feeding. Discrete lesions in the LHN of rats cause diminished feeding and weight loss. (Arden, 1992)

There is an evidence that hunger is a constant state in most animals and that satiety is merely the interruption of that cycle. This would explain why certain animals are continuous grazers (e.g. the sheep and the goat).

Ventro-medial hypothalamic nuclei (VHN) are the satiety centers, and discrete lesions cause continuous feeding and obesity in rats (Xavier, 1992). Also, injection

of anesthetic into the VHN produces feeding in satiated animals.

It is not yet clear how these two centers interact, but peptide neurotransmitters may play a role.

Cholecystokinin (CCK) is a neuropeptide widely distributed in the brain. It has been shown to inhibit feeding when administrated in the ventricular system of the brain in a variety of animals.

Because CCK antibody stimulate feeding when administration in the brain. It has been persumed that CCK acts physiologically in the brain to cause satiety.

Neuropeptide Y (NP_Y), another central peptide found in various brain centers, is a potent stimulant of feeding in the brain of rats and dog. The physiologic importance of NP_Y in the brain is not known, but interact with CCK at the hypothalamic centers have been hypothesized.

Bombesin also affects feeding when injected into the ventricular system of rats, but its physiological significance is not clear.

Peptides that control appetite in the brain do not function through change in gastric emptying. CCK a central inhibitor of feeding, accelerates gastric emptying and therefore its satiety effects cannot be dependent on changes in gastric emptying, Although brain NP_Y accelerates feeding, it slows gastric emptying when administered in the brain.

Neurotransmitters:

Catecholamines:

The Catecholamines, and, beta-adrenergic stimulation inhibits eating behavior (Olefsky, 1987).

Norepinephrine, Serotonin, Histamine, Gamma-amino-butyric acid and a number of peptides may be

involved in transmission of information that regulates food intake and nutrient stores. Infusion of norepinephrine into the ventromedial hypothalamus can increase food intake and food stores. (George, 1989)

Serotonin also plays an important role in the regulation of food intake and nutrient stores. Tryptophan and 5-hydroxy Tryptophan, two precursors of Serotonin that increase its concentration at neuroeffector junction, both decrease food intake. Thus both epinephrine and Serotonin play important roles in regulation of food intake through centers located in the medial and lateral hypothalamus (George, 1989, Blundell, 1984).

Adrenal steroids: The development or progression of experimental obesity is reversed or attenuated by adrenalectomy. (Bray, 1987)

Food intake returns nearly if not completely to normal levels after adrenalectomy in genetically obese animals. Energy expenditure is also increased after adrenalectomy. (George, 1989)

Fatty acid serves as afferent vagal signals. Increased fatty acid oxidation by the liver is associated with a decrease in food intake (Langhans et al, 1985).

Another potential regulatory process in the control of adipose tissue is involving adipose tissue lipoprotein lipase **(ATLPL).** ATLPL hydrolyses fatty acids from the triglycerides of circulating triglycerides- rich lipoproteins. The released fatty acids are taken up by adipocytes, converted to triglycerides and stored.

Thus ATLPL participate in the storage of excess fat calories in the adipose tissue.

The satiety center functions by inhibiting the feeding center. It appears that the feeding center is chronically active and that its activity is transiently inhibited by

activity in the satiety center after the ingestion of food. It is not certain that the feeding center and the satiety center simply control the desire for food. (Ganong 1995)

2- Stomach:

Gastric distension is the physiologic signal that the stomach uses to control satiety. Recently, it has been shown that the amount of gastric distension that occurs during a normal meal is sufficient to cause satiety, regardless of whether it is caused by nutrient, inert, or balloon distension.

Gastric distension acts on stretch receptors that increase firing rate during a gastric load. Signals from these receptors may travel through the vagi to the tractus solitarius. Further connections to the hypothalamic satiety centers have not been demonstrated.

Gastric distension is an early immediate satiety signal to stop active feeding. The mechanism that controls the inter-meal intervals is less clear, although changes in gastric emptying may be important. The rate at which the stomach empties its contents controls the rest of the gut.

As nutrients pass the pylorus, a variety of peptides are released that further slow gastric emptying. Both CCK and peptide YY (P_YY) are released by intestinal nutrients and are potent inhibitors of gastric emptying. This delay in gastric emptying may perpetuate the sensation of satiety associated with gastric distension.

(Power M., 1989)

3- Small intestine:

There are several mechanisms by which the small bowel is thought to mediate satiety. There are osmo-receptors in the duodenum that may cause satiation.

Recently there has been proof that peripheral CCK may be taken up by the brain to produce its satiety action.

The area postrema contains a deficient blood brain barrier that brings brain tissue in close proximity to the peripheral circulation. (Power M., 1989)

4- Other hormones that affect satiety:

Pancreatic polypeptide is produced by the delta cells of the pancreatic islets and is released in response to feeding. Whether pancreatic polypeptide deficiency is the cause of obesity or just a marker is not clear. (Power M., 1989)

Nutrient and hormonal signals may act on both the liver and the brain:

Glucose injected in the portal circulation decreases vagal afferent firing rate, probably through hepatic glucose receptors. (Geary et al, 1986)

Also the increase in plasma glucose after meal may activate the satiety center. (Olefsky, 1987)

Glucagon may act in the liver and other intestinal receptor system to initiate satiety, and this effect is partially abolished by vagotomy. (Geary et al, 1986)

It also suppresses the hypothalamic-feeding center. (Guyton, 1991)

Insulin Receptors are insulin sensitive. The satiety center may be activated by increase of glucose and insulin in plasma that follow a meal. (Olefsky, 1987)

Insulin response to carbohydrate would curtail the release of fat from the depot by its antilipolytic effect. (Flatt, 1987). Increased insulin levels are characteristic of obesity, moreover insulin injections can increase food intake probably by lowering glucose concentration (George, 1989). For unknown reason, presence of food in the stomach and duodenum increase secretion of insulin, which suppress the hypothalamic-feeding center. (Guyton, 1991)

Obesity

*Definition
*Prevalence of obesity
*Measurement of obesity
*Etiology of obesity
*Classification of obesity
*Differential diagnosis of obesity
*Complication of obesity