

Blessing, Chapter 7: Eating and Metabolism, Diagrams

[from Blessing, William W. (1997) *The Lower Brainstem and Bodily Homeostasis*. Oxford University Press. New York. pp. 323-372].

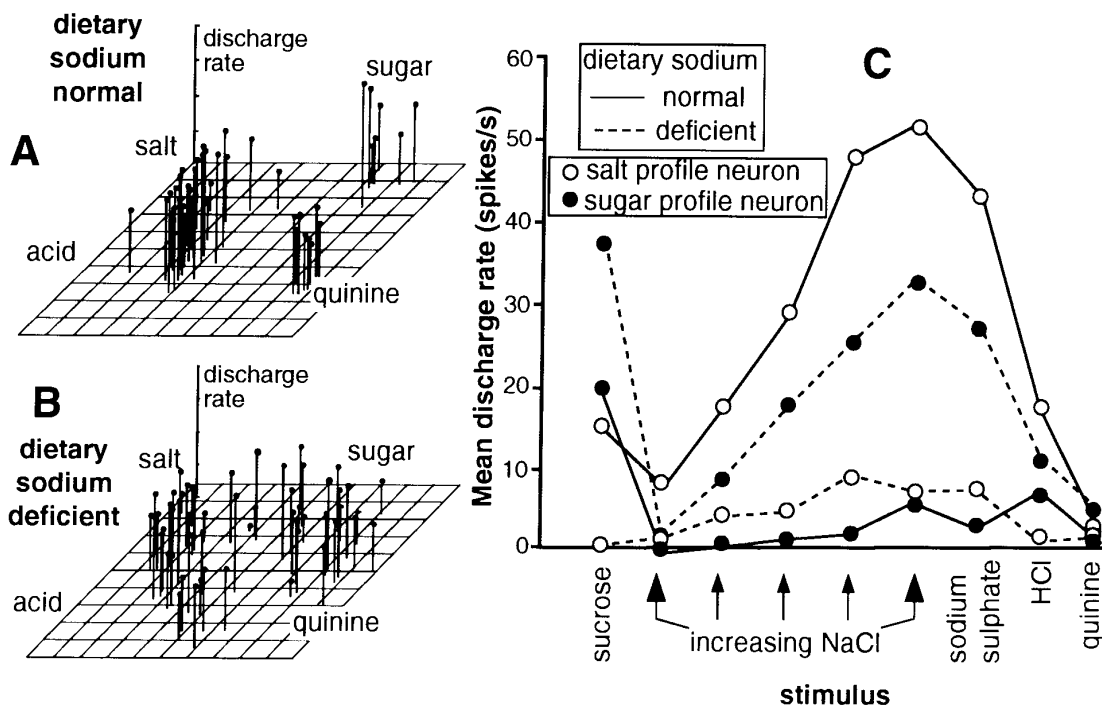


Figure 7.1 Effect of dietary sodium deficiency on nTS neuronal discharge to salt, acid, sugar, and quinine stimuli in rats. **A**, Multidimensional neuron space obtained in sodium-replete rats, demonstrating clusters of neurons discharging in response to specific stimuli. **B**, In sodium-deficient rats the clusters are less distinct, with neurons that respond to sodium also responding to sugar. **C**, Histogram showing mean net discharge rates across cells with salt and sugar profiles in sodium-replete and sodium-deficient rats. Sodium deficiency reduces the discharge of salt-profile neurons to salt stimuli and increases the discharge to salt of neurons that also respond to sugar. (Modified from Jacobs et al., 1988.)

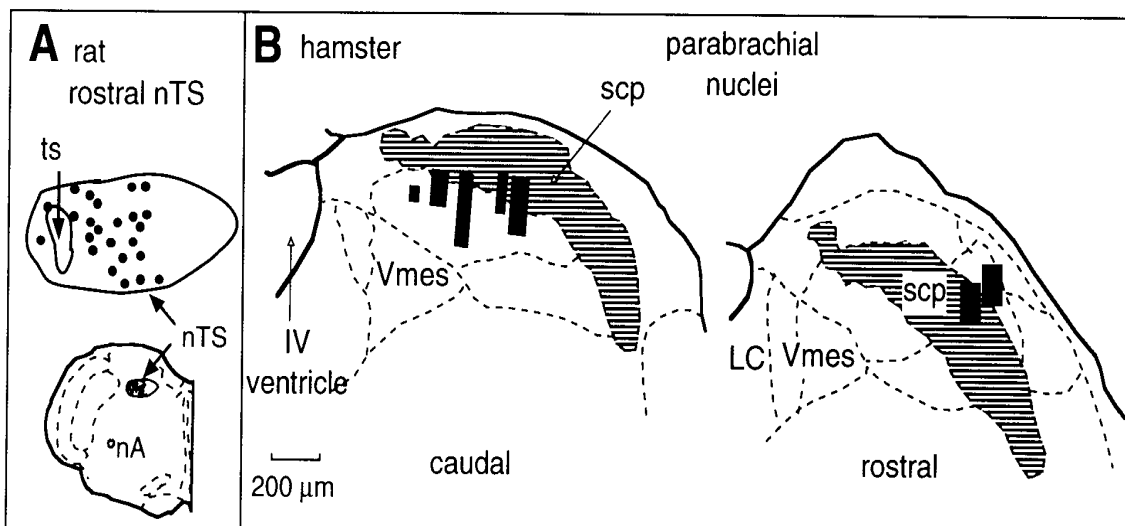


Figure 7.2 Location of taste responsive neurons in the nTS and in the parabrachial nucleus determined by extracellular recordings. (Modified from McPheeters et al., 1990, and Halsell and Frank, 1991.) Abbreviations listed on pages xiii-xiv.

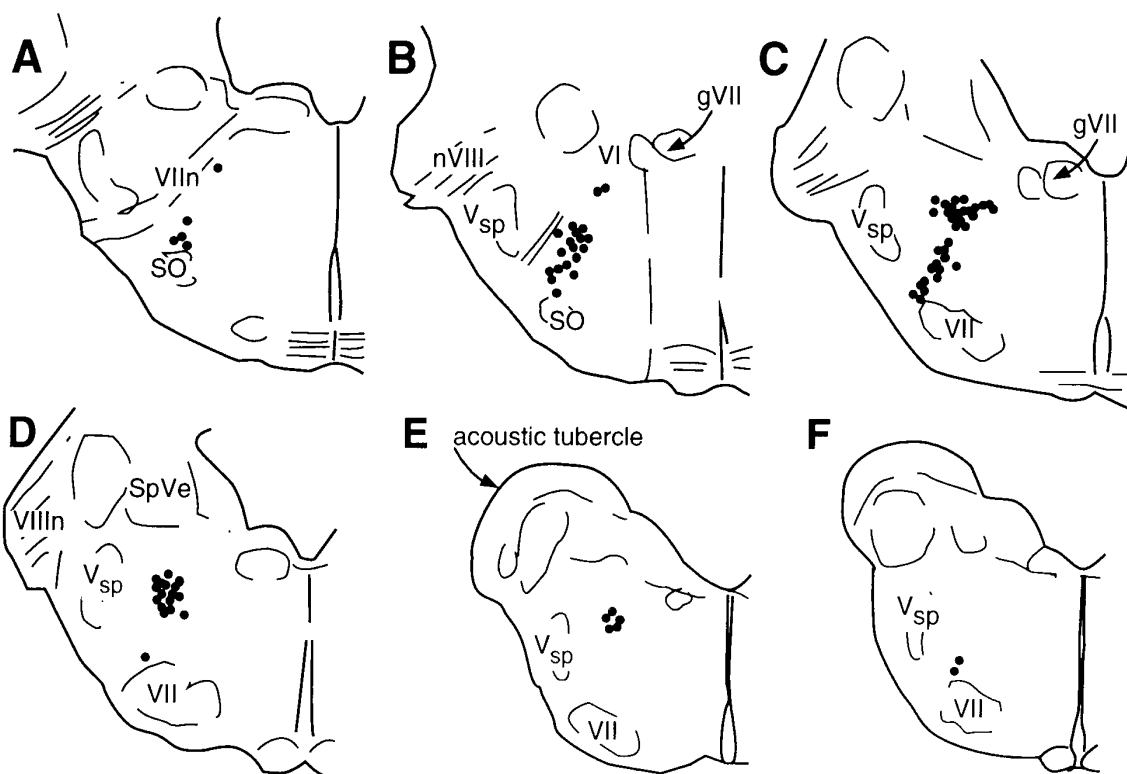


Figure 7.3 Location of parasympathetic preganglionic neurons projecting to submandibular salivary gland in rabbit. (Modified from Matsuo et al., 1980.) Abbreviations listed on pages xiii-xiv.

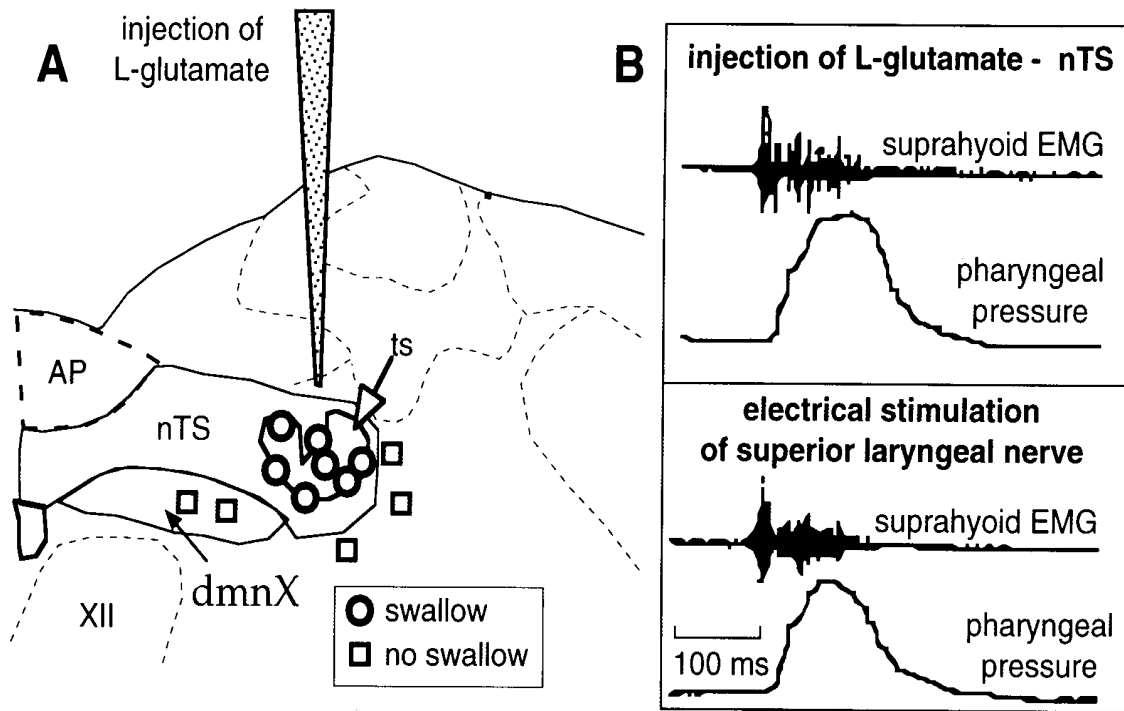


Figure 7.4 A, Regions of the rat nTS in which microinjection of L-glutamate induces swallowing. B, Similarity between swallowing elicited by injection of L-glutamate into the nTS and by electrical stimulation of the afferent superior laryngeal nerve. (Modified from Kessler et al., 1990.) Abbreviations listed on pages xiii-xiv.

Figure 7.5

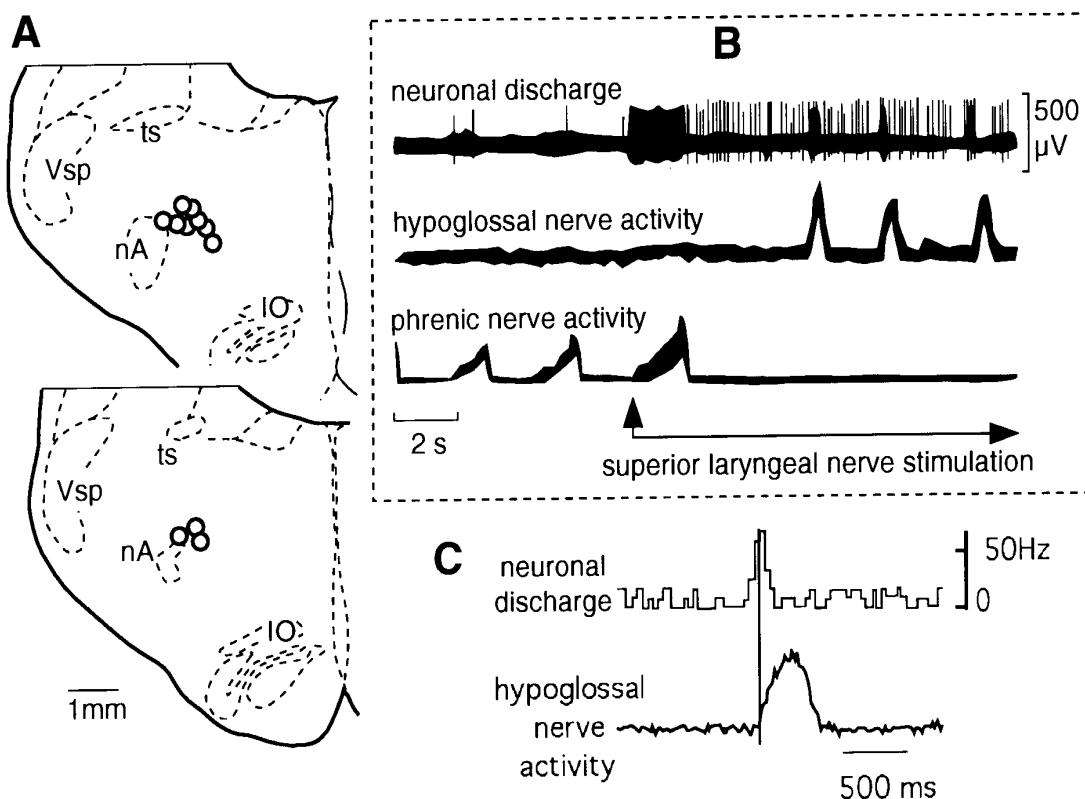


Figure 7.5 **A**, Distribution of ventrolateral medullary interneurons (open circles) activated orthodromically by stimulation of the superior laryngeal nerve (SLN) and active during the fictive swallowing that SLN stimulation induces. **B**, Discharge of one neuron orthodromically activated by electrical stimulation of the SLN. **C**, Relationship between discharge and hypoglossal nerve activity. (Modified from Ezure et al. 1993.) Abbreviations listed on pages xiii-xiv.

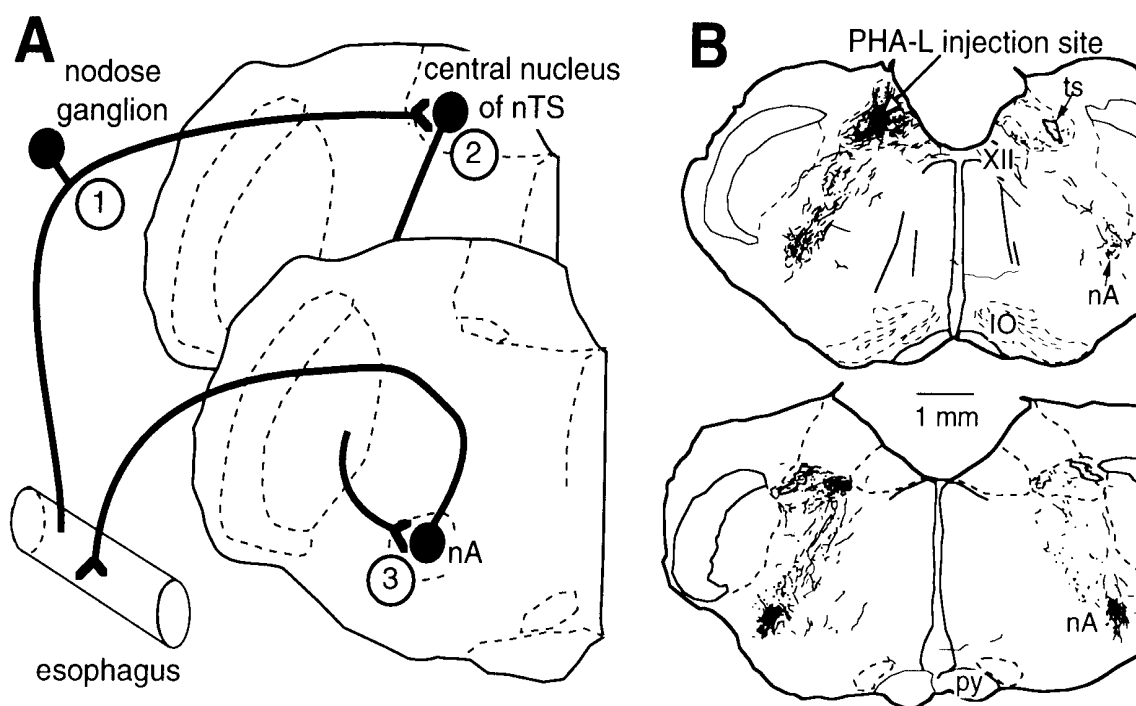


Figure 7.6 **A**, Diagram summarizing a single interneuron CNS pathway mediating esophagoesophageal reflexes. Neuron ①, the primary sensory neuron in the nodose ganglion, projects into the brain and synapses on neuron ②, a nitric oxide-synthesizing neuron in the central nucleus of the nTS. This neuron projects directly to a nucleus ambiguus motoneuron (neuron ③), which innervates the esophageal striated muscle. **B**, Neuroanatomical study in rabbit showing anterograde transport of *Phaseolus vulgaris* leucoagglutinin (PHA-L) from the central nucleus of the nucleus tractus solitarius to the nucleus ambiguus. (Modified from Yu et al., 1996a.) Abbreviations listed on pages xiii-xiv.

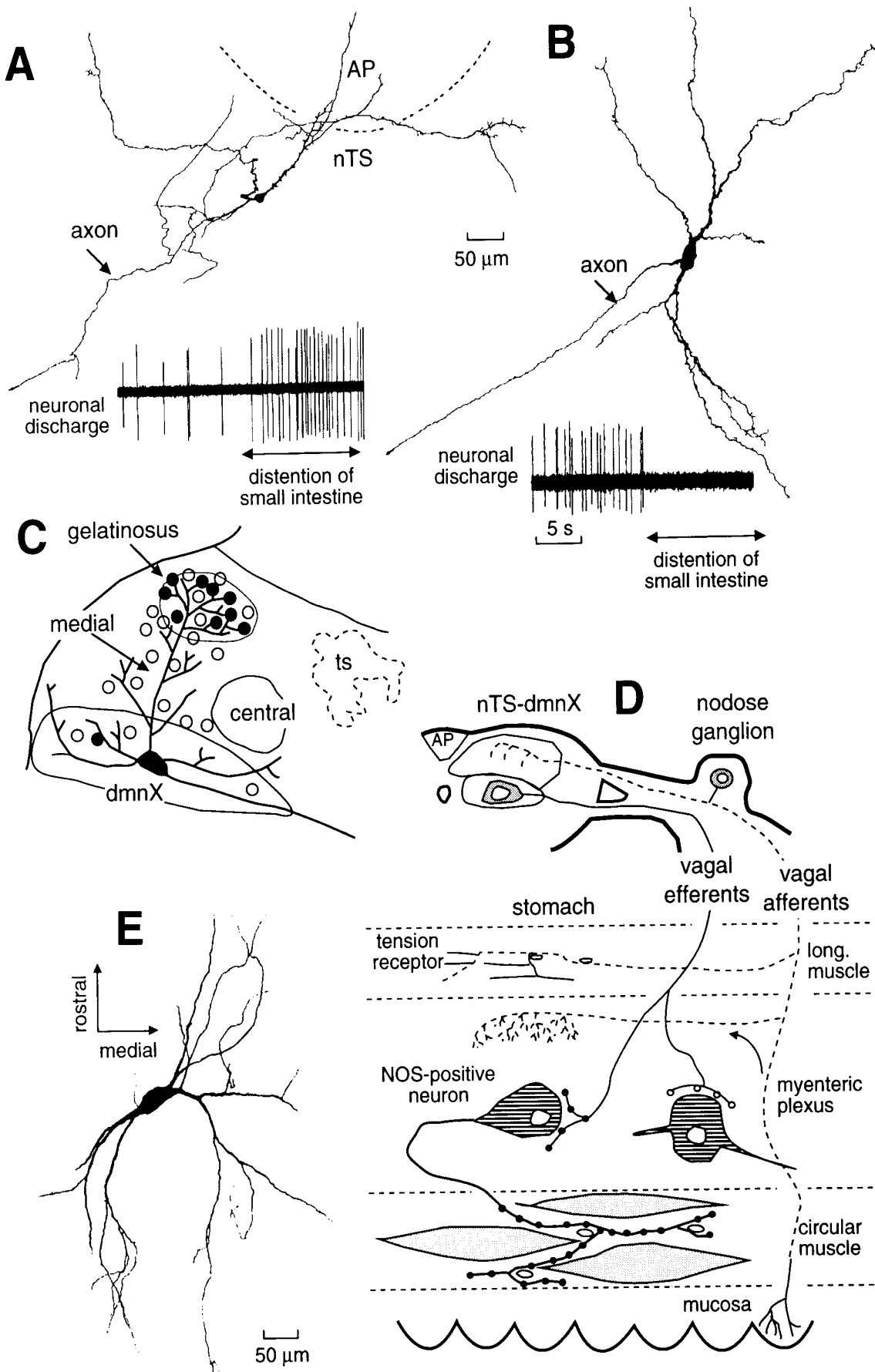


Figure 7.7 A,B, Intracellular fill of an nTS neuron with discharge is increased by intestinal distention and a dmNX neuron with discharge inhibited by intestinal distention. (Modified from Zhang et al., 1992.) C, Summary of the nTS-dmNX neuroanatomical interrelationships after injection

of cholera toxin-HRP into the stomach. Gastric vagal sensory axons terminate almost exclusively in the medial and gelatinous subnuclei of the nTS. Open circles represent vagal afferent boutons not demonstrated to be in synaptic relationship with dmX dendrites. Vago-vagal monosynaptic connections are indicated as closed circles. Some vagal afferent boutons form monosynaptic connections within the dmX. (Modified from Rinaman and colleagues, 1989.) **D**, Diagram of connections between enteric nervous system in the rat fundic stomach and the nTS-dmX. Vagal efferents innervate myenteric neurons, some of which contain nitric oxide synthase (NOS). Collaterals of vagal afferents also enter the myenteric plexus, possibly participating in local reflex activity. (Modified from Berthoud, 1995.) **E**, intracellularly filled dmX neuron, initially identified by retrograde transport of tracer applied to the celiac branch of the nerve in the abdomen. The soma and dendrites ramified principally in the horizontal plane. (Modified from Fox and Powley, 1992.) Abbreviations listed on pages xiii-xiv.

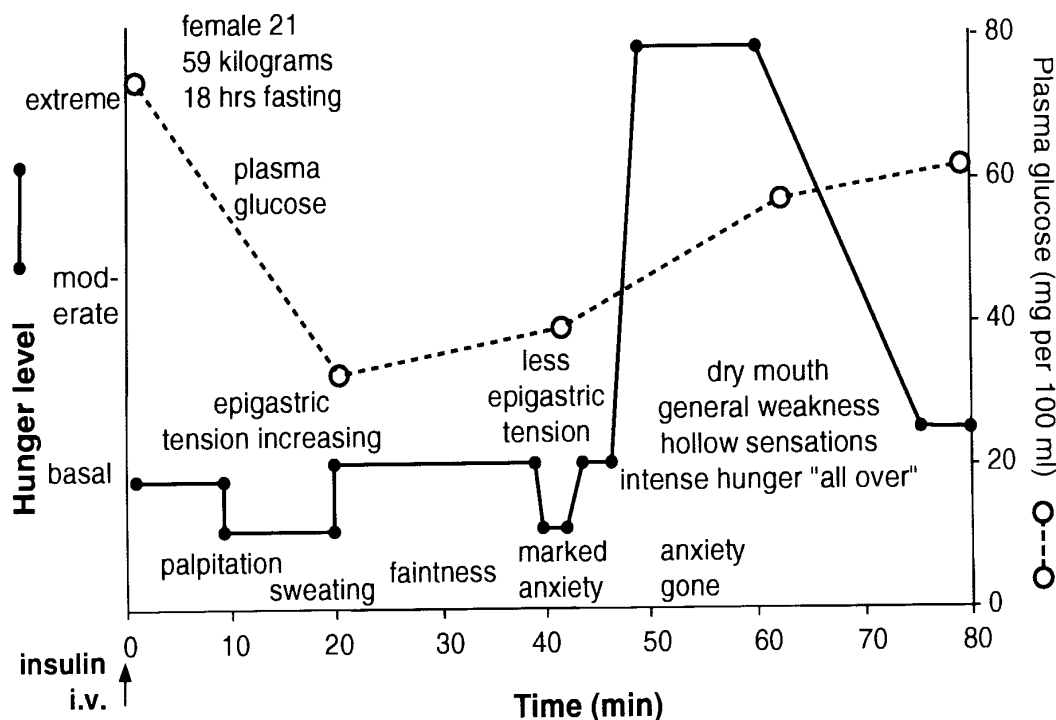
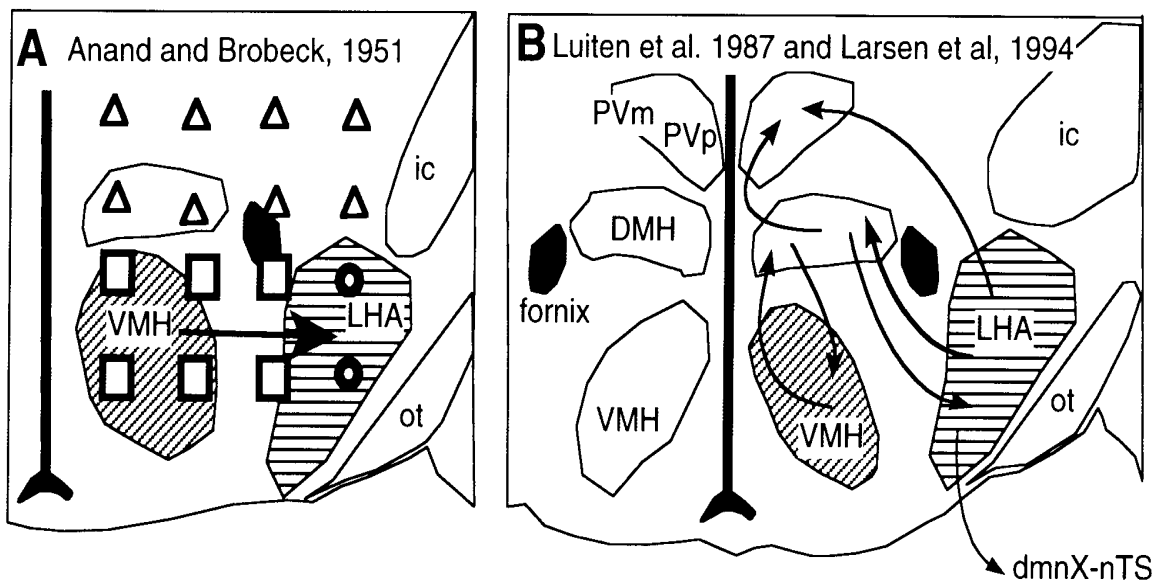
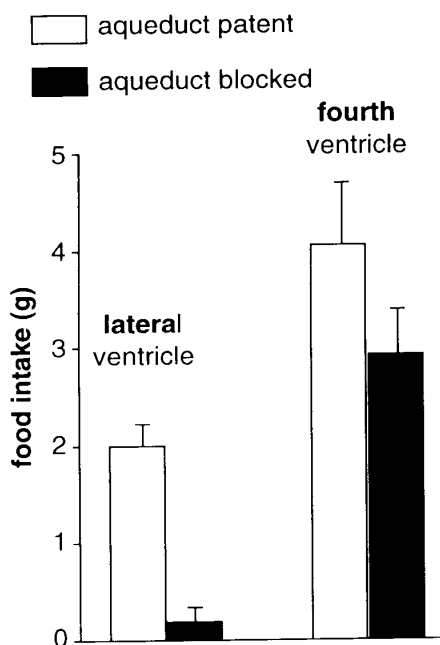


Figure 7.8 Relationship between change in blood glucose level and hunger sensation in a human subject after intravenous administration of insulin. (Modified from Janowitz and Ivy, 1949.)

Figure 7.9 A, Anatomical relationships in hypothalamus according to theoretical views of Anand and Brobeck, based on hypothalamic lesion studies. Triangles, normal food intake; squares, hyperphagia; circles, no food intake. **B**, Intrahypothalamic anatomical relationships based on axonal tracing experiments by Luiten et al. (1987) and Larsen et al. (1994.) Abbreviations listed on pages xiii-xiv.



A effect on food intake



B effect on blood glucose

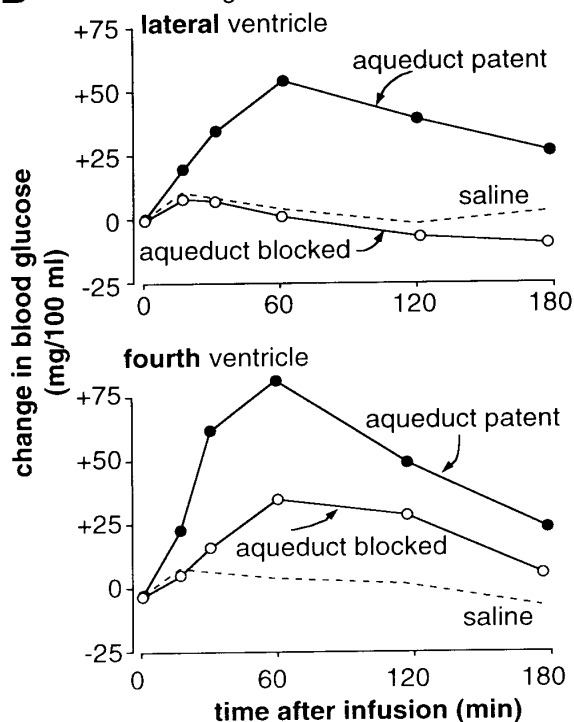
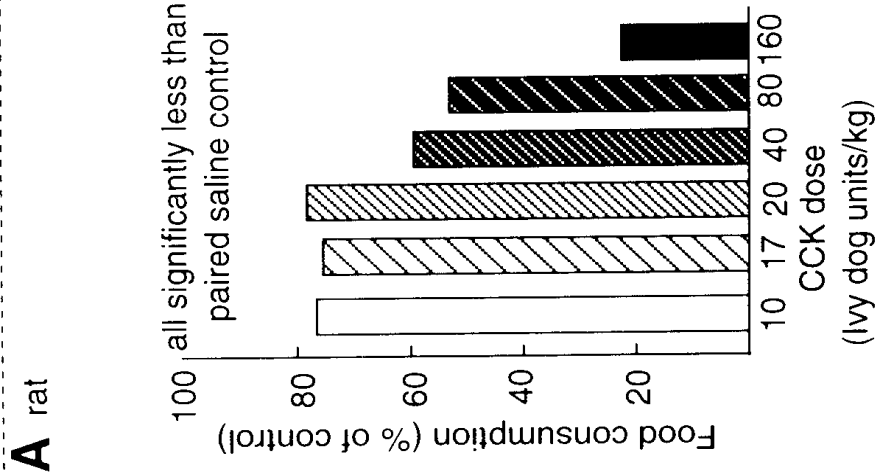
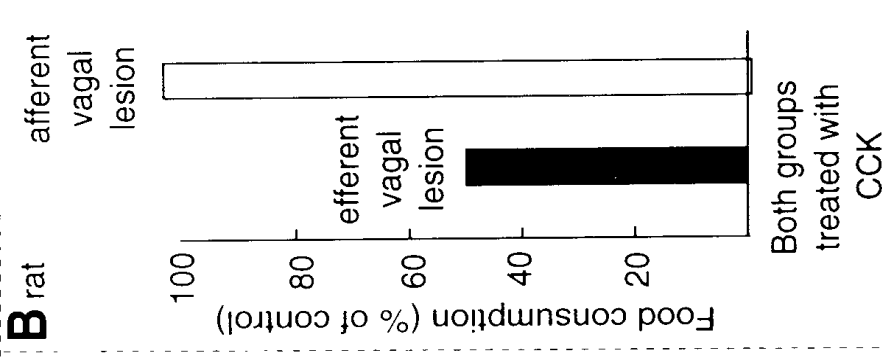


Figure 7.10 **A**, Infusion of 5-thioglucon into the lateral ventricle of rats increases food intake when the aqueduct is patent but not when it is blocked. Infusion of 5-thioglucon into the fourth ventricle increases food intake regardless of whether the aqueduct is patent or blocked. **B**, Blood glucose is affected in a similar manner. The aqueduct must be patent for an intraventricular infusion of 5-thioglucon to increase blood glucose, indicating that the relevant receptors are in the vicinity of the fourth ventricle and therefore probably in the hindbrain. (Modified from Ritter et al., 1981.)

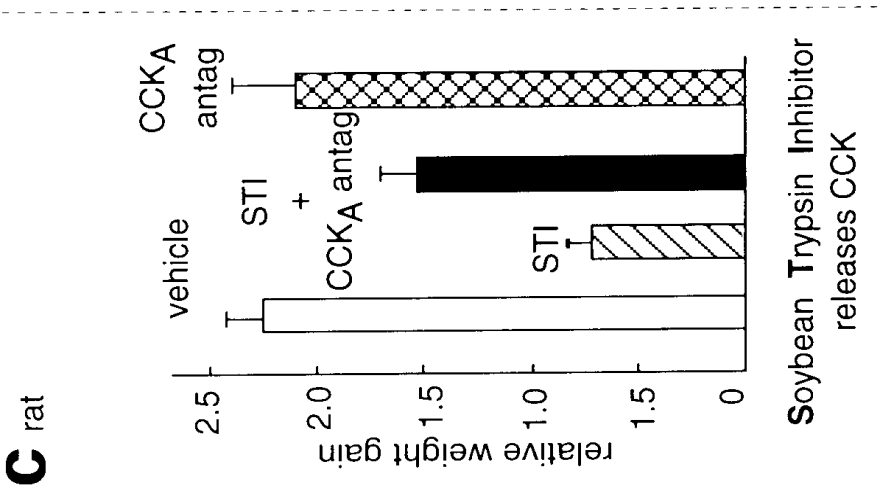
A rat
CCK reduces food intake
in rats with open gastric fistulae



B rat
CCK effect mediated by
vagal afferents



C rat
endogenous CCK-induced satiety
reversed by a CCK_A antagonist



D rhesus monkey
CCKA receptor antagonist
increases food intake

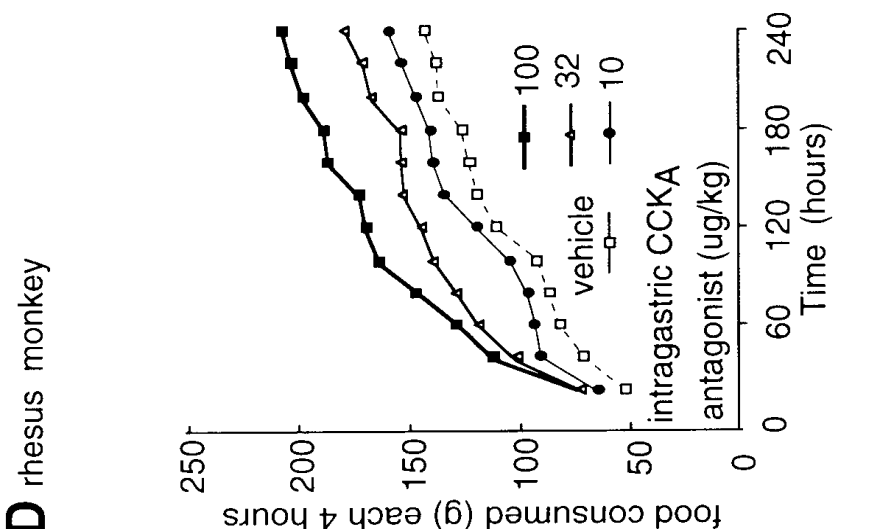


Figure 7.11 **A**, Intraperitoneal administration of CCK reduces the increased consumption of food (taken as 100%) in rats with chronic gastric fistulae. (Modified from Gibbs et al., 1973.) **B**, Effect on food consumption in CCK-treated rats with chronic gastric fistulae of selective bilateral sectioning of either the afferent or the efferent medullary rootlets of the vagus nerve. After an afferent lesion, the suppressive effect of CCK is abolished, and rats continue to eat an increased amount. After an efferent lesion the suppressive effect of CCK on food consumption is still present. (Modified from Smith et al., 1985.) **C**, Soybean trypsin inhibitor (STI) releases endogenous CCK from the upper gastrointestinal tract, thereby decreasing food intake, an action reversed by concurrent administration of a CCK_A receptor antagonist. (Modified from Weller et al., 1990.) **D**, Administration of a CCK_A receptor antagonist increases food intake in rhesus monkeys in a dose-dependent manner. (Modified from Moran et al., 1993).

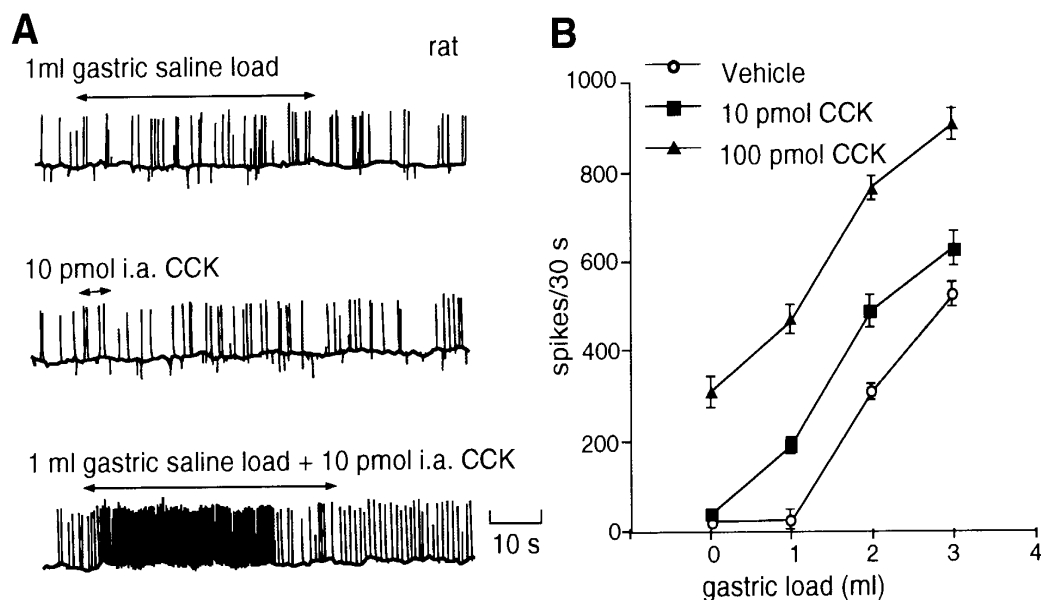


Figure 7.12 **A**, Electrophysiological recording from a representative gastric vagal afferent fiber in response to a 1 ml gastric saline load (top panel), a close arterial infusion of CCK (middle panel), or a combination of both these stimuli (lower panel). **B**, Interaction between CCK and gastric saline load in increasing the number of afferent vagal spikes. (Modified from Schwartz et al., 1993b.)

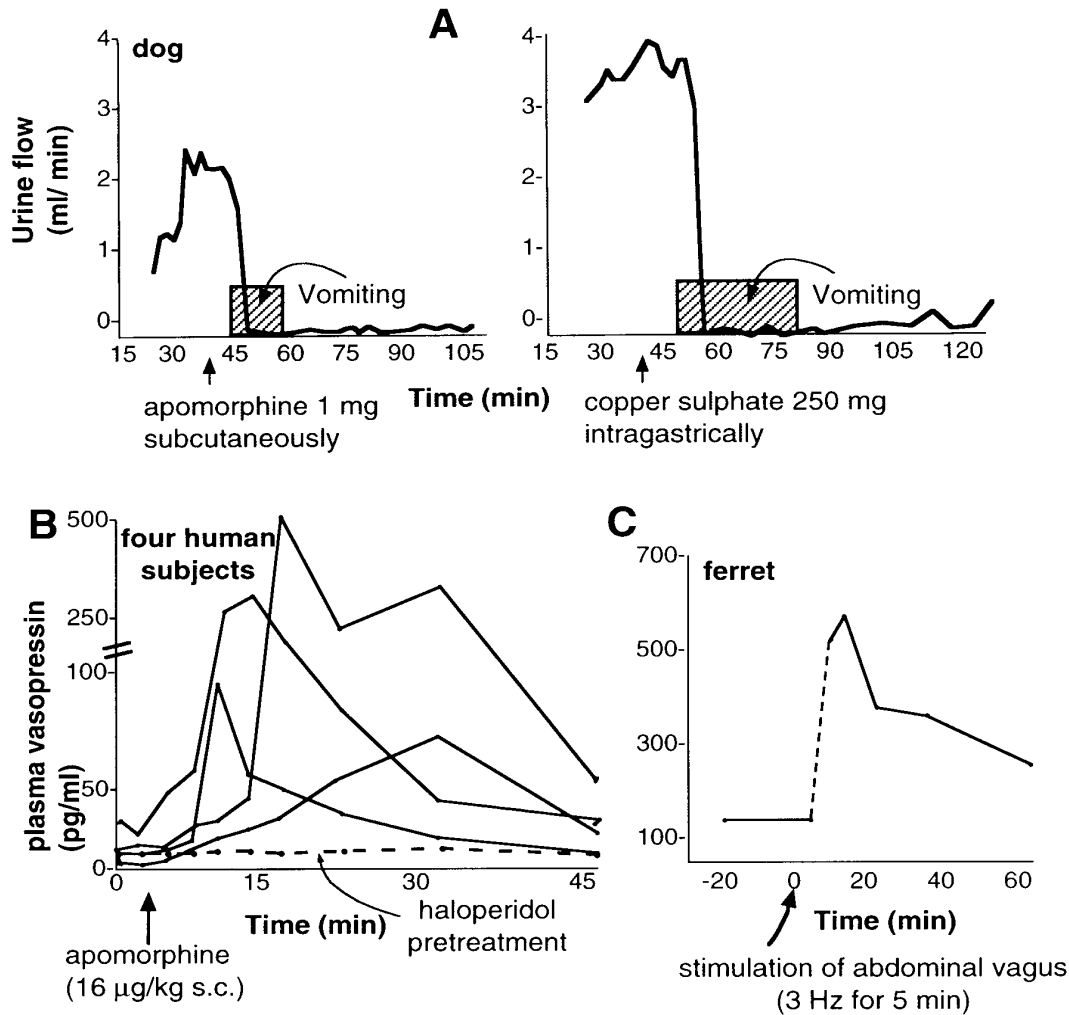


Figure 7.13 A, Effect of emetic agents on vasopressin secretion in the dog. (Modified from Andersson and Larsson, 1954.) B, Plasma vasopressin after administration of apomorphine in human subjects. (Modified from Rowe et al., 1979.) C, Plasma vasopressin after electrical stimulation of the afferent abdominal vagus nerve in anesthetized ferrets. (Modified from Hawthorn et al., 1988.)

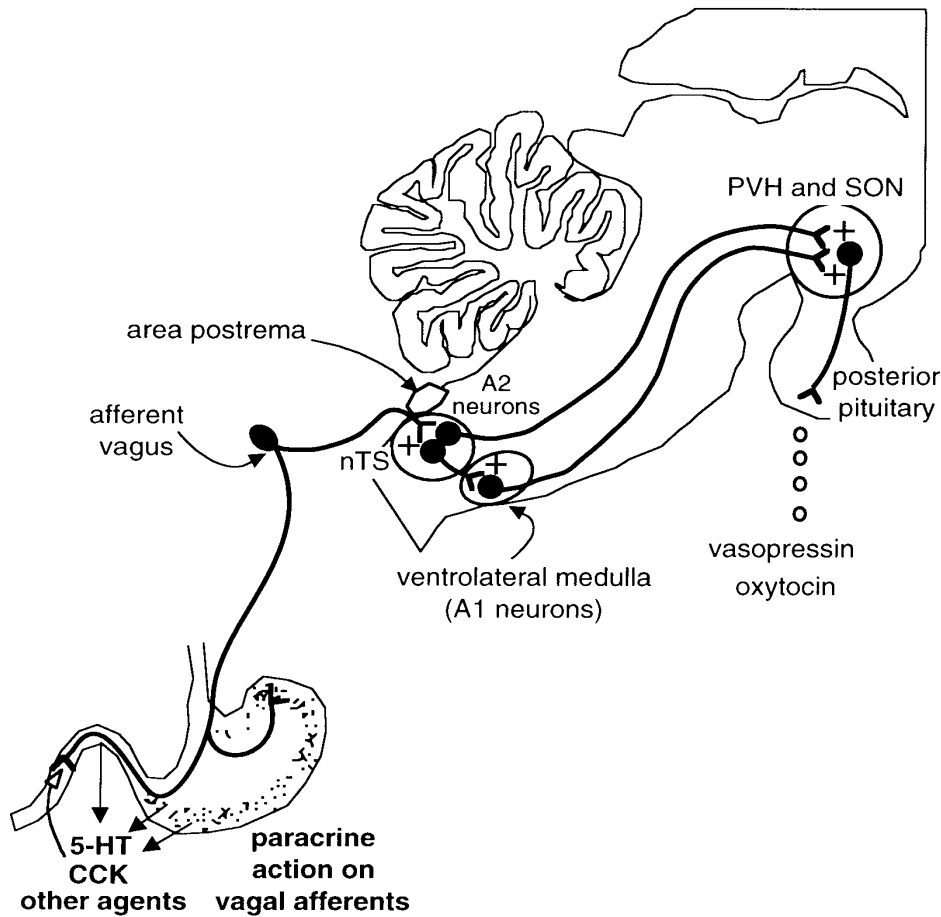


Figure 7.14 Pathways for secretion of neurohypophyseal hormones in response to gastrointestinal stimuli, including 5-HT and CCK. The A1 and A2 groups of catecholamine neurons are indicated. Abbreviations listed on pages xiii-xiv.

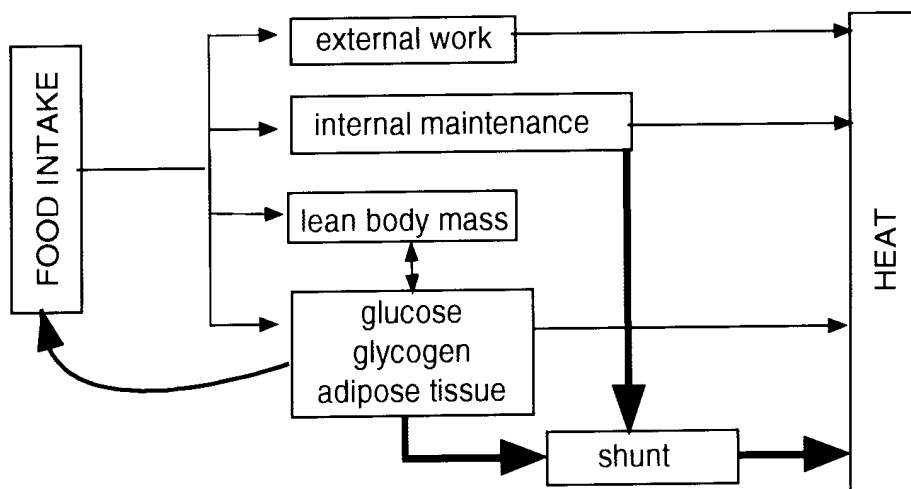


Figure 7.15 Possible metabolic and body temperature interrelationships that could involve sympathetic nerve activity in the regulation of body weight.