merulus and/or tubulus) which occurs in the rapid response to ischemia of kidney, remains to be elucidated. Even the localization of the different enzymes within the kidney has to be well clarified.

It is worthwhile to point out that the 2 enzymes studied had an equally rapid and simultaneous increase in the urine which may suggest a very near site of localization within the renal parenchima. The kininases, which have been described in normal urine, followed the same general pattern, namely, increased excretion after renal ischemia.

Measurements of serum enzymes were not made. However it is well known that increases in plasma GOT occur after renal infarction[7,12,13]. It indicates a leakage of enzymes through the plasma-cell barrier, besides the possible release of enzymes toward the tubular end of cell.

A similar study should be done concerning the serum bradykinolitic activity during and after renal ischemia. It might reveal whether or not the phenomenon of higher serum enzymes concentration following kidney injury is characteristic of some specific enzymes[14,15].

*Resumen.* Fue observado en los perros, nítido aumento en la excreción de transaminase glutamico-oxalo-acética, cininases y proteínas, inmediatamente después de una isquemia renal unilateral de 20 min. No fue observada ninguna alteración en la enzimuria o en la proteinuria en el riñon de control.

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## The Bundle of Schütz and its Relation to the Regulation of Food Intake

The experimental evidence for the presence of glucoreceptors in the ventromedial area of the hypothalamus (VMA) has recently been reviewed[1]. Briefly summarized, MARSHALL, BARRNETT and MAYER[2] have shown that mice injected with goldthioglucose (GTG) will develop lesions in VMA. These lesions will occur only if the glucose moiety is attached to the gold by the sulfur bridge. Other goldthio compounds, including goldthiogalactose, goldthiosorbitol, goldthiomalate, goldthioglycerol and goldthiocaproic or goldthiocapric acids will not cause VMA lesions[3]. Simultaneous injections of GTG with sodium thioglucose[3] or 2 glucose analogues[4] do not result in VMA lesions or hyperphagia and obesity, presumably because the injected glucose analogues compete with GTG for the sites of action. Mice made diabetic and subsequently injected with GTG also do not develop VMA lesions nor do they become hyperphagic and obese[5]. In the VMA, GTG-induced lesions are not due to a general increase in permeability in the blood-brain barrier[6].

Unlike electrolytically induced lesions in VMA, lesions made with GTG seem to be specific to the function of food intake regulation with minimal impairment shown to other functions. For example, GTG-obese mice, unlike mice made obese by electrolytic lesions do not show: (a) a range response[6], (b) gonadal atrophy but the mice can mate and rear their young[7], and (c) a water imbalance[8].

Recently, RIDLEY and BROOKS[9] demonstrated that destruction of VMA also eliminates the gastroacidity response to insulin hypoglycemia. In a previous study using rats, MAYER and SUDSANEH[10] had shown that destruction of VMA eliminates the inhibition of gastric hunger contractions by injections of glucagon and the consequent hyperglycemia and increase in glucose utilization. That gastric hunger contractions are not refractory to local agents is shown by the fact that they still respond to epinephrine and norepinephrine. On the basis of their results, MAYER and SUDSANEH[10] hypothesized that glucoreceptors in the ventromedial region of the hypothalamus, which may also include a portion of the periventricular system, involve fiber pathways that connect VMA to the lateral hypothalamic area (LHA) and the dorsal longitudinal fasciculus (bundle of Schütz) which is the primary efferent pathway from the hypothalamus to the mesencephalon and nucleus of the vagus.

Through the use of the FINK-HEIMER method[11] which is a stain for secondary degenerated fibers, AREES and MAYER[12] demonstrated connection from VMA to LHA. The purpose of the study reported here was to determine whether the bundle of Schütz would similarly degenerate when GTG was administered to animals.

Fifteen Charles River (CD-1) female mice weighing between 20–25 g were injected i.p. with goldthioglucose in concentrations equal to 0.5 mg/g body weight. 5 other mice were used as controls and injected with 0.5 mg/g body weight of glucose. 4 days after injection all mice were sacrificed, their brains removed and fixed in a manner similar to that described by AREES and MAYER[12]. Histological examination using the FINK-HEIMER method showed degeneration of the bundle of Schütz in 13 of the

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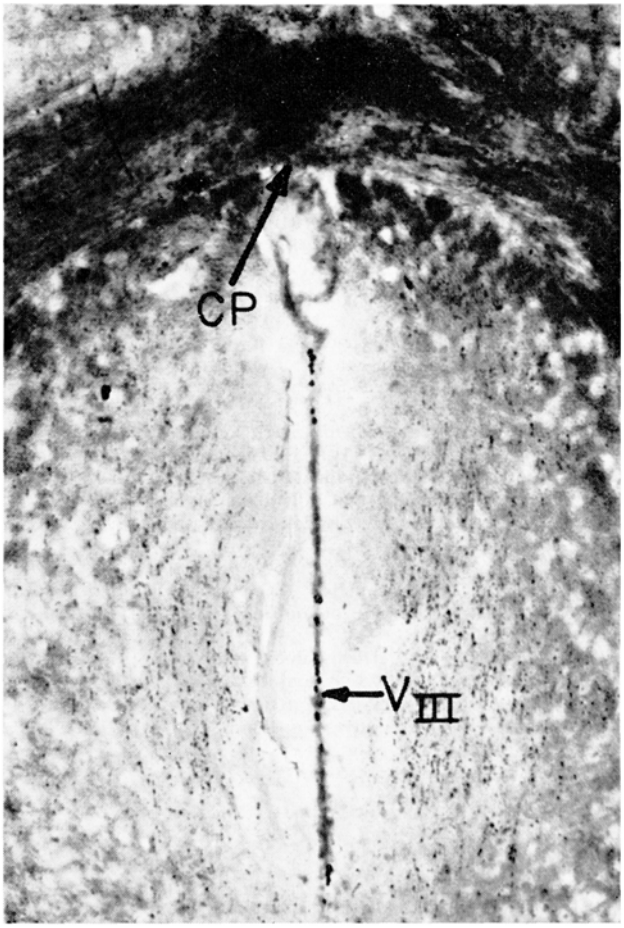


Terminal degeneration in central gray in mesencephalon at the level of the superior colliculus. $V_{III}$, third ventricle; CP, posterior commissure.

15 goldthioglucose-injected mice. The other 2 injected mice as well as the control mice showed no brain lesions. The pathway was shown to originate in the periventricular system, course dorsally and descend to terminate in the central gray region of the mesencephalon at the level of the superior colliculus. Terminal degeneration of the bundle of Schütz is illustrated in the Figure. No degeneration was shown below the level of the superior colliculus.

Thus, it appears that the bundle of Schütz like the fibers connecting VMA to LHA originate in neurons which are part of the glucoreceptive system as evidenced by the action of GTG[13].

*Résumé.* Les lésions provoquées chez des souris par une injection d'aurothioglucose dans la région ventromédiale de l'hypothalame sont suivies de la dégénérescence du fasciculus dorsalis longitudinalis (faisceau de Schütz). Cette observation indique que l'origine de cette structure efférente est glucoréceptive et fait partie du système glucorécepteur qui contrôle la satiété.

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## Hemodynamic Patterns During Fighting Behaviour in the Cat[1]

It is widely held that emotion results in a characteristic hemodynamic pattern consisting of increased arterial pressure, increased heart rate, augmented cardiac output and increased total peripheral resistance due to sympathetically mediated vasoconstriction in the visceral and cutaneous beds. This vasoconstriction, however, is thought to be associated with selective vasodilatation in muscle blood vessels, brought about through specialized sympathetic fibres which can be blocked by atropine[2].

Most of this information has not been derived from naturally behaving animals, but from experiments in which the so-called hypothalamic defence area was electrically stimulated in anaesthetized cats[2,3]. These investigations obviously suffer from all limitations inherent in electrical stimulation of the brain and use of anaesthesia. Data obtained in unanaesthetized animals are restricted to the demonstration of an increased muscle blood flow during pseudoaffective reactions in high decerebrate cats[4] and during simple alerting in intact conscious cats[5]. Investigation in man has seldom gone beyond tests such as a difficult mental arithmetic task[6], or has been performed with too limited or unreliable hemodynamic techniques[7,8].

The experiments, the preliminary results of which we are going to report, have been planned to obtain a faithful picture of hemodynamic patterns in unanaesthetized, unrestrained animals during a definite kind of emotional behaviour. The fighting behaviour of the cat was selected as particularly suitable. The experimental set-up consisted of a cage subdivided into 2 compartments by a movable opaque screen. In a compartment a cat was placed having an electrode chronically implanted in the mesencephalic grey, so that electrical stimulation through this electrode invariably elicited attack behaviour. This cat was only used as a stimulus for evoking natural fighting behaviour in another cat, the subject of the experiment, which was placed in the other compartment of the cage. The subject had previously been selected because of its constant responding with hissing and striking whenever attacked by the electrically stimulated animal. Cardiovascular reactions were repeatedly recorded in 6 subjects while fighting against the electrically stimulated attacking cat. About a week before recording, they were implanted with electromagnetic flow-probes (Statham) around the ascending aorta, the superior mesenteric artery, and an external