($p < 0.01$) in volume of the total gland and of its cortex was present only in the case of the left adrenal. For the right adrenal a less distinct difference ($p < 0.10$) existed. This discrepancy between the results of the volume measurements on the 2 sides can be explained by the right gland being much more severely shrunk. Nevertheless, because in the right adrenal gland at least the same tendencies are manifest, the results of the measurements in the left gland may be considered to be representative for both glands. The measurements show further that the medullae in both series of hamsters are of about the same size, but that the cortices in the hibernating, cold-exposed animals are significantly larger ($p < 0.01$) than those in the warmth-adapted individuals. Evidently, the adrenal cortex has been stimulated by the low environmental temperature; a phenomenon that is already well-known in cold-exposed, nonhibernating mammals such as the rat (cf. SMITH and HOIJER[5]).

It should be stipulated that changes in weights and/or volumes are only rough indications of changes in functions: Even the absence of such changes does not imply an unaltered function. This was clearly illustrated by the results of our previous histological studies on the testes. Nevertheless, from the results presented here the following conclusions can be drawn. The changes in organ size observed in the cold-adapted, hibernating hamsters must be attributed to cold-adaptation; this is in accordance with the findings in cold-adapted, homoiothermic mammals such as the rat. This means that our findings illustrate that the golden hamster behaves as a homoiothermic mammal, even when it shows hibernation; a conclusion similar to that of POHL[6] and POHL and HART[7], though with quite different argumentation. The preparation for hibernation occurs simultaneously with the trophic responses of the tissues to the stimulus to augmented heat generation.

Zusammenfassung. Von Dezember bis März wurden bei Goldhamstern im Winterschlaf und gleichzeitig bei wachen Kontrolltieren, die bei Zimmertemperatur gehalten wurden, Veränderungen von Organgewichten untersucht. Eine signifikante Zunahme des Gewichtes wurde bei Leber, Nieren, Herz, Pankreas, braunem Fettgewebe, Milz, Nebennieren und Nebennierenrinde der Winterschläfer gefunden. Bei Körpergewicht, Haut, Testes, Femora und Nebennierenmark waren keine Unterschiede zwischen beiden Tiergruppen festzustellen.

J. H. SMIT-VIS and G. J. SMIT

Anatomical-Embryological Laboratory,
University of Amsterdam and
Central Institute for Brain Research,
Amsterdam (Netherlands), 4 September 1968.

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The Excitation by Suxamethonium of Non-Proprioceptive Afferents from the Caudal Muscles in the Rat

The excitatory effect of suxamethonium on the muscle spindle is well known[1], but no attempt has been made to determine its action on unencapsulated muscle receptors. A possible sensitivity to suxamethonium of the free nerve endings is suggested by the response of some myelinated cutaneous fibres to related agents[2,3]. The problem has importance since suxamethonium evoked discharge from muscle spindles has been used as a form of selective input to the central nervous system[4,5].

Materials and methods. Adult male rats were anaesthetized with urethane (170 mg/100 g body weight) and the intertransverse caudal muscle, which comprises a number of small slips, was exposed and stretched over a light source which formed the bottom of a pool filled with Krebs' solution[6]. The spinal cord was exposed by laminectomy and the dorsal and ventral roots of the second and third caudal segments were cut, protected and prepared for electrical recording, or stimulation, under liquid paraffin.

The muscle selected for the experiments is the most distal of the group since it is usually innervated by a single branch from the lateral caudal nerve and contains between 4–8 receptors. The description of the afferent innervation is supported by Figure a. Before obtaining that record, neuromuscular transmission in the slip was blocked by addition of tubocurarine chloride (Tubarine, Burroughs Wellcome 1:10^6) to the muscle pool to ensure that confusing action potentials were not generated as a result of contraction of skeletomotor or fusimotor units in the muscle.

Results and discussion. Action potentials evoked by a single stimulus to the muscle nerve branch fall into 2 groups: those conducting at velocities between 25 m/sec and 11 m/sec which serve spindles and tendon organs, and slowly conducting afferents (7–4 m/sec), the majority of which do not respond to stretch. None of the slowly conducting afferents we used responded to stretch. When a suitable nerve branch was obtained, all others adjacent were cut, so the dorsal root used carried effective afferents only from the slip being studied.

Figure b was obtained from a dorsal root filament with 2 effective afferents, and their conduction velocities (19 m/sec and 6.3 m/sec) indicate that they are the first and fourth potentials of the compound of Figure a. When such a convenient filament had been obtained the tubocurarine was removed and the muscle was washed with Krebs' solution until neuromuscular transmission reappeared. 30 min later the action of suxamethonium on the afferents was investigated.

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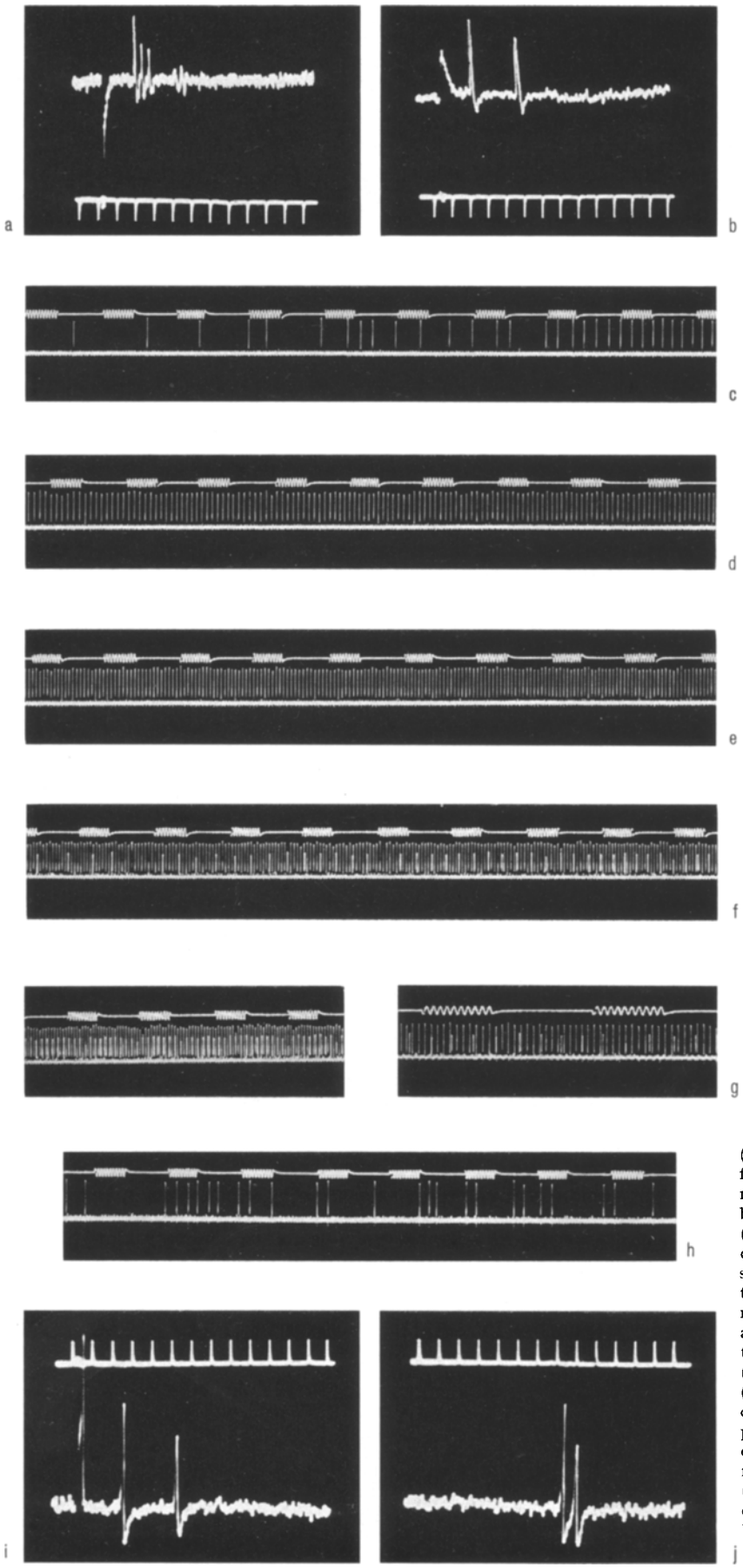

(a) The compound action potential recorded from the entire DR Cd3 and evoked by a maximally effective stimulus to the nerve branch innervating the intertransverse muscle. (b) Action potentials of fast and slowly conducting afferents recorded from a filament split from DR Cd3. (c–g) Records showing the excitation of the 2 afferents by suxamethonium ($1:10^4$). The records were obtained at the following times after application of the drug: (c) 45 sec, (d) 55 sec, (e) 60 sec, (f) 65 sec, (g) 5 min. Control recordings: (h) record obtained during repeated stretching of the muscle. (i) A comparison of the action potentials of the 2 afferents evoked by electrical stimulation, and, (j) by suxamethonium. Time scales: (a, b, i, j) 2 msec. (c–h) 50 cycles and 0.2 sec (amplification constant throughout). Conduction distance between stimulating and recording electrodes: 57 mm.

A minority of the slowly conducting afferents showed an expected[7], infrequent (less than 1 impulse/sec) and sporadic spontaneous activity. All spontaneously active, and a minority of non-spontaneously active afferents responded to suxamethonium by an increase or appearance of activity.

A powerful though threshold excitation of the slowly conducting afferent was obtained with a suxamethonium chloride (Scoline, Allen Hanburys) concentration of $1:10^4$ (f), a value 10 times the threshold concentration for the spindle afferent in the same muscle. The different sensitivity of the 2 forms of receptor was revealed also by the latencies of the 2 responses (c–f). A maximum activity of 45 impulses/sec was attained by the slowly conducting afferent 5 min after application of the drug (g). The remaining records of the Figure illustrate control procedures.

The sensitivity of the slowly conducting afferents to suxamethonium might be due to its depolarizing action on either the receptor ending or on a nodal region of the axon, so permitting impulse propagation in response to a natural but subthreshold depolarization of the receptor ending. Alternatively, the response could be a secondary effect due to evoked changes in the tissues in which the free endings lie[8]. The changes could be either a response to suxamethonium by smooth muscles of the vascular system, or a response by the ending to K^+ released from muscle cells by the suxamethonium[9,10].

Résumé. Des afférences myéliniques non-proprioceptives, à vitesse de conduction lente, en provenance des muscles de la queue du rat, sont excitées par le suxaméthonium d'une concentration de $1{,}0 \times 10^4$. On considère les mécanismes possibles de cette excitation, dont la signification est discutée.

G. L. Kidd and J. Kučera

The Physiological Laboratory, The University, Liverpool, 3 (England) and The Institute of Physiology, Czechoslovak Academy of Sciences, Prague 4 (Czechoslovakia), 12 September 1968.

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An in vivo Study of the Effect of Chronic Metabolic Acidosis on Renal Gluconeogenesis

It is well established that ammonia production by kidneys increases markedly in metabolic acidosis[1]. Pitts[1,2] has demonstrated that a major source of ammonia production is renal tubular glutamine. The exact mechanisms responsible for stimulation of increased glutamine deamidation are still unclear. At various times, increased synthesis or activity of glutaminase I[3,4] as well as inhibition[2] of the citrate condensing enzyme (citrate synthetase) have been implicated in augmented renal ammonia production. More recently Goodman, Fuiz and Cahill[5] demonstrated that in rats metabolic acidosis increased, whereas metabolic alkalosis decreased gluconeogenesis of renal cortical slices. Also, Goorno, Rector and Seldin[6] demonstrated that metabolic acidosis in dogs increased gluconeogenesis as well as ammoniagenesis of renal cortical slices. The purpose of the present study was to determine whether the increased renal gluconeogenesis described in in vitro studies for dogs with metabolic acidosis is in fact seen in vivo.

Seven mongrel female dogs weighing 9–15 kg were used in these studies. Each dog was fed a standard diet (Friskies dog food, Carnation Co., 16 oz/day). All animals were injected twice daily with 50 mg cortisone acetate (i.m.) to suppress endogenous steroid secretion[6]. Four of the dogs received 10 g NH_4Cl daily in their meal. The animals were maintained on this schedule for 10 days. Blood pH determinations were made periodically during this time. On the eleventh day, the dogs were anesthetized with sodium pentobarbital (30 mg/kg i.v.). Cannulae were placed in a femoral artery and vein for blood pressure monitoring and sampling as well as for infusion of drugs. The left renal artery was exposed by retroperitoneal approach and an electromagnetic flow probe (Medicon) placed around the vessel. The left renal vein was catheterized via the ovarian vein. Each animal was allowed to recover from the surgical procedure for 45 min before sampling was initiated. Samples of venous blood from the renal vein and arterial blood from the femoral artery were drawn at 10 min intervals for 90 min. At completion of each experiment, location of the catheter in the renal vein was verified and the electromagnetic flow probe on the renal artery calibrated in situ. Blood samples were placed immediately in cold 10% TCA, and stored overnight at 4 °C. Glucose determinations were made using a standard glucose oxidose method[7].

The data obtained in these experiments are summarized in Tables I and II. The data in Table I are reported as averages of the mean values for each animal for the first 60 min of sampling. Renal arterial blood glucose levels as well as blood flow are essentially the same for both groups. However, the A–V differences as well as the net renal uptakes or outputs of glucose for both groups are quite different. In contrast, data for the control animals show a mean uptake of glucose of 1.95 μmoles/g kidney · min.

An interesting change in renal glucose uptake and output appears to occur in time in the control animals. Table II shows that the glucose uptake by the control dogs decreases after 60 min and in fact changes to an

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