The present study shows that hypercapnia results in more than 50% depletion of the noradrenalin content of cat skeletal muscle. This depletion, which is dependent upon intact sympathetic outflow to the muscles, indicates that during the prevailing experimental conditions noradrenalin synthesis in the sympathetic nerves does not keep pace with impulse induced release. The phenomenon observed may indicate that adrenergic transmitter synthesis, during the initial phase of an abrupt increase in impulse activity, is only slowly and gradually stimulated. However, it is also conceivable that hypercapnia causes impairment of noradrenalin re-uptake and/or synthesis, as such effects only become apparent during sustained transmitter release[7].

Noradrenalin content of sympathetically innervated muscles (values expressed as % of content in decentralized control muscles)

	100% O_2		25% CO_2 + 75% O_2	
	Mean	Range	Mean	Range
Gastrocnemius (6)	78	100–58	44[a]	81–26
Tibialis anterior (3)	85	100–75	42	55–33

[a] Differs from control $p < 0.01$. Numbers within parentheses refer to number of cats used.

Zusammenfassung. Aktivitätserhöhung im sympathischen Nervensystem bei Katzen wurde durch Inhalieren von 25% CO_2 in O_2 hervorgerufen. Es wurde der Einfluss erhöhter sympathischer Aktivität auf den Noradrenalingehalt sympathischer Nerven geprüft, welche zu motorischen Muskeln verlaufen. Hyperkapnia rief keine Veränderung im akut dezentralisierten M. Gastrocnemius hervor. Bei intaktem sympathischem Fluss zum Muskel bewirkte Hyperkapnia eine erhebliche Erniedrigung des Noradrenalingehaltes. Die Wirkung hängt somit von einem intakten sympathischen Ausfluss ab.

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Spontaneous Activity of Rats on Diets Varied in Protein, Carbohydrate and Fat Content

Perusal of the literature reveals conflicting results on the relationship of spontaneous activity to diet. Some studies have indicated that low protein diets depress activity[1,2]. Other studies have suggested that dietary protein does not affect activity at all except at high protein levels, at which a decrease in activity occurs[3,4]. The ratio of protein to carbohydrate in the diet has also been investigated. Excitability level (number of standing up reactions) appears to be closely related to this ratio. As the level of proteins in the diet increases, the excitability level of rats decreases[5]. Activity level measured by the number of wheel turns has been claimed to decrease as the proportion of protein in the diet increased[6]. High fat diets have been said to depress activity initially at varying levels of dietary protein. However, upon prolonged administration, rats on high fat diets show an increase in the level of spontaneous activity above that for rats on low protein diets[7].

To try to clear up these contradictions, 6 male and 6 female Charles River albino rats were fed from weaning diets where the proportion of carbohydrate, protein and fat were varied systematically (Table I). The protein was casein, the carbohydrate sucrose, the saturated fat lard, and the unsaturated fat corn oil. The usual mineral and vitamin supplements were included. The animals were matched by sex and age at the time of running (28–82 days old). Each rat was placed in a stabilimeter cage for 6 consecutive days with free access to food and water. The cages were wire mesh with a closed top, 22.5 cm in diameter and 20.5 cm high, with a 6 cm diameter, 5 cm high food cup in the middle. Sawdust was placed in the bottom pan which rested on 4 switches which counted every time 2 switches were closed. A water bottle spout entered through an opening in the cage. The apparatus was turned off once daily, when counters were read and rats weighed.

The results showed a progressively stabler daily activity over the 6 day period for each group. The mean activity level was obtained by averaging activity scores

Table I. % calories of protein, carbohydrate and fat

Foodstuff	Diet				
	HP	SC	LP	HUF	HSF
Protein	59.8	24.5	9.8	24.5	24.5
Carbohydrate	19.1	64.5	79.2	–	–
Saturated fat	4.4	4.4	4.4	11.0	68.9
Unsaturated fat	6.6	6.6	6.6	64.5	6.6

[1] F. A. Hitchcock, Am. J. Physiol. *84*, 410 (1928).
[2] J. R. Slonaker, Am. J. Physiol. *96*, 547 (1931).
[3] T. S. Hamilton, J. Nutr. *17*, 565 (1939).
[4] E. A. Smith and R. M. Conger, Am. J. Physiol. *142*, 663 (1944).
[5] J. Lat, Physiologia bohemoslov. *5*, 38 (1956).
[6] G. H. Collier, R. L. Squibb and F. Jackson, Psychonomic Sci. *3*, 173 (1965).
[7] S. Frankova, Activitas nerv. sup. *4*, 471 (1962).

over the last 3 days (Table II). An analysis of variance using square root transformations was performed. No significant differences were found between sex or diet groups at the 0.05 level. The data suggest, however, that diet may have a differential effect on the activity level of male and female rats ($F = 2.14$, $df = {}^{5}/_{60}$, $p < 0.10$). When male and female activity scores were combined, differences between diet groups were not apparent[8].

Table II. Mean activity scores over the last 3 days for rats on varied diets

	Diet					
	Protein			Fat		Purina
	10%	25%	60%	29% unsaturated	31% saturated	
Male	411	604	497	600	329	579
Female	535	635	728	435	770	501
Total	946	1239	1325	1035	1099	1080

Résumé. Nous avons mesuré l'activité spontanée de rats mâles et femelles recevant des rations contenant 10%, 25% et 60% de protéine et 29% de graisses non-saturées ou 31% de graisses saturées. Les rats ont été maintenus isolés dans des cages à stabilimètre 6 jours de suite. On a comparé les données obtenues pendant les 3 derniers jours. On n'a pas trouvé de différences significatives dans l'effet des différents régimes, ni pour les mâles ni pour les femelles.

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Responses of Primary Muscle Spindle Endings at Constant or Changing Muscle Length to Variations in Fusimotor Activation

A systematic analysis has been undertaken of the responses of muscle spindle endings to defined changes in fusimotor activation. This study was prompted by the fact that the fusimotor and the skeletomotor systems are often found to be activated in parallel, as is the case in various reflex contractions[1-3] (for further references see e.g. MATTHEWS[4]) and in the physiologically-induced movements in respiration[5-8]. Recent work suggests that coactivation of fusimotoneurones may also occur in voluntary contractions in man[9]. In the case of strong fusimotor activation in parallel with the contraction of the main muscle, the discharge rate of both primary and secondary endings may increase during the phase of shortening in contraction[5,8].

The responses were studied of primary and secondary spindle endings to systematic variations of static and dynamic fusimotor activation, both with constant muscle length and during length change in pace with the variations in fusimotor stimulation. This preliminary note concerns results obtained with primary endings. A full report will be published elsewhere.

Methods. In 12 cats, stimulations were performed, in ventral root filaments, of single fusimotor fibres to muscle spindles in the soleus and lateral gastrocnemius muscles, the discharge of single muscle spindle endings being recorded in dorsal root filaments. Impulse frequency was measured by an 'instantaneous frequency meter'[10]. Periodic changes of muscle length with triangular waveform (see trace c in Figures 1 and 2) were generated and monitored as previously described[11]. The designation of a fusimotor fibre as 'dynamic' or 'static' followed the same criteria as in a previous paper[10]. In order to be able to subject single fusimotor fibres to increasing and decreasing stimulus frequencies, the stimulator was triggered from a voltage-to-frequency converter with a linear input-output relation. This device, in turn, was fed by a low-frequency generator (Hewlitt Packard Model 202 A) delivering triangular (see trace d in Figures 1 and 2) or sinusoidal voltage waves. The range of stimulus frequencies of the fusimotor fibres was chosen to lie either between 20–400 shocks/sec or between 60–180 shocks/sec.

Results. Primary endings at constant muscle length. Within the range of 60–180 shocks/sec, there was a rather linear relation between the frequency of stimulation of static and dynamic fusimotor fibres and the discharge rate of the primary endings. Stimulus frequencies below 30–40 shocks/sec did not produce any significant increase in discharge rate. When the rates of stimulation exceeded 180 shocks/sec, the corresponding increments in afferent discharge rate became successively smaller and approached its maximal level. Both static and dynamic fibres yielded their maximal responses at stimulus frequencies of 250–300 shocks/sec. However, considerably

[1] R. GRANIT and B. R. KAADA, Acta physiol. scand. *27*, 130 (1953).
[2] E. ELDRED, R. GRANIT and P. A. MERTON, J. Physiol. *122*, 498 (1953).
[3] C. C. HUNT and S. W. KUFFLER, J. Physiol. *113*, 283 (1951).
[4] P. B. C. MATTHEWS, Physiol. Rev. *44*, 219 (1964).
[5] V. CRITCHLOW and C. v. EULER, J. Physiol. *168*, 820 (1963).
[6] T. A. SEARS, J. Physiol. *174*, 295 (1964).
[7] G. EKLUND, C. v. EULER and S. RUTKOWSKI, J. Physiol. *171*, 139 (1964).
[8] C. v. EULER and G. PERETTI, J. Physiol. *187*, 59 (1966).
[9] K.-E. HAGBARTH and Å. B. VALLBO, Acta Soc. Med. upsal. In press (1967).
[10] P. B. C. MATTHEWS, J. Physiol. *169*, 58P (1963).
[11] G. LENNERSTRAND and U. THODEN, Experientia (1967).