be clearly seen, the average change in maintained discharge being greater than 6-fold.

In graph B), the reverse code is demonstrated with another cell (from the stratum griseum profundum layer of the superior colliculus) which sharply increased its maintained discharge in the bright environment above and decreased it in the dim one. This reversal, a 10-fold change in response, was repeated some 21 times with such consistency that even the longterm sinusoidal drift[3] inherent to this cell can be seen well represented and in phase at both luminance levels.

As reported earlier[4], certain specialty units with sharply localized receptive fields have been found to be wholly insensitive to changes of background luminance, even through contrast reversals. Yet, central cognizance of the surround luminance is clearly vital for purposes of pupillary control and adaptation state reference. Units, as described here, however, although poor in spatial definition, do exhibit the broad integrative properties required for such luminance assessments and thus appear uniquely suited to serve such needs.

Zusammenfassung. Nachweis, dass bestimmte Zellen im visuellen System, obwohl sie auf kurze transitorische Reize nicht reagieren, gleichmässig und langfristig auf Änderungen des Beleuchtungshintergrundes der Umgebung ansprechen.

G. D. Ritchie, A. A. Lee, T. J. Gast and R. M. Hill[5]

Comparative and Physiological Psychology, Biophysics, and Optometry, The Ohio State University, 338 West Tenth Avenue, Columbus (Ohio 43210, USA),
27 January 1975.

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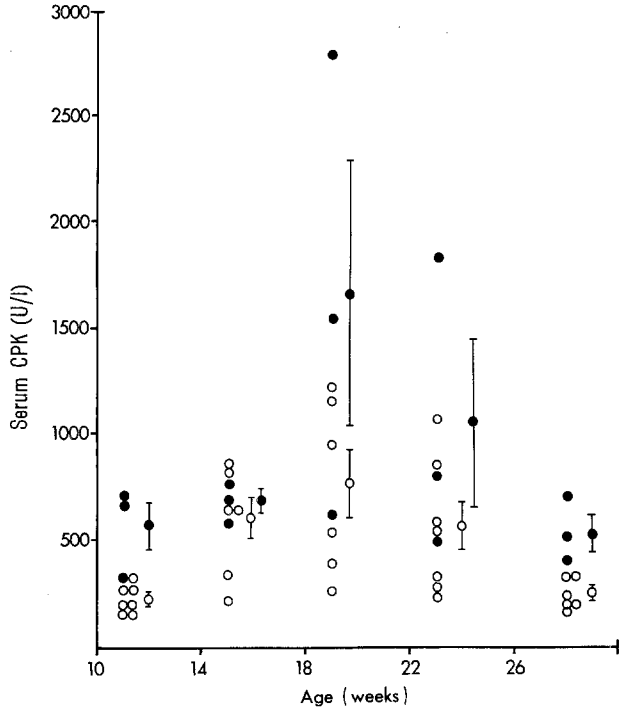
Age Dependent Variation of Serum Creatine Phosphokinase Levels in Pigs

Recently much has been written about the merit of elevated serum creatine phosphokinase (CPK) activity in diagnosing muscle disorders. The greatest increase in serum CPK activity occurs in Duchenne muscular dystrophy (DMD) while small increases may be observed in muscle atrophy of neurogenic origin[1]. The anaesthetic-induced malignant hyperthermia (MH) syndrome seen in man, a dominant-type inherited subclinical myopathy[2] can be detected by finding elevated serum CPK levels[3]. Occasionally, MH susceptible individuals may have normal serum CPK levels[4]. The clinically identical MH syndrome in pigs and the associated acute stress syndrome have also been correlated with elevated serum CPK levels by Woolf et al.[5]. The similarity of the syndrome in man and pigs makes the pig a valuable model for the MH syndrome and provides adequate tissue for biochemical investigation of the basic lesion which is apparently in the skeletal muscle[3]. Consequently, the possibility of using serum CPK levels to detect predisposition to the syndrome in pigs has been suggested[5,6] though the limitations were not fully recognized then. We have found that serum CPK activity in both stress susceptible and normal pigs vary enormously[7]. Although stress and exercise raise serum CPK levels in both man and pigs, we report here that unlike man[8] age is a major factor causing fluctuations in serum CPK activity in pigs.



Variation of serum CPK activity with age in 7 halothane-resistant and 3 halothane-sensitive German Landrace pigs. ○, halothane-resistant, $n = 7$; ●, halothane-sensitive, $n = 3$. Data are given as means ± S.E.M. for each group of pig. Halothane-sensitive pigs had elevated levels of CPK at 11 weeks ($t = 2.932$, d.f. 8, $p < 0.01$) and 28 weeks of age ($t = 3.000$, d.f. 7, $p < 0.01$).

Methods. We measured serum CPK levels in 10 pure-bred German Landrace pigs, a breed known to have a high incidence of the MH syndrome, between 11 and 28 weeks of age. There were equal numbers of males and females. The 10 pigs examined were taken from 2 litters selected randomly from a large herd. Blood samples were obtained every 4 weeks during the 11–28 week period. The pigs were housed in individual pens at 18–24 °C and were fed on a standard ration during the whole period. After 28 weeks of age all pigs were challenged with 3–4%

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halothane for positive identification of the susceptible animals. Serum CPK activity was assayecd by the coupled enzymatic method using the 'Monotest' CPK kit supplied by Boehringer GMBH, Mannheim, West Germany, and expressed as International Units per litre of serum (IU/l) at 30 °C.

Results and Conclusions. The Figure shows the variation of serum CPK activity with age for the 7 halothane-resistant and 3 halothane-susceptible pigs. CPK levels reached a peak at 19 weeks of age in both groups, and after 28 weeks the levels returned to values similar to those at 11 weeks of age. The 'normal' serum CPK activities were 217.3 ± 23.2 IU/l and 254.5 ± 24.5 IU/l (mean ± S.E.M.) at 11 and 28 weeks of age, respectively. There was no sex difference in serum CPK activity at any age. There were no statistically significant differences (p 0.05) between the CPK activities of the resistant and susceptible pigs at 15, 19 and 23 weeks of age, but significant differences were obtained at 11 and 28 weeks of age ($p < 0.01$; Student's t-test). These findings strongly indicate that age is a major factor determining serum CPK levels in both normal and MH susceptible pigs alike. In the group of pigs studied serum CPK activity reached a peak value at 19 weeks of age, an age correlating well with the period of maximum growth rate[9] and more specifically with the process of 'muscling'[10]. According to Hammond[10], 'muscling' refers to the period of maximal muscle protein anabolism and increase in muscle fibre size. We therefore infer that the increase in serum CPK activity observed during the 15th to 23rd week of age in the present study is directly related to the 'muscling' phenomenon. The increase in serum enzyme activity may be interpreted as an increase in the leakiness of the skeletal muscle fibres during the period of maximal muscle growth. The fact that the serum CPK levels in the MH susceptible pigs are significantly elevated at 11 and 28 weeks of age compared with the non-susceptible animals is in agreement with the increases in CPK activity observed in DMD and other myopathies[1] and in the human syndrome[2].

From these observations it is clear that there are numerous factors causing non specific increases in serum CPK levels. However, in the group of pigs studied, after minimizing these extraneous factors, we were able to detect those animals carrying the MH syndrome on the basis of significantly elevated serum CPK levels.

Zusammenfassung. Infolge Muskeltätigkeit steigt bekanntlich der Creatin-Phosphokinase-Spiegel (CPK) im Serum. Es wird nun eine Abhängigkeit des Serum-CPK-Wertes vom Alter festgestellt. Es ist daher wichtig, nichtspezifische Ursachen eines gesteigerten CPK-Spiegels auszuschalten, ehe eine Diagnose der Muskelerkrankung gestellt wird.

J. J. A. HEFFRON and G. MITCHELL[11]

Departments of Physiological Chemistry and Physiology, Medical School, University of the Witwatersrand, Hospital Street, Johannesburg 2001 (Republic of South Africa), 16 September 1974.

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Gastrointestinal Hormones and Neural Interaction Within the Central Nervous System

Obesity is one of the most common nutritional disorders in the world effecting millions of overweight people. Obesity is a major contributor to heart diseases. The underlying cause of obesity is unknown. Therapy of this disorder is generally unsatisfactory despite modern medical use of various drugs, operations and the popularity of fad diets. The basic problem for the fat person is that his appetite is not satisfied until he has consumed too much food.

BEAUMONT[1] noted that St. Martin required instillation of food into the upper gastrointestinal tract to experience satiety from hunger. Parenteral injection of intestinal mucosal extracts, presumably containing soluble polypeptide hormones, caused depression of hunger and weight loss in rabbits[2]. GIBBS et al.[3,4] injected unfed rats with purified cholecystokinin (CCK) or the synthetic octapeptide (CCK-OP) which possesses all biological activity of the entire CCK molecule; these agents evoked the satiety response. Other gastrointestinal hormones (pentagastrin and secretin) did not stimulate the satiety response in the rats. FARA et al.[5] noted that CCK induced somnolence in cats. Satiety was evoked by feeding, which is also known to produce release of gastrointestinal hormones, including CCK[6]. CCK is released normally when the upper small intestinal mucosa is exposed to ingested fat, amino acids and gastric juice[7]. The specific target areas of a satiety factor probably lie within the hypothalamus, since different lesions of the region can instigate or repress hunger[8,9].

The aim of this investigation was to determine electrophysiological response of various areas of the brain to the hormonal satiety factor. Central effects of CCK or other gastrointestinal hormones can be expected to result from modulation of neuronal activities, and such actions may be localized in one or more brain structures. The present study represents an initial attempt to elucidate the synaptic effects of gastrointestinal hormones in several structures within the brain which are presumably involved in controlling and regulating the appetite.

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