The results of the present study reveal that the glycogen content of both liver and muscle rises above the normal level concomitent with the occurrence of hypoglycaemia noticed earlier[4]. Compared to the liver, muscle possess a very small content of glycogen. The glycogen content of liver is more than 6 times the normal level in muscle. On the contrary, the rise is more pronounced in muscle as can be seen from the Figure, C and D, following the administration of any dose.

Following islectomy, the fish liver glycogen content was observed to fall[7]. On the contrary, massive doses of insulin injections of 40 IU were found to raise the muscle glycogen but not that of liver of certain marine fish[5]. Glycogen was found to reduce under those conditions.

It can be seen from Tables I and II that there is a concomitant fall in the blood glucose level with the corresponding rise in the glycogen content of the liver and muscle (Figure, A–D) at the various doses of insulin injected. The disappeared glucose might have been converted into glycogen in these tissues due to the injected insulin. Such a possibility of direct action of insulin on the liver and muscle, where glycogen is stored, has been demonstrated very clearly in mammals[8–12]. Further recent studies have also shown that the rise in glycogen could be due to the fact that insulin can inhibit the release of glucose from the liver[13]. Both increased acceleration of glycogen synthesis in these tissues and an inhibition of release of glucose could account for the high glycogen content in these tissues of *O. striatus*.

The rise of glycogen in the liver is not as fast as that observed for muscle, though the muscle glycogen level always remained far below that of liver. It is worth pointing out here that, though the normal liver glycogen was six times that of muscle, it was reduced to less than 4 times following heavy doses of insulin injection. This can be attributed to the very low glycogen present in the muscle at the beginning whose rate of increase has become more pronounced following insulin administration.

The results obtained by ROOT et al.[5] as pointed out earlier, are contradictory to the present observations. They, however, had remarked that their conclusions derived from the study of insulin action were very conflicting and they cannot be in agreement with all other investigations. On the other hand MCCORMIC and MACLEOD[7] showed that insulin was essential for the glycogen synthesis in liver and their results, though indirectly, seem to be in agreement with the present study[14].

Résumé. Des doses variées d'insuline administrées à l'*Ophicephalus striatus* ont augmenté la quantité de glycogène du foie et des muscles. Dans le foie, les doses étant fortes (40 IU et 60 IU), la quantité normale de glycogène a presque quadruplé et elle a plus que septuplé dans les muscles. Le mécanisme possible présidant à l'action de l'insuline est aussi discuté.

B. SESHADRI[15]

Department of Zoology, University of Udaipur (Rajasthan, India), 10th March 1967.

[7] N. A. MCCORMIC and J. J. R. MACLEOD, Proc. R. Soc. *98*, 1 (1925).
[8] J. P. BOUKAERT and C. DE DUVE, Physiol. Rev. *27*, 39 (1947).
[9] C. DE DUVE, Ciba Fdn Colloq. Endocr. *9*, 203 (1956).
[10] C. DE DUVE and H. G. HERS, A. Rev. Biochem. *26*, 149 (1957).
[11] J. P. BERTHET, P. JACQUES, H. G. HERS and C. DE DUVE, Biochem. biophys. Acta *20*, 190 (1956).
[12] P. J. SNYDER and C. F. CAHILL, Am. J. Physiol. *209*, 616 (1963).
[13] G. E. MORTIMORE, Am. J. Physiol. *204*, 699 (1963).
[14] The author expresses his thanks to Prof. R. V. SESHAIYA, Director, Marine Biological Station, Portonovo, India, for the help and encouragement shown in this work.
[15] Present address: Dept. Ob-Gyn. Biochem., K. U. Med. Center, Kansas City, Kansas 66103, USA.

Serum Desoxy-Ribose Compounds in Experimental Myocardial Infarction

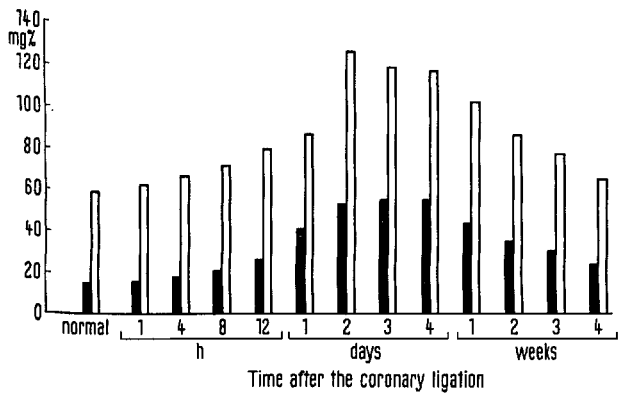
Myocardial infarction provides a unique opportunity to study biologic response to a relatively limited injury, in which certain changes are the consequence of a general biologic response rather than of the pathology, at least in the acute stage. Leakage of certain enzymes from the myocardium into the blood stream is already known under such circumstances. LYSENKO[1] was the first to notice DNA decomposition products in the serum of patients with myocardial infarction and angina pectoris. No other data dealing with observations based either on animal experiments or on patients have appeared in literature. The present investigation is a preliminary report of the work, and describes the experimental results obtained in dogs with experimental myocardial infarction at various time intervals after the operation.

Dogs weighing 10–15 kg were used. Myocardial infarction was produced by two stage coronary ligation following the technique of HARRIS[2]. DNA products were estimated by the method of Diphenyl amine reaction (DISHE[3]) for acid soluble ($HClO_4$ 5%) desoxyribose compounds and acid insoluble desoxy-ribose compounds. Blanks and standard solutions were used following the method of LYSENKO[1]. The investigations were made in 10 normal dogs and in the same dogs after experimental myocardial infarction. The estimations were continued only in those dogs which showed 90–100% ectopic beats 20–24 h after the ligation of the coronary artery.

The acid soluble and acid insoluble desoxy-ribose compounds in the serum of normal healthy dogs varied from 9–24 mg% and 42–77 mg% respectively with the mean values of 15 and 59 mg% (Figure). In dogs with myocardial infarction determinations were made at various intervals after the operation up to the fourth week as shown in the Figure. The concentration of these acid soluble compounds remained high for quite long and was significantly elevated up to the fourth week after the operation. Values not changing much up to 4 h, ranged

[1] L. T. LYSENKO, Fedn Proc. Fedn Am. Socs exp. Biol. *23*, 587 (1964).
[2] A. S. HARRIS, Circulation *1*, 318 (1950).
[3] W. DISHE, in *The Nucleic Acids* (Ed. E. CHARGAFF and J. N. DAVIDSON; Academic Press Inc., New York 1955), vol. 1, p. 287.

between 17–38 mg% 12 h after the operation and increased up to the third day when the values ranged between 45–67 with a mean value of 54 mg%, as compared to the normal value of 15 mg%. After showing a slight decrease, the serum levels of these acid soluble desoxy-ribose compounds after 4 days remained considerably higher even after 4 weeks of the operation, with the range of 16–34 mg%.



Serum concentration of desoxy-ribose compounds after the production of experimental myocardial infarction by two stage coronary ligation in dogs up to 4 weeks. Solid bars represent acid soluble desoxy-ribose compounds and blank bars acid insoluble desoxy-ribose compounds. The height of every bar represents the mean value obtained from 10 animals and the extreme left bars show control values obtained from 10 normal animals.

The acid insoluble desoxy-ribose compounds also increased and the maximum concentration was observed on the second day ranging from 98–146 mg%. The concentration after 1, 2, 3 and 4 weeks ranged between 73–134, 67–116, 54–101 and 47–89 mg%. Concentration of the desoxy-ribose compound both of soluble and insoluble products are increased greatly and remain higher at least up to 4 weeks after the production of experimental myocardial infarction, possibly due to the leakage from the nuclei of the cells of the infarcted area of the heart. Further work would establish the concentration of such compounds in the infarcted area of the heart tissue itself[4].

Zusammenfassung. Es wird nachgewiesen, dass eine koronare Unterbindung am Hund zu erhöhter Desoxyriboseverbindung im Serum führt.

PAWAN S. CHAUHAN[5] and R. B. ARORA

Department of Pharmacology, All India Institute of Medical Sciences, New Delhi 16 and Indian Institute of Experimental Medicine, Jadavpur, Calcutta 32 (India), 13th January 1967.

[4] This work was supported by a grant from Indian Council of Medical Research.

[5] Present address: Indian Institute of Experimental Medicine, Jadavpur, Calcutta 32 (India).

The Effect of Urethane on Some Electrical Properties of Molluscan Giant Neurons

Giant neurons of the mollusc *Onchidium verruculatum* in the presence of 2% urethane become incapable of producing an all-or-none type of action potential. The analysis with voltage clamp technique indicates that in these conditions Na-conductance diminished while K-conductance stayed at the same level. Similar results were obtained in Na-free solutions[1]. Thus urethane seems to act selectively on Na-conductance, which is probably connected with a Na-carrying mechanism. This point of view is shared also by authors using smaller concentrations of urethane on nerve and muscle fibres of other animals[2,3].

The present paper deals with the influence of urethane on some electrical properties of giant neurons of 2 species of Gastropodes: *Helix pomatia* and *Planorbis corneus*. These 2 species show distinct difference in excitability when placed in Na-free solutions[4]. A preliminary note of the present work has been published elsewhere[5].

Methods. The giant neurons from the visceral and parietal ganglia of *Helix pomatia* and *Planorbis corneus* were investigated. The ganglia were separated from the body and placed in the chamber with perfusing system. A 2% solution of ethyl-urethane was used in normal physiological saline. The experimental set-up for intracellular recording and stimulation and the physiological solutions used in this work are given in the authors' previous papers[6,7]. 12 experiments on 7 specimens of *Helix pomatia*, and 17 experiments on 10 specimens of *Planorbis corneus* were carried out.

Results. On the giant neurons of *P. corneus*, urethane exerts a rapid effect manifested by reduction of the amplitude of the spontaneous spike, increase of its duration and slowing of its rise-time (Figure 1 B). The action potentials were abolished 4–5 min after the beginning of perfusion with solution containing urethane. Nerve cells investigated in Na-free solution became incapable of producing action potentials in the same time (Figure 1 C).

The voltage-current relationship of the membrane after urethane is non-linear. Depolarizing currents evoked a smaller drop of the voltage as compared with the hyperpolarizing currents (Figure 1 D). Examples of electrotonic responses after urethane as a result of passing depolarizing and hyperpolarizing currents of the same intensity and duration are seen in Figure 1 E. The

[1] S. HAGIWARA and N. SAITO, J. Physiol. *148*, 161 (1959).
[2] I. TASAKI and A. F. BAK, J. Neurophysiol. *21*, 124 (1958).
[3] F. THESLEFF, Acta physiol. scand. *37*, 335 (1956).
[4] V. D. GIERASIMOV, P. G. KOSTYUK and V. A. MAISKI, Bull. exp. Biol. Med. U.S.S.R. *58*, 3 (1964).
[5] V. D. GIERASIMOV and L. JANISZEWSKI, *Cetvertoje naucnoje sovescanije po evolucjonnoj fisiologij, posviascennoje pamiati akademika L. A.*, Orbeli, SSSR *77*, (1965).
[6] L. JANISZEWSKI, (N. Copernicus University Press, Torun, 1965).
[7] V. D. GIERASIMOV, Fiziol. Zh. SSSR *50*, 457 (1964).