tion, did not develop palpable tumours. Of the twelve controls with established tumours, half were then treated with 5-tertiary benzyl benzimidazolone in the same dose. Partial regression occurred after seven days of treatment. These studies are being continued.

H. C. Stoerk
R. N. Arison
J. E. Hawkins, jun.*

Merck Institute for Therapeutic Research,
Rahway, New Jersey.

* Present address: Department of Otolaryngology, New York University—Bellevue Medical Center, New York 16.

Chemical Nature of Parathyroid Hormone

Most of the data accumulated in the past thirty-two years[1] has led to the conclusion that parathyroid hormone is a protein. In 1954, Handler, Cohn and Dratz[2] attempted to purify this hormone by means of ion-exchange chromatography, but found hormonal activity associated with a number of protein peaks. On the basis of their results, they suggested that the hormonal activity might reside in a smaller molecule, presumably not necessarily nitrogenous, which readily absorbs on proteins. Raoul, Mornay and Prelot[3] have recently claimed to have prepared, by two different lipid extraction methods, a highly active material which did not contain nitrogen. On the basis of unreported evidence, they suggested that this active substance was steroidal in nature. Since the D vitamins, which are steroid, closely mimic, in some respects, the action of parathyroid hormone on bone and kidney, the possibility that this hormone is a steroid merits consideration.

Upon undertaking the problem of the isolation of parathyroid hormone, it seemed imperative to us to perform experiments to confirm, if possible, the work of Raoul *et al.*[3]. They claimed that it was possible to obtain non-nitrogenous extracts containing a specific activity of 300 u./mgm. by three means: (1) extracting an acetone powder, prepared by the method of L'Heureux, Tepperman and Wilhelmi[4], with boiling absolute ethanol for at least 30 min.; (2) extracting the same active powder with a mixture of ethanol and ether (3 : 1) at − 40° C.; and (3) extracting a hydrochloric acid extract of 'defatted' glands with ethanol–ether (3 : 1) at − 40° C. They further claimed that the nitrogenous residue in each instance was inactive. We have followed their extraction procedures as closely as possible, using methods (1) and (3). In each instance, the lipid extract was concentrated to a few millilitres by vacuum distillation, and then assayed, as were the nitrogenous residues. All fractions were assayed by the method of Davies, Gordon and Mussett[4], which depends on the rise in concentration of plasma calcium in parathyroidectomized rats 18 hr. after the test injection. No hormonal activity was found in any of the lipid extracts and approximately 80 per cent of the estimated original activity was found in the nitrogenous residues. The results are completely contradictory to those of Raoul *et al.*[3].

The important difference in these two sets of experiments is in the assay method employed. Raoul *et al.*[3] utilized the method of Dyer[6], in which normal rabbits are the test animal. Dyer's own conclusions about this method were that rabbits were unsuitable assay animals because of the marked fluctuations in the normal concentration of serum calcium, and the wide variation in response to a test injection of parathormone. This has been the experience of others[1].

Our findings in conjunction with further work in progress lead us to believe that the hormone is a small protein, probably in the size-range of adrenocorticotrophic hormone and insulin. Our more important reasons for this conclusion arise from our unpublished observations that a partially purified, highly active (100 u./mgm.) hormonal preparation contained 14 per cent nitrogen (micro-Kjeldahl) and 97 per cent of its weight was accounted for by its amino-acid content.

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Howard Rasmussen*
Roland Westall

Medical Unit,
University College Hospital Medical School,
London, W.C.1.

* Present address: Rockefeller Institute for Medical Research, New York City.

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Toxicity of Erythromycin

In the course of studies on antibiotic therapy of rickettsial infection in the guinea pig, it was found that oxytetracycline, chlortetracycline and tetracycline administered intramuscularly were tolerated well in the dosages employed. The administration of erythromycin on an equivalent dose-weight basis, however, resulted in death of a portion of the test animals. The picture observed was comparable to that noted by Kaipainen and Faine[1] following oral, intraperitoneal or intravenous administration of the drug. Because the response of the guinea pig to erythromycin appears to differ from a simple toxicity phenomenon, the following observations are reported.

Hartley strain albino guinea pigs weighing 300–400 gm. were employed throughout this work. These animals were obtained from a single source and were free of intercurrent infection with bacterial pathogens. They were maintained in a constant-temperature environment on rabbit chow (without antibiotic supplement—Ralston Purina Co.) and fresh greens. Erythromycin lactobionate (Abbott Laboratories—Lot No. 650–8938), reconstituted daily in water for injection (USP), was used. Injections were made into the hamstring muscles.

Initially, the drug in a dose of 1·5 mgm. twice daily for 9 days (total dose 27·0 mgm.) was started simultaneously with an intraperitoneal rickettsial challenge. Nine of the ten animals on erythromycin died between the eighth and eleventh day post challenge, several days prior to death of the control animals receiving only the rickettsial challenge.

A second series of guinea pigs were then given drug alone twice daily for five days. The dosages admin