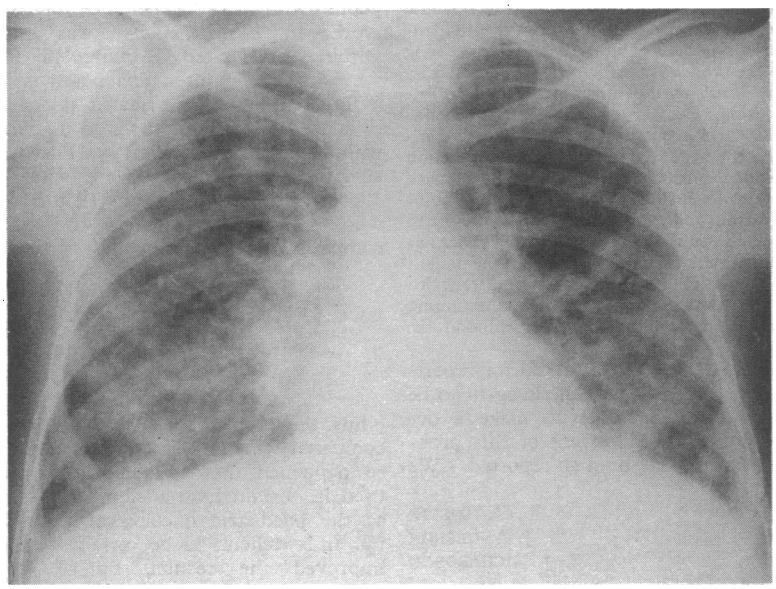

of opacity all over the right and left lung fields. The asthmatic attack was relieved only after giving 100 mg of hydrocortisone sodium succinate intravenously.—We are, etc.,

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1 Farid, Z., *et al.*, *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1972, **66**, 119.

Propranolol and the Hyperactive Carotid Sinus Reflex Syndrome

SIR,—The hyperactive carotid sinus reflex syndrome is a physiopathological condition in which syncope, originating in the carotid sinus and mediated through a cardiac and/or a peripheral vascular effect, develops on compression of the carotid sinus. The carotid sinus syndrome is an analogous physiopathological condition but of a higher grade as it manifests itself spontaneously.[1]

The existence of a carotid sinus syndrome is a contraindication for the administration of beta-adrenergic blocking agents as these drugs inhibit sympathetic stimulation, important whenever vagal tone is increased.[2] As the existence of a hyperactive carotid reflex syndrome might also be a contraindication for any therapy with beta-adrenergic blocking drugs, all the patients from our outpatient department who are to be put under treatment with propranolol are previously tested for a hyperactive carotid sinus reflex syndrome was diagnosed. A test dose Franke's procedure.[3,4]

A 62-year-old man with grade III chronic coronary insufficiency was to be treated with propranolol in an initial daily dose of 120 mg. On compression of the right carotid sinus he was found to develop a brief syncope and marked bradycardia resulting from sinus arrest followed by junctional escapes (see fig., A). Thus a hyperactive carotid sinus reflex syndrome was diagnosed. A test dose of 2 mg of propranolol was administered intravenously as a bolus injection and 10 minutes later hyperextension of the head provoked a more prolonged syncope and a more marked bradycardia than previously. Thus the hyperactive carotid sinus reflex syndrome had been transformed into a carotid sinus syndrome after propranolol. Digital compression of the carotid sinus for a shorter period than previously provoked a much more lasting sinus arrest 15 minutes after propranolol (see fig., B).

Twenty minutes after propranolol 1 mg of atropine was injected intravenously; five minutes later the right carotid sinus was massaged for several seconds. No effect was observed either on the electrical activity of the heart (see fig., C) or on the clinical condition of the patient. This last procedure and the corresponding result show the innocuity of our methods.

It is imperative to search for a hyperactive carotid sinus reflex syndrome in every patient who is to be treated with beta-adrenergic blocking drugs and this therapy avoided if the syndrome is diagnosed and the participation of a central cardiac mechanism in it demonstrated.—I am, etc.,

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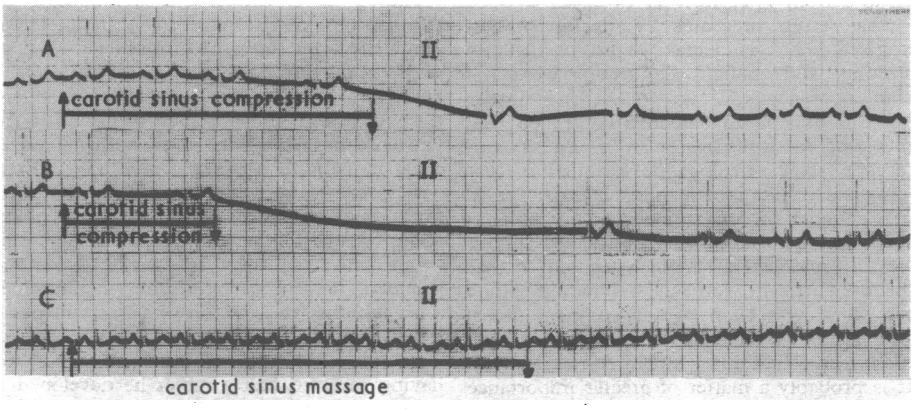
Fundacion Procardias,
Durazno 2025,
Montevideo, Uruguay

1 Thomas, J. E., *Mayo Clinic Proceedings*, 1969, **44**, 127.
2 Isasi, E. J., Reyes, A. J., and de Bayarres, M. A., *Arquivos Brasileiros de Cardiologia*, 1969, **22**, 215.
3 Franke, H., *Deutsche medizinische Wochenschrift*, 1967, **92**, 1155.
4 Grand, A., and Lapicorey, G., *Coeur et Médecine Interne*, 1972, **11**, 265.



Electrocardiograms (lead II) from patient with hyperactive carotid sinus reflex syndrome. A. Right carotid sinus compression: sinus arrest and junctional escapes. B. Carotid sinus compression after propranolol: cardiac inhibition is enhanced. C. Carotid sinus massage after atropine: no effect on cardiac activity.

New Ideas on Vitamin D

SIR,—Professor Iain MacIntyre (14 April, p. 120) referred to our point of view regarding the role of parathyroid hormone in the regulation of 1,25 dihydroxycholecalciferol (1,25-$(OH)_2D_3$) biosynthesis. The statement which he attributed to me is at best only partially correct.

Except under the abnormal hypophosphataemic state, we believe that the parathyroid glands do play an important determining role in the regulation of 1,25-$(OH)_2D_3$ synthesis. Our experiments[1] have been completely verified by Fraser and Kodicek,[2] by Hill and Mawer,[3] and by Rasmussen *et al.*[4] If animals are made hypophosphataemic, the parathyroid hormone is no longer required to stimulate 1,25-$(OH)_2D_3$ synthesis.[5] Of course we do not know the molecular events involved in the regulation, nor does anyone else to my knowledge. Though it is possible that the parathyroid hormone may stimulate 1,25-$(OH)_2D_3$ synthesis by changing ionic concentrations in the renal cell, the fact remains that under normal physiological circumstances (excluding hypophosphataemia), as far as we can presently surmise, the parathyroid hormone plays a major role in the stimulation of 1,25-$(OH)_2D_3$ synthesis. We know of no other points of view which have adequate experimental support.—I am, etc.,

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1 Garabedian, M., Holich, M. F., DeLuca, H. F., and Boyle, I. T., *Proceedings of the National Academy of Sciences of the U.S.A.*, 1972, **69**, 1673.
2 Fraser, D. R., and Kodicek, E., *Nature New Biology*, 1973, **241**, 163.
3 Hill, L. F., and Mawer, E. B., *Clinical Science*, 1973, **44**, 4p.
4 Rasmussen, H., Wong, M., Bikle, D., and Goodman, D. B., *Journal of Clinical Investigation*, 1972, **51**, 2502.
5 Tanaka, Y., and DeLuca, H. F., *Archives of Biochemistry and Biophysics*, 1973, **154**, 566.

Malignancy of Bronchial Adenoma

SIR,—With reference to your leading article on this subject (19 May, p. 378), there must be others like myself who were surprised to read that bronchial adenomas are white. Possibly some are in some circumstances,