internal and external feedback mechanisms would preclude R.F. measurements. This fact does not negate the hypothesis of Professor Hall and Dr. Warrick but simply makes the demonstration more difficult.

We have postulated that the constant release of R.F. in the L.D. syndrome may be due to a genetically transferred inborn error in metabolism. This defect may exist in the enzyme dopamine-β-hydroxylase which represents, because of a pathological gene, the rate-limiting step in the conversion of dopamine to noradrenaline (norepinephrine). This defect results in reduced amounts of noradrenaline in the brain and dopamine excess. In the L.D. patient, the combination of excess dopamine which normally drives the R.F. mechanism [7-11] and the reduction in noradrenaline that can inhibit the R.F. mechanism [12-14] effects R.F. hypersecretion. Collective evidence has underscored the important and unique role of dopamine in regulating the hypothalamic-endocrine axis. Consequently, selective inhibitors of this catecholamine may be useful in the treatment of L.D. and hopefully will ameliorate the symptoms of this disfiguring and inevitably fatal disease. Studies are presently under way using specific dopaminergic blocking agents to explore this possibility.

Persistence of L.D. in the absence of the pituitary indicates that increased blood R.F. may have extra-pituitary effects and provides a new physiological dimension. It is an important consideration that chronic secretion of R.F. may be secondary to generalised hypothalamic derangement, with the actual metabolic defect residing at the receptor sites of the various tissues (fat, muscle, bone, skin) where the disease is manifested. It is these suggestions that may provide the clue to the differences in Albright's and L.D., despite the fact that the end-result, R.F. hypersecretion, may be the same in both.

We would like to commend Professor Hall and Dr. Warrick for their conceptual foresightedness. Their hypothesis becomes more tenable in the light of our evidence demonstrating that chronic R.F. hypersecretion does exist in at least one other syndrome—lipoatrophic diabetes.

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TREATMENT OF ACUTE HYPERCALCÆMIA

SIR,—The editorial on this subject (Aug. 12, p. 314) rightly highlights the potential hazards of intravenous phosphate infusion. I suggest that mithramycin is an effective and safer alternative for most patients with hypercalcæmia associated with cancer.[15]

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7. McCann, S., Kalra, M., Donoso, P. S., Bishop, W., Schneider, H., Fawcett, P., Krulich, L. *in* Brain-Endocrine Interaction (edited by K. M. Knigge, D. E. Scott, and A. Weindl); p. 224. Basle, 1972.
8. Kamberi, I. A., Mical, R. S., Porter, J. C. *Endocrinology*, 1971, 88, 1003.
9. Kamberi, I. A., Mical, R. S., Porter, J. C. *ibid.* p. 1012.
10. Schneider, H. P. G., McCann, S. M. *ibid.* 1970, 86, 1217.
11. Porter, J. C., Kamberi, I. A., Ondo, J. G. *in* Brain-Endocrine Interaction (edited by K. M. Knigge, D. E. Scott, and A. Weindl); p. 245. Basle, 1972.
12. Ganong, W. F. *ibid.* p. 254.
13. Mayhew, D. A., Wright, P. H., Ashmore, J. *Pharmac. Rev.* 1969, 21, 183.
14. Iturriza, F. C. *Gen. comp. Endocr.* 1969, 12, 417.
15. Slayton, R. E., Shnider, B. I., Elias, E., Horton, J., Perlia, C. *Clin. Pharmac. Ther.* 1971, 12, 833.

BLOOD-LEAD LEVELS IN LONDON TAXI DRIVERS

SIR,—The interesting paper by Dr. Jones and his coworkers (Aug. 12, p. 302) does not appear to have taken account of the big difference between the behaviour of inorganic and organic lead compounds in the body. The only index that they have used to measure the absorption of lead was the blood-lead level, which, while being a useful indication of the amount of inorganic lead absorbed, is not so reliable an indicator for tetra-ethyl lead. It is this latter compound which one would presume to be the main source of lead in the bodies of taxi drivers. In this regard, Kehoe [1] goes so far as to say that the "absorption of tetra-ethyl lead does not produce an increase in the lead content of the blood that bears any apparent or proportional relationship to the quantity of lead absorbed". He advocates [1,2] the use of urinary lead estimations as an additional and perhaps more reliable check.

The unreliability of whole-blood-lead estimations themselves is further borne out by Beattie et al.,[3] who showed that in their cases of lead-alkyl poisoning the amount of lead in the lipid fraction of the blood showed a closer correlation with symptoms than total-blood-lead levels.

One would feel that the measurement of a single index is an insufficient basis upon which to build any firm conclusions. As the authors indicate they intend pursuing their investigations, might one suggest that they take into account both the urinary lead excretion and lead levels in the blood-lipid fraction, and also perhaps, in view of the work of Goldberg and his associates,[4] the activity of δ-aminolævulinic-acid-dehydrase in the blood?

Furthermore, one would also suggest that their figures could not stand in isolation and would require to be compared with a control group before any conclusions could be justifiably drawn.

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A NEW APNŒA ALARM FOR BABIES

SIR,—We wish to report early experience with a new and promising apnœa alarm for babies. The device* is essentially a mattress which senses the baby's movements, and thus his respiration, and sounds an alarm if no movement occurs over a pre-set time interval.

The mattress is of polyurethane foam impregnated with a conductive material so that its electrical resistance varies with very small changes in pressure. The material is arranged in a pattern of strips in such a way that alternate strips are in the same electrical circuit. Included between these sensing strips are strips of load-bearing material to allow the mattress to operate over a relatively wide range of baby-weights. The whole mattress is enclosed in an easily cleaned waterproof cover.

The control system, which is battery-operated, is connected to the mattress by one very thin but strong electric cable and at no time applies more than 4½ volts. The electrical output from the alternate strips in the mattress is fed into an integrated circuit amplifier, the response of which is varied by means of the sensitivity control. The output of this amplifier is used to produce an audible click for each breathing movement, and when the output is no longer present (the baby has ceased to breathe) an

1. Kehoe, R. A. *in* Industrial Hygiene and Toxicology (edited by F. A. Patty); vol. II, p. 955. New York, 1963.
2. Kehoe, R. A. *J. occup. Med.* 1972, 14, 390.
3. Beattie, A. D., Moore, M. R., Goldberg, A. *Lancet*, July 1, 1972, p. 12.
4. Millar, J. A., Thompson, G. G., Goldberg, A., Barry, P. S. I., Lowe, E. H. *Br. J. ind. Med.* 1972, 29, 317.

* The device is manufactured by Fleet Electronics Ltd., 30 Tite Street, London S.W.3.