postulated by other workers on the strength of clinical and experimental observations.[5–9]

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DIAGNOSIS OF MYOCARDIAL INFARCTION WITH LEFT BUNDLE-BRANCH BLOCK

SIR,—We have found the vectorcardiogram (Frank's method) useful in diagnosing myocardial infarction in patients with left bundle-branch block.

In left bundle-branch block, the efferent, non-delayed loop on the vectorcardiogram represents the activation of the right ventricle; the characteristically delayed afferent loop represents the (delayed) activation of the left ventricle. The activation of the left ventricle originates from the right ventricle and the cardiac vector proceeds in a posteroanterior direction (which is the opposite of the normal vector).

In combined left bundle-branch block and myocardial infarction, the afferent loop of the vectorcardiogram is deformed, representing the activation of the left ventricle.

These findings enable us to diagnose myocardial infarction in left bundle-branch block with reasonable accuracy. Translated into terms of ordinary electrocardiography the above criteria accord with the views of Chapman and Pearce.[10]

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THE CUP THAT CHEERS

SIR,—There have been a number of articles recently on postgraduate education in regional hospitals. Though the building of special postgraduate centres, as suggested by the Nuffield Foundation, is admirable, there are two much simpler measures that would do a lot to help straight away.

First, I would like to draw attention to the long-established practice of discussion over a cup of tea with visiting general practitioners or postgraduate students. This was common, especially before the Health Service started, in cottage hospitals when consultants visited on a specific day; and, during the visit, almost always there was a cup of tea and informal chat. The number of patients seen was not necessarily relevant to the value of the visit, because the doctors would discuss the various problems they faced both in the hospital and outside. Quite often the matron or sister took an interest in the discussion.

In the big teaching schools it was also not uncommon, especially on professorial units, to have similar informal staff meetings sometimes with students, and matters brought up in this way were often of far more value than formal lecture-demonstrations.

Equally essential is a room for the head of each department where he can meet his junior staff and visiting doctors. Such a room is very rarely provided for physicians and surgeons, although they do two-thirds of the work of the hospital, and it is these departments that will do most of the postgraduate teaching. When new departments are built there are always special rooms for consultants of special departments, but little thought is given to the general physicians and surgeons, who are expected to talk over problems in sister's office, corridors, and wards. A staff common-room, however magnificent, is no substitute for a room in the department.

Attention to these relatively small matters would be helpful in postgraduate teaching and in forming a satisfactory link between general practitioners and hospital staff.

Hitchin, Herts. P. J. W. MILLS.

5. Wassermann, F., Choquette, G., Cassinelli, R., Bellet, S. *Amer. J. med. Sci.* 1956, **231**, 502.
6. Weinberg, S. J. *California Med.* 1952, **77**, 253.
7. Weinberg, S. J., Fuster, J. M. *Ann. intern. Med.* 1960, **53**, 332.
8. Hugenholtz, P. G. *Amer. Heart J.* 1962, **63**, 451.
9. Melville, K. I., Blum, B., Shister, H. E., Silver, M. D. *Amer. J. Cardiol.* 1963, **12**, 781.
10. Chapman, M. G., Pearce, M. L. *Circulation*, 1957, **16**, 558.

BRACHMANN/DE LANGE SYNDROME

SIR,—Lawrence and Ishmael [1] and Hienz [2] could find no chromosomal aberrations in 3 patients with the Brachmann/de Lange syndrome; Geudeke et al.[3] and Jervis and Stimson,[4] however, observed chromosomal abnormalities in their patients, and suggested that these were relevant to the cause of the syndrome.

The almost simultaneous appearance of several articles [3–6] reporting 41 cases raises the suspicion that the syndrome is not as uncommon as was first thought. Since the publication of our report [6] in which a dominant mutation was postulated as "the most likely etiologic possibility", we have found 24 new cases, including five sibships with 14 affected sibs, and have accepted autosomal recessive inheritance as the most likely cause for the following reasons:

1. Multiple-anomaly syndromes in man (such as the Ellis/van Creveld and the Laurence-Moon-Biedl) are seldom inherited as dominant mutations, either autosomal or X-linked.
2. To date no evidence has been advanced for an environmental cause.
3. The overwhelming number of cases analysed cytogenetically—10 in our laboratory, 2 others by Geudeke et al.,[3] 2 by Polani for Schlesinger et al.,[5] 2 by Bernard et al., and 1 each by Uchida,[7] Summitt,[8] Giraud et al.,[9] Dumars and Gaskill,[10] in addition to those already mentioned [1 2]—have not revealed a chromosomal aberration. Geudeke et al.[3] and Ford [11] demonstrated an apparently balanced (euploid?) B/G translocation in a male with the Brachmann/de Lange syndrome; but none of their proposed genetic hypotheses (position effect, deletion, and break-induced mutation) seem very satisfactory. We believe, Ford [11] notwithstanding, that university hospitals tend to attract the "one out of a million" children in whom the conjunction of a chromosomal abnormality and a well-known clinical syndrome may be fortuitous. Even less convincing are the observations of Jervis and Stimson,[4] who report a non-satellited *acentric* fragment, smaller than chromosome 22, in a high proportion of cells in all of their cases. Acentric fragments do not generally perpetuate themselves to this extent; the technical quality of their chromosome preparation (their fig. 10) does not permit a meaningful karyotype analysis, and the illustrated supernumerary chromosome seems to be a metacentric F-group chromosome. Massimo and Vianello's case purporting to show mosaicism for a "small acrocentric or a fragment similar to that reported by Jervis and Stimson" remains obscure at the present time.[12]

This sometimes incompletely recessive gene may be fairly common judging by the absence of consanguinity among the parents and its occurrence in non-consanguineous first-cousin sibships (2 in our group and 1 reported by Jervis and Falek).[13]

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1. Lawrence, K. M., Ishmael, J. *Lancet*, 1963, i, 1426.
2. Hienz, H. A. *ibid.* 1963, ii, 585.
3. Geudeke, M., Bijlsma, J. B., De Bruijne, J. I. *Maandschr. Kindergeneesk.* 1963, **31**, 248.
4. Jervis, G. A., Stimson, C. W. *J. Pediat.* 1963, **63**, 634.
5. Schlesinger, B., Clayton, B., Bodsan, M., Vernon Jones, K. *Arch. Dis. Childh.* 1963, **38**, 349.
6. Ptacek, L. J., Opitz, J. M., Smith, D. W., Gerritsen, Th., Waisman, H. A. *J. Pediat.* 1963, **63**, 1000.
7. Uchida, I. Personal communication.
8. Summitt, R. Personal communication.
9. Giraud, P., Bernard, R., Giraud, F., Stahl, A., Lebeuf, M., Hartung, M. *Human Chromosome Newsletter*, 1963, **9**, 4.
10. Dumars, K. W., Gaskill, C. *ibid.* 1964, **12**, 2.
11. Ford, C. E. Autosomal Abnormalities. Second International Conference on Congenital Malformations; p. 28. New York, 1964.
12. Massimo, L., Vianello, M. G. *Human Chromosome Newsletter*, 1964, **13**, 19.
13. Jervis, G. A. Personal communication.