Early Ambulation after Myocardial Infarction

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Physicians of the calibre of Paul White and Sir Thomas Lewis advised several weeks of bed rest after acute myocardial infarction. Their advice was based not merely on clinical impression but upon knowledge of the pathological timetable[1-3] governing the healing process (Table 1). The timetable has not been shown to have been changed by any form of therapy currently in routine use.

Table 1. Time relationship of histological features of myocardial infarcts, modified from Mallory, White and Salcedo-Salgar[2].

| Necrotic muscle infiltration | Polymorph. infiltration | Vessels and connective tissue | Collagen |
|-----------------------------|-------------------------|-----------------------------|---------|
| 1 day                       | + +                     | +                            | 0       |
| 5 days                      | + + + +                 | + + + +                     | 0       |
| 7 days                      | + + + +                 | + + + +                     | 0       |
| 2 weeks                     | + + + +                 | + + + +                     | +       |
| 3 weeks                     | + + + +                 | + + + +                     | +       |
| 4 weeks                     | + + + +                 | + + + +                     | +       |
| 2 months                    | + + + +                 | + + + +                     | +       |
| 3 months                    | + + + +                 | + + + +                     | +       |

The essential features are the first appearance of collagen around the end of the first week and the continuation of healing towards a fully contracted scar over a period of several months[4]. The scar may be firm enough to withstand normal functional stresses by the fifth week[1].

Whereas ‘armchair’ nursing[5] may not increase the demand for cardiac output or call for more work from the damaged ventricle, early ambulation is likely to do so. Higher than necessary left ventricular pressures, heart rates and volumes might then impair the healing processes, stretch vulnerable myocardium and even favour the development of frank ventricular aneurysm, as was suggested long ago[6,7]. Whether or not there has been a real increase in the incidence of ventricular aneurysm in association with the present fashion for early ambulation is impossible to assess; diagnostic criteria are not agreed and the quoted incidence ranges from 4 per cent to 40 per cent[8]. However, any unnecessary expansion of the ventricular wall ought to be avoided since it places an additional haemodynamic and metabolic burden on the remaining myocardium. Hence the concept of ‘functional infarct size’, which is probably a more important determinant of haemodynamic impairment than the actual amount of infarcted myocardium[9].

The degree of expansion, after transmural infarction, increases with time for up to 7 days in rats[9] and this indicates that the critical time in humans may be about two weeks. Newer techniques such as scintigraphy, echocardiography and nuclear magnetic resonance imaging[10] should enable us to establish the in vivo healing timetable in man. Serial colour-encoded two-dimensional echocardiography may even allow us to follow the laying down of collagen in place of necrotic tissue as the lesion heals[11]. When we have such evidence it should be possible to evaluate the effects of therapeutic interventions during the critical period, particularly those aimed at minimising absolute and functional infarct size, with their implications for subsequent survival[12,13] and functional capacity. Then we shall know whether exercise during the early days favours thin scars and aneurysmal bulging, as it appears to do in dogs[14], and whether pharmacological means of ensuring rest for the damaged ventricle confer any of the long-term benefits which may have accrued from the intermediate periods of physical rest[15] that were advocated until a decade or so ago.

In the absence of such evidence, why have we, as a profession, been persuaded to ignore the basic pathological facts and to countenance or even encourage unnecessary physical activity while the sick left ventricle is still desperately in need of rest? The answer seems to lie in the misinterpretation of clinical trials that lacked the power to demonstrate any differences of outcome that may have attended somewhat different methods of management in selected sub-groups of patients (usually with ‘uncomplicated’ infarction). The fact that most of these trials were not large enough or sufficiently prolonged to reveal reasonably-to-be-expected differences of outcome at conventional levels of significance appears to have been overlooked. The consequent lack of significant differences has then been misconstrued as evidence of identity between the groups under observation, in spite of clear warnings about the dangers of the Type II error[16,17], and the data have been used to support the general notion that patients allowed or encouraged to walk about after very few days at rest do as well as those rested for rather longer. These days, authors reporting such trials would probably be asked to provide 95 per cent confidence limits which might make it abundantly clear that their negative findings did not by any means exclude the possibility that rather more rest is beneficial. The trial findings relate only to similar types of patient and it would be quite inappropriate to apply them to the management of many patients passing through coronary care units. They are also, as a rule, based on periods of follow-up as short as 6 weeks[18] or 8 months[19]. A notable exception was the randomised multi-centre trial reported by West and
Henderson[20] which suggested better survival during the second and third year if mobilisation was begun on the tenth rather than the fifth day. Further data on the 742 patients initially randomised in that large study would be very welcome.

The fundamental statistical error arising from the smaller trials may be illustrated by the study of Lamers et al.[21]. These authors followed-up 50 of 102 patients mobilised on the tenth day (but not ambulant until the fourteenth) for a mean period of 19 months, and observed 8 fatalities. This figure was compared with 9 fatalities, at a mean time of 18 months, among 48 of the 100 patients initially chosen randomly for twenty days' rest in bed. It is true that this difference is not significant but the findings are nonetheless consistent with a 50 per cent probability that a large difference of mortality between the two groups existed but was not shown by this trial[22]. On the basis of this and similar evidence, it is clearly wrong to conclude that longer rest during the acute illness confers no advantage (even during the relatively short follow-up periods of such trials). In fact, neither a beneficial nor a harmful effect can be excluded. The point has been made graphically, in relation to 'negative' trials of beta-blockade, by Baber and Lewis[23].

Whatever the outcome, the findings from Lamers' study could not have been safely used as the basis for a general policy because only a minority (203) of 555 patients with proven myocardial infarction were randomised to the trial groups. All were considered to be at low risk. All were treated by means of bed rest and anticoagulants for at least nine days. All were kept quietly in hospital for 30 days. Their low-risk status was confirmed during follow-up. Likewise, Bloch and colleagues[24] clearly selected a low-risk group for their study, in which 77 patients were allocated to ambulation at the beginning of the second week and 77 to strict bed rest for three or more weeks. No significant differences emerged during a follow-up period averaging 11.2 months, only four post-discharge deaths being observed. In the study reported by Abraham and colleagues[25] the only selection criterion was survival to Day 6 and a higher-risk population was thus recruited. No significant mortality differences emerged between the groups encouraged to walk around freely from the ninth or the sixteenth days, but more complications were recorded in the group mobilised later to whom anticoagulants were not administered routinely. Much earlier mobilisation (on Day 4), with discharge at eight days, was found by Ahlmark et al.[26] to be associated with higher fatal and non-fatal reinfarction rates even in 'uncomplicated' cases, although the differences (at three months) did not reach conventional levels of significance. Similarly, in the Hayes study[18], in which one group with uncomplicated infarction was mobilised after two days and another on the ninth day, there were three patients in the former group who died at home before their scheduled clinic attendance, whereas none of those rested for longer and not discharged home before the sixteenth day suffered that fate.

Sufficiently powerful trials will perhaps one day be mounted to provide reliable statistical evidence on which to base our management of the various categories of patients presenting with myocardial infarction, and particular attention will be paid to the possibility that early ambulation may impair long-term prognosis[27]. Until such a time, or until we have greater knowledge of the living pathology, we might serve our patients best by advising at least two weeks' rest (on anticoagulants) in bed and chair whenever they have suffered substantial (particularly transmural) cardiac necrosis. In uncomplicated cases a walk around the ward on the twelfth day appears to do no harm, the patient having rested for a full nine days and remaining under care in hospital for 21 days[28], but there is no sound basis for earlier ambulation. Suitable beta-blockade may provide additional protection against ventricular stress during this period and allow acceleration of the rehabilitation phase if this is felt to be desirable on psychological grounds or for employment reasons. Even so, it is probably unwise to encourage strenuous activity until two or three months have passed, by which time the myocardial scar should be firmly contracted.

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References

1. Levine, S. A. and Brown, C. L. (1929) Medicine (Baltimore), 8, 245.
2. Mallory, G. K., White, P. D. and Salcedo-Salgar, J. (1939) American Heart Journal, 18, 647.
3. Lodge-Patch, I. (1951) British Heart Journal, 13, 37.
4. Hudson, R. E. B. (1965) Cardiovascular Pathology, 1, p. 652. London: Edward Arnold.
5. Levine, S. A. and Lown, B. (1952) Journal of the American Medical Association, 148, 1365.
6. Ball, D. (1938) American Heart Journal, 16, 203.
7. Moyer, J. B. and Hiller, G. I. (1951) ibid., 41, 340.
8. Goldberg, M. J. (1982) British Journal of Hospital Medicine, 27, 143.
9. Hochman, J. S. and Bulkey, B. H. (1982) Circulation, 65, 1446.
10. Goldman, M. R., Brady, T. J., Pykett, I. L. et al. (1982) ibid., 66, 1012.
11. Parisi, A. F., Nieminen, M. O'Boyle, J. E. et al. (1982) ibid., 66, 764.
12. Norris, R. M., Caughley, D. E. and Mercer, C. J. (1974) British Heart Journal, 36, 786.
13. Cabin, H. S. and Roberts, W. C. (1982) American Heart Journal, 104, 216.
14. Sutton, D. C. and Davis, M. D. (1931) Archives of Internal Medicine, 48, 1118.
15. Friedberg, C. K. (1956) Diseases of the heart, 2nd edn, p. 566. Philadelphia and London: Saunders.
16. Freimen, J. A., Chalmers, T. C., Smith, H. and Kuebler, R. R. (1978) New England Journal of Medicine, 299, 690.
17. Editorial (1978) British Medical Journal, 2, 1318.
18. Hayes, M. J., Morris, G. K. and Hampton, J. R. (1974) ibid., 3, 10.
19. Harpur, J. E., Kellett, R. J., Conner, W. T. et al. (1971) Lancet, 2, 1331.
20. West, R. R. and Henderson, A. H. (1979) British Heart Journal, 42, 381.
21. Lamers, H. J., Drost, W. S. J., Kroon, B. J. M. et al. (1973) British Medical Journal, 1, 257.
22. Hanka, R. (1980) 'Minimal sample sizes required when comparing two independent proportions.' Internal report, School of Clinical Medicine, University of Cambridge.
23. Baber, N. S. and Lewis, J. A. (1980) British Medical Journal, 2, 59.
24. Bloch, A., Maeder, J.-P., Haissly, J.-C. et al. (1974) American Journal of Cardiology, 34, 152.
25. Abraham, A. S., Sever, Y., Winston, M. et al. (1975) New England Journal of Medicine, 292, 719.
26. Ahlmark, G., Ahlberg, G., Saetre, H. et al. (1979) Acta Medica Scandinavica, 206, 87.
27. Miller, A. J. (1976) American Heart Journal, 92, 547.
28. Medical Division, Royal Infirmary, Glasgow (1973) Lancet, 2, 346.

Book Review

Clinical Dermatology: Diagnosis and Therapy of Common Skin Diseases by P. Vasarinsh. Butterworth, Boston and London, 1983. Price £45.

In the English language there are two superb massive textbooks of dermatology, one British and one American, for the dermatologist in training and practice and as a reference source for others. At the other end of the spectrum there is a plethora of small slim elementary texts for medical students, which are also useful for house physicians and even those preparing for the MRCP examination. These latter books are supplemented by a number of colour atlases of skin disease, which are usually accompanied by a rudimentary text.

Dr Vasarinsh's book is different. It is a book for family practitioners, paediatricians and general physicians (internists) who do not have access to a dermatologist in the adjacent suite of the hospital outpatient department or in an office or hospital in the same town, or even in any town or city within a few hundred miles. In other words, this is a book for doctors who have had no postgraduate dermatological training who find themselves compelled to look after patients presenting with skin disease. Clearly, in the UK, which is small and has a highly developed NHS network of dermatological services, few practitioners are in this position. But in Canada, where Dr Vasarinsh practices and teaches, in much of the USA, in extensive English-speaking parts of Africa, the Indian sub-continent and South East Asia, many, if not most, generalists find themselves in this situation and have in general been badly served by the (predominantly teaching hospital) dermatologists who spew out books of all sizes.

Dr Vasarinsh's book is substantial, over 700 pages, printed on good paper, hardbacked and not too closely packed. It will stand thousands of short journeys from the bookshelf to the knee and back again and survive until another edition replaces it. To the dermatologist it is simplistic, even naive. It is bread-and-butter dermatology. But if the reviewer were a family practitioner in Butetown, Montana, or, flitting across the world, in Ooty with relics of the Raj in the waiting room, this book would rank with those other basic and precious volumes on which the isolated physician depends.

The sense of perspective is impressive. Many pages are devoted to detailed consideration of the commonest dermatoses in all their atypical and typical aspects. Differential diagnosis is emphasised and highlighted in short lists in windows of contrasting shade to help rapid identification and reference. Therapeutic advice is detailed and comprehensive. Dr Vasarinsh provides answers to the questions patients will ask. The fact that they are not always the right answers does not matter. They will do; they are sensible and safe.

The book is amply illustrated but only with black and white photographs. Now that colour reproduction has become much cheaper, one hopes that a future edition can include a colour atlas. Monochrome illustrations are useless to the reader whose dermatological mind's eye is innocent. A general practitioner friend to whom I showed this volume found only one in six of the photographs of any value.

One could make scores of minor criticisms, but they are irrelevant. Much more attention to the use of anthralin in psoriasis would be useful. Of the main ones, the pharmacopoeia is too orientated to North American trade names. It would be a very useful addition if a future edition could be internationalised from the therapeutic point of view.

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