Several tests have been devised based on the effect of A.L.S. on lymphocytes in vitro but, as with the anti-tumour assays mentioned previously, none of them provides an accurate forecast of the efficacy of ALS in vivo. It is not for want of trying that no in vitro assay exists; it has just proved intractably difficult and no amounts of exhortation or legislation would have altered the outcome.

ANTIBODY FORMATION

The first volume of the abstracts of *Alternatives to Laboratory Animals*, published by F.R.A.M.E., contains a section on immunology among which a quarter of the references (five of the 19) concern techniques for studying the formation of antibody in vitro.¹⁶ The selection is interesting because it illustrates the confusion about what constitutes an "alternative." Thus, anyone wishing to repeat the work described in each of these five papers would have to hold a Home Office licence since each requires the production of special reagents in living animals. The method which has excited the most recent interest is that given in abstract 1 1 G18.¹⁷ Though not stated in the abstract the method described in this paper requires the preparation of spleen cells from mice which have been actively immunized together with spleen cells taken from other mice six days after the administration of 800 rads of whole body radiation followed by an intravenous injection of thymus cells and antigen. As has already been mentioned, this technique promises to add considerably to our understanding of the mechanism of antibody formation but though it is an in vitro technique it is hardly an alternative; indeed, it is a great consumer of experimental animals and by its very popularity may actually increase rather than decrease their use.

I mention the five abstracts concerned with the production of antibody because my colleagues and I have been interested over several years in the cellular basis of antibody formation but we have only employed an in vitro method on one occasion.¹⁸ This is because in vitro methods for the production of antibody do not show a significant switch from 19S to 7S production, they result in the production of antibody of predominantly low affinity, and they do not generate immunological memory. This would be the reply if I were asked why the bulk of our work has been performed on living animals. I make the point in this way to illustrate that arguments

whether an alternative is acceptable would be *exclusively* technical; common sense and legal training could not decide the issue.

"A research institute should be set up to look into this matter"¹⁹

My proposal for the Institute for the Study of Alternative Methods is that to be effective it must have an enormous animal house. In vitro methods not only require a continual expenditure of animals to provide the ingredients for the techniques but to validate them as substitutes for in vivo studies they must be continually checked at all stages of development against the responses of the animals they are attempting to replace. The complexity of biological mechanisms and the intractable difficulty of many of the outstanding problems in medical research make it not altogether cynical to suggest that there are not enough animals available in the research laboratories of the world today to decide whether they can be replaced by alternative methods.

References

- ¹ Hansard, House of Commons, 20 July 1973, col. 1092.
- ² Hansard, House of Commons, 11 May 1973, col. 893.
- ³ Mishell, R. I., and Dutton, R. W., *Journal of Experimental Medicine*, 1967, 126, 423.
- ⁴ Feldmann, M., and Basten, A., *Journal of Experimental Medicine*, 1972, 136, 49.
- ⁵ Hansard, House of Commons, 11 May 1973, col. 896.
- ⁶ Hellstrom, K. E., and Hellstrom, I., *Annual Review of Microbiology*, 1970, 24, 373.
- ⁷ Fairley, G. Hamilton, *British Journal of Hospital Medicine*, 1971, 6, 633.
- ⁸ Mitchison, N. A., *Transplantation Proceedings*, 1970, 2, 92.
- ⁹ Harris, H., *Cell Fusion*. Oxford, Clarendon Press, 1970.
- ¹⁰ Watkins, J. F., and Chen, L., *Nature*, 1969, 223, 1018.
- ¹¹ Evans, R., and Alexander, P., *Immunology*, 1972, 23, 627.
- ¹² MacLennan, I. C. M., *Transplantation Reviews*, 1972, 13, 67.
- ¹³ Perlmann, P., Perlmann, H., and Wigzell, H., *Transplantation Reviews*, 1972, 13, 91.
- ¹⁴ Cerottini, J. C., Nordin, A. A., and Brunner, K. T., *Nature*, 1970, 228, 1308.
- ¹⁵ Lance, E. M., Medawar, P. B., and Taub, R. N., *Advances in Immunology*, 1973, 17, 1.
- ¹⁶ *Abstracts of Alternatives to Laboratory Animals*, 1973, 1, 74.
- ¹⁷ Feldmann, M., *Nature New Biology*, 1973, 242, 82.
- ¹⁸ Ford, W. L., and Gowans, J. L., *Proceedings of the Royal Society of London*, 1967, Series B, 168, 244.
- ¹⁹ Hansard, House of Commons, 11 May 1973, col. 941.

General Practice Observed

Home or Hospital Care after Myocardial Infarction: Is this the Right Question?

A. COLLING

British Medical Journal, 1974, 1, 559-563

Summary

This article attempts to lay down guide-lines for the general

Stockton-on-Tees, Teesside

A. COLLING, M.D., F.R.C.G.P., General Practitioner

practitioner faced with a patient with a myocardial infarction in his home. A too rigid distinction between home or hospital care should not be attempted. Rather the general practitioner should look at home and hospital care and decide which is more appropriate in a particular case. In particular, he must distinguish clearly between cases he sees very soon after an attack and those he sees some hours later. Two hours is suggested as a useful dividing time between the two groups of patients.

Introduction

Talking to general practitioners and vocational trainees during the last few years about the care of patients after myocardial infarction, I have become aware of the predicament of the primary physician who has to decide whether to send his patient to hospital or to treat him at home. This difficulty is reflected in the different admission rates reported in surveys from various parts of the country—for example, Oxford 74%,¹ Edinburgh 85%,² Bristol 77%,³ and Teesside 66%.⁴

Trainees ask for guidance. What are we to say? Over the last 25 years one treatment after another has been shown to reduce the mortality of those cases admitted to hospital⁵⁻¹⁰ only to be disproved or doubted later as a result of further inquiry.¹¹⁻¹⁹ With the advent of the coronary care unit it was not surprising that family doctors looked a little suspiciously at all the figures and wondered if, after all, it was not better to treat patients at home—a view apparently shared by at least one cardiologist.²⁰

Coronary care units have been established in many hospitals by physicians who argue that if patients with myocardial infarctions arrive in their hospitals this is the best treatment they can offer. They were not built as a result of a community studying available evidence and then deciding on a total policy for "coronary care." There is little guidance for the general practitioner faced with an ill patient in his home. For some doctors the argument remains an academic one for they practise many miles from the nearest hospital.²¹ Others perhaps have a cottage hospital near at hand, but what of the many doctors who practise within reach of a coronary care unit? How can they decide whether to send their patients to hospital?

Most commentators agree that a general medical ward offers no advantage over home care. But what of the coronary care unit itself? The early favourable reports from these came from large teaching hospitals and they warned of the danger of applying their results to the district, or community hospitals.²² Nevertheless, subsequent reports from general hospitals showed similar figures.^{3,26} But there is no way of assessing how significant these figures are, since randomization of cases has not seemed justified and physicians apply treatment as a matter of faith. They argue that some of the cases admitted to hospital develop cardiac arrest and are resuscitated, and a few of them survive to leave hospital. What is not known is whether so many arrests would have occurred if patients had been made comfortable in their own home surroundings and if they did occur how many would have recovered spontaneously.

The bold experiment at Bristol,³ in which some patients with myocardial infarction were treated either at home or in coronary care units, seemed to favour slightly those who stayed at home. But this investigation largely ignored the time factor. Many workers have shown that most deaths after myocardial infarction occur soon after the attack. About 40-50% of deaths occur within two hours and over 60% in men, middle aged and under.²⁷⁻²⁹ In any investigation it is important to say how quickly the cases come under care, because if the time is short then the mortality would be expected to be high. If there is long delay, then most of the deaths will have already occurred. In Bristol only 37% of patients were in contact with a doctor within the first two hours, by which time most of those who were going to die had done so. Another criticism was that only 28% of the cases of myocardial infarctions in the Bristol survey were randomized, the bulk of patients and doctors electing for hospital or home care on other criteria. Thus there were more random cases admitted early to hospital than kept at home, a feature which would tend to raise the hospital mortality, though the cases may not have been compatible in other ways.

A general practitioner may have about 10-12 myocardial infarctions a year in his practice, but usually he is called after the time of greatest mortality. In only about 25-40% of cases

will he be called within the first hour,^{3,26,31} though possibly doctors are being called earlier as the public becomes more aware of the problem.³¹ From these figures it is apparent that the general practitioner may only be in a life-saving position in coronary care two to four times a year.

Because each case seen by the general practitioner has a different series of pathological, social, geographical, and personal factors, it is impossible to lay down precise rules of management. Nevertheless, it is important to try to establish guide-lines for doctors undertaking vocational training for general practice. Many general practitioners now use electrocardiographs (40% in a recent survey on Teesside⁴) and entrants into general practice are now expected to have a working knowledge of cardiology.³² Vocational trainees and candidates for the R.C.G.P. examination seem to have a good knowledge of drugs used in acute coronary care. Therefore it seems reasonable when working out guide lines that we assume that the future general practitioner will be able to use the cardiograph and drugs appropriate to the coronary emergency.

Suggested Approach

Of all deaths occurring after myocardial infarction, about half occur within two hours.²³ There would seem to be a case for a general practitioner to differentiate between those patients he sees soon after the onset of the attack and those first visited some hours after an infarction. There is extreme urgency in reaching the earlier group as this is when patients die and when first aid is most needed. The general practitioner is unlikely to possess a defibrillator, but there is good evidence that the presence of a doctor, the relief of pain, and the correction of arrhythmias can considerably benefit the patient, and some of these measures are well within the competence of the general practitioner. Ventricular fibrillation will occur in about 14% of cases he reaches within the first two hours.³⁰ Apart from external cardiac massage he may be able to do little for these patients until help arrives or he can take his patient to hospital. In other dysrhythmias, however—such as bradycardia, heart block, ventricular ectopic beats, supraventricular and ventricular tachycardia—then the use of drugs such as atropine, lignocaine, and practolol may help. In the remainder of patients, the general practitioner is called several hours after the attack, often the next morning. Different rules then apply and there is less urgency.

Let us take two hours as the dividing line between these two groups of patients. It is an arbitrary figure and in practice should not be taken too literally, but it is a useful cut-off point to pursue this approach.

If a doctor is called within two hours of an attack he should remain with the patient until two hours from the onset, establishing a diagnosis, giving treatment, and monitoring the patient with his electrocardiograph. The electrocardiograph should remain attached to the patient for this purpose, though not actually using paper, the doctor being kept aware of the rhythm, either by the sight or sound of the needle. Short tracings can be taken from time to time. A simple monitor/defibrillator will soon be available which could be used by a group practice or health centre but it will be relatively expensive.

Though two hours may seem a long time for the general practitioner to remain in the patient's home, it is not so forbidding as it sounds. There is always some delay in the patient or family sending for the doctor and also in the general practitioner receiving the message and travelling to the patient's home. By the time the general practitioner has made his assessment, given analgesics, waited for them to relieve pain, taken a cardiograph, perhaps also blood for enzyme estimation, then much of this time will have elapsed. If conditions were right a doctor might decide to transfer his patient to

Summary of Guide Lines

	Patient Seen Within Two Hours of Attack	Patient First Seen Two Hours after Attack
Urgency	Drop everything. Go at once. (This is when people die)	Go as soon as possible, but use discretion
Care of Patient	Ease pain quickly and keep patient as still as possible. <i>Give enough analgesic to ease pain.</i> Patient must remain under medical supervision for two hours from onset of attack. Do not move to hospital without a doctor. Monitor on E.C.G. If in doubt do not move the patient.	Ease pain quickly and keep patient as still as possible. <i>Give enough analgesic to ease pain.</i> Arrange treatment at home or hospital. Criteria are social except (1) when heart block (2) unstable rhythm. When hospital treatment is indicated for medical reasons then transfer to hospital should be under a doctor's supervision.
Treatment (Use with Caution. Give all injections slowly)	<i>Diamorphine</i> 5 mg intramuscularly (intravenously if necessary). Repeat if pain unrelieved. <i>Atropine</i> * 0.6 mg aliquots (intravenously) if bradycardia (less than 55) or heart block. Suggested not more than two aliquots in general practice. Duration of action two to four hours. <i>Practolol</i> * if supraventricular tachycardia (more than 140) 5 mg aliquots (intravenously). May take 20 minutes to work. Suggested not more than two aliquots in general practice. Do not use in patients with asthma or obstructive airway disease. <i>Lignocaine</i> * 50-mg aliquots (intravenously) if (1) Ventricular ectopic beats more than 5/min. (2) Coupling of ectopic beats. (3) R on T ventricular ectopic beats. (4) Runs of ventricular tachycardia. (5) Ventricular tachycardia. Suggested not more than two aliquots in general practice. Contraindicated in heart block and bradycardia. Duration of action about 10-20 min.	The same
Later	After 2 hours arrange treatment at home or hospital, depending on social factors and clinical state of patient	

*A general practitioner will rarely need to use these drugs, but they could prove life saving. It is recommended that he should keep a special box of drugs for use in cardiac emergencies, which could also contain makers' leaflets or other instructions to remind him of the indications and contraindications for their use.

hospital before two hours have elapsed but the patient would need to have been stabilized, a coronary care unit should be near at hand, and good transfer facilities must be available.

After two hours the general practitioner must decide whether to continue to treat the patient at home or whether to transfer him to hospital. If the patient is free of pain, in stable rhythm, and with some one to care for him, then he can safely be cared for at home. There are risks of course, but on available evidence they are balanced by unknown harmful factors in those patients who are transferred to hospital. The situation may change when more is known about which patients are in danger of developing cardiac arrest later. Minor degrees of shock and heart failure are not contraindications to home care. If the patient is very shocked or has recently had a dysrhythmia causing concern to the doctor, then the decision becomes more difficult. He must continue to remain under continuous medical supervision and it is unlikely that the general practitioner will be able, or willing, to devote further time to the patient. He must be transferred to hospital, and under a doctor's supervision. The general practitioner must accept responsibility for seeing that this is done, either by coronary ambulance, consultant, or himself. Indeed, those who advocate the use of the coronary ambulance emphasize that a

patient should not be transferred to hospital until he is free of pain and in a stable rhythm.

It is easier to decide how to treat the second group of patients, in which the general practitioner has been called some some hours after the attack. From the evidence at Bristol,³ which largely dealt with this group of patients, and which compared home and hospital treatment, there seems to be no overall benefit in hospital care. If anything, it is slightly better to remain at home. Hospital admission, therefore, is indicated only for social reasons or when there is some persistent medical problem which requires hospital supervision—for example, heart block, unstable rhythm, or loud murmurs, which may suggest ruptured papillary muscles or aneurysms.

Severe Shock

The general practitioner will occasionally be faced with a severely shocked patient after myocardial infarction who could be in either of the two groups. The results of treatment for these patients are very bad in hospital, but it is difficult to believe that a general practitioner would often be prepared to undertake their care at home. Nevertheless, a confident general practitioner, or one many miles from a coronary care unit might decide that the risks of transfer were too high. He would be justified in keeping his patient at home. Much would depend on the age of the patient and other factors, such as whether a coronary ambulance was available and the wishes of the relatives.

Two cases demonstrate this approach.

Case 1

At 12.50 p.m. on 10 January 1970 my surgery called me on the radio telephone to say that an old woman of 80 had collapsed at home. I arrived within five minutes and found her lying semi-conscious on the floor, groaning, and unable to answer questions. There were no obvious signs of a stroke. Her daughter told me that her mother had been drinking a glass of sherry before lunch when she suddenly developed an epigastric pain and fell backwards (12.30 p.m.). While I assessed the patient, her son collected the cardiograph from my surgery. An E.C.G. (1.15 p.m.) showed a massive inferior infarction. She was given an analgesic and nursed on the floor where she had collapsed. B.P. 100/60. The electrocardiograph was left attached to the patient and the tracing was monitored intermittently every few minutes, when the needle appeared to show or sound any change. As will be seen from the tracing, the complex (lead II) changed considerably over the following hour (fig. 1). At times the needle was scarcely moving and I thought the patient was dying. However, her condition stabilized and by 2.30 p.m., two hours after the infarction, I considered her fit to leave for a while. By 4.00 p.m. she was fully conscious, a bed was brought to where she lay, and she was nursed at home. B.P. 160/90. She made a good recovery and has since travelled abroad on her own, a sprightly 83-year-old woman.

Case 2

On 23 September 1972 an unmarried man of 39 suddenly sweated and collapsed with chest pain while showing films at a club. His father, who was with him, blamed the hot atmosphere, took him home by car and sent for me when he didn't rapidly improve.

He was pale, cold and clammy, and complained of pain across his chest. The blood pressure was not recordable. He was given diamorphine 5 mg intravenously immediately. A cardiograph showed an inferior infarction. He was considered too ill to move to the coronary care unit 10 miles (km) away. Monitoring was continued as in Case 1 (see Lead II fig. 2).

After some runs of ventricular tachycardia, he was given intravenous lignocaine. At 00.40 (2 hours 10 minutes after the attack) he was still extremely ill with an unstable rhythm and clearly unfit to be transferred to hospital. I asked a consultant physician for a domiciliary visit. Before he arrived the patient developed bradycardia and heart block, and was given atropine, the second aliquot giving a reasonable rate and clearing the block.

FIG. 1—E.C.G. in Case 1.

FIG. 2—2-E.C.G. in Case 2.

The physician arrived at 00.55 hours, by which time the heart block had gone and, though still very ill, the patient had stabilized. Because of his recent dysrhythmias it seemed unwise to continue his treatment at home. An ambulance was called by the physician and we accompanied the patient into hospital.

He made a complete recovery and when last seen 11 months after the attack, he was running two miles (3 km) every evening without disability.

Discussion

Over the last 10 or 15 years, most general practitioners have come to regard hospital treatment as obligatory in nearly all cases of myocardial infarction, impressed by the conviction with which cardiologists pressed their case for various forms of treatment. These views are now being questioned and the trend is perhaps beginning to reverse. General practitioners are becoming preoccupied with the question: home or hospital? Is it better to keep patients at home rather than risk transport to hospital? I believe there is sufficient evidence now

to put this question into perspective, which would lead to general practitioners using home *and* hospital for the treatment of infarction along the guide lines above.

It must be assumed that the general practitioner is familiar with the electrocardiograph and the use of a few basic drugs. This is not too much to ask of our vocational trainees entering general practice, as it is known to be accepted by many older practitioners. It is important to formalize this kind of teaching and for the general practitioner to know with conviction what can be considered correct treatment on our present knowledge. Myocardial infarction is dangerous and deaths will occur at home just as they do in hospital. A young doctor losing a patient at home could easily believe it might not have happened if he had sent him to hospital. So could an older doctor who had decided to try and treat patients at home.

I must emphasize that these are only guide lines for treatment. They are not meant to be strict criteria. Each general practitioner must look at the situation in his area. Is there a coronary ambulance and a coronary care unit? How far are his patients away from the coronary care unit? He should

not assume that the conditions existing in his local coronary care unit are as good as those in highly specialized cardiology units described in the journals. Each general practitioner should decide on a policy and carry it out with full conviction.

Because of staffing difficulties the coronary ambulance is unlikely to become widely available and the general practitioner must learn to give primary care of high quality. In addition, there is disquiet about the delay sometimes experienced in finding a doctor in an emergency and, though outside the scope of this article, it is a problem to be examined by every practice.

In future possibly the whole medical community of an area will look at the total situation and formulate a community policy for treating myocardial infarction, but this is only in the early stages of development.⁴

I would like to thank those doctors who kindly read the draft of the paper during its preparation and, particularly, Dr. A. Dellipiani and Dr. J. Whewell who have made helpful suggestions.

References

- ¹ L. J. Kinlen, *A Community Study of Myocardial Infarction and Sudden Death*, Doctor of Philosophy Thesis. Oxford, 1969.
- ² A. Armstrong, *et al.*, *British Heart Journal*, 1972, 34, 67.
- ³ H. G. Mather, *et al.*, *British Medical Journal*, 1971, 3, 334.
- ⁴ *Teesside Coronary Survey*. In press.
- ⁵ A. M. Master, *American Heart Journal*, 1936, 12, 545.

- ⁶ I. S. Wright, C. D. Marple, and D. F. Beck, *Myocardial Infarction. Its Clinical Manifestations and Treatment with Anticoagulants*. New York, 1954.
- ⁷ A. Pitt, H. Lipp, and S. T. Anderson, *Lancet*, 1971, 1, 612.
- ⁸ European Working Party, *British Medical Journal*, 1971, 3, 325.
- ⁹ J. V. Rosso, G. C. Friesinger, S. Margolis, and R. S. Ross, *Lancet*, 1970, 2, 1271.
- ¹⁰ B. Mitra, *Lancet*, 1965, 2, 607.
- ¹¹ Medical Research Council, *British Medical Journal*, 1969, 1, 335.
- ¹² P. G. Nelson, *British Medical Journal*, 1970, 3, 735.
- ¹³ M. A. Bennett, J. M. Wilner, and B. L. Pentecost, *Lancet*, 1970, 2, 909.
- ¹⁴ N. Dioguandi, *et al.*, *Lancet*, 1971, 2, 891.
- ¹⁵ J. D. Eddy, and S. P. Singh, *Lancet*, 1969, 2, 1378.
- ¹⁶ B. L. Pentecost, N. M. Mayne, and P. Lamb, *Lancet*, 1968, 1, 946.
- ¹⁷ D. McC. Boyle, J. M. Barber, M. J. Walsh, G. Shivalingappa, and N. C. Chaturvedi, *Lancet*, 1972, 2, 57.
- ¹⁸ H. J. Laners, *et al.*, *British Medical Journal*, 1973, 1, 257.
- ¹⁹ G. E. Honey, and S. C. Truelove, *Lancet*, 1957, 1, 1155.
- ²⁰ D. Evan Bedford, *British Medical Journal*, 1968, 4, 273.
- ²¹ D. P. Black, *Management of Myocardial Infarction in a Rural Area*. In press.
- ²² M. F. Oliver, D. G. Julian, and K. W. Donald, *American Journal of Cardiology*, 1967, 20, 465.
- ²³ B. L. Pentecost, and N. M. C. Mayne, *British Medical Journal*, 1968, 1, 830.
- ²⁴ C. P. Aber, R. W. Portal, and M. P. Chopra, *British Medical Journal*, 1969, 1, 209.
- ²⁵ P. C. Reynell, *British Medical Journal*, 1969, 2, 502.
- ²⁶ G. J. MacKenzie, B. C. Ogilvie, T. L. Turner, M. Fulton, and W. Lutz, *Lancet*, 1971, 2, 200.
- ²⁷ R. H. McNeill, and J. Pemberton, *British Medical Journal*, 1968, 2, 139.
- ²⁸ M. Fulton, D. G. Julian, and M. F. Oliver, *Circulation*, 1969, 40, suppl. No. 4, 182.
- ²⁹ C. R. Bainton, and D. R. Peterson, *New England Journal of Medicine*, 1963, 268, 569.
- ³⁰ A. A. J. Adgey, *et al.*, *Lancet*, 1971, 2, 502.
- ³¹ H. C. Smyllie, M. P. Taylor, and R. A. Cunningham-Green, *British Medical Journal*, 1972, 1, 32.
- ³² Personal observation.

Medical History

Some Social and Forensic Aspects of Exhumation and Reinterment of Industrial Revolution Remains

E. J. DUFF, J. S. JOHNSON

British Medical Journal, 1974, 1, 563-567

Summary

The aetiological aspects of exhumed remains from two burial sites were examined using 1839 and 1879 as years of comparison. We tried to discover whether the sample of recovered remains was representative of those buried. The state of the remains varied according to the type of soil and coffin material in which they were buried. At the earlier date most deaths were caused by infectious lesions rather than degenerative ones and 76% of those who died were below employable age—whereas in 1879 the commonest causes of death were tuberculosis ("phthisis") and bronchitis, and 42% died before they could be employed. The registration of deaths were recorded more accurately at the later date, and it was easier to build up a picture of the age, sex, and occupation of the people who died.

Departments of Preventive Dentistry and Orthodontics, University of Manchester
E. J. DUFF, B.Sc., PH.D., Honorary Lecturer in Dental Crystallography
J. S. JOHNSON, PH.D., D.ORTH., Senior Lecturer in Orthodontics

Introduction

Reports on exhumed remains from burial sites have usually been either on those found on ancient sites or they have been on recent deaths, and it was a unique opportunity when a road-widening scheme in Ashton-under-Lyne enabled us to examine a number of 19th century remains. As several thousand remains were to be removed from one burial site to another we expected that the findings resulting from anthropometric and dental examinations would lend themselves to a statistical analysis.

The people in this area (now the town centre of Ashton-under-Lyne) had a period of social and economic change, when the rudiments of community health measures were modifying an environment of squalor, inadequate accommodation, rudimentary sanitation, long working hours, and malnutrition. The 1834 Poor Law Amendment Act, the repeal of the Corn Laws in 1846, and numerous factory and public health acts affected every household directly or indirectly. Though malnutrition was common, the diet increased in refinement—particularly in the intake of carbohydrates—but there was little evidence of extensive dental treatment. The overall investigation of the remains was primarily orientated towards the aetiological aspects of the latter and not directed towards forensic details.

The road widening eroded the margins of two burial sites near the town centre, separated from each other by about 400 yards (370 metres). St. Michael's parish churchyard (fig. 1) was the