without proportionate increase in length may reflect adverse influences in late gestation.²¹

Our findings are further evidence for the in utero origin of raised adult blood pressure. Fingertip whorls and a narrow palmar angle are indelible markers of impaired fetal development at different stages in pregnancy. Both are associated with high blood pressure in adult life.

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- Barker DJP, Bull AR, Osmond C, Simmonds SJ. Fetal and placental size and risk of hypertension in adult life. *BMJ* 1990;301:259-62.
 Barker DJP, Meade TW, Fall CHD, Lee A, Osmond C, Phipps K, Stirling Y.
- 2 Barker DJP, Meade TW, Fall CHD, Lee A, Osmond C, Phipps K, Stirling Y. Relation of fetal and infant growth to plasma fibrinogen and factor VII concentrations in adult life. *BM* 91992;304:148-52.
- Barker DJP, Osmond C, Golding J, Kuh D, Wadsworth MEJ. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. *BMJ* 1989;298:564-7.
 Law CM, Barker DJP, Bull AR, Osmond C. Maternal and fetal influences on
- Law CM, Barker DJP, Bull AR, Osmond C. Maternal and fetal influences on blood pressure. Arch Dis Child 1991;66:1291-5.
 Barker DJP, Godfrey KM, Bull AR, Osmond C. The relation of fetal length,

- 6 Mulvihill JJ, Smith DW. The genesis of dermatoglyphics. J Pediatr 1969;75: 579-89.
- Iacroix B, Wolff-Quenot M-J, Haffen K. Early human hand morphology: an estimation of fetal age. *Early Hum Devel* 1984;9:127-36.
 Penrose LS. Medical significance of finger-prints and related phenomena.
- BMJ 1968;ii:321-5.
 9 Reed T, Uchida IS, Norton JA, Christian JC. Comparisons of dermatoglyphic
- patterns in monochorionic and dicharly to comparison of units of grant and grant patterns in monochorionic and dicharly to compare the set of the set of
- 1968;iii:141-3. 11 Jain PK, Sharma BK, Mathur BD. Dermatoglyphics in essential hypertension.
- JAsso Physicians India 1984;32:335-7. 12 Pursnani ML, Elhence GP, Tibrewala L. Palmar dermatoglyphics in essential
- hypertension. Indian Heart J 1989;41:119-22.
 13 Rashad MN, Mi MP. Dermatoglyphic traits in patients with cardiovascular disorders. Am J Phys Anthropol 1975;42:281-3.
- usorders. Am J Fray Animopol 1913;42:01-3. 14 Cherrill FR. The fingerprint system at Scotland Yard. London: HMSO, 1954. 15 Dawes GS, Borruto F, Zacutti A, Zacutti A, Jr. Fetal autonomy and adaptation.
- Chichester: Wiley, 1990:27. 16 Kinmonth JB, Negus D. Arteriovenous fistula in the management of lower
- limb discrepancy. J Cardiovasc Surg (Torino) 1974;15:447-53. 17 Young AE. Maldevelopments of the vascular system: clinical conundrums. In:
- Nugent J, O'Connor M, eds. Development of the vascular system. London: Pitman, 1983:222-43. (Ciba Foundation Symposium No 100.)
 18 Barry A. The aortic arch derivatives in the human adult. Anat Rec 1951;111:
- 221. 19 Rudolph AM. The fetal circulation and its response to stress. J Dev Physiol
- 1984;6:11-9. 20 Robinson SM, Wheeler T, Hayes MC, Barker DJP, Osmond C. Fetal heart
- Robinson Sin, Witcher J., Hayes Web, Barker D.J., Ositolia C., Fedarikart rate and intrauterine growth. Br J Obstet Gynaecol 1991;98:1223-7.
 Barker DJP, Gluckman PD, Godfrey KM, Harding J, Owens JA, Robinson JS. Fetal nutrition and cardiovascular disease in adult life. Lancet 1993;341:
- JS. Fetal nutrition and cardiovascular disease in adult life. Lancet 1993;341 938-41.

Earliest electrocardiographic evidence of myocardial infarction: implications for thrombolytic treatment

Jacqueline Adams, Roger Trent, John Rawles on behalf of the GREAT Group

Abstract

Objectives—To determine the incidence of the earliest electrocardiographic changes in patients with suspected myocardial infarction and their sensitivity and specificity for predicting the final diagnosis of acute myocardial infarction.

Design—Retrospective study of paired electrocardiograms recorded at home and on admission to hospital.

Setting-29 rural practices in Grampian and teaching hospitals in Aberdeen.

Patients—137 patients participating in the early anistreplase trial in the Grampian region, who received placebo at home and for whom paired electrocardiograms were available.

Main outcome measures—Classified electrocardiographic abnormalities and diagnosis at discharge.

Results—Electrocardiograms recorded were immediately before injection of placebo at home and anistreplase in hospital at median times of 110 and 240 minutes after the onset of symptoms. Definite or probable myocardial infarction was later confirmed in 93 (68%) patients. Of these, 66 (71%) had the same findings on both electrocardiograms of either ST elevation, bundle branch block, or a non-specific abnormality, while 27 (29%) showed a major change of classification between home and hospital recordings; 21 (23%) had ST elevation or bundle branch block on only one of the paired recordings. Although ST elevation was the commonest abnormality in the 93 patients with myocardial infarction, in only 51 was it recorded at home (sensitivity 55%) and in 49 on admission (sensitivity 53%). Of 57 patients with ST elevation at home, six did not have infarction (specificity 86%), while of 51 with ST elevation on admission, two did not have infarction (specificity 95%).

Conclusions-Elevation of the ST segment is a

transient electrocardiographic abnormality that has high specificity but low sensitivity for predicting the diagnosis of acute myocardial infarction; it is an unsatisfactory precondition for giving thrombolytic treatment to patients with suspected acute myocardial infarction.

Introduction

It is now generally accepted that patients with acute myocardial infarction should receive thrombolytic treatment, provided that they present sufficiently early and there are no contraindications. Several clinical trials¹⁻³ support the experimental evidence⁴⁵ that such treatment should be given as soon as possible after the onset of infarction. As thrombolytic treatment has the potential to cause serious adverse events, cerebral haemorrhage being the most feared, it is considered important to establish the diagnosis of myocardial infarction rapidly so that only those patients who might benefit are exposed to the risks of treatment.

The use of an electrocardiograph remains the quickest, simplest, and most reliable method of diagnosing myocardial infarction.⁶ There are, however, varying estimates of the specificity of electrocardiographic abnormalities in patients with chest pain,⁷⁻¹⁰ and little work has been published on the very earliest changes and how these evolve in acute myocardial infarction.

The early anistreplase trial in the Grampian region³ was a randomised, double blind, parallel group trial of anistreplase, given either at home by general practitioners or later in hospital. The purpose of the trial was to assess the feasibility, safety, and efficacy of domiciliary thrombolysis by general practitioners. Provided that there were no contraindications to thrombolytic treatment, patients were entered into the trial if there was strong clinical suspicion of acute myocardial infarction when they were seen by their general practitioners within four hours after onset of symp-

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BMJ 1993;307:409-13

⁵ Barker DJP, Godfrey KM, Bull AR, Osmond C. The relation of fetal length, ponderal index and head circumference to blood pressure and the risk of hypertension in adult life. *Paediatr Perinat Epidemiol* 1992;6:35-44.

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toms. General practitioners were required to record an electrocardiogram but not to interpret it. These electrocardiograms and those subsequently recorded on admission to hospital in patients who received placebo at home and anistreplase in hospital are the subject of this report.

Our objectives were (a) to establish the incidence of the earliest electrocardiographic abnormalities in patients with acute myocardial infarction to see how they change spontaneously before thrombolytic treatment is given and (b) to determine their sensitivity and specificity for predicting the diagnosis of myocardial infarction.

Methods

Patients—A total of 311 patients entered the trial, from whom paired electrocardiograms were available for coding in 280 cases. Of these, 137 patients received placebo at home and are considered here. Their mean age was 63 years; 98 (72%) were men.

Classification of electrocardiograms—Two observers (JA and RT) coded the paired electrocardiograms for each patient entered into the trial, according to the scheme shown in table I. If more than one feature was

TABLE 1—Coding scheme for electrocardiographic abnormalities and numbers (percentages) of patients with various abnormalities recorded at home and on admission to hospital*

Electrocardiographic abnormality	Code	Home	Hospital
Bundle branch block	1	6 (4)	9 (7)
ST elevation†:			
Anterior alone (anterior, lateral, or			
anterolateral)	2	19 (14)	18 (13)
Inferior alone	3	35 (26)	28 (20)
Anterior and inferior	4	3 (2)	5 (4)
Other ST elevation (not covered by codes		.,	• • •
2-4)	5	11 (8)	11 (8)
ST depression without ST elevation	6	18 (13)	18 (13)
Q waves	7	$1(1)^{'}$	4 (3)
T wave inversion	8	10 (7)	11 (8)
Normal	9	17 (12)	19 (14)
Other abnormality	10	17 (12)	14 (10)
Total		137 (100)	137 (100

*After fourth international study of infarct survival (ISIS 4). $\pm T$ elevation ≥ 1 mm in two limb leads or ≥ 2 mm in two precordial leads.

present then the lowest appropriate code number was allocated. The electrocardiograms were coded without reference to the other tracing for each patient. Each observer was blinded to the other's results and to the nature of the treatment which the patient received at home or in hospital. The two observers then compared their results and tried to resolve their differences. When agreement could not be reached a third person (JR) independently coded the disputed electrocardiogram and negotiated a consensus.

Discharge diagnosis-At the time of discharge from hospital each patient was assigned to one of seven diagnostic categories by using data collected in hospital.3 Definite myocardial infarction was defined as a convincing history plus new pathological Q waves on the electrocardiogram and a peak myocardial fraction of creatinine kinase exceeding the upper limit of normal. Probable myocardial infarction was defined as a convincing history plus either new pathological Q waves or a raised myocardial fraction of creatinine kinase. For this study, patients were considered to have had a confirmed infarct if they were assigned to either of these categories. Patients in other diagnostic categories of possible myocardial infarction, ischaemic heart disease, chest pain of unknown cause, or alternative diagnosis were designated non-infarct.

Receiver operator characteristic curves are a way of representing the overall performance of a diagnostic test in terms of sensitivity and specificity as the ratio of true positive to false positive results. They may be used for a continuous variable, such as a cardiac enzyme assay, or, as in this paper, with a discontinuous variable, the electrocardiogram. To construct the curves codes 5, 7, and 8, representing electrocardiograms with minor ST elevation, Q waves, or T inversion, were aggregated. The eight resulting categories of electrocardiographic abnormalities were then ranked in order of specificity for prediction of the diagnosis of myocardial infarction. Eight points on the receiver operator characteristic curve were plotted as the cumulative percentage of true positives (ordinate, sensitivity) and the cumulative percentage of false positives (abscissa, 100-specificity) for each of the eight categories and for electrocardiograms recorded at home and in hospital.

Results

Electrocardiograms were recorded immediately before injection of placebo at home and anistreplase in hospital at median times of 110 and 240 minutes after the onset of symptoms, respectively. Of the 137 electrocardiograms recorded at home, 135 (99%) were performed within four hours, and 127 (93%) of the 137 electrocardiograms recorded in hospital were performed within six hours of onset of symptoms. The median interval between recordings at home and hospital was 125 minutes.

After the first attempt at coding the electrocardiograms the two observers disagreed in 74 (27%) cases. After discussion there were 36 (13%) traces on which the third observer had to arbitrate.

Table I gives the numbers of patients with various electrocardiographic abnormalities. In both home and hospital electrocardiograms the commonest abnormality was definite ST segment elevation. In the home electrocardiograms the next commonest findings were ST depression and a normal recording, but in the hospital recordings the order was reversed, with normal tracings and ST depression ranking second and third.

The diagnosis of myocardial infarction was confirmed in 93 (68%) cases; 60 were categorised as definite and 33 as probable infarction. There were 44 cases of non-infarction.

RECEIVER OPERATOR CHARACTERISTIC CURVES

Figure 1 shows the receiver operator characteristic curve for the electrocardiograms recorded at home. Elevation of the ST segment in both the anterior and inferior leads was the most specific indicator of infarction, followed by ST elevation in the inferior and then the anterior leads. Definite ST elevation in any location was present in 51 (55%) of those with confirmed infarction (ordinate) and six (14%) of those without infarction (abscissa). The specificity of this abnormality is 100-14=86%. The area under the curve is 81% of the area of the graph.

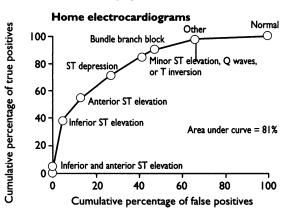


FIG 1—Receiver operator characteristic curve for electrocardiograms recorded at home

Figure 2 shows the receiver operator characteristic curve for the electrocardiograms recorded in hospital. The rank order of specificities is slightly different from that for electrocardiograms recorded at home. As in the electrocardiograms recorded at home, the most specific abnormalities were definite ST elevations but followed by bundle branch block rather than ST depression. For hospital electrocardiograms, patients with definite ST elevation in any location comprised 49 (53%) of those with infarction and two (5%) of those without infarction; specificity was 95%. The area under the curve is 86% of the area of the graph.

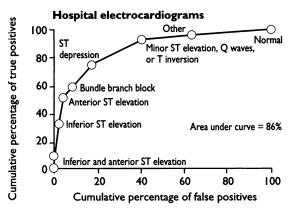


FIG 2—Receiver operator characteristic curve for electrocardiograms recorded on admission to hospital

Considering home electrocardiograms, 91 (98%) patients with infarction had some abnormality compared with 29 (66%) patients without infarction. Two (2%) patients with infarction and 15 (34%) without infarction had normal recordings at home.

CHANGES IN READINGS FROM HOME TO HOSPITAL

The electrocardiographic code differed between home and hospital recordings in 39 (42%) patients with infarction and 15 (34%) patients without infarction.

Electrocardiograms with 10 different codes were aggregated into three larger categories: codes 1, 2, 3, or 4 to "ST elevation and bundle branch block," codes 5, 6, 7, 8, or 10 to "non-specific," and code 9 to "normal." Table II shows how the results from electrocardiograms recorded in hospital had changed from those recorded at home in patients with and without confirmed myocardial infarction.

Sixty six (71%) of the 93 patients who had a confirmed myocardial infarction showed no change of category between the electrocardiogram recorded at home and in hospital; of these patients, 45 (48%) had ST segment elevation or bundle branch block on both traces.

Of the 93 with infarction, 27 (29%) showed a major change in category between home and hospital recordings. Of particular interest are 10 (11%) patients who had bundle branch block or ST segment elevation at home but only non-specific changes by the time of admission; at that time five had minimal ST segment elevation, two had developed Q waves, two had T wave inversion, and one had ST segment depression.

TABLE II—Number of patients with various electrocardiographic abnormalities recorded at home and on admission to hospital in patients with myocardial infarction (numbers of patients without infarction in parentheses)

Hospital electrocardiogram	Home electrocardiogram				
	ST elevation or bundle branch block	Non-specific changes	Normal	Combined	
ST elevation or bundle branch block	45 (3)	11(1)	0 (0)	56 (4)	
Non-specific changes	10 (4)	21 (19)	2 (2)	33 (25)	
Normal	0 (1)	4(1)	0 (13)	4 (15)	
Combined	55 (8)	36 (21)	2 (15)	93 (44)	

Cumulative percentage of true positives

tests

100

80 ·

60

40

20

0

20 40

FIG 3—Hypothetical receiver

non-discriminatory diagnostic

ideal and completely

operator characteristic curves for

Ideal curve

Useless curve

Cumulative percentage

of false positives

60 80 100

Progression from the non-specific category to ST elevation or bundle branch block occurred in 11 patients, the commonest sequence being additional elevation of minimally raised ST segments, which was found in six patients. Two patients with infarction progressed from a normal electrocardiogram when first seen to non-specific abnormalities in hospital, while four patients with infarction showed regression of nonspecific abnormalities to a normal electrocardiogram.

Discussion

A previous paper from this department dealt with the earliest electrocardiographic evidence of myocardial infarction. Short reported that of 150 patients with myocardial infarction in only "27 (18%) did the tracing show a pattern regarded as diagnostic of acute infarction—that is, localised ST elevation of 1 mm or more."¹¹ Ninety eight of these electrocardiograms, however, were recorded more than 24 hours after the onset of chest pain, and the symptoms were slight and subacute.

The advent of thrombolytic treatment has accentuated the need for rapid diagnosis of acute myocardial infarction when the patient first comes under medical care. Analysis of an electrocardiogram remains the best way to do this, but there is a dearth of information about the accuracy of predictions based on recordings made in the community when patients with suspected acute myocardial infarction are first seen.

RECEIVER OPERATOR CHARACTERISTIC CURVES

The receiver operator characteristic curve is a way of representing the overall performance of a diagnostic test in terms of sensitivity and specificity as the ratio of true positive to false positive results. A completely non-discriminatory test would result in a diagonal line from bottom left to top right of the graph, all points on the line representing a ratio of true to false positives of 1:1, the area beneath the line would be half the total area. An ideal diagnostic test would inscribe a rectangular line passing from the origin to the top right hand point by way of the top left hand corner; the area beneath such a curve would be nearly 100% of the total area of the graph (fig 3). The receiver operator characteristic curve may be used to identify a threshold value for a diagnostic test that gives the optimal balance between true and false positives.

The curves plotted from the results in this study show that neither home nor hospital electrocardiograms are ideal for predicting infarction, but hospital electrocardiograms have a slightly better performance and larger area under the curve than those of electrocardiograms recorded at home (86% v 81%); these curves are similar to previous published results.¹²¹³ The only abnormality that was completely specific for acute myocardial infarction was ST elevation in inferior and anterior leads, but that was present in only 3% and 5% of the electrocardiograms recorded at home and hospital, respectively, in patients with infarct. Less specific abnormalities are seen in a greater proportion of infarct cases, but these same abnormalities are also to be found in some non-infarct cases.

Thrombolytic treatment is commonly recommended if the electrocardiogram shows distinct ST segment elevation or bundle branch block.¹⁴⁻¹⁶ Based on the electrocardiograms recorded in hospital this recommendation would have included 56 (60%) of 93 patients with subsequently confirmed infarction and four (9%) of 44 patients without infarction; four (7%) of 60 patients to whom thrombolytic treatment would have been given would not have had myocardial infarction confirmed. By using electrocardiograms recorded at home, the corresponding figures are 55 (59%) and eight (18%), so that eight (13%) of 63 patients given thrombolysis would not have had infarcts.

BENEFIT:RISK ANALYSIS

The receiver operator characteristic curves (figs 1 and 2) illustrate clearly the dilemma faced when thrombolytic treatment is considered—if the presence of the electrocardiographic abnormalities that are most specific for myocardial infarction is the prerequisite for treatment, then only a small proportion of patients with infarction will benefit. On the other hand, if the presence of non-specific abnormalities possessed by a high proportion of infarct patients is considered a sufficient precondition for treatment then an increasing proportion of patients who do not have infarction will receive it and will derive no benefit but will be at risk of adverse events.

The threshold on the receiver operator characteristic curve that is used to determine the administration of thrombolytic treatment should therefore be based on a consideration of the benefit:risk ratio at various points on the curve; benefit accruing to those with infarction while the main risk is carried by those without infarction.13 It should be noted that the thresholds for thrombolytic treatment based on electrocardiograms recorded at home and hospital will be different because treatment administered at home at an earlier time than is possible in hospital has greater efficacy in cases of infarction' but no greater risk in cases of noninfarction. For the same benefit:risk ratio that applies in hospital, thrombolytic treatment may be given at home for less specific electrocardiographic abnormalities than are required in hospital.

RELIABILITY OF ELECTROCARDIOGRAPHIC INTERPRETATION

Although experienced at reading electrocardiograms, two observers in this study disagreed at the first run of coding in 74 (27%) of the 274 cases. Difficulty in categorising a single electrocardiogram without reference to preceding or subsequent recordings was experienced by all three observers.

TRANSIENCE OF ELECTROCARDIOGRAPHIC ABNORMALITIES

In the early stages of acute myocardial infarction the electrocardiogram may change rapidly: ST elevation may fluctuate or subside permanently,¹⁷⁻¹⁹ ST depression may occur,^{20 21} or T waves invert.²² In addition, Q waves may appear as early as the second hour after the onset of chest pain.²³

In this series the electrocardiographic codes of 39 (42%) of 93 patients with evolving infarction changed between recordings at home and hospital, and this resulted in a major change of category in 27 (29%)

Clinical implications

• In acute myocardial infarction thrombolytic therapy is commonly withheld from patients without ST elevation on the presenting electro-cardiogram

- In this study ST elevation was found in only about half of patients with infarction
- In the early stages of infarction ST elevation may be transient and difficult to measure precisely

• Rigidly defined ST elevation is an unsatisfactory precondition for thrombolytic therapy

• Particularly in patients presenting very early, thrombolytic therapy should be considered in all with an abnormal electrocardiogram cases. In 21 (23%) cases ST elevation or bundle branch block was present in only one of the paired recordings, non-specific abnormalities being found in the other. In these patients with evolving infarction it would seem regrettable to forego the opportunity to use thrombolytic treatment soon after the onset of symptoms—at a time when it is most efficacious—merely because the qualifying abnormalities have not yet appeared. It would seem equally mistaken to deny patients thrombolytic treatment in hospital because the qualifying abnormalities previously present are no longer there because of rapid development of the electrocardiographic abnormalities.

The presence of rigidly defined electrocardiographic abnormalities that are transient and difficult to recognise is an unsatisfactory basis for determining whether or not thrombolytic treatment should be given.

CONCLUSION

The currently recommended electrocardiographic preconditions for the administration of thrombolytic treatment are arbitrary, difficult to recognise, and fairly insensitive so that a substantial proportion of patients with acute myocardial infarction do not receive treatment from which they might benefit. The use of less specific electrocardiographic preconditions, however, would result in administration of thrombolytic treatment to a greater proportion of patients without infarction.

It has been argued elsewhere¹³ that acceptable benefit:risk ratios would be obtained if thrombolytic treatment were to be given in hospital to all patients with a convincing clinical history of acute myocardial infarction, none of the standard contraindications and any abnormality in an electrocardiogram. As thrombolytic treatment has enhanced efficacy when given at an earlier time, these recommendations for the administration of treatment would have even greater force in the community.

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 Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986;i:397-401.

2 Second International Study of Infarct Survival Collaborative Group. Random-

-----**-**-----

ised trial of intravenous streptokinase, or aspirin, both or neither among 17187 cases of suspected acute myocardial infarction: ISIS-2. Lancet 1988;ii:349-60.

3 GREAT Group. Feasibility, safety, and efficacy of domicillary thrombolysis by general practitioners: Grampian region early anistreplase trial. BMJ 1992;305:548-53.

4 Baughman KL, Maroko PR, Vatner SF. Effects of coronary artery reperfusion on myocardial infarct size and survival in conscious dogs. *Circulation* 1981;63:317-23.

5 Bergmann SR, Lerch RA, Fox KAA, Ludbrook PA, Welch MJ, Ter-Pogossian M, et al. Temporal dependence of beneficial effects of coronary thrombolysis characterised by positron tomography. Am J Med 1982; 73:573-81.

6 Timmis A. Early diagnosis of acute myocardial infarction. BMJ 1990;301: 941-2.

7 Lee TH, Rouan GW, Weisberg MC, Brand DA, Cook F, Acampora MPH, et al. Sensitivity of routine clinical criteria for diagnosing myocardial infarction within 24 hours of hospitalization. Ann Intern Med 1987;106: 181-6.

8 Brush JE, Brand DA, Acampora D, Chalmer B, Wackers FJ. Use of the initial electrocardiogram to predict in-hospital complications of acute myocardial infarction. N Engl J Med 1985;312:1137-41.

9 Yusef S, Pearson M, Sterry H, Parish S, Ramsdale D, Rossi P, et al. The entry electrocardiogram in the early diagnosis and prognostic stratification of patients with suspectd acute myocardial infarction. Eur Heart J 1984; 5: 690-6.

 Rude RE, Poole WK, Muller JE, Turi Z, Rutherford J, Parker P, et al. Electrocardiographic and clinical criteria for recognition of acute myocardial infarction based on analysis of 3 697 patients. Am J Cardiol 1983;52:936-42.
 Short D. The earliest electrocardiographic evidence of myocardial infarction. Br Heart 7 1970:32:6-15.

12 Rouan GW, Lee TH, Cook EF, Brand DA, Weisberg MC, Goldman L. Clinical characteristics and outcome of acute myocardial infarction in patients with initially normal or nonspecific electrocardiograms (a report from the multicenter chest pain study). Am J Cardiol 1989;64:1087-92. 13 Rawles JM. Risk benefit analysis of thrombolytic therapy for acute myocardial infarction: a perspective. *Coronary Artery Disease* 1992;3:1153-61.

14 Wilcox RG. Choice of agent: optimal efficacy vs side-effects. In: de Bono D, ed. Thrombolysis: current issues and future directions. Oxford: The Medicine Group (Education) Ltd, 1992:9-15. (MEDICINE Publishing Foundation Series, 31.)

15 Chamberlain DA. Relationship of trials to the general population: who should get thrombolysis and when? In: de Bono D, ed. *Thrombolysis: current issues* and future directions. Oxford: The Medicine Group (Education) Ltd, 1992:17-20. (MEDICINE Publishing Foundation Series, 31.)

16 Schweitzer P. The electrocardiographic diagnosis of myocardial infarction in the thrombolytic era. Am Heart § 1990;119:642-54.

17 Essen R, Merx W, Effert S. Spontaneous course of ST segment elevation in anterior myocardial infarction. Circulation 1979;59:105-12.
18 Zmyslinski R, Akiyana T, Biddle TL, Shah PM. Natural course of ST segment

and QRS complex in patients with acute anterior myocardial infarction. Am 7 Cardiol 1979:43:29-34.

19 Reid D, Pelides L, Shillingford J. Surface mapping of RST segment in acute myocardial infarction. Br Heart \$ 1971:33:370-4.

myocardial infarction. Br Heart § 1971;33:370-4. 20 Quyyumi A, Crake T, Rubens MB, Levy RD, Rickards AF, Fox KM. Importance of reciprocal electrocardiographic changes during occlusion of left anterior descending coronary artery. Lancet 1986;1:347-50. 21 Gibson RS, Crampton RS, Watson DD, Taylor GJ, Carabello BA, Holt ND,

21 Gibson RS, Crampton RS, Watson DD, Taylor GJ, Carabello BA, Holt ND, et al. Precordial ST-segment depression during acute myocardial infarction; clinical scintigraphic and angiographic correlations. *Circulation* 1982;66: 732-41.

22 Klainman E, Sclarovsky S, Lewin RF, Topaz O, Farbstein H, Pinchas A, et al. Natural course of electrocardiographic components and stages in the first twelve hours of acute myocardial infarction. *J Electrocardiol* 1987;20: 98-109.

23 Selwyn A, Fox K, Welman E, Shillingford J. Natural history and evaluation of Q waves during acute myocardial infarction. Br Heart J 1978;40:383-7.

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Minor surgery by general practitioners under the 1990 contract: effects on hospital workload

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Abstract

Objective—To determine the extent to which minor surgery undertaken by general practitioners after the introduction of the 1990 contract substituted for hospital outpatient workload.

Design—Before and after observational study.

Setting—Four English family health services authorities.

Subjects—Patients in 22 practice populations who were operated on by their general practitioner or referred to hospital for minor surgery during April to June 1990 or April to June 1991.

Main outcome measures—Numbers of minor surgical procedures undertaken in general practice and in hospital, numbers of referrals to hospitals for conditions treatable by a minor surgical procedure, and the mix of diagnoses and procedures undertaken in each setting.

Results—General practitioners claimed reimbursement for 600 minor surgical procedures during April to June 1990 and for 847 during April to June 1991, an increase of 41%. Referrals to hospital for comparable conditions showed no compensatory decrease (385 during April to June 1990 and 388 during April to June 1991, 95% confidence interval for change in referrals -51 to 57), and the number of hospital procedures resulting from those referrals also remained constant (187 in the first period, 189 in the second, 95% confidence interval for change in procedures -36 to 40). The mix of procedures did not change significantly from one study period to the next in either setting.

Conclusions—Many or all of the additional patients receiving minor surgery under the terms of the 1990 contract may not have previously been referred to hospital. General practitioners seem not to have systematically shifted towards treating the more trivial cases. The overall increase in minor surgical activity may reflect an improvement in accessibility of care or changes in patients' perceptions and attitudes.

Introduction

Waiting times are shorter and costs to the NHS lower when minor surgery is performed in general practice rather than in hospital.¹⁴ Quality of care, insofar as it has been measured, is broadly comparable in the two settings, and patients' satisfaction with minor surgery performed by general practitioners is universally high.

After many calls for general practitioners to be reimbursed for minor operations,15-7 the 1987 white paper on primary care recommended such payments on the grounds that "Patients would benefit from a rapid and more convenient service, and minor surgery cases would not take up time in out-patient departments which might be needed for more serious problems."8 Item of service payments for general practitioners performing minor surgery were introduced in the 1990 contract to encourage a shift from hospital to general practice.9 Since April 1990 general practitioners listed by family health services authorities as willing to perform minor surgery have been entitled to a fee of \pounds 20 per procedure for a specified list of minor operations ranging from cauterisation of warts to excision of small lesions.

Two reports have estimated the savings when minor surgery is performed in general practice by extrapolating from calculations of costs in the two settings.¹² Others have pointed out that resources are saved only when a patient who would have been treated in hospital is treated instead in general practice.³⁴ Whether minor surgery by general practitioners substitutes for minor surgery in hospital or whether it offers a complementary service to patients who would otherwise not have been treated at all has, so far, been explored only superficially. One small study in 1990 observed a

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