PAPERS AND ORIGINALS

Prognosis of new and worsening angina pectoris

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Summary

The natural history of new and worsening angina pectoris was studied in 251 men aged under 70 years. Most were ambulant and all were referred by selected general practitioners to a special hospital clinic over two and a half years. Heart attacks developed in 39 patients, nine of whom died. Seventy-two per cent of the attacks occurred within six weeks of the onset or worsening of angina. Of the 212 patients who did not suffer myocardial infarction and who were clinically reviewed six months after their first attendance 66 had been pain free for the previous three months and 14 had experienced only infrequent attacks of angina. Of the 128 men aged under 65 years who were previously in employment 81% had returned to full-time work six months after their first attendance.

A discriminant function analysis using many variables was made to develop a predictive index that would allow patients with new or worsening angina who were likely to develop serious cardiac complications to be identified.

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This did not prove possible, and the only predictive factor of significance was an increased cardiothoracic ratio.

The syndrome of new and worsening angina has a low risk of early death, and many patients are symptom free six months later. In general, emergency coronary arteriography and surgery is not indicated.

Introduction

A prospective study of new and worsening angina based on patients seen by general practitioners was carried out in Edinburgh. A preliminary report¹ indicated that 14% of 167 ambulant patients with new or worsening angina developed myocardial infarction within six months of its onset, and a further 2% died suddenly within the same period. The present report, based on the completed study, amplifies these findings, examines the predictive value of several clinical and laboratory observations, and outlines the long-term prognosis.

Design of study

Selected general practitioners were asked to refer to the Royal Infirmary, Edinburgh, all men aged under 70 who had experienced, during the previous four weeks, chest pain suggestive of myocardial ischaemia that had either occurred for the first time (new cases), recurred after an interval of freedom at of least one month (recurrences), or abruptly and inexplicably increased in frequency or severity (exacerbations) in the absence of objective evidence of definite recent myocardial infarction.

Patients were referred by 71 general practitioners practising within easy access of the hospital, and all those who were approached agreed to co-operate. A population of about 28 400 men aged 35 to 69 was served by these doctors and included a wide range of social groups. Full details of the definitions and methods used in the study have been published.¹ The study began in June 1970 and the intake of patients continued for two and a half years.

Methods

A special clinic was established in the cardiology department of the Royal Infirmary, and patients were seen daily (except Sundays). Almost all attended within 24 hours of referral.

At the first visit a cardiologist took a full history and carried out a complete clinical examination. Blood pressure was recorded in the recumbent position. A 6 ft (2 m) x-ray picture of the heart was made to record the cardiothoracic ratio. Laboratory tests included measurements of haemoglobin, packed cell volume, and casual blood glucose, serum cholesterol, serum aspartate aminotransferase (SGOT), and serum hydroxybutyrate dehydrogenase (SHBD) concentrations. Serum cholesterol and triglycerides were measured and a modified glucose tolerance test was carried out after an overnight fast, usually at the second visit. The SGOT and SHBD were measured again subsequently if there was any suggestion that myocardial infarction had occurred. Other investigations (such as barium meal studies) were carried out when clinically indicated.

Electrocardiogram—A 12-lead electrocardiogram (ECG) and a three-minute record of lead 2 was made at each visit. Patients seen during the last year of the study also had their cardiac rhythm recorded on tape for 30 minutes at the first visit. Exercise tolerance tests, carried out with a bicycle ergometer in accordance with World Health Organisation recommendations,² were done when the diagnosis was in doubt. ECGs and three-minute monitoring strips were coded according to the Minnesota code³ by two independent observers—one doctor and one technician. Disagreements were discussed and referred to a third person if they could not be resolved. The same routine was used for reading the tape recordings.

Assessment of angina—Angina was finally diagnosed clinically by an experienced cardiologist who took into account the results of all the investigations. In addition, a score based on the system developed by the staff of the Coronary Heart Study of the Health Insurance Plan (HIP) of greater New York was used as a diagnostic aid.⁴ There were three categories: definite angina (score over 10); possible angina (score 1-10); no angina (negative score). Coronary angiography was not undertaken during the study.

Patients

A total of 616 patients were referred to the clinic; 251 fulfilled the criteria for new or worsening angina and were accepted into the study. Among these were 21 (8.4%) patients who formed a special subgroup with "possible myocardial infarction." They either reported recent prolonged pain (30 minutes or more) or showed equivocal ECG changes at their first visit.

The remaining 365 patients did not satisfy the criteria for new or worsening angina. Twenty-six were excluded because of recent definite myocardial infarction, and 31 had chronic angina with no exacerbation within the previous four weeks. The final diagnoses of the remaining patients were: musculoskeletal pain (125), gastrointestinal disease (59), chest infection (14), pericarditis (3), herpes zoster (1), and others (106). Their progress was checked by writing to

TABLE I—Numbers of serious complications developing among 251 patients with new and worsening angina

	Sudden death	Circulatory arrest and survival	Definite myocardial infarction	Probable myocardial infarction	Total
No (%) of cases	6 (2.4)	1 (0.4)	26 (10.4)	6 (2.4)	39 (15.5)

TABLE II—Proportions of patients with serious complications according to category of new and worsening angina

Category:	New	Exacerbation	Recurrence	Total
Total No of patients	129	103	19	251
No (%) developing serious complications	18 (14.0)	17 (16.5)	4 (21.1)	39 (15.5)

TABLE III—Interval from onset of new or worsening angina to development of serious complications

Weeks from onset:	<1	1-2	<3	<4	<5	<6	<7	<8	<9	<10	<11	<12	>12	Total
No with definite and probable myocardial infarction	2	8	3	5	1	2	0	2	1	1	1	1	5	32
No of sudden deaths (including one cardiac arrest)	0	2	2	0	2	1	0	0	0	0	0	0	0	7
Total	2	10	5	5	3	3	0	2	1	1	1	1	5	39

their general practitioner six months after their first attendance. Our diagnosis was probably wrong in two patients who subsequently developed myocardial infarction, one within 24 hours of being seen (he had given an atypical history suggestive of gastrointestinal pain) and one after five months (his presenting symptoms were attributed to a hiatus hernia). A third patient who had chronic angina without an exacerbation had a myocardial infarct two weeks after his referral to the clinic.

Management—Patients who fulfilled the criteria for acceptance were seen one, two, four, six, and 12 weeks and six months after the first visit. Glyceryl trinitrate was the only drug used routinely in the first three months. After this beta-adrenergic blocking drugs were prescribed when necessary. Anticoagulants were not given. Patients were advised to avoid strenuous physical activity but most were allowed to remain at work provided it was not unduly heavy or associated with pain. General advice about stopping smoking and weight loss was given when indicated. Patients with recent chest pain lasting 30 minutes or longer, a crescendo anginal pattern, an ECG pattern of acute ischaemia at rest (new appearance of Minnesota codes 4-1, 4-2 : 5-1 or 7-1), or acute myocardial infarction were admitted to hospital.

Results

Of the 251 patients accepted for study 129 had angina for the first time, 103 had exacerbations of chronic angina, and 19 had recurrences. Forty-nine (19.5%) of these patients—10% of those with new angina and 30% of the 122 with exacerbations or recurrences—had a history of myocardial infarction.

MORBIDITY AND MORTALITY

Serious cardiac complications were classified as follows: (a) sudden death (death within an hour of the onset of symptoms or within an hour of being seen alive); (b) definite myocardial infarction and (c) probable myocardial infarction (defined in terms of symptoms, enzyme levels, and ECG changes*); and (d) circulatory arrest with survival.

During the study 39 (15.5%) patients developed serious cardiac complications (table I); the complication rates did not differ significantly between the three angina groups (table II). Twenty-eight (72%) of the 39 patients suffered complications within six weeks and 34 (87%) suffered them within 12 weeks of the onset of symptoms (table III). There were nine (3.6%) deaths in the six-month follow-up period; six were sudden and three were associated with definite myocardial infarction.

Eighty-seven patients were admitted to hospital because of prolonged chest pain or progressive worsening of angina. Eight had recurrent episodes of pain after admission, and four of these developed myocardial infarction. Of the remaining 79 patients six developed myocardial infarction, which made a total of 10 (11.5%) patients who had myocardial infarction during their admission. A further six (6.9%) patients had serious cardiac complications after discharge—two within seven days and the remainder two to 18 weeks later. These rates were not significantly different from those of patients who were not admitted to hospital: 14% (23) of the 164 not admitted developed serious complications. Two (9.5%) patients among those with possible myocardial infarction subsequently developed definite myocardial infarction.

ECG FINDINGS AND HIP SCORE

There were 171 patients with S-T depression or T-wave changes on the ECG, and only 28 with a completely normal record. The ECG findings were graded according to severity: (a) serious abnormality, (b) less serious but unequivocal abnormality, (c) equivocal abnormality,

*Full details available on request.

(d) minor abnormality, and (e) normal ECG. Full details are given in the Appendix.

The distribution of these grades was similar among patients with a HIP score over 10 and among those with HIP scores of 10 or less: 66% of the former and 59% of the latter group had serious or unequivocal ECG abnormalities. The subsequent development of a serious cardiac complication was not related to the presence of serious or unequivocal ECG abnormalities: such abnormalities were present in 64% of patients who did not develop a serious complication compared with 51% of those who did.

The 251 patients were grouped in seven mutually exclusive categories according to history, ECG findings, and HIP score. Group 1 were those with ischaemic heart disease while group 7 had normal ECGs, and there were decreasing probabilities in between (table IV). Altogether 86% of patients had either a history of myocardial infarction or definite ECG abnormalities or a HIP score over 10. In the remaining 14% the diagnosis was less certain but the incidence of serious cardiac complications in this group was almost the same as that in the definite group.

In 52 patients the diagnosis was in doubt and they had an exercise tolerance test during their clinic attendance: 14 developed unequivocal ischaemic changes,* a further 17 developed equivocal changes,* six developed an arrhythmia without other changes, and 15 were normal. These 15 patients remained in the study because an experienced cardiologist thought that their histories were compatible with angina. Seven of them had a HIP score over 10 and a further patient subsequently developed significant T-wave inversion on a resting ECG.

Ventricular premature beats (VPBs)—The 21 patients who presented initially with a recent possible myocardial infarction were excluded from this analysis. Examination of the resting ECGs of the remaining 230 patients showed that 42 had single VPBs and a further 12 had complicated VPBs (runs of two or multifocal complexes). Seven serious cardiac complications occurred among these 54 patients—three sudden deaths and four myocardial infarctions—but this was not significantly more than expected. VPBs were significantly more common ($P < 0.005$) in patients with a history of myocardial infarction than in those with no previous infarction. An analysis of the three-minute rhythm strips both separately and in conjunction with the resting ECG showed no significant association between the presence of VPBs and the subsequent development of a serious cardiac complication. Thirty-minute monitoring of heart rhythm was recorded on tape from 81 patients seen during the last year of the study. They included eight patients who subsequently developed myocardial infarction and four who died suddenly. Again, there was no significant association between the presence of VPBs and the subsequent development of a serious cardiac complication.

FREQUENCY OF ANGINA

Information on death and the development of myocardial infarction was available for all 251 patients at six months. In assessing the

TABLE IV—Grouping of patients according to history, ECG findings, and HIP score

Group	Definition	No of patients	Cumulative % of patients	No with serious complications
1	History of myocardial infarction	49	20	6
2	Serious ECG abnormality	36	34	4
3	Unequivocal ECG abnormality	75	64	9
4	HIP Score > 10	56	86	15
5	Equivocal ECG abnormality	25	96	3
6	Minor ECG abnormality	7	99	1
7	Normal ECG	3	100	1
	Total	251		39 (15.5%)

*On the Minnesota code, unequivocal changes: 11-1, 11-2, 12-1, 12-2; and equivocal changes: 11-3, 11-4, 12-3, 12-4.

TABLE V—Outcome in 251 patients at 6 months and prevalence of angina in 212 patients who survived without myocardial infarction at 6 months

No (%) of patients	Died	Developed myocardial infarction but survived	Remaining patients (n = 212)				
			No angina	Infrequent angina	Angina		Incomplete information
					On beta-blockers	Not on beta-blockers	
	9 (4)	30 (12)	66 (31)	14 (7)	25 (12)	75 (35)	32 (15)

TABLE VI—Prevalence of angina six months after first visit in 180 patients with complete information

Frequency of attacks:	$> 1/\text{day}$	$> 1/\text{week}$	$> 1/\text{month}$	$< 1/\text{month}$	No pain	Total
No not on beta-blockers	28	28	19	14	66	155
No on beta-blockers	11	10	1	1	2	25
Total	39	38	20	15	68	180

prevalence of angina the 39 patients who had experienced serious cardiac complications were excluded. Of the remaining 212 patients 66 had been pain free for the previous three months; 14 had experienced only infrequent attacks of angina, and 75 had angina of effort (table V). A further 25 had angina of sufficient severity to warrant treatment with beta-adrenergic blocking drugs.

Twenty-five of the patients for whom information on angina was incomplete failed to attend at six months, mainly because of intercurrent illness or unwillingness to continue attending hospital. A further seven were unable to give an accurate account of their pain frequency. Details of the frequency of pain at six months in the 180 patients with complete information are shown in table VI.

No significant differences were found between the characteristics of patients who continued to experience pain throughout the six months' follow-up and the characteristics of those in whom symptoms resolved. The two groups comprised, respectively, 79 patients who had experienced pain within a week of their final clinic visit and 75 patients who had been free of pain for at least eight weeks before their final visit. The frequency of pain recorded at the first visit bore no significant relation to the subsequent development of a serious cardiac complication.

EMPLOYMENT STATUS SIX MONTHS LATER

The employment status of 128 men (aged under 65 years) who had been working full-time was assessed at six months; 104 (81%) had returned to full-time work. Of those not receiving beta-adrenergic blocking drugs (109 patients) 85.3% were working full time at six months, 2.7% were working part-time, 8.3% were on sickness absence, and 3.7% were unemployed. The record of those taking beta-adrenergic blocking drugs (19 patients) was not so good, with 57.9% in full-time work and 26.3% on sickness absence.

PREDICTION OF OUTCOME: DISCRIMINANT FUNCTION ANALYSIS

Since 16 of the 39 serious cardiac complications occurred between the first and second clinic visits only the information obtained at the first visit was used in this analysis. Initially many individual characteristics* were examined to determine those which might have an association with the outcome. Only the cardiothoracic ratio showed a significant relationship (it was on average higher among those with serious cardiac complications), and this was still true when patients with a history of myocardial infarction were excluded from the analysis.

Discriminant function analysis was then applied using the University of California statistical package (programme BMD 07M).⁵ After preliminary trials using factors known to be implicated in the development of ischaemic heart disease the following variables were retained in all subsequent analyses: (a) history of myocardial infarction; (b) cigarette smoking habits; (c) ECG Minnesota codes 4 (S-T segment depression) or 5 (T-wave items); (d) cardiothoracic ratio; and (e) age. Other variables included in the analysis were an exacerbating pattern of pain, ECG Minnesota codes 3 (high amplitude R waves) and 8 (arrhythmias), systolic blood pressure, heart rate, weight gain over the previous year, height-weight index (W/H^2), packed cell volume, and

*Full list available on request.

serum cholesterol concentration. To satisfy the requirements of the statistical method, the qualitative variables, such as a history of myocardial infarction, were coded and given numerical scores. Actual values were used in the remainder. Some new variables were derived in the hope that they might prove more important as "predictors" than the original variables. These included the square of the patient's smoking score, and "interaction" variables given by age $\times$ myocardial infarction score, age $\times$ smoking score, and age $\times$ cardiothoracic ratio.

Discriminant analysis derives a score for each patient from the linear combination of variables: a score falling within certain ranges classifies or predicts a patient as belonging to a particular group. The effectiveness of the discriminant analysis is then measured by the percentage of cases in which the statistically predicted result coincides with the clinically observed result.

The discriminant function scores were derived from 197 cases and then used to predict the outcome of these cases. In only 38.6% did the statistically predicted result coincide with the clinically observed result. When the scores were applied to an additional group of 30 cases the agreement was of the same order, at 30%. No improvement in prediction was obtained by using different combinations of variables.

Discussion

Our main new findings are (a) that angina, even when severe and of recent onset, is transient in many patients (table V), and (b) that it has not proved possible to develop a predictive index that would allow prior identification of patients likely to develop a heart attack. These findings also confirm that new or worsening angina has a low risk of serious cardiac complications, with 3.6% dying and 12% developing non-fatal myocardial infarction.

We have provided information on the prevalence of angina six months after patients were first examined. Of the 212 patients who did not develop a heart attack 38% had either no pain or less than one episode of angina per month (table V). This low frequency of angina resulted from neither treatment with beta-adrenergic blocking drugs, since only 12% were on these drugs, nor major changes in activity or responsibility at work, since most of these patients had returned to full-time work at the time of the six months' assessment. These findings emphasise that angina is not uncommonly transient. The results of a survey using the London School of Hygiene questionnaire on chest pain were similar.⁶ Fewer than half the men classed at the first examination as having angina still experienced pain a year later. Gazes *et al*⁷ reported that 74% of their patients had less angina after one year, and half had resumed normal activity, while the Framingham study⁸ noted the spontaneous disappearance of angina in 30% of patients.

Discriminant function analysis indicated that the routine measurements recorded in patients with new or worsening angina do not afford a means of determining those who are likely to develop complications. The results of discriminant analysis were disappointing, and agreement between the predicted and observed results was poor. The number of patients, especially those developing serious cardiac complications, was small, and possibly certain untried combinations of variables might improve the predictive value of the discriminant function. But the results obtained were sufficiently discouraging to make this unlikely. The only feature that showed a significant positive association with the development of serious cardiac complications was an increased cardiothoracic ratio. The finding of the Coronary Drug Project that heart size on chest x-ray film is an independent prognostic factor in survivors of myocardial infarction⁹ provides some support for this apparent association. Further evidence from Alabama indicates that cardiac enlargement based on heart volume calculated from posteroanterior and lateral chest x-ray films is also significantly related to mortality in patients presenting with chest pain.¹⁰

It is difficult to compare these findings with those from other studies since many terms have been used to describe the syndrome that falls between angina and myocardial infarction: changing patterns of angina pectoris,¹¹ impending myocardial infarction,¹²⁻¹⁴ acute coronary insufficiency,^{15 16} intermediate

coronary syndrome,^{17 18} unstable angina pectoris,¹⁹⁻²¹ and preinfarction angina.^{7 21 22} This multiplicity of names immediately draws attention to the difficulty in defining this condition. The description of the pain and its associated features and the extent of supporting evidence from ECG and coronary angiography vary greatly between series. The syndrome is often a changing one, which adds to the problems as patients may first be seen at different stages of the disease.

The composition of the groups reported has also varied widely. Though some patients had angina severe enough for them to be admitted to hospital, most were ambulant and few had pain when they attended hospital. Ours is the only study to have collected data from patients referred by general practitioners; others have drawn patients from private practice,^{7 11 12 13 16} from referrals to hospital,^{18 20 22} and from admissions to coronary care units.^{16 19} Other features which vary are age and the proportion of patients with a history of infarction or congestive cardiac failure. These characteristics have an important influence on the prognosis of ischaemic heart disease.^{23 24}

Coronary angiography was not undertaken routinely in our study because it was thought to be an extra hazard in patients with exacerbating symptoms,¹⁹ and the facilities for coronary artery surgery were limited at the start of the survey. Possibly our group included some patients without coronary disease, but it is unlikely that in a series of this kind the error exceeded 10%.²⁰

The choice for management lies between medical treatment or surgical intervention with saphenous vein bypass. The relative merits of these alternatives are under intensive study in randomised controlled trials,^{25 26} and full results are awaited with interest. Preliminary figures from one study²⁵ showed a higher mortality and morbidity rate in the first month after surgical treatment and little difference on longer follow-up. The other study²⁶ has so far shown a high mortality in medically treated patients in the subgroup with prolonged recurrent angina pectoris unrelated to effort. There was no difference in the other subgroup with progressive angina. These studies relate to patients who were generally more seriously ill than ours.

Our study was designed to explore the natural history of new and worsening angina and we did not attempt to assess the effectiveness of medical or surgical treatment. It seems, however, that the prognosis in our patients was such that an aggressive approach to investigation and surgery would not in general be justified.

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Appendix

Classification of ECG findings according to Minnesota code³: serious abnormality: 1-1, 1-2, 4-1, 5-1, 7-1, and 8-2; unequivocal abnormality: 3-1, 4-2, 5-2, 6-1, 6-2, 7-2, 7-4, 7-6, 8-3, 9-6,* 11-1, 11-2, 12-1, and 12-2; equivocal abnormality: 1-3, 2-1, 2-2, 3-2, 3-3, 4-3, 4-4, 5-3, 5-4, 6-3, 6-4, 6-5, 7-3, 7-5, 8-1, 8-6, 9-2-2,* 9-5, 9-7-2,* 11-3, 11-4, 12-3, and 12-4; minor abnormality: 2-3, 2-4, 2-5, 8-4, 8-5, 8-7, 8-8, 8-9, 9-2-1,* 9-3, 9-4-1, 9-4-2, 9-7-1,* and 9-8; normal ECG: 1-0.

*Additional codes used in the study; definitions available on request.

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Influence of maternal obesity on subcutaneous fat in the newborn

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Summary

Skinfold thickness measurements were made at biceps, triceps, subscapular, and suprilliac sites on both sides on 265 full-term newborn infants. The mothers were classified as thin (25), normal (179), or obese (61) on the basis of their triceps skinfold thicknesses. At all skinfold sites the babies of the obese mothers were significantly fatter than the babies of normal mothers, and the babies of the normal mothers were significantly fatter than those of the thin mothers. There was a highly significant positive correlation between maternal triceps thickness and the baby's sum of skinfold thicknesses. Hypertension was common during the pregnancies of the obese mothers and was associated with a significant reduction in the babies' skinfold thicknesses. When each of the 61 obese mothers were matched for parity, blood pressure, and smoking habit with 61 non-obese mothers the babies of the obese mothers still had significantly greater skinfold thicknesses than the babies of non-obese mothers.

Introduction

Both genetic and environmental factors contribute to the well-recognised familial tendency to obesity.¹ Both excessive maternal weight in the first trimester and excessive weight gain in pregnancy are associated with increased birth weight.^{2,3} Ounsted and Taylor⁴ found that small-for-gestational-age babies had mothers who, at delivery, were lighter than mothers of normal weight infants, and large-for-gestational-age babies had mothers who were taller and heavier than mothers of normal weight infants. Body weight, however, does not necessarily indicate the amount of fat in the body. People may be heavy without being fat if they are tall or have heavy bones, big

muscles, or water retention. In the newborn, differences in head size may appreciably affect body weight. There are no reports linking actual fatness at birth with maternal fatness, and in this study skinfold thickness was measured to determine whether a correlation exists between these two variables.

Subjects and methods

Two-hundred-and-sixty-five mothers and their babies born after at least 37 completed weeks' gestation were examined within 48 hours of birth. The mothers were randomly selected from the list of deliveries in the labour ward, apart from some of the obese mothers, who were found by searching the wards, but without knowledge of the baby's size. Mothers with abnormal glucose tolerance test results were excluded, as were those with multiple births, babies with multiple or severe congenital malformations, and babies assessed as less than 37 completed weeks' gestation.

Skinfold thickness was measured with a Harpenden caliper, which can be read to 0.1 mm and has surfaces small enough to be used on newborn babies. Brans *et al*⁵ suggest that when the caliper is used in the newborn it should be left on the skinfold for 60 seconds because otherwise the presence of oedema in the neonate leads to a falsely high estimate of subcutaneous fat. I have confirmed their finding that the shorter the period of gestation and the more oedematous the baby the greater is the percentage drop in the skinfold reading over the first 60 seconds after application of the caliper. Measurements were recorded after 60 seconds' pressure at four sites on both sides: biceps skinfold,⁶ triceps skinfold,⁷ subscapular skinfold,⁷ and suprilliac skinfold.⁶ A repeatability study of 104 pairs of duplicate measurements by one observer gave 95% confidence limits at the triceps site of ± 0.25 mm ($\pm 6.9\%$ of mean triceps skinfold) and at the subscapular site of ± 0.35 mm ($\pm 9.1\%$ of mean subscapular skinfold). The sum of eight readings (four on each side of the baby) had better repeatability, with 95% confidence limits of ± 0.94 mm (3.5% of mean sum of eight skinfolds), and also represented both trunk and limb fat. Like Farr,⁸ I found that skinfold thickness in newborn babies had a Gaussian distribution and could therefore be analysed statistically without logarithmic transformation.

Birth weight was recorded by midwives on beam scales. Crown to heel length was measured on a Cardiff neonatometer and occipito-frontal head circumference with a papertape measure.

Each mother's left triceps skinfold was measured and compared with the standards for young women.⁷ Mothers with triceps skinfold thicknesses above the 90th centile (24 mm) were classified as obese, those with readings between the 10th and 90th centile as normal, and those with readings below the 10th centile (10.2 mm) as thin. The mothers gave their informed consent to the study. The application of