reduces the second, and evidently has little effect on the third: thus there is no major penalty to the heart in terms of oxygen uptake for the increased flow that is generated. With the exception of other vasodilators most agents capable of augmenting cardiac output do so at greater metabolic cost.

As with most clinical studies, some compromises were made in design to safeguard the overriding interests of the patients. We were anxious to establish the most appropriate treatment for each patient with little delay and were therefore unable to return to control conditions after each infusion rate to distinguish drug effect from possible spontaneous improvement. We thought it justifiable, however, to interrupt treatment once after the 40 μg/min dose regimen in eight of our 11 patients to show that the haemodynamic variables returned to the pretreatment values; only mean arterial pressure remained appreciably different from control readings in most patients. We chose to use pulmonary artery end-diastolic pressure as a measure of left ventricular filling pressure to avoid repeatedly wedging the Swan-Ganz catheter, since frequent changes in position or balloon inflation carry small risks. Though the measurement tends to underestimate true left atrial pressure in the presence of heart failure,[15] the correlation between the two indices is close and changes are faithfully reflected.

Salbutamol has little effect on "backward failure" as shown by the increased pulmonary artery end-diastolic pressure in our patients and therefore cannot be recommended for the treatment of pulmonary oedema in acute myocardial infarction. It is not useful if an increase in blood pressure is believed to be essential. Our results suggest, however, that salbutamol is a useful and safe method of increasing cardiac output in those patients with myocardial infarction in whom poor perfusion is the most important haemodynamic disturbance.

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The Scottish Perinatal Mortality Survey

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Summary and conclusions

Perinatal deaths in single births that occurred in Scotland during 1977 were investigated by case-record analysis. Causes of death were divided into nine categories, an extended version of the Aberdeen classification being used. Out of 1012 single perinatal deaths, 265 were due to fetal abnormality, which in 140 cases was malformation of the central nervous system. Of the 747 normally formed infants, 446 weighed 1500 g or more, of whom 82 died intra partum and 154 were born alive. The largest single cause of death was low birth weight in normally formed babies whose mothers had no complications of pregnancy (302 cases). Of these babies, 103 (34%) were growth-retarded. Rhesus incompatibility (16 deaths) and maternal diabetes (seven deaths) were not major causes of perinatal loss.

These results were thought to be valuable in illustrating the main causes of perinatal mortality and directing attention to important issues. Hence a modified version of the study is being continued to see whether yearly audit by regional assessors is a feasible and practical way of monitoring trends in perinatal mortality.

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Introduction

Perinatal and neonatal mortality rates are higher in the United Kingdom than in many other Western countries. Anxiety about this was highlighted in an investigation by the Employment and Social Security Subcommittee of the House of Commons. In Scotland the perinatal mortality rate has always been higher than in England and Wales, but during 1966-76 there was a dramatic fall—namely, from 29/1000 total births to 18/1000. An English study showed that social and biological changes in the maternal age, parity, and social-class distribution of births accounted for a quarter of the overall improvement in the population.[1] During the decade there were many changes in obstetric and neonatal practice—for example, screening for central nervous system deformities; increased use of fetal monitoring during pregnancy and labour; greater use of induction of labour and caesarean section; and, on the neonatal side, increased use of ventilatory support and blood-gas monitoring despite little increase in medical and nursing staff. The effect of medical intervention was debatable, but greater awareness of the problems of pregnancy and recourse to induction of labour to prevent postmaturity definitely contributed to lower mortality rates.[2]

Similarly, changes in neonatal paediatric practice resulting in improved intensive care for babies of low birth weight changed the outlook for the small baby.[3]

The inadequacies of using crude perinatal mortality statistics to study the effectiveness of the perinatal health services have long been recognised, and the desirability of routine provision of standardised perinatal statistics for non-malformed singleton infants has been proposed.[4] As a method of obtaining information on non-malformed infants and determining events leading to death a study was undertaken of all perinatal deaths occurring in Scotland during 1977. The aims of the study, which was conducted from an obstetric standpoint, were (1) to determine the feasibility of obtaining information on all perinatal deaths during one year; (2) to analyse the case record of each perinatal death and classify that death according to the Aberdeen classification[5]; and (3) to analyse area differences in perinatal death rates with reference to maternal characteristics, type of care, and cause of death.

Method

Before the study began all the obstetric divisions in Scotland were asked to permit access to the records of all perinatal deaths that occurred in cases under their care. General practitioners were approached via the General Practitioner Subcommittee of the National Medical Consultative Committee. When a perinatal death occurred we were notified by the Registrar General for Scotland. Some 99% of births occurred in hospital, so the study was conducted as a retrospective case-record review. Information on domiciliary deliveries was obtained from the general practitioner concerned. The hospital medical records officer was requested to make the appropriate case notes available to a member of the team, who visited the hospital and completed a form for each death. Information obtained comprised the social, medical, and past obstetric history of the mother together with full details of her antenatal care, labour, and mode of delivery in the pregnancy that resulted in perinatal death. Neonatal records were abstracted for information on the baby. Necropsy findings and the registered cause of death were also recorded. The information was coded and transferred to punch cards.

Each death was classified according to an extended version of the Aberdeen classification, which categorises death according to the predisposing obstetric event. The nine main categories are: (1) low birth weight—that is, birth weight <2500 g, with no maternal complication; (2) "normal" birth weight—that is, birth weight ⩾2500 g, with no maternal complication; (3) trauma—that is, in babies over 1800 g; (4) pre-eclampsia; (5) antepartum haemorrhage; (6) maternal disease—that is, existing before pregnancy; (7) fetal abnormality; (8) rhesus incompatibility; and (9) other.

Growth retardation was assessed with tables based on the Aberdeen population, which were standardised for parity, sex of the infant, and gestational age.[6] Baseline data on all births in Scotland were obtained from the Information Services Division of the Common Services Agency (S Cole, personal communication, 1979).

TABLE I—*Birth weights and times of death (in relation to labour) of normally formed infants, and birth weights of infants dying from fetal abnormality*

	Birth weight				Total
	<1000 g	1000–1499 g	1500–2499 g	⩾2500 g	
Antepartum death	37	60	101	108	306 (41%)
Intrapartum death	9	6	33	49	97 (13%)
Postpartum death	80	93	83	73	329 (44%)
Total	126 (17%)	159 (21%)	217 (29%)	230 (31%)	732 (98%)*
Fetal abnormalities	21	34	91	111	257†
Grand total	147	194	308	341	989

*Birth weight was not stated in 15 cases.
†Birth weight was not stated in eight cases.

Results

During 1977, 62 895 births occurred in Scotland, of which 1150 resulted in a perinatal death; this represents a perinatal mortality rate of 18/1000 total births.[7] There were 110 deaths in multiple pregnancies, but these are excluded from the present analysis. Six babies were found abandoned, one abortion had been wrongly classified as a stillbirth, seven babies were born to women not normally resident in Scotland, and no details were available for 14 others. This report therefore analyses the remaining 1012 perinatal deaths in single births.

Table I gives the birth weights and times of death (in relation to labour) of normally formed infants and the birth weights of infants dying from fetal abnormalities. Of the 747 normally formed infants, 126 (17%) weighed under 1000 g, 159 (21%) weighed 1000-1499 g, 217 (29%) weighed 1500-2499 g, and 230 (31%) weighed 2500 g and over. A total of 306 babies (41%) died before labour, 97 (13%) died during labour, and 329 (44%) died after delivery. There were more postpartum than antepartum deaths among babies weighing under 1500 g, whereas the reverse was true for bigger babies. Of all 1012 perinatal deaths, 265 (26%) resulted from fetal abnormality.

Table II lists the distribution of categories of death at different times among normally formed infants of various birth weights. A total of 302 (40%) deaths among these infants occurred in those of low birth weight whose mothers had no obstetric complications.

TABLE II—*Categories of perinatal death at different times among normally formed infants of various birth weights*

Time of death	Low birth weight*	Antepartum haemorrhage	Normal birth weight*	Pre-eclampsia	Trauma (birth weight >1800 g)	Maternal disease†	Rhesus incompatibility	Other	Total	
				Weight <1000 g						
Antepartum	25	0	0	6	0	6	0	0	37	126 (17%)
Intrapartum	2	3	0	4	0	0	0	0	9	
Postpartum	57	15	0	4	0	4	0	0	80	
				Weight 1000–1499 g						
Antepartum	26	14	0	13	0	7	0	0	60	159 (22%)
Intrapartum	0	4	0	2	0	0	0	0	6	
Postpartum	68	12	0	7	0	6	0	0	93	
				Weight 1500–2499 g						
Antepartum	61	21	0	10	1	5	3	0	101	217 (30%)
Intrapartum	11	8	0	10	3	1	0	0	33	
Postpartum	44	20	0	6	7	2	4	0	83	
				Weight ⩾2500 g						
Antepartum	0	16	65	13	1	9	4	0	108	230 (31%)
Intrapartum	0	6	17	6	18	1	1	0	49	
Postpartum	0	7	24	7	18	3	2	12	73	
				All weights						
Antepartum	112	51	65	42	2	27	7	0	306	732 (100%)
Intrapartum	13	21	17	22	21	2	1	0	97	
Postpartum	169	54	24	24	25	15	6	12	329	
Total	294	126	106	88	48	44	14	12	732	
Weight not stated	8	0	0	1	2	1	2	1	15 (2%)	
Grand total	302 (40%)	126 (17%)	106 (14%)	89 (12%)	50 (7%)	45 (6%)	16 (2%)	13 (2%)	747 (100%)	

*No maternal complications.
†Existing before pregnancy.

Antepartum haemorrhage was the predisposing factor in 126 other cases (17%), and in 106 cases (14%) in which birth weight was normal no cause could be found. Pre-eclampsia occurred in 89 cases (12%), traumatic delivery was the cause of death in 50 (7%), and pre-existing maternal disease was present in 45 (6%). Rhesus incompatibility and other conditions accounted for 16 (2%) and 13 (2%) of the deaths respectively. Death before the onset of labour occurred in 112 infants (37%) in the low birth weight group, 51 (40%) in the antepartum haemorrhage group, 65 (61%) in the normal birth weight group, 42 (48%) in the pre-eclamptic group, and 27 (60%) in the maternal disease group. Intrapartum neonatal death occurred in 22 cases of pre-eclampsia (24%) and 21 cases of trauma (42%). Only 17 deaths in the normal birth weight group (16%) occurred intra partum.

Of the deaths in the low birth weight group (table III), the largest single subgroup comprised preterm babies who were not growth-retarded (132 cases; 44%). Nevertheless, growth retardation had occurred in 103 cases (34%), and 27 mothers (9%) gave a history suggestive of cervical incompetence; in 37 cases (12%) there was a history of threatened abortion earlier in pregnancy. Interestingly, though three-quarters of the preterm infants were alive at birth, the same proportion of growth-retarded infants had died before labour.

Table IV gives an extended version of the Aberdeen classification. The greatest problem was that of low birth weight babies without maternal complications (30% of all deaths), preterm labour accounting for almost half of the deaths in this group. In the normal birth weight group only 13 babies were growth-retarded. Poor maternal weight gain—that is, less than 1 kg in the last four weeks of pregnancy—was evident in 36 mothers (12 of whom were obese), and fetal distress was recorded in 23 babies who died during or after labour. Cord complications resulted in 14 deaths. Difficult instrumental delivery accounted for a further 20 deaths, and complications with breech delivery was the cause of 16. Deaths from pre-eclampsia were complicated by growth retardation in 32 cases.

Antepartum haemorrhage accounted for 126 deaths. Abruptio placentae was responsible for 85 of these, and 30 babies weighed under 1500 g. Of the bigger babies, only nine were born alive, six weighing under 1500 g. There were 29 deaths to mothers with a history of antepartum haemorrhage but in whom no retroplacental clot was found; of these babies, 17 weighed 1500 g or more, 10 of whom were born alive.

TABLE III—*Analysis of deaths in normally formed infants of low birth weight*

Time of death	Preterm	Growth-retarded	Cervical incompetence	Threatened abortion	Other	Total
			Weight <1000 g			
Antepartum	2	21	0	2	0	25
Intrapartum	0	0	1	1	0	2
Postpartum	28	0	12	17	0	57
			Weight 1000–1499 g			
Antepartum	11	12	0	3	0	26
Intrapartum	0	0	0	0	0	0
Postpartum	40	9	10	8	1	68
			Weight 1000–1999 g			
Antepartum	10	16	1	1	0	28
Intrapartum	1	3	1	1	0	6
Postpartum	18	6	2	1	1	28
			Weight 2000–2499 g			
Antepartum	9	24	0	0	0	33
Intrapartum	1	4	0	0	0	5
Postpartum	7	8	0	0	1	16
			All weights			
Antepartum	32	73	1	6	0	112
Intrapartum	2	7	2	2	0	13
Postpartum	93	23	24	26	3	169
Total	127	103	27	34	3	294
Weight not stated	5	0	0	3	0	8
Grand total	132 (44%)	103 (34%)	27 (9%)	37 (12%)	3 (1%)	302 (100%)

TABLE IV—*Distribution of all 1012 perinatal deaths in single births categorised according to extended Aberdeen classification*

Classification	No of deaths in single births	Death rate/ 1000 total births
Low birth weight (no maternal complications)	302	4·8
Preterm	127	
Growth-retarded	103	
Threatened abortion	37	
Cervical incompetence	27	
Other	8	
Normal birth weight (no maternal complications)	106	1·7
Normal	93	
Growth-retarded	13	
Fetal abnormality	265	4·3
Central nervous system	140	
Congenital heart disease	38	
Renal	26	
Alimentary	19	
Multiple	19	
Chromosomal	11	
Skeletal	5	
Other	7	
Antepartum haemorrhage	126	2·0
Abruptio placentae	85	
Placenta praevia	12	
Other	29	
Pre-eclampsia	89	1·4
Trauma (birth weight >1800 g)	50	0·8
Breech	16	
Cord	14	
Other	20	
Maternal disease	45	0·7
Essential hypertension	12	
Diabetes	7	
Abdominal operation during pregnancy	6	
Cervical operation before pregnancy	3	
Jaundice	3	
Road-traffic accident	2	
Other	12	
Rhesus incompatibility	16	0·3
Other	13	0·2

Various maternal conditions contributed to 45 perinatal deaths, the greatest number (12 cases) occurring in women with essential hypertension. Diabetes was held to be the predisposing factor in seven cases. Rhesus incompatibility accounted for 16 perinatal deaths, nine of the women having been sensitised before the introduction of anti-D immunoglobulin.

Fetal abnormality was the cause of 265 perinatal deaths, of which 140 were due to malformation of the central nervous system. Sixty-one mothers had attended the hospital antenatal clinic before 20 weeks of gestation, but α-fetoprotein estimation had not been carried out.

Thirteen normally formed babies died during the first week of life from various neonatal complications unconnected with the antenatal and intrapartum periods.

A total of 675 infants (67%) came to necropsy.

Discussion

This case-record analysis of perinatal deaths occurring in Scotland during 1977 was made possible by the complete co-operation of all concerned with maternity care throughout the country. This first report highlights the main problems.

With good obstetric and paediatric care normally formed babies weighing at least 1500 g would be expected to survive, yet in our survey 446 normally formed babies weighed at least 1500 g and two-thirds were dead before delivery. These deaths constituted 44% of the perinatal deaths in the year studied; had these infants survived the perinatal mortality rate would have been 9/1000 total births.

The largest single cause of death was low birth weight in

babies whose mothers had no obstetric complications. In the preterm group three-quarters of the babies were born alive and presented problems in the neonatal period, requiring specialised paediatric care. In the growth-retarded group the problem was that of intrauterine death, and if such babies are to survive the obstetrician must be able to detect that the fetus is compromised and deliver it before fetal death occurs. This is also true for babies of "normal" birth weight, 82 such infants dying before delivery. Postmaturity is not now an important cause of fetal death; the problem is that of unexplained intrauterine death before term. Such babies were not growth-retarded.

From the retrospective analysis we could not discern whether many of the deaths caused by trauma were preventable. Traumatic breech deliveries would, of course, be prevented by earlier recourse to caesarean section. This operation is not without complications, however, and undiagnosed breech presentations will continue to occur. In the antepartum haemorrhage group the fetal heart was not heard on admission in 57% of cases in which death occurred before delivery. We cannot see how these deaths could have been averted. Earlier antenatal attendance, so that screening for central nervous system abnormality could be carried out and the patient offered termination of pregnancy, would reduce the number of abnormal births, although clearly would not prevent the occurrence of the abnormality. In the pre-eclamptic group 39 babies died before delivery and weighed 1500 g or more. The mothers of these babies did not have fulminating pre-eclampsia, and the diagnosis that the fetus was stressed had not been made. In all but a few cases of maternal disease the condition had been recognised by the obstetrician and appropriate care given. Hence these deaths could probably not have been prevented. The problems of rhesus incompatibility are now only a small part of obstetric and paediatric practice, partly because of the introduction of anti-D immunoglobulin and partly as a result of changes in family size.

We believe that analysing perinatal deaths in the manner described is of value in illustrating the main causes of perinatal mortality and of directing attention to issues of contemporary importance. Hence we are now studying perinatal deaths that occurred in Scotland during 1979 to see whether yearly assessment by regional assessors (obstetricians and paediatricians) is a feasible and practical way of monitoring trends in perinatal mortality that, in turn, will lead to a reduction in preventable deaths.

We are grateful for the help we received from all concerned in perinatal care throughout Scotland. We acknowledge the help we received from Sir John Brotherston and the Scottish Home and Health Department in launching the survey. Frances Dunn is supported by a grant from the Scottish Home and Health Department.

Copies of the survey report may be obtained from Frances Dunn, price £1.20 (including postage).

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Essential hypertension: effect of an oral inhibitor of angiotensin-converting enzyme

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Summary and conclusions

Captopril, a specific oral inhibitor of angiotensin-converting enzyme, was given to 18 unselected patients with moderate essential hypertension. Mean blood pressure fell by 14·5% at the maximum dose given, and this fall was significantly correlated with the initial plasma renin activity. The main fall in blood pressure occurred two hours after the first dose of captopril.

These results suggest that captopril effectively lowers blood pressure in patients with essential hypertension and that the renin-angiotensin aldosterone system may maintain blood pressure in essential hypertension. This does not necessarily imply that the renin-angiotensin system is the cause of the high blood pressure.

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Introduction

The importance of the renin-angiotensin aldosterone system in maintaining blood pressure in essential hypertension is disputed.[1 2] Infusions of competitive inhibitors of angiotensin II, such as saralasin, cause no change in blood pressure in most patients with essential hypertension receiving their normal sodium intake.[3] Saralasin, however, is a potent agonist,[4-6] and when given as a short-term infusion blocks only the immediate effects of circulating angiotensin II. Both of these factors will tend to underestimate the role of the renin-angiotensin aldosterone system in maintaining blood pressure. The converting-enzyme inhibitor teprotide, which blocks the enzyme that converts angiotensin I to angiotensin II, has, when given as a single injection, a slight lowering effect on blood pressure in some patients with essential hypertension studied while receiving their normal diet.[3] More recently an oral drug, captopril, that specifically inhibits the angiotensin-converting enzyme has been developed.[7 8] With this drug long-term inhibition of the formation of angiotensin II is now possible.