Classification of perinatal mortality

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Summary: A classification based upon pathophysiological mechanisms has been applied to perinatal mortality. The approximate relevance of each group was: fetal insufficiency 40%, respiratory failure 30%, developmental abnormality 20%, and the specific mechanisms 10%. Secondary subclassification of each category according to the maturity of the gestation emphasizes the importance of prematurity in the total perinatal mortality (66%) and the relevance of each pathophysiological mechanism in each phase of maturity. The principal mechanism in the mature group is fetal insufficiency and in the premature group, particularly between 20 and 28 weeks, it is neonatal respiratory failure. Perinatal death due to developmental abnormalities occurs most frequently in the mature infant and due to specific mechanisms in the premature infant, particularly between 29 and 36 weeks. It is suggested that greater emphasis should be placed on perinatal mortality rather than the separate consideration of stillbirth and neonatal mortality.

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A classification of perinatal morbidity and mortality based upon the pathophysiological mechanisms which affect the fetus and newborn infant has previously been proposed.[1] The application of the classification to perinatal mortality has been assessed using data derived from a review of the perinatal mortality from one service, for the five year period 1966-1970. The clinical course and pathological findings in each perinatal death between 20 weeks of gestational age and the 28th day of the neonatal period have been discussed in conference.

The classification comprises four groups: Group I—Chronic fetal insufficiency, Acute fetal insufficiency; Group II—Neonatal respiratory failure; Group III—Developmental abnormality; Group IV—Blood group incompatibility, infection, trauma and miscellaneous.

Results

The proportion of perinatal deaths due to each mechanism is shown in Table I.

Relationship of gestational maturity to perinatal mortality

A classification of perinatal mortality should be subclassified on the basis of maturity to demonstrate the role of prematurity in total perinatal mortality, and the relevance of variations in maturity to each pathophysiological mechanism.

In the total series there were 112 perinatal deaths in the premature group, representing 66% of the total perinatal mortality. Fifty-one perinatal deaths occurred between 20 and 28 weeks and 61 occurred between 29 and 36 weeks, representing 30% and 36% of the total perinatal mortality respectively. There were 56 perinatal deaths in the mature group, representing 34% of the perinatal mortality, (Table II).

Table III demonstrates the relevance of each group of pathophysiological mechanisms to the perinatal mortality in the two phases of the premature group and the mature group. Group I (chronic and acute fetal insufficiency) is the principal mechanism of perinatal mortality in the mature infant, accounting for 55% of the perinatal mortality in this group. However, Group I contributes significantly to the perinatal mortality in the premature infant, to the extent of 32% of the perinatal mortality between 20 and 28 weeks and 33% between 29 and 36 weeks. Group II (neonatal respiratory failure) is the principal mechanism of

perinatal mortality in the premature group accounting for 50% of the perinatal mortality between 20 and 28 weeks and 28% between 29 and 36 weeks. There are occasional deaths in the mature infant due to respiratory failure, accounting for 7% of the mature deaths in this series. Pregnancies associated with major developmental fetal abnormalities (Group III) tend to continue toward maturity, accounting for only 8% of the perinatal deaths between 20 and 28 weeks, 21% between 29 and 36 weeks, and 32% in the mature group. Perinatal deaths due to the specific mechanisms of Group IV occurred most frequently in the premature infants between 29 and 36 weeks, accounting for 16% of the perinatal mortality in this group.

Table I
The relevance of each group of pathophysiological mechanisms in respect of perinatal mortality

Group	Mechanisms		No.	%	Relevance
I	Fetal insufficiency	Chronic	26	16 %	40%
		Acute	40	24 %	
II	Neonatal respiratory failure		49	29 %	30%
III	Developmental abnormality		35	21 %	20%
IV	Blood group incompatibility		9	5.4%	10%
	Infection		6	3.6%	
	Trauma		2	1.2%	
	Miscellaneous		1	0.6%	

Table II
The relationship of maturity to perinatal mortality

	Premature		Mature
	20-28 weeks	29-36 weeks	37-44 weeks
Number	51	61	56
Percentage	30%	36%	34%

Table III
The relevance of each group of pathophysiological mechanisms in respect of perinatal mortality in each phase of maturity

			Premature		Mature
Group	Mechanism		20-28 weeks	29-36 weeks	37-44 weeks
I	Fetal insufficiency	Chronic	4%	18%	25%
		Acute	28%	15%	30%
II	Neonatal respiratory failure		50%	28%	7%
III	Developmental abnormality		8%	21%	32%
IV	Blood group incompatibility		2%	10%	3.5%
	Infection		4%	5%	1%
	Trauma		2%		
	Miscellaneous			1%	
	Total		100%	100%	100%

Table IV
The characteristics of the obstetric patients with an intrauterine death due to acute fetal insufficiency

		Premature		Mature
	Total	20-28 weeks	29-36 weeks	37-44 weeks
Obstetrical complications	17			
Antepartum hemorrhage		9	2	0
Acute toxemia		0	1	2
Labour complications		0	1	2
"Normal"	23	6	5	12

Acute fetal insufficiency

Acute fetal insufficiency is a fetal state due to failure of maternal-fetal blood-gas exchange in which fetal asphyxia leads to metabolic acidosis; the latter, when sustained or severe, results in an intrauterine death. There were 40 intrauterine deaths attributed to acute fetal insufficiency. The characteristics of the patients in whom this occurred are outlined in Table IV. Eleven occurred in patients with an antepartum hemorrhage. This was associated with premature placental separation in eight cases, placenta praevia in two cases, and a circumvallate placenta on one occasion. Three occurred in patients with acute toxemia, one in the premature and two in the mature group; there was one case of eclamptic toxemia, one of severe and one of mild pre-eclamptic toxemia. Three occurred in classical complications of labour, one in the premature and two in the mature group; one patient had a transverse lie with a prolapsed arm, one a ruptured uterus and one a prolapsed cord. Fifty-five percent of the intrauterine deaths[2, 3] attributed to acute fetal insufficiency occurred in patients with no apparent major medical or obstetric complication, six between 20 and 28 weeks, five between 29 and 36 weeks, and 12 in the mature group of whom six were beyond 42 weeks of gestational age.

Chronic fetal insufficiency

Chronic fetal insufficiency is a fetal state in which retardation of fetal growth during the intrauterine period leads to a newborn infant weighing significantly less than the norm for gestational age with or without gross tissue wasting. The criterion followed in this study was an infant below the 10th percentile for the gestational age on the basis of the weight-gestational age scale of Gruenwald.[2] At the present time, infants below the 10th percentile with major developmental abnormalities and intrauterine infections such as rubella are excluded from this group. There were 26 intrauterine deaths classified as being due to chronic fetal insufficiency. The characteristics of the patients in whom these occurred are outlined in Table V. Six intrauterine deaths occurred in patients with major medical disease, one between 20 and 28 weeks, three between 29 and 36 weeks, and two in the mature group. This group comprised one patient with lupus erythematosis and renal disease, two patients with hypertension and three patients with diabetes. Six intrauterine deaths occurred in patients with major obstetric complications, one between 20 and 28 weeks, four between 29 and 36 weeks and one in the mature group. Two of the patients had severe pre-eclampsia, two had premature placental separation, and two had monozygotic twin pregnancies in which the second twin was sustained by a small fraction of the total placenta. Again, 55% of the intrauterine deaths (14)

Table V
The characteristics of the obstetric patients with an intrauterine death due to chronic fetal insufficiency

		Premature		Mature
	Total	20-28 weeks	29-36 weeks	37-44 weeks
Medical complications	6			
Lupus		1		
Hypertension			2	
Diabetes			1	2
Obstetric complications	6			
Toxemia			2	
Antepartum hemorrhage		1	1	
Multiple pregnancy			1	1
"Normal"	14		3	11

attributed to chronic fetal insufficiency occurred in patients with no major medical or obstetric complication, three in the premature and eleven in the mature group of whom five were beyond 42 weeks of gestational age.

Neonatal respiratory failure

There were 48 neonatal deaths of newborn infants due to respiratory failure. The two principal categories identified were central respiratory failure and hyaline membrane disease (Table VI).

Central respiratory failure accounted for 32 neonatal deaths (66%), 23 between 20 and 28 weeks, seven between 29 and 36 weeks and two in the mature group. These newborns were characterized by a low Apgar score (1-3 in 27 cases) difficult resuscitation followed by persistent respiratory difficulty with apneic episodes. Survival was brief, less than one hour in 14 cases, one to six hours in 14 cases and slightly in excess of six hours in three cases. Hyaline membrane disease accounted for 12 neonatal deaths, one between 20 and 28 weeks, 10 between 29 and 36 weeks, and one in the mature group. These newborn infants were characterized by a variable Apgar score, later onset of respiratory difficulty and neonatal death between 15 and 51 hours after birth. There was one case of aspiration syndrome and one of pulmonary hemorrhage.

The established importance of prematurity in respect of respiratory failure is clearly demonstrated by the large proportion of immature newborn infants in the central respiratory failure category and of premature newborn infants in the group with hyaline membrane disease. Chronic fetal insufficiency was present on six occasions, two in association with central respiratory failure, and four in association with hyaline membrane disease.

Table VI

The principal categories of respiratory failure leading to a neonatal death

	Total	Premature		Mature
		20-28 weeks	29-36 weeks	37-44 weeks
Central respiratory failure	32	23	7	2
Hyaline membrane disease	12	1	10	1
Aspiration syndrome	1			1
Pulmonary hemorrhage	1		1	
Miscellaneous	2			

Table VII

Classification of the perinatal mortality in Kingston General Hospital for the five-year period 1966-1970

Mechanisms	Total No. patients	Percentage of total	Maturity (weeks) Premature 20-28	Premature 29-36	Mature 37-44
I Fetal insufficiency Chronic	26	40%	2	11	13
I Fetal insufficiency Acute	40		14	9	17
II Neonatal respiratory failure	49	29%	27	17	5
III Developmental abnormality	35	21%	4	13	18
IV Blood group incompatibility	9		1	6	2
Infection	6	10%	2	3	1
Trauma	2		1	1	0
Miscellaneous	1		0	1	0
Total	168	100%	51	61	56

Acute fetal insufficiency and fetal asphyxia were frequently inferred because of the associated obstetric complication and the low Apgar scores; however, biochemical confirmation is as yet rarely available and pathological interpretation is confused by the effects of neonatal asphyxia

Developmental abnormality

Developmental abnormalities resulting in an intrauterine or neonatal death occurred in 35 cases. Central nervous system abnormalities occurring in 16 cases composed the principal group, the remainder included seven major cardiac abnormalities, four renal anomalies and a miscellaneous group.

Specific mechanisms

Major Rh blood group incompatibilities led to intrauterine or neonatal death in nine cases. Infection, septicemia or pneumonia resulted in neonatal death in six cases while trauma at delivery associated with intracranial hemorrhage occurred in two cases.

Comments

It is proposed that perinatal mortality be summarized in the manner outlined in Table VII. The primary classification of each perinatal death should be on the basis of the relevant pathophysiological mechanism. The assignment of each perinatal death to these broad categories is not difficult in most instances. This provides an overall perspective of the importance of each mechanism. The approximate relevance of each group in this perinatal mortality experience was: fetal insufficiency 40%, respiratory failure 30%, developmental abnormality 20%, and the specific mechanisms 10%.

Secondary subclassification of each category according to the maturity of the gestation emphasizes the importance of prematurity in the total perinatal mortality. The association of prematurity in 66% of cases corresponds in general to previously reported experience. However, the recent reminder by Gruenwald,[3] that a significant proportion of perinatal mortality occurs in mature gestations is particularly timely in the continuing endeavour to further reduce perinatal wastage.

The relevance of each pathophysiological mechanism in each phase of maturity is identified by such a display of perinatal mortality. Fetal insufficiency is significant in each phase of maturity and the principal mechanism in the mature group. Neonatal respiratory failure is the principal mechanism in the premature group, particularly between 20 and 28 weeks, but is occasionally the cause of death in the mature group. Perinatal deaths associated with developmental abnormalities occur most frequently in the mature infant while those due to specific mechanisms are most common in the premature infant, particularly between 29 and 36 weeks.

Chronic fetal insufficiency is defined by weight-gestational age standards. Although major progress has been made in respect of these standards a number of outstanding questions remain in respect of the accurate definition of gestational age and the weight standards for different patient populations. The weight-gestational age should be supplemented by further sophisticated indices of intrauterine growth retardation to permit accurate identification of this entity.

Fifty-five percent of perinatal mortality due to chronic and acute fetal insufficiency occurred in "normal" obstetric patients, demonstrating very clearly a need for more refined or specific indices to identify these high risk

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the onset of secondary impotence in other than advanced states of diabetic neuropathy, this fact is but another example of the multiple etiological aspects of secondary impotence." The present study shows that obstruction to blood flow in vessels supplying the penis must be considered as a possible cause of impotence in diabetic patients.

I am grateful to the normal subjects who volunteered for measurements of penile pressure and to the patients who cooperated in this study. I also wish to thank Mr. David Powell for his technical assistance.

Résumé

L'étude de la pression systolique normale du pénis, mesurée par le méthode streptoscopique, a montré qu'elle est égale ou supérieure à celle calculée par la pression brachiale moyenne. Une pression nettement inférieure à celle-ci chez des impuissants indique l'existence d'un obstacle à la circulation dans les vaisseaux irriguant le pénis. En partant de ce principe, on a découvert un obstacle à la circulation chez des malades qui ne présentaient pas ou peu de signes d'une pathologie des vaisseaux périphériques.

References

1. GASKELL P: The rate of blood flow in the foot and calf before and after reconstruction by arterial grafting of an occluded main artery to the lower limb. *Clin Sci* 15: 259-269, 1956
2. IDEM: Laboratory tests of circulation in the limbs. *Manit Med Rev* 45: 540-546, 1965
3. WINSOR T: Influence of arterial disease on the systolic blood pressure gradients of the extremity. *Am J Med Sci* 220: 117-126, 1950
4. GASKELL P: The measurement of blood pressure, the critical opening pressure, and the critical closing pressure of digital vessels under various circumstances. *Can J Physiol Pharmacol* 43: 979-993, 1965
5. STRAUSS EB: Impotence from the psychiatric standpoint. *Br Med J* 1: 697-699, 1950
6. STAFFORD-CLARK D: The etiology and treatment of impotence. *Practitioner* 172: 397-404, 1954
7. HASTINGS DW: *Impotence and Frigidity,* Boston, Little, Brown, 1963
8. MASTERS WH, JOHNSON WE: *Human Sexual Inadequacy,* Boston, Little, Brown, 1970
9. JOHNSON J: *Disorders of Sexual Potency in the Male,* Oxford, Pergamon 1968, p 28
10. RUBIN A, BABBOTT D: Impotence and diabetes mellitus. *JAMA* 168: 498-500, 1958
11. SIMPSON SL: Impotence. *Br Med J* 1: 692-696, 1950
12. SCHÖFFLING K, FEDERLIN K, DITSCHUNEIT H, et al: Disorders of sexual function in male diabetics. *Diabetes* 12: 519-527, 1963

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Recent Advances in Blood Coagulation, ed. Poller L, London, Churchill, 1969
4. LOELIGER EA, MEUWISSE-BRAUN JB, MUIS H, et al: Laboratory control of oral anticoagulants. *Thromb Diath Haemorrh* 23: 569-584, 1970
5. ZUCKER S, CATHEY MH, SOX PJ, et al: Standardization of laboratory tests for controlling anticoagulant therapy. *Am J Clin Path* 53: 348-354, 1970
6. CECIL RL, LOEB RF: *Textbook of Medicine,* ed Beeson PB, McDermott W, twelfth ed, Philadelphia, Saunders, 1967
7. MIALE JB: *Laboratory Medicine Hematology,* third ed, St. Louis, Mosby, 1967
8. GOODMAN LS, GILMAN A: *The Pharmacological Basis of Therapeutics,* fourth ed, Toronto, Macmillan, 1970
9. POLLER L: Standardization of anticoagulant treatment: the Manchester regional thromboplastin scheme. *Br Med J* 2: 565-566, 1964
10. THOMSON JM, CHART IS: A system for laboratory control of oral anticoagulant therapy using British comparative thromboplastin. *J Med Lab Technol* 27: 207-212, 1970
11. BIGGS R, MACFARLANE RG: *Human Blood Coagulation,* third ed, Oxford, Blackwell, 1962
12. HILL AB: *Principles of Medical Statistics,* eighth ed, New York, Oxford University Press, 1966
13. BLAKELY JA: Unpublished observations
14. BIGGS R, DENSON KWE: Standardization of the one-stage prothrombin time for the control of anticoagulant therapy. *Br Med J* 1: 84-88, 1967
15. MIALE JB, LAFOND DJ: Prothrombin time standardization, *Plasma Fractions for Treatment of Hemophilia and Anticoagulant Therapy: Standardization of Tests,* ed Owren PA, Stuttgart, Schattauer, 1969
16. IDEM: Prothrombin time standardization. *Am J Clin Path* 52: 154-160, 1969
17. DOUGLAS AS: Current status of anticoagulant therapy, *Recent Advances in Blood Coagulation,* ed Poller L, London, Churchill, 1969, pp 107-135
18. POLLER L, THOMSON JM: National survey of value of plasma standards for anticoagulant therapy. *Br Med J* 2: 88-90, 1968

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patients in the obstetric population. There is, in addition, the need for specific measures to provide for fetal welfare if perinatal mortality due to chronic and acute fetal insufficiency is to be avoided in the high risk patients identified.

The relationship of respiratory failure to prematurity has been demonstrated and the importance of the prevention of premature labour persists as a foremost problem to the obstetrician. The relevance of other mechanisms of perinatal pathophysiology as predisposing or complicating factors in newborn respiratory failure has often been implied but has not yet been accurately identified. The true incidence particularly of acute fetal insufficiency as a predisposing factor to central respiratory failure or hyaline membrane disease requires further assessment.

Rh blood group incompatibility particularly during the critical premature period continues to be associated with some perinatal loss in spite of current understanding and resources of management. Similarly the importance of infection to the premature infant is emphasized by the mortality in this category.

Finally, it is proposed that statistically greater emphasis should be placed on perinatal mortality rather than on the separate consideration of stillbirth and neonatal mortality. The fundamental unity of the perinatal period is evident from the continuity of some pathophysiological mechanisms through the antenatal and neonatal periods and the likely inter-relationship of mechanisms distinctive to the antenatal and neonatal phases such as fetal insufficiency and neonatal respiratory failure.

Résumé

La mortalité périnatale: essai de classification

Les auteurs ont appliqué à la mortalité périnatale les principes d'une classification basée sur les mécanismes physiopathologiques. La composition approximative de chaque groupe était la suivante: insuffisance foetale 40%, insuffisance respiratoire 30%, anomalies du développement 20%, divers mécanismes spécifiques 10%. Les sous-classes de chaque catégorie, suivant l'état d'avancement de la grossesse, font état de l'importance de la prématurité dans la mortalité périnatale globale (66%) et du rôle de chaque mécanisme physiopathologique à chaque stade de la maturité. Dans le groupe des enfants nés à terme, le mécanisme principal est l'insuffisance foetale et, dans le groupe des prématurés, particulièrement entre 20 et 28 semaines, c'est l'insuffisance respiratoire néonatale. Le décès périnatal par anomalies du développement survient plus souvent chez le nourrisson à terme. Le décès causé par un mécanisme spécifique survient chez le prématuré, particulièrement entre 29 et 36 semaines. Les auteurs estiment qu'il faudrait considérer davantage la mortalité périnatale, plutôt que de considérer séparément les naissances d'enfants mort-nés et la mortalité néonatale.

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two groups was not significant. A second observation raising doubt about the effectiveness of antiarrhythmic therapy concerns the timing of deaths. A year-by-year comparison of mortality in the Coronary Unit at the Toronto General Hospital showed a decrease from 36% in the first year to 14% in the eighth year (Table VIII). This reduction was due mainly to the occurrence of fewer deaths during the first 48 hours. Since this is the period when arrhythmias are most frequent and because of the yearly increase in the use of antiarrhythmic drugs, it might seem likely that the reduction in mortality was due to the treatment of cardiac arrhythmias. It was surprising, therefore, to find in the present study that early deaths were not more numerous on the medical wards where cardiac arrhythmias were not detected as frequently or treated as precisely as in the Coronary Unit.

The monitoring of the ECG which encouraged the use of antiarrhythmic drugs had the reverse effect on the use of digitalis. The fear that digitalis would aggravate arrhythmias has led to the restriction of the drug in the Coronary Unit. Mild congestive heart failure was treated often with diuretics alone. On the other hand digitalis was prescribed in the usual manner on the medical wards. If digitalis is a dangerous drug and increases the incidence of cardiac arrhythmias, the monitor in the Coronary Unit might have proved to be an important safeguard for patients admitted there. However, mortality for patients on digitalis in the Coronary Unit was not significantly different from that for patients on the drug in the medical wards.

It is generally believed that the primary role of the Coronary Unit approach is to prevent deaths due to cardiac arrhythmias. However, except in the case of primary ventricular fibrillation, it is usually impossible to decide whether arrhythmias play a primary role in determining the outcome or are merely secondary to other factors related to the size of the infarct.[5]

In the present study we have been unable to establish that there is any clear-cut benefit from the use of antiarrhythmic drugs in the Coronary Unit. Moreover, in a recent report of 20 patients with primary ventricular fibrillation no antecedent warning arrhythmia was observed in five patients, and another eight of the 20 were receiving antiarrhythmic drugs at the time that ventricular fibrillation occurred.[6]

A definite advantage offered by the Coronary Unit was the immediate resuscitation following cardiac arrest. This was responsible for only a small reduction in the mortality of the Coronary Unit group. If the observed difference between the two groups of patients was not largely due to the greater number of high-risk elderly patients admitted to the medical wards it would appear that several factors may have interacted to save lives in the Coronary Unit. For example, the closer supervision in the Unit may have resulted in the early recognition of congestive heart failure and more effective treatment of this complication with diuretics. Similarly the detection of heart block may have led to the use of pacemakers more frequently. In the absence of a controlled trial these possible benefits remain unproven.

Table VIII

Mortality in patients with proven acute myocardial infarction treated in Toronto General Hospital coronary unit March 13, 1962 to March 12, 1971

Year	No. of patients	Hospital mortality	Deaths first 48 hours	Deaths after 48 hours	Patients given antiarrhythmic therapy
1962-63	146	52(36)	34(23)	18(13)	21(14)
1963-64	176	65(37)	32(18)	33(19)	30(17)
1964-65	161	44(27)	22(14)	22(13)	15(31)
1965-66	170	44(26)	20(12)	24(14)	85(50)
1966-67	136	30(22)	13(10)	17(12)	78(57)
1967-68	191	35(18)	13(7)	22(12)	134(70)
1968-69	196	23(12)	6(3)	17(8)	126(64)
1969-70	239	34(14)	16(7)	18(7)	147(61)
1970-71	197	29(14)	17(9)	12(6)	143(73)
Total	1612	356(22)	173(11)	183(11)	779(48)

Figures in parentheses are per cent

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Résumé

Comparaison du traitement de l'infarctus aigu du myocarde dans une unité spéciale pour coronariens et dans une salle de médecine générale

Durant l'année 1968, 400 cas d'infarctus aigu du myocarde ont été hospitalisés au Toronto General Hospital (mortalité 25.0%). La moitié environ des malades qui ont survécu à leur séjour au service d'urgence ont été envoyés à la section des coronariens (mortalité 15.6%), alors que l'autre moitié, par manque de lits disponibles dans la dite section, ont dû être envoyes dans une salle de médecine générale (mortalité 26.5%). Il faut cependant noter qu'un plus grand nombre de malades (de plus de 70 ans) ont été admis dans les salles de médecine, où leur grand âge a certes contribué à augmenter la mortalité.

References

1. SLOMAN G: Changing concepts in the care of patients with acute myocardial infarction. *Med J Aust* 1: 1157, 1969
2. WAHLBERG F: A study of acute myocardial infarction at the Seraphimer Hospital during 1950-1959. *Am Heart J* 65: 749-757, 1963
3. ROCKWELL MA: A summary of coronary care-unit literature. Rand Corporation, Memorandum RM—5944 RC April 1969
4. LOWN B, KOSOWSKY B, KLEIN M et al: Pathogenesis, prevention and treatment of arrhythmias in myocardial infarction. *Circulation* 29 (supp 4); 261, 1969
5. MACMILLAN RL, BROWN KW, PECKMAN GB, et al: Changing perspectives in coronary care. *Am J Cardiol* 20: 451, 1967
6. DHURANDHAR RW, MACMILLAN RL, BROWN, KW, et al: Primary ventricular fibrillation complicating acute myocardial infarction. *Am J Cardiol* 27: 347, 1971

Continued from page 1051

References

1. LOW JA, BOSTON RW, CERVENKO FW: The clinical classification of perinatal wastage. *Can Med Assoc J* 102: 365, 1970
2. GRUENWALD P: Growth of the human fetus 1. Normal growths and its variations. *Am J Obstet Gynecol* 94: 1112, 1966
3. *Idem:* Perinatal death of full-sized and full term infants. *Am J Obstet Gynecol* 107: 1022, 1970