The Relationship Between Family Functioning, Life Events, Family Structure, and the Outcome of Pregnancy

Christian N. Ramsey, Jr., MD, Troy D. Abell, PhD, MPH, and Lisa C. Baker, PhD
Oklahoma City, Oklahoma

A prospective study was undertaken to evaluate the relationship of family functioning, family structure, and life events with pregnancy outcome. Family functioning was assessed utilizing the Family Adaptability and Cohesion Evaluation Scales (FACES) and the Family APGAR. One hundred twenty-five infants were delivered with a mean birth weight of 3,283 g and a mean gestational age of 281.2 days. Listwise deletion of missing data reduced the study sample to 102 mother-infant pairs with no bias in the dependent or predetermined variables in the subsample. Birth weight was regressed on medical, anthropometric, risk-behavior, sociodemographic, and life-events variables, which together explained 42 percent of the variance. Family functioning was found to contribute an additional 7 percent of the variance (adjusted $R^2=0.49$). Family structure accounted for 4.5 percent of the variance in birth weight, and life events added 5 percent. As a complementary analysis, infant birth weight was regressed on the medical, anthropometric, sociodemographic, risk-behavior, and life-events variables, and the residuals from this equation were then regressed on the measures of family functioning. Again, abnormal family functioning proved to be a powerful and significant contributor to the explained variance.

The World Health Organization has declared low birth weight "the single most important determinant of the chances of the newborn to survive and to experience healthy growth and development,"[1] The association of low birth weight with mortality,[1] congenital malformations,[2] mental retardation,[3] and other physical and neurological impairments[4] is well established.

A relatively large number of anthropometric, medical, behavioral, and sociodemographic variables have been identified as risk factors for low birth weight: maternal age[5]; maternal height, weight, and weight gain[6]; parity[7]; menstrual history and infertility[8]; prior pregnancy history[9]; maternal health[5]; prenatal care[10]; ethnicity[11]; socioeconomic status[11]; smoking[12]; consumption of alcohol and other drugs[13,14]; and marital status.[15] While these known determinants account for a substantial part of the variance in birth weight, there is still a significant proportion of unexplained variance.

Pregnancy being a complex biopsychosocial process, researchers have turned to psychosocial factors, including behavior, perception, and attitudes, as a logical place to improve their ability to predict adverse outcome. Maternal anxiety,[16] pregnancy planning,[17] and stress or social support[18,19] have been shown to be related to the outcome of pregnancy. For example, in a study of wives of enlisted men, Nuckolls and colleagues[20] showed that women with low psychosocial assets and high life-event scores experienced more complications of pregnancy than women without this combination. Though these studies have identified variables that increase the ability to predict pregnancy outcome, they have also just begun to suggest a theoretical understanding of pregnancy as a biopsychosocial process. The tradition that addresses this issue most directly is the stress and social support literature. Cassel's conceptualization,[21] representative of this line of thinking, views stress as a direct negative influence on health, and this effect may be "buffered" by social support. Recognition of relationships, events, and feel-

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From the Department of Family Medicine, Health Sciences Center, University of Oklahoma, Oklahoma City, Oklahoma. Requests for reprints should be addressed to Dr. Christian N. Ramsey, Jr., Department of Family Medicine, 800 NE 15th, Suite 503, PO Box 26901, Oklahoma City, OK 73910.

ings as an integral part of an individual's health and illness opens the door to giving more attention to the social context within which a person lives.

The structure and functioning of the family system are the primary context for the psychosocial variables mentioned above. Family functioning has been shown to be related to depression[22] and infection,[23] and is a principal form of intimate social support in society. However, the presence of a family does more than simply provide positive support for its members. A number of studies have shown a relationship between family functioning and biologic processes,[24] illness,[25] and health care behaviors.[26] Results show that family relationships can have a positive or negative influence on health, and, in fact, it is probable that the complexity of family relationships may allow numerous types of biologic interactions that could influence the outcome of pregnancy.

A number of theories of family functioning have been used in relating family systems to biologic systems, including stress and adaptation theories,[27] developmental theory,[28] constructivist theory,[29] and such family typologies as the Beavers' model[30] and the Circumplex Model of Marital and Family Systems.[31] The Circumplex Model proposed by Olson and colleagues is particularly useful as a research model because it synthesizes the work of a number of investigators and has been operationalized into a self-report questionnaire.

The Circumplex Model posits that cohesion and adaptability are two of the most important dimensions of family systems. Cohesion is defined as "the emotional bonding that family members have towards one another," while adaptability is defined as "the ability of a marital or family system to change its power structure, role relationships, and relationship rules in response to situational and developmental stress."[31] These two dimensions are hypothesized to be related curvilinearally to family health; that is, the extremes of cohesion—enmeshment and disengagement—are theorized to be unhealthy, while the midrange is thought to be healthy. The same is hypothesized for adaptability, with the extreme ends of the continuum being labeled as rigid and chaotic.

In this study it is hypothesized that extremely low or extremely high cohesion or adaptability (as reported by the mother) will be significantly related to low infant birth weight. Furthermore, it is possible that each end of both continua may have distinct interactions with other predictor variables and with birth weight; that is, it may be more productive to treat the four extremes as separate variables rather than simply to test for the effects of extreme vs moderate responses. For example, it is possible that enmeshed families play a different role in influencing fetal growth than do disengaged families, and the collapsing of these two dysfunctional patterns together in the analysis might cloud the issues and diminish the extent to which their unique contributions can be discerned.

This study was undertaken to estimate the contribution of family structure and function to infant birth weight after adjusting for known medical, sociodemographic, anthropometric, and behavioral determinants.

METHODS

SUBJECTS AND PROCEDURE

One hundred thirty-two patients were recruited at their initial pregnancy visit at the Family Practice Center in Waco, Texas, or at the McLennan County Health Department, Waco, Texas, from May 1 through December 31, 1981. The study sample cannot be considered a random sample of the Waco Family Practice Residency Program's obstetric patient population, but the average levels for infant birth weight and gestational age (Table 1) are comparable to large studies focusing upon low-income mothers birthing on a public service.[6] Description of the mother-infant pairs in terms of anthropometric, sociodemographic, behavioral, and medical factors (Table 1) provides a foundation for decisions concerning generalizability. Study data were collected on three occasions: (1) during the initial prenatal visit, (2) during subsequent prenatal visits, and (3) during hospitalization for delivery.

At the initial visit comprehensive data were collected pertaining to medical history and sociodemographic status. The usual prenatal physical examination and laboratory data were also collected at this time. Two instruments were administered to measure family functioning: FACES[32] and the Family APGAR.[33] FACES is the acronym for Family Adaptability and Cohesion Evaluation Scales and is a written inventory of the subject's view of the family according to the Circumplex Model described above. The Family APGAR is a five-item questionnaire that asks for the respondent's satisfaction with her family's adaptability, partnership, growth, affection, and resolve. Each item is rated on a five-response scale.

Anthropometric measurements and routine laboratory work were carried out at each subsequent prenatal visit. At the time of delivery, information was collected as to life events before and during pregnancy using the Schedule of Recent Events (SRE) of Holmes and Rahe,[34] and anthropometric measurements of the infant were obtained.

STATISTICAL ANALYSIS

Infant birth weight was regressed on known medical, sociodemographic, and anthropometric determinants. Hierarchical inclusion of variables was used, as opposed to stepwise inclusion; that is, a model based on prior research and theoretical ordering of variables was built rather than using the combination of variables that account for the highest level of variance.

TABLE 1. BASIC CHARACTERISTICS OF SAMPLES

	Original Sample			Listwise Study Sample (n=102)	
	No.	Mean	Standard Deviation	Mean	Standard Deviation
Infant's birth weight (oz)	125	115.66	17.96	115.43	18.06
Gestational age (d)	125	281.17	21.83	280.46	22.85
Birth weight/gestational age ratio	125	0.41	0.06	0.41	0.06
Gender (male=1)	125	0.48	0.50	0.50	0.50
Emergency cesarean section	125	0.13	0.34	0.14	0.35
Maternal age (m)	121	265.59	53.41	256.85	52.01
Parity	118	1.02	1.34	0.99	1.35
Prepregnancy weight (lb)	118	132.21	30.07	133.07	30.38
Weight gain (lb)	117	25.76	13.50	25.43	13.20
Maternal height (in)	117	63.50	2.85	63.55	2.87
Highest diastolic blood pressure	125	73.11	8.79	72.40	8.34
Uterine bleeding third trimester	125	0.10	0.31	0.11	0.31
Maternal smoking	125	0.27	0.45	0.32	0.47
Number of prenatal visits	125	6.10	2.96	6.24	2.99
Hispanic	116	0.27	0.44	0.28	0.45
Homeowner	120	0.23	0.43	0.26	0.44
Married	125	0.51	0.50	0.52	0.50
Living alone	124	0.12	0.33	0.13	0.34
Planned pregnancy	124	0.24	0.43	0.23	0.43
Family stress before pregnancy	116	1.07	1.50	0.82	1.02
Family stress during pregnancy	120	1.60	1.66	1.53	1.51
Money-related stress before pregnancy	125	0.20	0.48	0.18	0.46
Enmeshed family	122	37.69	6.19	37.56	6.35
Disengaged family	123	37.59	7.76	37.10	7.27
Rigid family	122	27.41	5.23	27.24	5.38
Chaotic family	122	27.41	6.90	30.68	6.85

Once these known determinants were included in the model, then measures of family structure, stress, and family functioning were added. Thus, the final model represents the effect of family structure, stress, and family functioning on birth weight, while first controlling for the effects of known medical, sociodemographic, anthropometric, and behavioral determinants. Also, as a focused analysis of the effects of family functioning, birth weight was regressed on the 12 determinant variables excluding family functioning, and the residuals from this equation were regressed on family functioning.

The two curvilinear scales in FACES, adaptability and cohesion, were transformed into four linear scales: enmeshed, disengaged, rigid, and chaotic.[35] The total number of life events on the Schedule of Recent Events were grouped according to content into the following categories: family, money, lifestyle, and employment.[35]

RESULTS

While information was obtained on 132 mother-infant pairs, seven pairs were extremely atypical regarding age of the mother, weight or height of the mother, or birth weight of the infant (including one stillborn); these seven outliers were excluded from the analysis. Additionally, listwise deletion of subjects due to missing data on any one variable reduced the sample from 125 to 102. It is assumed that the distribution of missing variables is normally distributed; a comparison of the variables in the original and revised samples (Table 1) reveals no detectable bias in the final sample with regard to the dependent variable itself or to the variables determinant of birth weight.

Given that infant birth weight is highly dependent upon length of gestation, several alternatives exist for modeling the relationship between birth weight and gestational age. For this analysis birth weight was chosen as the dependent variable with gestational age as the first predetermined variable in the regressing equation. Table 2 shows the regression model. Variables were included if they were theoretically crucial and added approximately 0.5 percent in additional explained variance, regardless of whether they were statistically significant at the 0.05 level.

As can be seen, birth weight is positively associated with gestational age, emergency cesarean section, maternal age less than or equal to 30 years, parity, diastolic blood pressure, lack of uterine bleeding,

TABLE 2. DETERMINANTS OF INFANT BIRTH WEIGHT

	B (regression coefficients)	R^2	Adjusted R^2
Intercept	(84.797)		
Gestational age	0.273*	0.096	0.087
Emergency cesarean section	10.859**	0.134	0.117
Maternal age > 30 years	−16.590***	0.135	0.108
Parity	3.497**	0.160	0.125
Maternal height	−1.045	0.203	0.162
Highest diastolic blood pressure	0.654*	0.288	0.243
Third trimester bleeding	−16.948*	0.340	0.291
Maternal smoking	−4.424	0.358	0.303
Hispanic	5.891	0.392	0.332
Married	8.662**	0.439	0.378
Living alone	−6.611	0.444	0.376
Money-related stress before pregnancy	−11.011*	0.493	0.425
Enmeshed family	−0.828*	0.560	0.495

*$P < .001$
**$P < .01$
***$P < .05$
Dependent Variable: Infant Birth Weight (oz) n=102

nonsmokers, Hispanic ethnicity, and marital status.

Race is operationalized as a binary variable indicating the presence or absence of Hispanic ethnicity. In this triethnic patient population, it was the Hispanic mothers who delivered heavier infants. Black mothers and white mothers differed little in the birth weight of their infants; consequently, inclusion of a binary variable representing black or white ethnicity resulted in no additional contribution to explained variance.

It should be noted that the relationship between infant birth weight and maternal height is atypical. Hispanic women were shorter, gained no more weight than their white or black counterparts, but still gave birth to larger infants. Consequently, maternal height is negatively correlated (although nonsignificantly) with infant birth weight, while prepregnancy weight and maternal weight gain make no significant additions to the explained variance.

Socioeconomic status was operationalized in four ways: educational attainment, the Duncan socioeconomic index,[36] the Nam socioeconomic index,[37] and home ownership. None of these approaches made significant contributions to the explained variance in outcome. It is no surprise that socioeconomic status is not a powerful determinant, since the participants were all on the lower end of any socioeconomic continuum. Two other factors related to socioeconomic status failed to add to the explained variance in infant birth weight: number of prenatal visits and participation in a federal supplemental feeding program (WIC).

Family structure was operationalized into three categories: (1) women living with their husbands, (2) women living within extended families but not with their husbands, and (3) women living alone or with only their children. The second group (extended family with no husband) was the reference category, with (1) "married" and (3) "alone" entered as the two binary indicator variables. Thus, the married women living with their husbands were delivered of infants weighing, on the average, 8.6 ounces more than those women living without their husbands in an extended family. Those women living alone were delivered of infants weighing, on the average, 6.6 ounces less than the women living in extended families with no husband and 15.2 ounces less than women living with their husbands.

As can be seen, money-related stress and family enmeshment are powerful determinants of birth weight, contributing 5 percent and 7 percent, respectively. Total life events (SRE) for these two time periods account for 1.2 percent of the variance; however, prediction is improved by entering the events related only to financial issues. Similarly, prediction is greatly improved by using the transformed enmeshment scale as a single predictor. (The variance accounted for by the linear scores of adaptability and cohesion was only 1 percent.) The transformed disengagement scale—the opposite end of the cohesion continuum from enmeshment—accounts for 1.0 percent of the variance in infant birth weight when enmeshment is not entered into the regression model. When both variables enter the model, the disengagement scale loses statistical significance. Thus, as predicted by theory, infants delivered to mothers perceiving their families as disengaged *or* as enmeshed weighed less than those from moderately cohesive families; enmeshment, in this study, was the more powerful determinant. Neither of the adaptability subscales (rigid and chaotic) added significantly to the explained variance in

birth weight. Substitution of the Family APGAR in place of the enmeshment scale from FACES results in Family APGAR being a statistically significant contributor to infant birth weight, but less so than enmeshment (1 percent additional variance vs 7 percent).

In summary, the variables assessing family structure, changes in life events, and family functioning account for approximately one third of the explained variance in infant birth weight after adjusting for other known determinants.

By using only statistically significant variables ($P < .05$), the number of predictor variables is reduced from 13 to nine, with a resultant drop in explained variance (adjusted R^2) of 8 percent (from 0.495 to 0.415). In this reduced model, money-related stress and family enmeshment continue to be powerful determinants and together add 9 percent in explaining the variance in infant birth weight. It would be premature to suggest that these nine variables would be the best determinants in another sample, given the sample size of 102; thus, the larger model (including theoretically sound, but statistically nonsignificant variables) should be retained as the basis for further research.

Because of the possibility that the powerful contribution of family functioning might be an artifact of the diminishing degrees of freedom in the models (a relatively large number of determinants for a sample size of 102), birth weight was regressed on the 12 variables excluding family functioning, and the residuals from this equation were then regressed on family functioning. The results of this analysis are shown in Table 3. The model presented in Table 2 was used in creating the residuals as a conservative measure: any variance contributed by a theoretically sound, even if statistically nonsignificant, determinant of birth weight was "controlled" before family functioning variables were given an opportunity to contribute to the explained variance. As can be seen in Table 3, even after all other variables that contribute to birth weight are held constant, family enmeshment makes a powerful contribution (an additional 9 percent explained variance).

TABLE 3. BIRTH WEIGHT RESIDUALS REGRESSED ON FAMILY FUNCTIONING

	B (regression coefficient)	R^2	Adjusted R^2
Intercept	(24.362)		
Enmeshed family	−0.649*	0.103	0.094

*$P < .01$
Dependent Variable: Infant Birth Weight (oz) n=102

DISCUSSION

The data from this study are congruent with the findings of other studies with regard to the contribution of such factors as maternal age, smoking, medical history, and parity. Taken together, these factors account for approximately one third of the total variance in birth weight. The failure of gender, maternal height, and prepregnancy habitus (weight-height ratio) to make significant contributions to the variance in infant birth weight is consistent with Miller's[38] recent finding on this topic. More important, the data show that family dysfunction, as reported by a pregnant mother, is a significant determinant of infant birth weight. The findings will be discussed from three viewpoints: (1) relation to family theory, (2) possible biologic mediators of family system functioning, and (3) clinical implications.

The data show that family structure and family functioning are both significantly and independently related to birth weight. From the standpoint of family structure, the woman who lives alone is at risk for having a smaller baby; living with her extended family improves the chances of having a heavier baby, but not so much as does living with her husband. The presence of family members may be helpful in facilitating better health practices, such as regular, planned meals and clinic visits, or it may play a part by emotionally easing the transition in the face of society's pressure to bear children into a home with a traditional family structure, particularly one in which the husband is present.

Along with the question of who lives in the home is the issue of what happens in the family. The data go beyond the concept of the family acting solely as a protector from stress; enmeshment, a particular type of family interaction, is a powerful determinant of birth weight independent of other determinants, including family structure and life events. The data point to a role in which the family acts as a stress producer instead of as a stress absorber. The common notion of the family as social support is called into question. Rather than the family failing to provide adequate support, the family's overinvolvement (enmeshment) may be interpreted as lack of privacy, autonomy, and psychological space to make room for the new member.

These data suggest that family functioning is having an effect on the fetus and that the child has become a member of the family system even before birth. Minuchin[39] theorizes that in some enmeshed families the sick child becomes the "symptom bearer" of the dysfunctional family system. In a similar way, a low birth weight baby may also be a symptom bearer of an enmeshed family system.

Several pathways may explain the biologic mechanisms that mediate family functioning and infant birth weight. At least two areas of research may be relevant: work on nutrition and the maternal immune system. Metcoff and others[6] have shown that maternal nutri-

tion is a predictor of birth weight. Since eating habits are an integral part of family routines and may be subject to dysfunctional family patterns, it is reasonable to hypothesize that at least a part of the family's contribution to infant birth weight may be through its influence on the mother's nutrition. A striking example of the family system's relationship to nutritional disturbance is seen in the work by Minuchin and colleagues[40] that links family interaction patterns, including enmeshment, with anorexia nervosa.

Studies concerning aspects of family functioning and the immune system suggest that the family system may act to compromise immune protection from infectious agents.[23,41] Since there is considerable evidence that intrauterine infections may account for some of the variance in prematurity and intrauterine growth regardation,[42-45] it is possible that the dysfunctional family system may interact with the maternal immune system to compromise immune states and allow for intrauterine infection. High levels of secretory immunoglobin A (IgA) in cervical mucus are necessary to protect the entrance to the uterine cavity from infectious agents, and normally high secretory IgA levels are lowered in certain intrauterine infections.[46,47] Jemmott et al[48] have shown that stress can lower salivary IgA levels. It seems possible, therefore, that dysfunctional families may play a part in the risk of low birth weight by contributing to compromised immunologic functioning through a lowering of cervical secretory IgA levels, which allows for intrauterine infection and causes fetal growth retardation. Such a hypothesis remains to be tested.

From the standpoint of clinical implications, a major goal of this research is to generate information that can improve the outcome of pregnancy. Recently, Sexton and Hebel[49] have reported on an increase in birth weight following the institution of a smoking cessation program for mothers. Herron and colleagues[50] have presented initial results of an intervention with mothers that looks promising in lowering the incidence of preterm deliveries. In a similar manner, as understanding of the contribution of the family system to pregnancy outcome increases, it should be possible to intervene early in pregnancy to modify risk factors associated with family systems.

Much work is needed to improve the understanding of family variables and biomedical determinants of birth weight and to delineate the specific biologic pathways by which family interaction affects birth weight. This study reveals family structure and function to be powerfully involved in the complex interactions of biologic, social, and behavioral factors affecting infant birth weight.

References

1. World Health Organization: The incidence of low birth weight: A critical review of available information. Geneva, Switzerland, WHO, Division of Family Health, 1980
2. Taffel S: Congenital anomalies and birth injuries among live births. United States. 1973-74. In National Center for Health Statistics (Hyattsville, Md): Vital and Health Statistics, series 21, No. 31. DHEW publication No. (PHS) 79-1909. Government Printing Office, 1978
3. Churchill JA, Neff JW, Caldwell DF, et al: Birth weight and intelligence. Obstet Gynecol 1966; 28:425-429
4. Lubchenco LO, Horner FA, Reed LH: Sequelae of premature birth. Am J Dis Child 1963; 106:101-115
5. Miller HC, Merritt TA: Fetal Growth in Humans. Chicago, Year Book Medical, 1979
6. Metcoff J, Costiloe P, Crosby W, et al: Maternal nutrition and fetal outcome. Am J Clin Nutr 1981; 34:708-721
7. Kaminski M, Goujard J, Rouquette-Rumeau C: Prediction of low birth weight and prematurity by a multiple regression analysis with maternal characteristics known since the beginning of pregnancy. Int J Epidemiol 1973; 2:195-204
8. Berkowitz GS: An epidemiologic study of preterm delivery. Am J Epidemiol 1981; 113:81-92
9. Butler NR, Alberman EA: Perinatal Problems: The Second Report of the 1958 British Perinatal Mortality Survey. Edinburgh, Churchill Livingstone, 1969
10. Gortmaker SL: The effects of prenatal care upon the health of the newborn. Am J Public Health 1979; 69:653-660
11. Bross DS, Shapiro S: Direct and indirect associations of five factors with infant mortality. Am J Epidemiol 1982; 115:78-91
12. Meyer MB, Tonascia JA: Maternal smoking, pregnancy complications, and perinatal mortality. Am J Obstet Gynecol 1977; 128:494-502
13. Little RE: Moderate alcohol use during pregnancy and decreased infant birth weight. Am J Public Health 1977; 67:1154-1156
14. Zelson C, Rubio E, Wasserman E: Neonatal narcotic addiction: 10 year observation. Pediatrics 1971; 48:178-189
15. Jansson I: Etiological factors in prematurity. Acta Obstet Gynecol Scand 1966; 45:579-600
16. Crandon AJ: Maternal anxiety and neonatal well being. J Psychosom Res 1979; 23:113-115
17. Laukaran VH, Van Den Berg BJ: The relationship of maternal attitude to pregnancy outcomes and obstetric complication. Am J Obstet Gynecol 1979; 136:34
18. Norbeck JS, Tilden VP: Life stress; social support, and emotional disequilibrium in complications of pregnancy: A prospective, multivariate study. J Health Soc Behav 1983; 24:30-46
19. Smilkstein G, Helsper-Lucas A, Ashworth C, et al: Prediction of pregnancy complications: An application of the biopsychosocial model. Soc Sci Med 1984; 18:315-321
20. Nuckolls VK, Cassel J, Caplan VH: Psychosocial assets, life crisis, and the prognosis of pregnancy. Am J Epidemiol 1972; 95:431
21. Cassel J: The contribution of the social environment to host resistance. Am J Epidemiol 1976; 104:107-123
22. Widmer RB, Cadoret RJ: Depression in family practice: Changes in patterns of patient visits and complaints during subsequent developing depression. J Fam Pract 1979; 9:1017-1021
23. Meyer RJ, Haggerty RJ: Streptococcal infections in families: Factors altering individual susceptibility. Pediatrics 1962; 29:539-549
24. Schleifer SJ, Keller SE, Camerino M, et al: Suppression of lymphocyte stimulation following bereavement. JAMA 1983; 250:374-377
25. Neser WB, Tyroler HA, Cassel JC: Social disorganization and stroke mortality in the black population of North Carolina. Am J Epidemiol 1971; 93:166-175
26. Pratt L: Family Structure and Effective Health Behavior: The Energized Family. Boston, Houghton Mifflin, 1976
27. McCubbin HI, Patterson JM: The family stress process: The double ABCX model of family adjustment and adaptation. In

McCubbin H, Sussman M, Patterson JM (eds): Advances and Developments in Family Stress Theory and Research. New York, Haworth, 1983
28. Carter E, McGoldrick M (eds): The Family Life Cycle: A Framework for Family Therapy. New York, Gardner Press, 1980
29. Reiss D: The Family's Construction of Reality. Cambridge, Mass, Harvard University Press, 1981
30. Beavers WR: A systems model of family for family therapists. J Marital Fam Ther 1981; 299-307
31. Olson DH, Sprenkle DH, Russell CS: Circumplex model of marital and family systems: 1. Cohesion and adaptability dimensions, family types, and clinical applications. Fam Proc 1979; 18:3-28
32. Olson DH, Bell R, Portner J: FACES. St. Paul, Department of Family of Social Science, University of Minnesota, 1978, privately published
33. Smilkstein G: The family APGAR: A proposal for family function test and its use by physicians. J Fam Pract 1978; 6:1231-1239
34. Holmes TH, Rahe RH: The social readjustment rating scale. J Psychosom Res 1967; 11:213-218
35. Baker L, Ramsey C, Abell T: Measuring family functioning: Refining the approach in a study of pregnancy outcome. Paper presented at the 11th Annual Meeting of the North American Primary Care Research Group, Banff, Canada, April 18-20, 1983
36. Reiss AJ, Duncan OD, Hatt PK, North CC: Occupational and social status. Glencoe, NY, The Free Press, 1901
37. Nam CB: Occupational status scores: Stability and change. Proc Am Stat Assoc 1975; 570-575
38. Miller HC: A model for studying the pathogenesis and evidence of low birth weight infants. Am J Dis Child 1983; 137:323-327
39. Minuchin S: Psychosomatic Families. Cambridge, Mass, Harvard University Press, 1978
40. Minuchin S, Baker L, Rosman BL, et al: A conceptual model of psychosomatic illness in children. Arch Gen Psychiatry 1975; 32:1031-1038
41. Ramsey CN, Baker L, Campbell J: Family functioning and stress as predictors of influenza infection, abstracted. In Proceedings of the 11th Annual Meeting of the North American Primary Care Research Group, Banff, Alberta, April 17-20, 1983. Richmond, Va, NAPCRG, 1983
42. Kass EH: Pregnancy, pyelonephritis and prematurity. Clin Obstet Gynecol 1970; 13:239-254
43. Naeye RL: Causes of the excessive rates of perinatal mortality and prematurity in pregnancies complicated by maternal urinary tract infections. N Engl J Med 1979; 300:819-823
44. Regan JA, Chao S, James LS: Premature rupture of membranes, preterm delivery, and group B streptococcal colonization of mothers. Am J Obstet Gynecol 1981; 141:184-186
45. Sweet RL, Schachter J, Landers DV: Chlamydial infections in obstetrics and gynecology. Clin Obstet Gynecol 1983; 26:143
46. Brunham RC, Martin DH, Kuo C-C, et al: Cellular immune response during uncomplicated genital infection with Chlamydia trachomatis in humans. Infect Immun 1981; 34:98-104
47. Brunham RC, Kuo C-C, Cles L, et al: Correlation of host immune response with quantitative recovery of Chlamydia trachomatis from the human endocervix. Infect Immun 1983; 39:1491-1494
48. Jemmott JB III, Borysenko M, Chapman R, et al: Academic stress, power motivation, and decrease in secretion rate of salivary secretory immunoglobulin A. Lancet 1983; 1:1400-1402
49. Sexton M, Hebel JR: A clinical trial of change in maternal smoking and its effect on birth weight. JAMA 1984; 251:911-915
50. Herron MA, Katz M, Creasy RK: Evaluation of preterm birth prevention program: Preliminary report. Obstet Gynecol 1982; 59:452-456