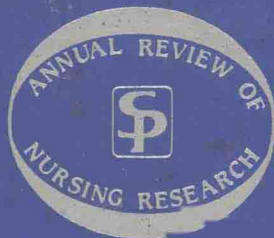


ANNUAL REVIEW OF NURSING RESEARCH

Volume 4, 1986



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Harriet H. Werley, Ph.D.
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Editors



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Preface

This is the fourth volume in the *Annual Review of Nursing Research (ARNR)* series. Volume 1 appeared in December 1983, Volume 2 in October 1984, and Volume 3 in May 1985; this volume in early 1986. Volumes 5 through 7 are currently in the planning and production stages, and soon we will be initiating discussions with potential authors for Volume 8.

We have received a very positive response from the scientific community regarding the launching of this landmark series. We have introduced our work at various forums through presentations, symposia, and posters. Many of our colleagues have indicated that the *ARNR* series is an important addition to their libraries.

The present volume is the first in which we have repeated chapter themes from previous volumes. As they did in Volume 1, the chapters under Nursing Practice for Volume 4 pertain to human development along the life span. Volume 2 chapters in this area were focused on the family and Volume 3 chapters on the community. Volume 5 chapters will be focused on human responses to health problems, and Volume 6 on nursing interventions related to patient or client responses to health.

As in previous volumes, research reviewed for Volume 4 follows the established format of five major parts: Nursing Practice, Nursing Care Delivery, Nursing Education, the Profession of Nursing, and Other Research. In the nursing practice area, with a focus on human development, Regina Placzek Lederman examines the fetal and newborn dimensions of maternal anxiety in pregnancy, Juanita W. Fleming reviews research on preschool children, Ann M. Voda and Theresa George examine research on menopause, Mary Adams reviews research on aging, and Alice Sterner Demi and Margaret Shandor Miles examine grief research. In the area of nursing care delivery Phyllis Giovannetti deals with evaluation of primary nursing and Joyce E. Thompson presents midwifery research from a care-delivery perspective. In the section on nursing education, Kathleen G. Andreoli and Leigh Anne Musser examine the area of faculty productivity, Carrie B. Lenburg reviews research on nontraditional nursing education programs, and Betty L. Chang examines the area of computer-aided instruction in nursing education. Research on the profession of nursing includes a chapter by Irene Sabelberg Palmer on nursing's heritage. In the area of other research, we have continued the efforts to include a chapter on nursing research in another

country, this time on nursing education research in Canada, by Margaret M. Allemang and Margaret C. Cahoon.

Another significant change has occurred with this volume: We have added a third editor, Roma Lee Taunton, who has worked diligently with us on the previous volumes. With Volume 4 she has assumed direct editorial responsibility, and we are pleased to welcome her to our team.

We acknowledge most gratefully the work of the authors, the advice and involvement of the Advisory Board members, the critiques of anonymous reviewers, and the editorial and clerical assistance provided by Lois Schweitzer at Case Western Reserve University, Margaret M. Sprung at the University of Wisconsin-Milwaukee, and Beverly Warren at the University of Kansas. Further, we are particularly indebted to Nikki Polis, Ph.D. student at Case Western Reserve University, for her assistance with the final editing of this volume.

We welcome readers' comments and suggestions for shaping the upcoming volumes, including identifying potential contributors. Authors should be recognized authorities in their area, having completed significant research. Please let us know your interests.

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Forthcoming

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Part I

Research on Nursing Practice

Chapter 1

Maternal Anxiety in Pregnancy: Relationship to Fetal and Newborn Health Status

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This chapter includes a review of the literature on maternal anxiety and psychosocial stressors that may have an impact on delivery outcomes and the future health and development of the newborn infant. Of particular interest are those fetal and newborn conditions that are not explained adequately by physical causes or disease processes. This review is focused on the psychosocial conditions, relationships, and developmental conflicts that give rise to maternal anxiety, which in turn may have an adverse physiological effect on the intrauterine environment and on fetal and newborn health and development.

The nurse, by virtue of providing continuous and comprehensive psychosocial, physical, and physiological care throughout the maternity

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cycle, is in a unique position to recognize and assess maternal anxiety and to appreciate the impact of stress or anxiety on fetal and newborn development and on the sensitive, evolving maternal relationship to the fetus and the newborn. The objective of this review is to increase the nurse's awareness of conditions precipitating maternal anxiety, of maternal and fetal responses to anxiety, and of research designs and issues pertinent to future nursing investigation in this emerging area of psychophysiological research. The literature chosen for review was obtained from computerized searches of medical, psychological, and sociological indexes, as well as the author's own reference collection of the past several years. It was assumed that the reader has knowledge of common obstetrical terms and a beginning acquaintance with human and animal research on the psychophysiology of reproductive events.

DEFINITION OF TERMS

The terms *anxiety* and *stress* often are used interchangeably. In order to provide a conceptual framework, the following operational definitions are stated. Spielberger, Gorsuch, and Lushene (1970) defined *state anxiety* as a transitory emotional state or condition of the human organism that is characterized by subjective, consciously perceived feelings of tension and apprehension and heightened autonomic nervous system activity. *Trait anxiety* is used to refer to relatively stable individual differences in general anxiety proneness, that is, to differences between people in the tendency to respond, with elevations of state anxiety intensity, to situations perceived as threatening. Cox (1978) defined *stress* as a perceptual phenomenon arising from a comparison between the demand on the person and his ability to cope; an imbalance in this mechanism gives rise to the experience of stress and thus to the stress response.

The definitions of anxiety and stress are similar in a number of ways, particularly in giving recognition to individual behavioral responses and differences in response. While the definition of anxiety is focused on intrapersonal responses in any anxiety-producing situation, the definition of stress appears to be related to discrepancies between individual aspirations and actual events, as well as the adequacy of coping responses. These differences in definition are perhaps relative rather than absolute, however, and may not be consistently evident in the research literature on anxiety. Definitions of anxiety and stress vary among different researchers and usually are defined by the instruments and methods of the specific research project.

COMPLICATIONS OF THE FETUS AND NEWBORN

A few animal and human studies provided some evidence for an important relationship between maternal anxiety and fetal distress or asphyxia. Myers (1975) observed substantial fetal bradycardia and hypotension in primates in response to maternal psychological stress of both contrived (clapping hands, shouting) or unintended stress (entry into darkened room by the experimenter). Myers suggested that maternal stress also may aggravate further an already hypoxic fetus. Morishima, Pedersen, and Finster (1978) also reported a significant decrease in fetal heart rate, arterial oxygenation, and uterine blood flow in primate fetuses with induced maternal stress, as well as late fetal heart rate decelerations in some fetuses with an existing asphyxia. All subjects in the Morishima et al. study received general anesthesia, so questions remain concerning the possible confounding effects of anesthesia on the observed response.

Goodlin and Lowe (1974) reported abnormalities in fetal heart rate patterns during human labor that were associated with stress of the gravida. Engstrom, af Geijerstam, Holmberg, and Uhrus (1964) found that fetal asphyxia in labor occurred in 33% of the sample when gravidas reported five or more negative factors associated with pregnancy. Lederman, Lederman, Work, and McCann (1981) reported that maternal epinephrine ($r = .37, p < .05$) and anxiety ($r = .33, p < .05$) measured at the onset of active labor were correlated significantly with subsequent fetal heart rate patterns (of normality or deceleration) in active phase labor. The fetal heart patterns also correlated significantly with Apgar scores at 1 and 5 minutes ($r = -.50, p < .01$; $r = -.47, p < .01$), respectively.

In a prospective study of 504 gravid subjects, Crawford (1968) found the newborn Apgar rating also was associated significantly with a measure of maternal emotional tension in labor ($p < .001$). Crandon (1979a, 1979b) found that the mean Apgar score at 5 minutes was significantly lower ($M = 5.83$) in gravidas scoring high on the IPAT anxiety scale (Cattell & Scheier, 1963) than those with lower IPAT scores ($M = .9$). Erickson (1976a, 1976b) also reported that psychological stress during pregnancy was associated with low Apgar scores.

The mechanisms leading to fetal heart rate deceleration in labor were reconstructed as follows: Maternal anxiety, associated with increased sympathetic nervous system activity and catecholamine release from the adrenal medulla and nerve terminals, resulted in the constriction of uterine blood vessels with a concomitant decrease in uterine blood flow, placental intervillous space perfusion, and fetal oxygenation. Studies with healthy primates, dogs, rabbits, and ewes showed that reduced uterine blood flow and fetal

distress were related to catecholamine effects (Adams, Assali, Cushman, & Westersten, 1961; Adamsons, Mueller-Heubach, & Myers, 1971; Ahlquist & Woodbury, 1947; Carter & Olin, 1972; Dalton, Dawes, & Patrick, 1977; Greiss, 1963; Greiss, Jr. & Gobble, 1969; Levinson & Shnider, 1979; Roman Ponce, Thatcher, Caton, Barron, & Wilcox, 1978; Rosenfeld, Barton, & Meschia, 1976; Svihovec, Stulc, & Rychter, 1978).

Several other investigators found relationships between prenatal stress and newborn complications in humans (Blau et al., 1963; Davids & DeVault, 1962; McDonald, Gynther, & Christakos, 1963; Stott, 1959). Physical environmental factors such as overcrowding, geographical moves during pregnancy, emotionally stressful factors involving disturbed personal relationships, and economic instability were among the stress factors cited as affecting human neonatal well-being (Downs, 1977; Rose, 1961). Downs (1977) found significant differences between stressed and nonstressed pregnant subjects and the incidence of neonatal pathological conditions such as prematurity, stillbirth, congenital malformations, and neonatal death.

In the realm of animal research, as in human studies, prenatal anxiety from various induced psychological and physiological stressors has been associated with fetal and newborn complications. In addition, deviations in animal infant behavior in areas of emotional responsiveness, reflex development, learning capacity, and sexual functions have been reported; these complications are discussed in the next section.

NEWBORN, INFANT, AND CHILDHOOD COMPLICATIONS ASSOCIATED WITH MATERNAL ANXIETY AND FETAL STRESS

Animal Studies

The question of the relationship between prenatal anxiety and neonatal development is one that has been investigated amply in animal research. Animal studies are highly relevant to the study of environment and behavior, even if they are not applicable and transferable immediately to the human condition. In particular, animal research offers advantages for controlling the stress environment that often are not possible in human research. For example, in animal research it is possible to study the effects of prenatal stress, while controlling for the possible continued effects of stress in the postnatal environment through the technique of cross-fostering, that is, distributing some offspring among other experimental and control mothers

while other offspring remain with their natural mothers. Thus, the influence of prenatal treatment can be detected apart from the influence of the postnatal environment. Such designs also allow for the control of genotype or genetic influences on behavior. Another considerable advantage is the opportunity to investigate relationships among reproductive organ activity, reproductive and sexual functioning, brain tissue physiology, and hormone concentrations in the brain and in plasma.

Reproductive functioning and sexual behavior in relation to prenatal stress have been studied extensively in animal research. Ward and Weisz (1980) measured plasma testosterone in the male rat fetuses of stressed and control mothers. The male offspring of both stressed and control mothers failed to show a surge of circulating testosterone on days 18 and 19 after conception, a period postulated to be critical in the development of the central nervous system and normal sexual behavior of the male rat. Dahlof, Hard, and Larsson (1977) exposed rats during the last trimester of pregnancy to crowded living conditions or to immobilization combined with intense illumination, all known stressors. In adulthood, the male offspring exposed to either condition of stress showed increased readiness toward feminine sexual behavior (lordosis), while no deficits were observed in masculine sexual behavior. The researchers concluded that perinatal stress adversely affects sexual differentiation by causing feminization, but not masculinization, of male offspring; other investigators have reported demasculinization (Herrenkohl & Whitney, 1976; Whitney & Herrenkohl, 1977).

Moyer, Herrenkohl, and Jacobowitz (1978) studied the influence of prenatal stress on catecholamine concentrations in discrete brain regions of offspring in adulthood, since relationships were reported in the literature among prenatal stress, brain catecholamines, and sexual response. At 90 days of age, the brains of sacrificed mice showed a reduction of norepinephrine in the medial preoptic nucleus and the median eminence, regions known to be target areas for gonadal steroids and to be involved with neuroendocrine control mechanisms, including gonadatropin release and sexual behaviors. The researchers suggested further that prenatal stress in rats may have resulted in hormonally induced permanent neurochemical changes that were responsible for the feminization of male offspring. The researchers speculated that prenatal stress could alter reproductive function and maternal behavior in female offspring as well.

While Beckhardt and Ward (1983) were unable to substantiate such a relationship, Herrenkohl (1979) found that female rats stressed prenatally with restraint and illumination experienced fewer conceptions, more spontaneous abortions and vaginal hemorrhaging, longer pregnancies, and fewer viable young than nonstressed rats. Offspring of stressed rats were lighter in weight and less likely to survive the neonatal period. Herrenkohl suggested that

prenatal stress might have influenced the balance of adrenal and gonadal hormones during a critical stage of fetal hypothalamic differentiation, thereby producing a number of reproductive dysfunctions later in female adulthood.

Thompson (1957) conducted many of the early studies on the influence of prenatal maternal anxiety on emotional behavior of offspring. High emotionality was indicated by high latency in response and low activity in the test condition (the time needed to reach food after 24 hours of food deprivation). Thompson stressed prenatal rats by blocking an expected escape route from a shuttle box. Postnatal effects were controlled by cross-fostering. Significant differences in activity and response latency were obtained at both 30 to 40 days (infancy) and 230 to 240 days (adulthood). The results indicated that maternal anxiety increased emotionality in offspring, that these effects were not related to the postnatal environment, and that the differences persisted into adulthood. These results were supported in subsequent experiments by other investigators (Ader & Belfer, 1962; Hockman, 1961; Morra, 1965). More recently, Holloway, Dollinger, and Denenberg (1980) convincingly demonstrated that an abnormal labor experience in rats was associated with inappropriate maternal nesting and lactational behavior and asynchronous maternal and newborn interaction, which threatened pup survival and long-term development. These researchers (Dollinger, Holloway, & Denenberg, 1980) also showed the considerable learning ability and behavioral modifiability of the newborn in the earliest hours after birth by examining progressive changes in maternal and newborn attachment and responses to deprivation.

Corson, Heseltine, and Smith (1970) found that 79-day-old rat pups of prenatally stressed females showed a decrease in learning ability when compared to those from nonstressed females, but these changes were not seen at 100 days of age. Similar results were reported in a study by Lyle, Johnson, Edwards, and Penny (1973) with guinea pigs, wherein the effects of stress at middle and late gestation resulted in reduced learning performance of offspring of stressed rats compared to controls. Chapman and Stern (1979), however, failed to produce significant results with rat offspring in open field and cage emergence tests, the traditional measures of emotionality. The researchers attributed this difference in findings to their control for litter effect by using a substantial number of litters and testing with only one or two offspring of each gender per litter.

Reviews of the literature on socioenvironmental factors affecting animal reproduction have been published by Rosenblatt (1967) and Archer and Blackman (1971). In their review about offspring behavior of prenatally stressed rats and mice, Archer and Blackman offered several relevant comments regarding research design, in addition to those offered by Chapman