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Lewis, Keeta DeStefano

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Prenatal Drug Exposure: Assessing Risk

by

Keeta De Stefano Lewis

DISSERTATION

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This work is dedicated to my mother, my children, and their children:

Kathleen DeStefano, Lisa, Art, and John;

DaNaii and Dante;

and in memory of Stretch Lewis.

Acknowledgments

My deepest appreciation is extended to the numerous people who gave me their time, talent, encouragement, trust, and support. Without these individuals my dissertation would have remained a dream and would not have come to fruition. I am indebted to the children and their families who gave me the opportunity to enter their homes and share their struggle to live a full life. Their arduous life journey has not only humbled my life, but has given me many reasons to strive to make a positive difference in their lives as well as the lives of others.

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Last, however not least, my mother who has always been proud of me, and who believes I can do anything I choose. Thanks, Mom, I love you.

Abstract

PRENATAL DRUG EXPOSURE: ASSESSING RISK

Keeta DeStefano Lewis, RN, Ph.D.

University of California, San Francisco, 1998

The purpose of this study was to further evaluate and refine the Lewis Neurobehavioral Assessment Scale (LNAS), a tool to identify neurobehavioral problems of infants with prenatal drug exposure (PDE). The first aim was to determine reliability of the LNAS through assessment of interrater agreement, internal consistency testing using Cronbach Coefficients, and test-retest reliability after a one-week interval. Aim 2 was to determine the construct and predictive validity of the LNAS. Construct validity testing assessed the measure's ability to differentiate infants with PDE from those without, as well as low risk from high risk infants. Predictive validity testing examined the tool's utility as a predictor of a) intent, clarity of cues, and responsiveness to a parent as well as b) early indicators of cognitive difficulties at six months of age.

A cross-sectional design was employed using a convenience sample of 80 newborns, including forty infants with PDE and 40 infants without PDE who were assessed and compared during their second week of life. The LNAS, Nursing Child Assessment Feeding Scale, and Parmelee Complications Scale were administered to all newborns. Sixteen of the newborns were reassessed at six months using the LNAS and the Mullen Scales of Early Development.

This study provided evidence for the overall validity and reliability of the LNAS in the early identification of neurobehavioral problems for PDE infants. The total LNAS score appears robust. Interrater reliability was excellent across the entire scale. Test-retest for short term stability was impressive as well as its long term stability at six months, with only one subscale changing significantly

over time. Three of the subscales show excellent validity. However, two subscales did not demonstrate adequate internal consistency, and both their construct and predictive validity were questionable.

The findings, although limited by sample size, contribute valuable data regarding neurobehavioral characteristics of PDE infants at birth and their implications for later development. The instrument has multidisciplinary application and strengthens nursing's contribution to the care and understanding of high risk infant populations.

Keeta DeStefano Lewis, RN, Ph.D.
Candidate

Sandra Weiss, RN, Ph.D., D.N.Sc., FAAN
Chairperson

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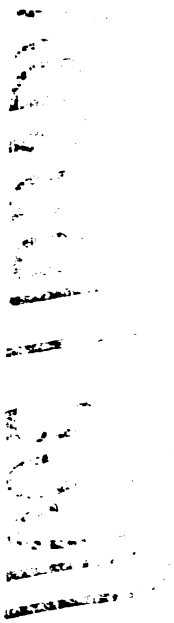


CHAPTER ONE

The Study Problem

Introduction

Infants with in-utero drug exposure continue to be a major concern to health and educational professionals around the world (International Network Parenthood-Drug Abuse, 1996). Prenatal drug exposure (PDE) can have a wide range of effects that are associated with infant development. The effects range from mild speech and language problems to severe malformations. Specific physical deficits may not be observed as frequently as was originally reported, but instead the infant's capacity for using motor, language, cognitive, sensory, and affective (attention, play) domains are compromised (Greenspan, 1991). The atypical and qualitative neurobehavioral differences observed in this population can interfere with normal infant development and healthy adaptation. These neurobehavioral differences may be indicators for later difficulties with learning skills and appropriate social behavior. How these atypical neurobehaviors are assessed in infancy is varied, inconsistent, and without appropriate standardized measures.

Purpose of the Study

The purpose of this research study was to determine the validity and reliability of the revised Lewis Neurobehavioral Assessment Scale (LNAS), with newborn infants known to have PDE. The LNAS is an instrument used to assess the neurobehavioral characteristics of infants (birth through 12 months) who have been exposed prenatally to the teratogenic effects of drugs such as cocaine, opiates, amphetamines, marijuana, phencyclidine (PCP), benzodiazephines and barbiturates. Preliminary reliability and content validity were determined in previous research and served as the basis for revisions of the measure. No comprehensive psychometric testing of the revised instrument has yet occurred. The LNAS can provide a highly sensitive developmental screening instrument

that is also cost efficient, relatively quick to administer, and easy for regular nursing staff and other professionals to master at high levels of interrater reliability. If this instrument is found to be valid and reliable, it can assist with early detection of risk, a determination crucial for the appropriate intervention, treatment, and follow-up care. There were two specific aims of this research, each of which had related research questions.

Aim 1

To determine the reliability of the LNAS in a sample of ethnically diverse newborn infants with prenatal drug exposure (PDE).

1.1 Does the LNAS show interrater reliability?

1.2 Is the LNAS internally consistent?

1.3 Does the LNAS have test-retest reliability?

Aim 2

To determine the construct and predictive validity of the LNAS in a sample of ethnically diverse newborn infants with PDE .

2.1 Does the LNAS differentiate infants with PDE from those without PDE?

2.2 Does the LNAS significantly differentiate low and high risk infants with PDE?

2.3 Does the LNAS predict an infant's clarity of cues and responsiveness to parent/caregiver during an infant feeding situation?

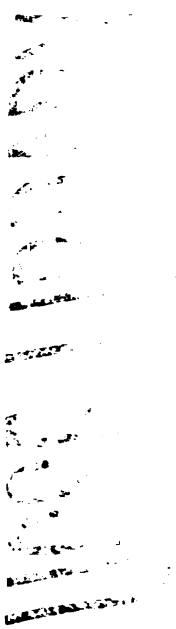
2.4 Does the LNAS predict early indicators of cognitive difficulties at six months of age?

Background and Significance

Prenatal drug exposure is a national health focus (U.S. Dept of Health & Human Services, 1990) as well as a Maternal Child nursing priority. Alcohol and other drug abuse have been frequently cited as primary factors contributing to the increase in child maltreatment (U.S. Advisory Board, 1990) and have major

implications for medical care and costs. In 1991 it was estimated that up to 739,200 women used one or more illegal substances during their pregnancies (Gomby & Shiono, 1991). In a recent California statewide survey (1993), approximately 69,000 births out of the total 607,000 births in California in 1992 were born to mothers who used alcohol and/or drugs prior to delivery; this translates to an overall prevalence rate of 11.35 percent (Vega, Noble, Kolady, Porter, Hwang & Bale, 1993). This drug use by pregnant women exceeds $500 million annually for the medical care of cocaine exposed infants alone (Phibbs, Bateman, Schwartz, 1991).

Research on neonates and young infants who are prenatally drug exposed and the relationship to developmental outcomes has grown dramatically over the last 20 years. The bulk of the initial work focuses on prenatal narcotic exposure, followed by great interest in alcohol, with documentation of Fetal Alcohol Syndrome, consideration of marijuana and nicotine, and the predominance of literature on the effects of cocaine and its various alkaloid forms. The specific teratogenic effects of drugs on the developing infant have not been well established, with the exception of maternal alcohol use and the recognition of fetal alcohol syndrome and fetal alcohol effects.



However, the use of drugs by the pregnant woman has been associated with a wide range of significant neurobehavioral effects in the neonate as well as growth and developmental concerns. These reported effects are consistent with pharmacologic actions on the vascular and central nervous systems in adults, but in the developing fetus may manifest as a chronic condition. It is unknown whether the adverse effects in the infants are part of a withdrawal syndrome associated with physical health problems secondary to PDE such as prematurity or intrauterine growth retardation, or if there are permanent neurological alterations secondary to maternal drug usage and exacerbated by maternal and

environmental factors as the infant matures.

It is known from the research that drugs taken during pregnancy do have an impact on the developing fetus, with evidence from many studies that this impact may place the infant at risk. However, a controversy exists about infant effects and outcome, which is dependent upon the drug, i.e., marijuana (Dreher, Nugent & Hudgins, 1994; Hayes, Dreher & Nugent, 1988).

Measures to Assess Neurobehavioral Impact

The following demonstrate the lack of a valid and sensitive assessment instrument to discriminate the subtle neurobehaviors of infants with PDE during the first year of life. What is needed is a comprehensive assessment tool which captures the infants' subtle neurobehaviors associated with maternal drug use, regardless of the drugs taken by the pregnant women.

A number of standardized but global developmental assessments exist. These include the Brazelton Neonatal Behavioral Assessment Scale, Mullen's Scale of Early Learning, Denver II, and the Bayley Scales of Infant Development. While these are screening and assessment measures for infant development, none of these tests have the specificity or sensitivity alone to measure the subtle neurobehavioral characteristics of infants withe PDE. An analysis by Meisels and Wasik (1990) points out the fact that "very little is known regarding the epidemiology or risk and disability in the first three years of life" (p. 606). By examining data from the state of Michigan they point out that of children entitled to comprehensive special education services from birth through age 21, only 0.7% are identified by age two, 7.5% are identified by age six, and 12.6% by age nine. One of the primary causes they cite for the lack of early identification is that there are few valid assessment tools available for use with infants and children birth to three years (Cicchetti & Wagner, 1990; Meisels, 1988).

The neurobehavioral characteristics of PDE infants reported in most studies

were noted by anecdotal observation or scored on the Finnegan Neonatal Abstinence Scale (NAS) or the Brazelton Neonatal Behavioral Assessment Scale (BNBAS). The NAS is used with newborns, and the BNBAS is used with infants from birth up to 30 days of age. These assessments, while helpful in many ways, have not been developed to address the specific neurobehavioral problems known to exist for PDE infants. They do not provide important information regarding visual or communicative social skills such as initiating and maintaining eye contact, visual tracking, social smile or laugh, or reciprocal vocalizations. These behaviors have been noted in the LNAS for their clinical importance in PDE infants not only in the neonatal period but as the infant matures. These are all skills involved in later learning and social success and are important to the development of appropriate infant intervention strategies.

The Bayley Scales of Infant Development are used later in infancy (2 to 30 months) and also do not provide information on subtle neurobehaviors which continue to be observed in this population and which can interfere with the infant's performance. When the Bayley's composite score is used in research, or an overall poor score is reported, the results do not pinpoint whether there is a specific area of impaired function, whether it reflects global or generalized functioning, or whether strong scores in one area are masking scores that reflect impaired functioning in another area.

A study by Morrison and Villarreal (1993) indicates that children with PDE who have difficulty in particular domains are not identified by the overall total score. Thus the Bayley score alone may not be helpful in planning specific interventions in areas of need. For instance, it does not provide information regarding the integrity of the self-regulatory system or the age appropriateness of the infant's attention orientation. Furthermore, assessments like the BNBAS and the Bayley allow for influence by the examiner who facilitates the infant's focus

and state regulation during item administration. Chasnoff and colleagues (1992) suggest that the examiner's actions may mask self-regulatory difficulties in the infant.

Some traditional methods of infant assessment alone, such as the Bayley and BNBAS, may not be the best way to provide a complete picture of the infant's developmental status (Clark, Paulson & Conklin, 1993). They are not targeted enough to the unique neurobehaviors of these high-risk infants.

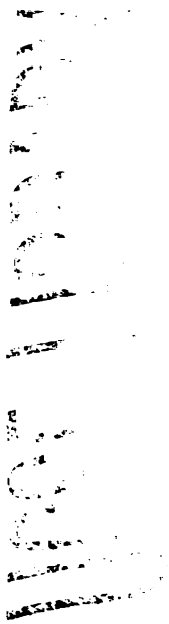
New assessment instruments that expand on neurobehavioral categories and extend them into later age ranges are needed to better evaluate the high-risk infant's developmental progression of neurobehavioral sequelae, both for the purposes of health planning and resource allocation.

A New Assessment Measure

The LNAS was developed specifically from known data and clinical nursing observations to assure a more comprehensive and specific focus on problems unique to PDE. It is the first nursing based assessment for this population and it offers the following strengths which existing tools do not.

The revised LNAS provides for continuity across the age span from birth to 12 months. This allows for comparison across early developmental periods. Regardless of the drug or combinations of drugs taken by the pregnant woman, the assessment tool captures the infants' subtle neurobehaviors associated with PDE, as impaired behaviors are known to effect social, cognitive and learning abilities. The results of the Scale can be used by the clinician to develop specific intervention strategies related to particular areas of risk observed in the individual infant, as each item identifies a particular atypical behavior which may be amenable to intervention. A number of behaviors can be assessed with the LNAS during early infancy such as distractibility, ease of frustration, and

hyperactivity. These behaviors are often observed later in the school age child with attention deficit hyperactivity disorder, learning disabilities and/or attention deficits. The assessment allows for monitoring of infants who display these early warning behaviors to determine whether they resolve, persist over time, and — if they do persist — whether they are antecedent to later learning disabilities or later behavioral and social problems.



CHAPTER TWO

Literature Review And Conceptual Framework

The literature review for this research was organized around three major propositions which served to guide the study. First, there is evidence of neurobehavioral problems for infants who are exposed to drugs in utero. Second, there is a need for improved assessment of neurobehavioral status in drug exposed infants. Third, systems theory provides a conceptual model which can serve as an effective foundation for measuring the neurobehavioral status of these infants. Support for each of these propositions is presented in this chapter.

The Effects of Intrauterine Drug Exposure on Infant Neurobehavioral Problems

The particular neurobehavioral effects attributed to certain drugs are documented in the literature. Studies have focused primarily on the Central Nervous System (CNS) stimulants cocaine and amphetamine, CNS depressants heroin and methadone, and psychoactive drugs marijuana and phencyclidine.

Cocaine

Mechanisms of fetal transfer and effects. CNS drugs are lipid soluble and of relatively low molecular weight which facilitates their movement across biologic membranes, including the placental-fetal barrier and the blood-brain barrier. Cocaine blocks the re-uptake of norepinephrine, dopamine, and serotonin at the nerve terminals thereby increasing their levels at postsynaptic receptor sites (Hall, Talbert & Ereshefsky, 1990). Excessive stimulation of the sympathetic nervous system is due to the drug's peripheral effects, and causes vasoconstriction, mydriasis, and increases in respiratory and heart rates. Cocaine's vasoconstrictive effects reduce blood flow through the placenta, decreasing both the needed nutrients and oxygen flow to the fetus. The vasoconstrictive effect combined with increased heart rate and increased blood

pressure can lead to myocardial or cerebral infarction in the adult. In the pregnant woman, vasoconstriction can decrease blood flow and stimulate uterine contractions. Pregnant cocaine users have an increased rate of abruptio placentae, acute onset of labor, immediately after intravenous cocaine use and spontaneous abortion (Chasnoff, Burns, Schnoll & Burns, 1985).

The appetite suppressant action of cocaine can cause an inadequate maternal diet, which can play a role in retardation of fetal growth. Placental vasoconstriction can lead to fetal hypoxia and inadequate fetal nutrition. Furthermore, the fetal liver is immature and drugs are not excreted efficiently. In one study benzoylecgonine, a metabolite of cocaine, remained in urine samples of neonates for five days after delivery when the mothers used cocaine one to two days prior to delivery (Chasnoff, Bussey & Savich, 1986). Norcocaine is a water-soluble substance which is more potent than cocaine, and is reported to persist in the urine of neonates for up to 4 days (Chasnoff, 1988). This cocaine metabolite is excreted by the fetus into the amniotic fluid and than re-swallowed by the fetus, creating repeated exposure of the fetus to the drug (Chance & Watts, 1993).

Effects on pregnancy and the infant. Cocaine affects brain chemistry and development three ways. First, fetal brain development is influenced directly by cocaine exposure due to its effects on the developing and mature neurotransmitter systems; second, it affects neuronal differentiation and brain structure formation; and third, it indirectly affects blood flow to the developing fetal brain (Mayes, 1994). Cocaine readily crosses the placenta and the blood brain barrier of the fetus affecting three major neurotransmitters, including dopamine, norepinephrine, and serotonin. This occurs at the level of neurotransmitter release, recognition, and reuptake at the synaptic gap (Mayes & Bornstein, 1995). Blocking of reuptake of the three neurotransmitters leaves

more of the chemical available in the synaptic gap, which results in enhanced activity of these agents in the Central Nervous System (CNS). This activity is associated with physiologic reactions such as tachycardia and vasoconstriction and also creates a neurological vulnerability expressed behaviorally and developmentally in a variety of ways during the first few years of life (Mayes, 1994). These neural systems are involved in functions such as attention, arousal, motivation, social interaction or motor activity.

Cocaine effects are reported to be gender specific. Dow-Edwards (1995) found that male rats exposed to cocaine have altered metabolism , while female rats exhibit more atypical neurobehavioral characteristics. Postnatal cocaine exposure appears to affect brain metabolism in female rats, while both genders exhibit behavioral abnormalities. Animal studies suggest a behavioral vulnerability when cocaine-exposed rat pups respond differently to environmental cues and stressors (Dow-Edwards, 1995). This helps to support the notion that human infants with PDE to cocaine have decreased capacity for self-regulation and the ability to regulate their emotional state (Chasnoff, Burns, Schnoll, Burns, 1985; Griffith, 1995; Kammel, Gardner, Freedland, 1996). Neurobehaviors displayed by cocaine exposed infants include irritability, hyperactivity, high-pitched cry, difficulty feeding, tremors, difficulty initiating and maintaining eye contact, and gaze aversion (Bear, B., 1995; Chasnoff, Griffith, & Freier & Murray, 1992; Lewis, K., 1991). In addition, the decrease in blood flow creates vasoconstriction, which if chronic can result in fetal hypoxemia and decreased nutrient transfer which has adverse effects on fetal growth (Woods, Plessinger, Clark, 1987). These effects are documented as low birth weight, decreased length and head size in the newborn infant exposed to cocaine (Chasnoff, Burns, Schnoll & Burns, 1985; Lewis, 1991).

Some studies demonstrate an association between cocaine exposure and an

increased risk for spontaneous abortion, prematurity, decreased fetal growth, low birth weight, shorter birth length, small head circumference (Chasnoff, Burns, Schnoll & Burns, 1985; Oro & Dixon, 1987, Zuckerman et al, 1989), small size for gestational age (MacGregor, et al, 1987), and implicated in visual abnormalities including optic nerve abnormalities, delayed visual maturation, and prolonged eyelid edema (Good, Ferriero, Golabi, Kobori, 1992). A study by Stafford, Rosen, Zaider and Merrian (1994) of cocaine exposure in 40 cocaine exposed infants and 40 control infants found no differences in the incidence of eye abnormalities in the infants. Some researchers report findings of numerous congenital deficits as a result of cocaine exposure in utero, including facial, cranial, skeletal, cardiac, and renal/genital malfunctioning (Bingol, et al, 1986; Chasnoff, Griffith, MacGregor, Dirkes & Burns, 1989; Dixon & Bejar, 1989; Fries, et al, 1993; Lawton, 1992). Although a variety of malformations have been documented, no classic description — such as the neonatal abstinence syndrome or fetal alcohol syndrome — has been identified.

Studies using the Brazelton Neonatal Behavioral Assessment Scale (BNBAS) create a profile of newborn cocaine-exposed infants as having increased irritability, tremulousness, startle responses, increased muscle tone, retention of primitive reflexes, poor state regulation, less consolability, and depressed interaction ability (Chasnoff, Burns, Schnoll & Burns, 1985; Chasnoff, 1988; Griffith, 1988).

In a case series study of 39 neonates in which the opiate abstinence scoring approach was used, tremulousness, irritability, and increased muscle tone were found to be neurobehavioral problems (Doberczak, Shanzer, Senie & Kendall, 1988). Infants exposed to both cocaine and amphetamines had abnormal sleep patterns, tremors, poor feeding, hypertonia, vomiting, sneezing, high-pitched cry, frantic fist sucking, tachypnea, loose stools, fever, yawning, and hyperreflexia.

This was a cross-sectional study comparing three groups; 1) cocaine (13), methamphetamine (28), cocaine and methamphetamine (5) (n=46); 2) Heroin (17), methadone plus heroin (32), (n=49); and 3) Drug-free control group (n=45). These groups studied were in a hospital obstetric population. The groups were matched by maternal factors, i.e., age gravidity, pariety, abortion, ethnicity and prenatal care, and no difference was found on neonatal abstinence scores (Oro & Dixon, 1987). Still another study of drug-exposed infants in which observation descriptions were employed revealed tremulousness, irritability, and muscular rigidity in the neonate (LeBlanc, Parekh, Naso & Glass, 1987). These studies use a scale derived specifically from a neonatal opiate withdrawal model, when cocaine-induced symptoms may be long-term drug effects, not neonatal withdrawal symptoms. Furthermore, this model does not capture the complete range of atypical neurobehaviors observed in this population.

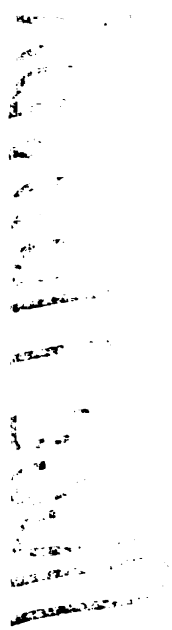
The reported incidence of Sudden Infant Death Syndrome (SIDS) in this population is inconsistent and controversial. Some researchers found an increased incidence of SIDS, 1%-5% (Chasnoff, Hunt, Kletter & Kaplan, 1989; Durand, Espinoza, & Nickerson, 1990), while others found no association (Bauchner, Zuckerman, McClain, Frank, Fried & Kayne, 1988).

Cocaine intoxication in a breast feeding two-week-old infant has been documented by Chasnoff and colleagues (1987). The mother used intranasal cocaine, and the infant symptoms included irritability, tremulousness, vomiting, diarrhea, tachycardia, tachypnea, high pitched cry and hypertension. From this study the American Academy of Pediatrics Committee on Drugs recommends that cocaine-using mothers not breast feed their infants (Committee on Drugs, 1994). Another nursing infant developed apnea and seizures after her mother used topical cocaine to relieve nipple soreness (Chaney, Franke & Wadington, 1988).

Effects on the developing child. Research examining the long-term effects of cocaine are not conclusive and need further verification. Rodning, Beckwith & Howard, 1989, report significantly lower intellectual functioning and quality of play in a group of 18-month-old drug exposed infants; however, the study has been highly criticized for a lack of control for confounding, nonblinded exams, and no toxicological assays or drug reporting for the comparison group (Neuspiel & Hamel, 1991). Chasnoff, Griffith, Freier & Murray (1992) followed a group of 151 infants through two years of age who were prenatally drug exposed. They were divided into three groups; 1) cocaine and usually marijuana and/or alcohol (n=106); 2) marijuana and/or alcohol, but no cocaine (n=45), and; 3) non-drug exposed (n=81). The researchers reported that most of the infants in group 1 and 2 were functioning in the normal range of cognitive and motor development; however, 30% to 40% were having behavioral and language problems, which included low frustration tolerance, distractibility, difficulty with behavioral organization, and language delays. At 2 years of age the drug exposed children, both cocaine/polydrug exposed or polydrug/noncocaine exposed, had a significantly smaller head size than the non drug exposed, a trend falling below the 10th and 5th percentile. A significant relationship was found between small head size and cocaine exposure, but at age 3 neither cocaine nor any other study drug was predictive of small head size.

The follow-up study (Griffith, Azuma & Chasnoff, 1994) demonstrated smaller mean head sizes for the drug exposed group at age 3, although the findings were not statistically significant. There were a larger number of drug exposed infants below the tenth and fifth percentiles for head size than the control group, which may be an indicator for predicting poor long term development. The children from all three groups were performing within the normal range of IQ using the Stanford-Binet Intelligence Scale. However, the

cocaine/polydrug group scored significantly lower on the verbal reasoning tasks, and the polydrug/noncocaine group scored significantly lower on abstract/ visual reasoning subscales. Examiners rated the two drug groups as having difficulty sustaining attention, and caregivers rated both drug groups as more aggressive than the control group. The more destructive group was the cocaine/ polydrug exposed children. A limitation of the study is that the women who participated were in drug treatment programs during pregnancy, and involved in subsequent pediatric and developmental follow-up for their children, although the study does not address parenting skills. Medical involvement and routine follow-up can be interpreted as intervention, the study does not address this phenomenon. Furthermore, some of the children in the study were living with drug-using mothers, others with mothers who had stopped using drugs; still others were in foster home care. Research needs to examine both maternal and environmental factors which infants and children live that may influence and/or have an effect on child learning and neurobehavior, not only intellectual development.



Amphetamines

Mechanisms of fetal transfer and effect. Cocaine and amphetamines are structurally different but pharmacologically similar (Galanter & Kleber, 1994). Amphetamine, like cocaine, is a CNS drug and demonstrates similar physiological effects. These physiological effects are caused by the stimulation of the release and blocking of reuptake of the neurotransmitters dopamine, serotonin, and norepinephrine (Dixon, 1989; Kandall, 1991). Both drugs produce direct cardiovascular actions causing vasoconstriction and hypertension, which in turn may cause fetal hypoxia and cardiac and vascular accidents.

It is unknown whether the amphetamines are teratogenic. This question has been explored in a number of animal studies, both abroad and in the United States;

however, studies on humans are limited. This may change with the recent widespread abuse of methamphetamine by childbearing-aged women. Methamphetamine is structurally similar to amphetamine, but is more potent (Martin, 1992).

Effects on pregnancy and to the infant. Amphetamine/methamphetamine is reported to stimulate the release of and inhibit monoamine oxidase (MAO) and block reuptake of dopamine, norepinephrine and serotonin, thus increasing the availability of these neurotransmitters to the post synaptic receptors. This action also occurs with cocaine, but the mechanism by which it occurs may be different than that of amphetamines (Middaugh, 1989). High does of amphetamine given over a three-day period to rats cause degeneration of dopamine neurons, beginning at the terminals (Ricaurte, Seiden & Schuster, 1984) and continues to the cell bodies (Middaugh, 1989). Lower doses of amphetamine injected into adult rats stimulate motor activity with an intact nigrostriatal tract while large dose chronic administration depletes catecholamine stores and reduces motor activity. This supports the notion that effects on brain chemistry of the developing fetus may depend upon the amount of drug reaching the fetus, the duration of the drug exposure and time of exposure during uterine brain development (Middaugh, 1989). The neostriatum is the principal site of dysfunction in Parkinson's and Huntington's disease, and is believed to be involved in both motor and cognitive functions of the human brain (Groves, Ryan, Linder, 1987). Motor and cognitive dysfunction observed in infants with PDE to amphetamine have tremors, poor feeding, hypertonia, hypotonia, irritability, high-pitched cry, lethargy, and hyperactivity (Oro & Dixon 1987; Dixon & Bejar, 1989; Lewis, 1991).

Because amphetamine and cocaine have similar central physiological effects, some researchers theorize that similar effects can occur to infants exposed to this

drug in utero. An early retrospective research study by Ericksson, Larsson, Winbladh and Zetterstrom (1978) studied 23 women, retrospectively, who were chronic amphetamine users, six of whom reported they gave up amphetamine use after becoming pregnant. They found an increased rate of prematurity, small infant size for gestational age, and perinatal complications for women who used amphetamines throughout pregnancy as compared to those who stopped early. Amphetamine use was also associated with decreased prenatal visits and an increased occurrence of low birth weight infants. Neurological findings included one infant with unexplained seizures and two infants who were lethargic and unable to feed. Ericksson and Zetterstrom (1981) later studied 69 amphetamine-abusing women, 52 of whom used amphetamine throughout pregnancy. The researchers reported a high incidence of pregnancy-related complications, perinatal mortality rate, congenital malformations, and neonatal neurological abnormalities. However, this second study had multiple methodological weaknesses, including nonblind examination, no control group, drug use by maternal report, and no control for confounding maternal factors.

Oro and Dixon (1987) report research results similar to those of Ericksson and colleagues in Sweden, both suggesting a dose-related morbidity. Oro and Dixon compared 46 neonates exposed to cocaine, methamphetamine, and cocaine with methamphetamine using the Finnegan Neonatal Scoring System. Neonates with cocaine and methamphetamine exposure, both alone or in combination, displayed an increased rate of intrauterine growth retardation, prematurity, and perinatal complications compared to a control group. These complications included placental hemorrhages and abruptions, lack of prenatal care, decreased birth weight length and head circumferences. Even with these increased risk factors, they found no statistically significant differences among infants on selected perinatal variables. Neonatal neurobehaviors exhibited included

tremors, poor feeding, abnormal sleep patterns, abnormal cry, vomiting, state disorganization, and hypertonia. This study was limited by the lack of a drug-free control group, nonblind examinations, and use of a scale designed for opiate exposure.

There is a small number of early research reports of related complications or defects related to prenatal amphetamine exposure. The researchers report findings of heart defects (Nora, Vargo, Nora, Love & McNamara, 1970; Gilbert & Khoury, 1970,) biliary atresia (Levine, 1971), and withdrawal consisting of agitation alternating with fatigue, apnea during feedings, diaphoresis, vomiting, glassy-eyed stare and seizure activity (Ramer, 1974).

A study of term infants exposed to cocaine (37), methamphetamine (27), or to cocaine and/or heroin and methadone (18) (n=82) were assessed blind within three days of birth by cranial ultrasonography (Dixon & Bejar, 1989). The drug-exposed group was compared to both a high-risk group (n=87) and a healthy non-drug exposed group (n=19) of infants. Thirty-five percent (26) of the infants with PDE had cranial abnormalities, which was similar to the high-risk group with 27.6% (24), but different than the healthy non-drug exposed group with 5.3% (1) abnormalities. These data indicated that prenatal exposure to cocaine and methamphetamine was associated with a significant increased incidence of echoencephalogram (ECHO) abnormalities in full term newborns that had no other known cause of cerebral injury. Areas suggestive of "old" infarction with cavitation indicating the possibility of brain injury occurrance prior to birth. The location, type, and location of documented lesions may not be clinically evident in infancy or early childhood (Dixon & Bejar, 1989). Damage occurring in the frontal lobes and the basal ganglion may be reported after the first year of life when more complex visual-motor and social cognitive tasks are required by the young child (Volpe, 1987). Drug withdrawal symptoms may be evidence of

permanent CNS injury to the infant. This study has not been replicated.

Amphetamine was detected in the urine of a nursing infant who exhibited irritability and poor sleeping patterns in a study by Steiner and colleagues (1984). Based on this case report, breast feeding by mothers using amphetamines is contraindicated by the American Academy of Pediatrics (Committee on Drugs, 1994).

Effects on the Developing Child. Billing and colleagues (1994) followed a cohort (N=65) of infants exposed to amphetamine in utero to age 8. The researchers found a significant relationship between the amount and duration of amphetamine exposure in utero and later behavioral problems of these children, specifically aggressive behavior. Children exposed during the whole pregnancy were more aggressive and had more peer-related difficulties than those children exposed early in pregnancy.

Marijuana

Mechanism of fetal transfer and effects. Marijuana readily crosses the placenta. Transfer appears to be higher in early gestation and to decrease as the pregnancy advances (Zuckerman & Bresnahan, 1991). Marijuana reduces the amount of oxygen passing into the maternal bloodstream, which in turn reduces oxygen levels to the fetus. Marijuana increases maternal carbon monoxide levels, which can also cause fetal hypoxia.

Effects on the pregnancy and to the infant. Perinatal cannabinoid (chemical compound found in marijuana) exposure may be involved in alterations in brain functioning with long term developmental consequences, such as effects on receptor sensitivity observed in rats prenatally (Walters & Carr, 1986). Dalterio and colleagues (1984) also found effects on the male reproductive system, i.e., reduced testicular weight and elevated luteinezing hormone levels and changes in the concentrations of dopamine, norepinephrine and serotonin in cannabinoid

exposed male mice. Tetrahydrocannabinol (THC), the active component in marijuana, binds to specific receptors of which many coordinate movement. This can be observed in animals when they are given large doses and they collapse and cannot move . Marijuana decreases the activity of neurons in the hippocampus, a relay center, which is involved with memory storage and learning and contains many THC receptors, which may account for poor short-term memory and problems processing information in heavy users of marijuana (Society for Neuroscience, 1996). Lester and Dreher performed high-speed computer voice analysis to assess the maturity of newborns born to marijuana smoking mothers in Jamaica. The infants had a much higher frequency of voice anomalies suggesting possible impairment of fetal brain development.

Research to date fails to provide sufficient data about the effects of prenatal exposure to marijuana on the developing fetal brain, the neonate, infant and the developmental outcomes of the young child. What is available is inconsistent and limited. This is surprising, as marijuana use and abuse by adolescents and adults is not a new phenomenon. Zuckerman and colleagues (1989) propose that inconsistencies or missing an association between maternal marijuana use and outcomes can occur when urine drug screening is not utilized for identification of drug abuse. They studied 1,226 mothers, and determined 27% used marijuana during pregnancy and 18% used cocaine as determined by either interviews or urine screenings. When they used only urine drug screenings for identification, the corresponding values were 16% and 9%. The exposed infants with positive drug assays demonstrated a greater decline in birth weight and birth length. Deficits such as lower weight and head circumference were reported by Hingson and colleagues in 1982, and were confirmed by Hatch and Bracken (1986) and Lester and Dreher (1989). However, other researches reported that when confounding variables were controlled statistically, no anthropometry deficits

were associated with maternal marijuana use, or the outcomes were varying (Tennes et al, 1985; Fried, Watkinson, & Willian, 1984; Linn, Schoenbaum, Monson, Rosner, Stubblefield & Ryan, 1983).

Fried and Makin (1987), using the Brazelton Neonatal Behavioral Assessment Scale, reported a significant relationship between prenatal marijuana exposure and increased neonatal tremors, startles, and poorer habituation to visual stimuli. The tremors, which were both spontaneous and in response to stimuli, were often accompanied by exaggerated startles. These behaviors persisted through the first 30 days of life, after which time the BNBAS cannot be administered. These findings were similar to a previous study (Fried, 1980; Fried, 1985). However, Hayes, Dreher and Nugent (1988) suggest that their ethnographic field study findings of no significant difference between the marijuana exposed group (n=30) and the non-exposed group (n=26) in the neonatal period reflects the maternal cultural characteristics of Jamaican women, which has the capacity to subdue the potentially adverse effects of marijuana. "Ganja" smoking women display as a major characteristic, independence, while their lifestyle is one of an active household with many caregivers, relaxed motherhood, and infant stimulation and playfulness. These mothers believed the use of ganja increased their appetites and decreased nausea, which allowed them to better accomplish childcare and household tasks. Certainly cultural as well as environmental differences need to be considered when examining these findings in Jamaican infants. Studies at later age ranges would be useful to examine social behavior and educational adjustment. A recent comparative study by Dreher, Nugent, Hudgins (1994), to identify neurobehavioral effects of prenatal marijuana exposure on infants in Jamaica, using the BNBAS, found no differences between drug exposed infants and non- drug exposed infants. In Jamaica, marijuana is legal, which may alleviate one of the methodological

concerns; that is, that the use of marijuana was not confounded by maternal use of alcohol, tobacco, or other drugs. The population studied was from the lower socioeconomic (SES) areas. However, in Jamaica, heavy use of marijuana by women is associated with a higher level of education and greater financial independence. Pearson's correlations were performed to determine whether there was indeed an association between maternal education and the infant's outcome at one month. They revealed that maternal education was significantly correlated with the Autonomic cluster at one month. Ethnographic observation of the environment reveals a higher number of single mother households, fewer children in the home, and more adults living in the households. Drug use was identified by maternal report only, and assessments were nonblinded.

Lester and Dreher (1989) assessed the maturity of neonates with high speed computer voice analysis. The cries of infants born to mothers who smoked marijuana (n=20) demonstrated a higher number of voice anomalies than the cries of infants born to non smokers (n=20) living in Jamaica. They argue that this phenomenon may indicate injury to fetal brain development by exposure to marijuana.

Marijuana was found in breast milk in a study by Perez-Reyes & Walls (1982). As a result of that study, breast feeding is contraindicated by the American Academy of Pediatrics, Committee on Drugs (1994).

Effects on the developing child. Few studies have followed the developmental outcomes of children with prenatal marijuana exposure. Day and colleagues (1994) followed 829 women and their children. Half of the infants were Caucasian and half were African-American, which reflected the composition of the prenatal clinic where the study was conducted. The children were assessed at delivery, 8, 18, and 36 months with the Stanford-Binet Intelligence Scale and a variety of other measures. At 3 years of age the final

cohort was 655. There were no significant negative effects on the overall composite score of the 3-year-olds using the Stanford-Binet. When subscale scores were used as outcome variables, there were negative effects from maternal marijuana use which was dose-related and affected by the trimester of use. In the Caucasian group, no significant effects were noted on the composite scores or subscale scores. Caucasian children who did not attend daycare showed a decrease in IQ scores. This effect was offset by the increased IQ scores of those Caucasian children who attended daycare/preschool. The African-American group demonstrated significant negative effects on composite scores, short-term memory, and verbal reasoning subscales regardless of preschool/ daycare attendance. It is unclear whether urine screens were performed or if only maternal report of drug use was used, and it is unclear whether assessments were conducted blind.

Fried and Watkinson (1990), in their Ottawa Prenatal Prospective Study (OPPS), using the McCarthy Scales of Children's Abilities, found a significant association between prenatal marijuana exposure and lower scores in both verbal and memory domains at 48 months of age. A later report from the OPPS on 5- and 6-year-old children found no significant effects of fetal marijuana exposure on cognitive or receptive language development; however, significant effects were noted in omission errors on a vigilance task and of impulsive and hyperactive behavior documented by maternal report. (Fried, Watkinson & Gray, 1992).

Phencyclidine

Mechanisms of fetal transfer and effects. The mechanisms of fetal transfer and the effects on the human fetus and neonate have not been well studied. PCP is known to cross the placenta into the fetal circulation, is found in neonatal blood, urine, and breast milk (Harry & Howard, 1992; Kaufman,

Petrucha, Pitts & Kaufman, 1983; Kaufman, Petrucha, Pitts & Weekes, 1983; Strauss, Modanlow & Bosu, 1981).

Effects on pregnancy and to the infant. Phencyclidine (PCP) alters several neurotransmitter systems, although the mechanisms of how this occurs are unclear. Prenatal transfer of PCP was documented to occur in the pig (Cummings, 1979), mouse, rabbit (Nicholas & Schreiber, 1983) and human (Petrucha, Kaufman, & Pitts, 1982). Brain concentration levels of PCP are much higher in fetal tissue than in maternal brain levels in the pig, mouse and rabbit. Researchers report that rats administered PCP throughout gestation and lactation had permanent increases in dopamine and serotonin concentrations in discrete brain areas of male offspring. The functional significance of these chemical alterations is unknown, as short- or long-term behavioral assessment of the offspring was not assessed (Fico & VanderWende, 1988). Some neurobehaviorals reported in infants with prenatal PCP exposure are flapping tremors, hyperactivity, irritability, diarrhea, vomiting, temperature instability, as well as prematurity and decreased anthropometry measures.

Golden, Sokol, and Rubin (1980) were the first researchers to document the effects of prenatal PCP exposure by describing the neurobehavioral characteristics exhibited by a neonate whose mother smoked PCP daily during pregnancy. The neurobehavioral symptoms exhibited after birth, by observation, included jitteriness, coarse flapping movements in response to slight tactile or auditory stimuli, hypertonicity, nystagmus and poor visual tracking. Dysmorphic features were also present. The infant at 2 months of age continued to display coarse tremors, hypertonicity and roving eye movements.

Strauss, Modanlou and Bosu (1981) reported on two infants with prenatal PCP exposure. These infants exhibited jitteriness, irritability, and hypertonicity. Chasnoff, Burns, Hatcher and Burns (1983) described the behavior of seven

infants whose mothers used PCP on a daily basis throughout pregnancy as well as using various other drugs on occasion. These infants were compared to a control group (N=27) that was determined on the basis of maternal report and lack of physical evidence using the BNBAS, with examiners blind to drug exposure. The PCP exposed neonates exhibited irritability alternating with lethargy, rapid changes in the level of consciousness, tremors, facial grimacing and were sensitive to auditory stimuli.

Howard, Kropenske, and Tyler (1986) observed 12 neonates exposed to PCP and described their growth and development at birth and at 9 and 18 months of age. The average gestational age was 37.8 weeks with a range of 33-42 weeks. The mean birth weight was 2,481 (10th percentile), mean head circumference was 31.5 cm (10th percentile) and the mean length was 47 cm (25th percentile). All infants or mothers had positive toxicology reports. Within 24 hours of life all infants developed atypical neurobehavioral symptoms which included tremors, hypertonicity, irritability, bizarre eye movement and staring spells. Observed less frequently were lethargy, diarrhea, poor sucking and facial twitching. These beginning studies have numerous methodological concerns, including small sample size, no control group, lack of control for confounding factors, and no appropriate assessment scales.

In 1990 Tabor, Smith-Wallace and Yonekura conducted a retrospective study which compared 37 infants with prenatal PCP exposure to 37 infants with prenatal cocaine exposure. Both groups of infants had a high incidence of intrauterine growth retardation, precipitate labor, symptoms of neonatal drug intoxication and prolonged neonatal hospitalizations. The PCP infants were less premature than the cocaine exposed, but the infants with PCP exposure were born smaller for gestational ages (32.4%) than the cocaine exposed infants (18.9%). The neurobehavioral characteristics described for the infants with

prenatal PCP exposure included coarse flapping tremors, hypertonicity, irritability, continuous crying, poor feeding, diarrhea, disorganized sucking, vomiting, restlessness and temperature instability. This retrospective study was limited to the information documented on the hospital chart, which may not have had important data such as pattern of drug use, maternal health history, and socioeconomic background.

Rahbar, Fomufod, White and Westney, (1993) studied 505 newborns who were prenatally exposed to illicit drugs. Of the 505 neonates 370 were exposed to a single drug only, which included 83 who were exposed to PCP and 287 neonates who were exposed to cocaine. Drug exposure was determined by a positive urine toxicology in mother or neonate or by maternal report. Intrauterine growth retardation was evidenced when 42% of the PCP exposed infants displayed birth weight for gestational age (GA) below the 25th percentile, 37.3% had birth heights for GA below the 25th percentile and 45.7% of the neonates displayed head circumference below the 25th percentile, 12% of whom had head circumferences below the 10th percentile. No dysmorphic features were noted. Neurobehavioral symptoms noted most frequently included high pitched cry, poor tracking, and decreased attention. These symptoms were measured on a special drug withdrawal scoring sheet. Study limitations include non-blind examiners and lack of consideration of confounding effects.

Effects on the developing child. In the Howard & colleagues (1986) study, eight infants at 8.5 and 17.8 months of age were given the Gesell Developmental Evaluation to determine gross motor, fine motor, adaptive, language, and personal- social development. Most infants scored within the normal or low normal range of development in the individual scales with an overall score of 95 for the 8.5 month old infants and 94 for the 17.8 month old toddler. The developmental scores on the Gesell are not able to reveal whether there is any

difficulty in performing the task, or whether any atypical quality of movements are exhibited by the infants and toddlers in performing the tasks. Fine motor development was in the low normal range, but many of the infants were observed to display abnormal movement patterns while attempting to grasp small objects, arm movements were awkward and hand positioning was peculiar, palmar approach was used to pick up a cube, and some infants were persistent, while others were easily frustrated. Developmental milestones were obtained within the normal range. Again, this study has limitations due to its lack of non-blind examinations and no specific assessment scale for neurobehavioral symptoms.

Heroin

Mechanisms of fetal transfer and effects. Heroin readily crosses the placenta and can affect the fetus directly and/or indirectly. The fetus can develop a tolerance for drugs in utero and after birth the infant appears to go through a withdrawal commonly referred to as neonatal abstinence syndrome (NAS).

Effect on the pregnancy and to the infant. Prenatal exposure to opiate drugs such as heroin, methodone, Demerol and morphine may result in atypical structural organization of the fetal brain (Smith, Hui, & Crofford, 1977; Sakellaridis, Mangoura, & Veradakis, 1986). Neuron density is decreased in particular areas of the hypothalamus and in the cerebral cortex by morphine (Hammer, Ricalde, & Seatriz, 1989). As receptors affect dentritic growth and mature at different rates in different parts of the brain, prenatal exposure may produce altered effects dependent upon the timing of the drug exposure. This provids support for the fact that atypical neurobehavioral, i.e. neonatal abstinence syndrome, observed in narcotic exposed infants may be attributable in part to underlying structural changes in brain development (Zuckerman & Brown, 1993). Opiate drugs mimic the effects of a number of natural

neurotransmitters which are known as the endogenous morphines, or endorphins (Society for Neuroscience, 1996) and are related substances that bind to opioid receptors in numerous areas of the body. The effects that occur when an opiate binds to opioid receptors depend upon the area of brain involved, i.e., binding to receptors in the medulla cause nausea and vomiting, in the reticular activating system causes sedation, and in the limbic system causes euphoria (Novitt-Moreno, 1996).

Studies have documented that prematurity and intrauterine growth are associated with prenatal heroin exposure (Naeye, Blanc, Leblanc & Khatamee, 1973; Wilson, Desmond & Verniaud, 1973). However, studies on subsequent growth and development differ in outcome. A group of researchers found a significant difference in mean birth anthropometry in heroin exposed newborns (n=22). Group means were similar to non-drug exposed infants (n=28) after adjustments (maternal smoking, education, race, prenatal care, and weight gain) were made (Lifschitz, Wilson, Smith, Desmond, 1983). At one year of age there were no differences between groups. The researchers state that the effect of drugs could not be differentiated from the factors associated with maternal drug abuse lifestyle. These same children at 3 years of age were not different in growth factors from the high risk, drug-free control group, but all groups were below the fiftieth percentile.

Neurobehavioral symptoms, often referred to as NAS, develop within the first day of life, and often require medication and very structured intervention strategies. The drug treatment course is determined by the results of the Finnegan Neonatal Assessment Scoring system. The major drugs to treat NAS are phenobarbital and opium, in the form of either paregoric or denatured tincture of opium (DTO) (Zuckerman & Brown, 1993). The neurobehavioral symptoms reported in the literature include irritability, stuffy nose, sweating,

difficulty in feeding due to an uncoordinated suck (Kron, Litt, & Phoenix, 1976), increased muscle tone, high pitched cry, frantic sucking on hands (Kaltenback & Finnegan, 1989), restlessness, tremors, agitation, and brief sleep periods (Desmond & Wilson, 1975). One study using the BNBAS on infants who were exposed to opiates found there were significant differences in motor maturity, state control, and interactive ability (Chasnoff, Burns, Burns & Schnoll, 1986). Desmond and Wilson examined the same heroin exposed infants (n=13) exhibiting neurobehavioral symptoms (1975) one year later and found them to have high activity levels, tantrums, sleep disturbances, and low frustration levels. These early studies demonstrate numerous limitations due to sample size, lack of consistency in identification of drug exposure, lack of control for confounding factors, and reliable assessment scales.

Sudden infant death syndrome has been reported to occur with infants born to narcotic addicted mothers (Householder, Hatcher, Burns, & Chasnoff, 1982). The exact mechanism of this phenomenon is unknown. Kandall and Gaines (1991) agree on an increased risk, but stress that studies have not separated opiate use from maternal life style factors that may influence the risk of SIDS.

Heroin is reported to be found in breast milk and produced tremors, restlessness, vomiting, and poor feeding in infancy (Cobinik, Hood & Chusid, 1959). The American Academy of Pediatrics (Committee on Drugs, 1994) contraindicates breast feeding by mothers who use this drug.

Effects on the developing child. In another study by Lifschitz and colleagues (1985), the birth head size of children prenatally heroin exposed (n=25) was significantly below that of the drug free control group (n=41). At age three the head circumference did not differ significantly from the control group matched on the same environmental variables. The drug exposed children exhibited an increased incidence of low average and mildly retarded intellectual

performance as measured by the McCarthy Scale of Children's Abilities. The variables most predictive of intellectual performance were prenatal care, prenatal risk score and home environment. There was no heroin dose-related association found.

Wilson (1989) at Baylor College of Medicine have studied the long-term effects of children with prenatal heroin exposure. A follow-up study with a group of school age children with in-utero heroin exposure indicated 30% of the children had repeated one or more grades, 35% needed special educational services, and 13% had language disabilities. There were no particular neurologic findings, but a weakness in the area of motor coordination and visual motor perceptual function was identified. Limitations of the studies of older children include difficulty in measuring the maternal drug use and extent, a comparison group, and limitations of instrumentation.

Methadone

Methadone is structurally different than opiates, but is pharmacologically similar. Research studies from the 1960s and 1970s established the framework for methadone treatment as the standard of care for opiate dependent pregnant women (Jarvis & Schnoll, 1994). There continues to be controversy over the drug. A clinical perspective supports the view that the appropriate dose combined with prenatal care and a supportive comprehensive maternal program can make a positive difference in fetal and infant outcomes, as the incidence and severity of neonatal withdrawal may be altered by methadone treatment (Jarvis & Schnoll, 1994).

Methadone is administered via the oral route. It has a longer duration than opiates, and the therapeutic action lasts 2-3 days (Bertacchi, 1987). The half-life of methadone in an adult is 23 hours, and the half-life in a newborn is 32 hours (Brown & Zuckerman, 1991). Methadone is used in the treatment of narcotic

dependence. Methadone is substituted for heroin and is given to pregnant women on a consistent basis. This will curtail repeated episodes of heroin intoxication and withdrawal to the fetus. The drug alone will not reduce perinatal complications, but it does reduce the risk. In a comprehensive program, maternal nutrition is improved, as are prenatal care, early identification of complications, and decreased infections such as Hepatitis B and Human Immunodeficiency Syndrome. The therapeutic action lasts 2-3 days (Bertacchi, 1987). Although methadone was developed for drug withdrawal treatment, it is now being sold on the streets for illicit drug use.

Effects on the pregnancy and to the infant. Methadone is the treatment of choice for pregnant women who are opioid dependent, even through there are potential physiologic and teratologic consequences for the fetus and infant. Studies indicate that opioids have varying effects on rat brain chemistry and physiology which depends on the drugs used, the sites examined and the timing of exposure (Jarvis & Schnoll, 1994). Methadone is an oral opioid that helps to prevent withdrawal symptoms in the opioid addictive pregnant woman, and therefore the same type of atypical neuroanathomical brain chemistry as heroin is anticipated. Birth weights of infants born to women who use methadone are higher than those infants whose mothers use heroin (Kandall, Albin, Gartner, Lee, Eidelman, & Lowinson, 1977). Seventy to 90 percent of methadone exposed infants (40% to 50% heroin) display NAS, which requires pharmacological intervention (Zuckerman & Breshanan, 1991). NAS in the methadone exposed neonate is more severe than in the newborn with opiate withdrawal. Methadone has a long half-life of 32 hours in the newborn, thus the drug can be found in the infant's system for days after birth. This may be one reason why infants withdrawal symptoms are not observed before 24 to 48 hours after birth, and can last 10 to 14 days (Zuckerman & Brown, 1993).

Neurobehavioral symptoms such as restlessness, agitation, and sleep disturbance have been documented as long as 3 to 6 months after birth (Hutchings, 1982). Hans (1989) found the behavior of infants exposed to methadone at 1 day and 4 weeks of age using the BNBAS were typical of other reports of NAS, including hypertonicity, jerkiness, tremulousness, hyperactivity and general irritability. Methodone exposed infants (n=42) were reported to be poorer than the control group (n=44) in physical growth — height, weight, and head circumference.

Methadone is found in breast milk in concentrations close to plasma levels and is thought to prevent withdrawal symptoms in addicted infants (Briggs, Freeman & Yaffe, 1990). However, at least one infant death has been reported from methodone delivered via breast milk (Smialek, Monforte, Aronow & Spitz, 1977). The American Academy of Pediatrics, Committee on Drugs (1994) judges methadone to be compatible with breast feeding when the clinical recommendations are followed — 20mg/24 hours or less per nursing mother.

Effects on the developing child. Findings from a longitudinal study comparing cognitive development of methadone exposed infants (n=30) to a control group (n=44) at 24 months of age from comparable socioeconomic and racial/ethnic/educational backgrounds, consistently showed no differences in cognition between the groups, and mean IQ scores were about 90 on the Bayley Scales of Infant Development (Hans, 1989). These study findings suggest that the effects of an adverse environment is as important as the effects of drugs on the developing infant and toddler. The infants with methadone exposure were shorter, had smaller head circumferences, were more tense, and displayed poorer fine and gross motor coordination on the Psychomotor Development Indexes than the control group. It was noted that infants exposed to methodone displayed deficits in the physical, motor, and mental areas of development. It

suggests that methodone may influence a delay susceptibility in children living in impoverished environments.

Summary

There is growing evidence that prenatal drug exposure results in abnormal structural organization and neurochemical changes of the fetal brain. Most of the research studies are done with animal models with findings extended to the human infant. Drugs change the activity of neurons by altering the way neurons communicate with each other through neurotransmission, thus creating a neurological vulnerability in the infant and young child. How the indirect and direct effects of drug use occur in the developing fetal brain are as yet not clear (Mayes, 1994). Also unknown is whether the subtle neurobehavioral characteristics displayed can be minimized by positive effects of the environment or ongoing CNS maturation or whether the neurobehavioral characteristics will become more apparent as the child matures and behavioral and educational demands are required.

Neurochemical alterations exhibited as neurological dysfunction or as neurobehavioral abnormalities, although subtle, are observed in the human newborn and infant with prenatal drug exposure. How these changes occur is not totally apparent, but what is apparent from the research is that timing of the drug use, amount of drug, duration of the exposure and susceptibility of the fetus plays an important part in the outcome. Neurobehaviors in the infant have been documented and overlap with each drug the fetus is exposed to and can be related to three neurotransmitter actions. The monoaminergic neurotransmitters serotonin, norephinephrine, and dopamine are important brain chemicals that play a role in central control of basic processes such as regulation of attention, response to sensory stimuli, and modulation of mood states (Jacobs, 1985). Serotonin—regulates body temperature, onset of sleep, pain perception, mood

and appetite; dopamine—regulates emotional responses and movements; norepinephine—is involved in arousal, motivation, regulation of sleep and mood, motor activity, excitement, fight or flight response. Drug effects on these regulatory activities can produce behaviors such as hyperactivity or tremors that are observed in drug-exposed infants.

With the advent and recognition of maternal polydrug use of legal (tobacco and caffeine), and illegal (cocaine and heroin) drugs, it is difficult — if not impossible — to separate out the often overlapping effects of these drugs on the developing fetus and young infant. As can be seen in Table 2.1, the neurobehaviors exhibited by the infants with PDE are not unique to any one drug, but are observed in infants exposed to different drugs or combinations of drugs.

Tremors are reported in cocaine, amphetamine, methadone, marijuana and PCP-exposed infants; irritability in cocaine, heroin, methadone, and PCP exposed infants; increased startle response in cocaine and marijuana exposed infants, hypertonicity in cocaine, amphetamine, methadone and PCP exposed infants; difficulty feeding in cocaine, methamphetamine, heroin and PCP exposed infants; poor tracking in PCP exposed infants; decreased organizational ability and decreased interactive behavior in cocaine and heroin exposed infants; difficulty being consoled with cocaine and marijuana exposed infants, and lethargy in amphetamine and PCP exposed infants. There are numerous other neurobehavioral symptoms observed and reported with different maternal drug use such as abnormal sleep patterns, stuffy nose, sweating, uncoordinated suck, fever, frequent yawning, vomiting, frantic fist sucking, tachypnea, no reaction to visual stimulus, decreased attention, roving eye movement, and staring spells. Decreased birth head size, low birth weight and shortened birth length were

Table 2.1. Summary of Studies Describing the Neurobehavioral Effects of Various Drugs on Infants With PDE

AUTHORS & YEAR	PDE SAMPLE SIZE	CONTROL SIZE	AGE AT TESTING	DRUG EXPOSURE	LOCATION OF STUDY	ASSESSMENT SCORING METHOD	NEURO-BEHAVIORS
Chasnoff, Burns, Hatcher & Burns, 1981	38	0	0-4 mo	Cocaine	Special care nursery	Observation	Tremulousness, irritability, muscular rigidity
Doberczak, Shanzer, Senie & Kendall, 1988	39	0	1-10 days	Cocaine	NICU	Scoring system for opiate abstinence	EEG abnormalities, irritability, increased muscle tone, tremors, poor feeding, abnormal sleep
Chasnoff, Burns, Burns & Schnoll, 1986	38	15	Birth & 3 days	Cocaine	Nursery	BNBAS	Tremulousness, startle responses, depressed interactive behavior, impairment in organizational abilities, irritability
Oro & Dixon, 1987	46	45	Birth	Cocaine & Amphetamines	Hospital	Finnegan Abstinence Scale observations	Tremors, poor feeding, hypertonia, abnormal sleep patterns, vomiting, sneezing, high-pitched cry, frantic fist sucking, tachypnea, loose stools, fever, yawning, hyperreflexia
Erickson, Larsson, Winbladh & Zetterstrom, 1987	23	0	Birth	Amphetamines	Hospital	Observation from medical records	Seizures, lethargy, unable to feed
Kron, Litt & Phoenix, 1976	43	10 non	48 hours or less	Heroin		Observations	Irritability, stuffy nose, sweating, difficulty feeding, uncoordinated suck, Methadone more depressed.
Chasnoff, Burns, Burns & Schnoll, 1986	51	27	2 days	Heroin	Hospital	BNBAS	Decreased interactive ability, motor maturity, state control
Hans, 1989	42	47	1 day/4 wks	Methadone	Hospital/ clinic	BNBAS	hypertonicity, jerkimess, tremulousness, hyperactivity, general irritability
LeBlanc, Parekl, Naso & Glass, 1987			Birth	Cocaine		Scoring system for opiate abstinence	Tremulousness, irritability, muscular rigidity

Table 2.1. Summary of Studies Describing the Neurobehavioral Effects of Various Drugs on Infants With PDE, continued

AUTHORS & YEAR	PDE SAMPLE SIZE	CONTROL SIZE	AGE AT TESTING	DRUG EXPOSURE	LOCATION OF STUDY	ASSESSMENT SCORING METHOD	NEURO-BEHAVIORS
Fried, 1980	52	0	Neonate	Marijuana	Nursery	BNBAS	No reaction to visual stimulus, tremors, startle, less successful at self-quieting
Fried & Makin, 1987	47	0	Birth	Marijuana	Nursery	BNBAS	Increased tremors & startles, irritibility, poor habituation to visual stimuli
Hayes, Dreher & Nugent, 1988	30	26	3 days & 30 days	Marijuana	Jamaica — hospital and home	BNBAS	No significant differences
Lester & Dreher, 1989	20	20	Birth	Marijuana	Jamaica — hospital	Computer voice analysis	Cries shorter, higher frequency, more variable in tone.
Dreher, Nugent, Hudgins, 1994			3 days & 30 days	Marijuana	Jamaica	BNBAS	No significant differences
Golden, Sokal & 1980	1	0	Birth, 2 months	PCP	Hospital	Observation	Jitteriness, coarse flapping movements, hypertonicity, nystagmus, poor visual tracking. At 2 mo displays coarse tremors, ghpertonicity, roving eye movements
Strauss, Modanlow & Bosu, 1981	2	0	Birth	PCP	Hospital	Observation	Jitteriness, irritability, hypertonicity
Chasnoff, Burns, Hatcher & Burns, 1983	7	27	2 days	PCP & other drugs	Hospital	BNBAS	Irritability/lethargy, rapid changes in level of consciousness, tremors, facial grimacing, sensitivity to auditory stimuli
Howard, Kropenske & Taylor, 1986	12	None	24 hours	PCP	Hospital	Observation	Irritability, tremors, hypertonicity, abnormal eye movements, staring spells, poor sucking, lethargy, diarrhea & facial twitching
Tabor, Smith, Wallace & Yorekura, 1990	37	37 cocaine exposed	Neonate	PCP	Hospital	Observation	Hypertonicity, irritability, continuous crying, coarse flapping tremors, poor feeding, diarrhea, disorganized sucking, vomiting, restlessness, temperature instability
Rahbar, Fomufod, White & Westney, 1993	83	0	Birth	PCP	Hospital	Drug withdrawal scoring sheet	High-pitched cry, poor tracking, decreased attention

demonstrated in studies involving maternal drug use of cocaine, marijuana, heroin and PCP. Decreased head size can affect infant/child behavior, development and learning.

The Need for Improved Assessment of Neurobehaviors in PDE Infants

Conspicuously absent from the literature are assessment instruments to determine the non-normative neurobehaviors of PDE infants. Such assessment instruments are needed in order to develop, implement, and evaluate early intervention strategies.

Developmental assessment of infants and young children is part of the nursing process. Other disciplines are concerned with developmental assessment, and each discipline conceptualizes the process in a slightly different way (Lynch, 1995). For nurses, the developmental assessment includes the child's current and past health status, physical measurements, and screening of traditional developmental areas such as the child's performance on tasks in the self-care, adaptive, cognitive, fine and gross motor, perceptual, speech and language, or social emotional domains. To determine the need or eligibility for special services or intervention strategies, a comprehensive formal assessment of health status, development, and behavior is required. Four primary measures have been used to assess the neurobehavioral status of infants with PDE. These include the Bayley Scales of Infant Development, the Brazelton Neonatal Behavioral Assessment Scale, the Mullen Scale of Early Learning, and the Movement Assessment of Infants.

The Bayley Scales of Infant Development

The Bayley (Bayley, 1969) measures an infant/child's developmental status between 2 to 30 months of age and provides an assessment index of both mental development (MDI) and psychomotor development (PDI). The scales are considered a way of assessing "infant intelligence". Besides the MDI and the

PDI, the scales provide an age equivalence for both mental and motor development. The scales were developed and standardized 30 years ago with a normal population of infants and toddlers across the United States. The studies included infants and toddlers with white, black, and Puerto Rican ethnic background who lived in rural and urban communities. The scales are divided into three parts: the Mental Scale (163 items), the Motor Scale (81 items), and the Infant Behavior Record (30 items). The MDI includes skills such as perception and auditory stimuli, memory, discrimination, early verbal skills and problem solving skills. The PDI assesses various fine and gross motor abilities. The Infant Behavior Record is a rating scale that complements the Scales by providing a convenient form for reporting particular behaviors observed by the examiner during the assessment. Average testing time to administer the MDI and the PDI is about 45 minutes, but it can take up to 90 minutes. There is a manual and test kit necessary for proper administration and scoring of the scale, and formal examiner training is required. The training consists of participation in a workshop that provides an opportunity for each participant to assess a child while the instructor makes ongoing comments of the child's responses and method of examination, and further unsupervised assessment of children of various ages. Ongoing examiner administration is necessary to maintain an acceptable skill level.

Validity testing is not reported in the manual (Bayley, 1969). Reliability data is available in the manual and includes internal consistency and inter-observer agreement. Internal consistency was estimated by using split-half reliability coefficients for each of 14 age groups, and ranged from .81 to .93 on the Mental Scale and from .68 to .92 on the Motor Scale. Inter-observer agreement between the examiner and an observer for 90 infants was 89% for the Mental Scale and about 93% for the Motor Scale. Test-retest reliability was collected one week

apart on a subsample of 28 infants from the above 90 infants. The agreement between the two scores of 76% for the Mental Scale and 75% for the Motor Scale was reported. No reliability estimates were provided for the Individual Behavior Record. No separate reliability testing was provided for the neonatal period.

The Bayley has been used in research studies with infants and toddlers who have had prenatal drug exposure to alcohol (Harris, Osborn, Weinberg, Loock & Junaid, 1993; Streissguth, Barr, Martin & Herman, 1980), cocaine and/or polydrugs (Chasnoff, Griffith, Freier & Murray, 1992). It is suggested that infant ability is not a unitary trait, but a compilation of abilities (Morrison, 1992). If the PDE infant's Bayley score is within normal range as has been reported in several studies (Chasnoff, Burns, Hatcher, Burns, 1983; Chasnoff, Griffith, Freier & Murray, 1992; Griffith, Azuma & Chasnoff, 1994; Hans, 1989; Harris, Osborn, Weinberg, Loock & Junaid, 1993), then this may lead to a failure to provide necessary services and intervention, which is assessed by individual scores in subsets of development. Conversely, when the test score is outside of normal range, very little information is available regarding which domains of functioning are impaired (Jacobson & Jacobson, 1991). The Bayley clusters the test items into two scores, the MDI and the PDI. Enough is known about morbidity to not cluster all development into one or two scores if the outcome is to provide services and intervention strategies. The scales have been used in cross cultural research, with premature infants and with infants with malnutrition and anemia. The Bayley is the most commonly used measure of infant cognition and appears to be well standardized and reliable (Francis, Self & Horowitz, 1989).

There is a revised edition of the Bayley, but the original version has been used in the research studies. The studies indicate that the children assessed usually score within the normal range of intelligence quotients, (IQ) but then indicate

particular domains of deficit. Dr. Delbert Morrison (personal communication, August 6, 1997) contends that developmental domains are more important scores to report than the IQ score, as they focus on particular areas of development deficit.

A longitudinal study by Chasnoff and colleagues, (1992) using the Bayley, followed 3 groups of infants from birth. The three groups included Group l, which consisted of cocaine/polydrug exposure; Group 2 consisted of non—cocaine/polydrug exposure; and Group 3 was nondrug exposed. At 2 years of age the two PDE groups' mean developmental scores did not vary significantly from the control group. However, a larger number of infants from the two PDE groups scored greater than two standard deviations (SD) below the standardized mean score on both the MDI and the PDI when compared to the control group. Birth and 2 year old head circumference measurements of the 2 PDE groups was smaller than for the control group. The best single predictor of head circumference of the study group was cocaine exposure in utero. In the follow-up study (Griffith, Azuma & Chasnoff, 1994), the 3 year old group continued to show a decrease in mean head circumference when the 2 PDE groups were compared with the control group; however, it was not statistically significant. The 3-year-olds with head circumference below the 5th percentile made up 20% of the cocaine/polydrug group, 12% of the non cocaine/polydrug group, and 0% of the control group. In other studies of very low birth weight, infants' poor head growth was found to be a more powerful indicator of developmental outcome than birth head circumference (Eckerman, Lynne, Gross,1985; Gross, Oehler, Eckerman, 1983) Hack, Breslan, Weissman, Aram, Klein & Borawski, 1991). Hack and colleagues (1991) found decreased head circumference at 8 months predictive of poorer verbal and performance IQ scores at 8 years of age in very low birth weight infants.

Chasnoff and colleagues (1992) found that at 2 years of age there was no statistically significant difference in mean scores of the 3 PDE groups and the control group in the study. However, a significant number of 6 month and 24 month old infants were more than 2 SD below the Bayley Mental Scale mean on both the MDI and PDI. There were no significant differences found among the PDE groups and the non PDE group at age 3 on the Stanford-Binet Intelligence Scale. It was noted, however, that the cocaine/polydrug group scored lower on verbal reasoning tasks and the non- cocaine/polydrug group scored lower on abstract/visual reasoning. Both PDE groups were rated by caregivers as displaying more destructive behavior than the control group, and they were found to be more aggressive and destructive on the Child Behavior Checklist (Griffith, Azuma & Chasnoff, 1994). This study did not provide data on interrater reliability, training, or on test-retest reliability. This may undermine the construct validity testing conducted.

In summary, the Bayley Scale and Stanford Binet showed no overall differences in PDE groups as compared with the control groups. PDE status did not significantly effect IQ scores. It may be significant that Hack and colleagues (1991) found head circumference at 8 months predictive of poorer verbal and performance IQ scores at age 8 in very low birth weight infants. The problem represented by small head size may not show up until more complex series of learning are required. It might also be hypothesized that the deficit due to PDE may not be apparent in IQ testing until school age. Research by Almli and Finger (1984) indicates that deficits are frequently delayed until later stages of cognitive development.

Brazelton Neonatal Behavioral Assessment Scale

The BNBAS (Brazelton, 1984) is the most widely known and utilized neonatal assessment scale, and is the most commonly used scale with neonates with PDE

in the published research. It was designed for use with the normal neonate and scores their available responses to the environment. The scale was developed and tested in 1973 and reevaluated in 1984 with items added for high risk and fragile neonates. It is used with neonates 1-3 days old up to one month old that were 36 to 44 weeks gestation at birth. However, the first day of birth is not felt to be an appropriate time for assessment, as the newborn is recovering from the immediate stress of birth. Two or more assessments are recommended to develop an infant profile of post birth stress and environmental adaptation.

The BNBAS assesses the neurological and social interactive capacities of the neonate, and is divided into two sections. The first section is comprised of 20 elicited response items, and includes basic reflex measures of neuro-intactness. The second section consists of 28 behavioral items scored on a 9 point scale and is grouped into four categories: interactive capacities, motoric capabilities, organization of state and physiologic organization. Lester (1984) developed a cluster system of organization for data analysis purposes that is both conceptually and empirically based. The data is organized into seven cluster scores which are reflective of neonatal behavior constructs. The seven clusters include six behavioral clusters: habituation, orientation, motor, range of state, regulation of state, autonomic stability, and one reflex cluster, reflexes. Nine supplementary items were added to the second edition of the BNBAS (1984) for high risk and fragile neonates, and are used at the examiner's discretion. These additional items are designed to provide knowledge about the cost to the infant's nervous system as it organizes after a stressful labor, delivery or peri-postnatal event.

Psychometric properties are not provided in the BNBAS manual. However, many research studies testing the scale are available, one of which indicates that the BNBAS is not formally standardized, although this is reported to be in

progress (Francis, Self &Horowitz, 1989). Horowitz & Brazelton (1973) report inter-observer reliability to be consistently within acceptable limits, .85 to 1.00. The BNBAS demonstrates high inter-observer but test-retest or stability has not been demonstrated.

Reliability is established by extensive training for administration and interpretation of the scale and an Inter-observer agreement level of .90. Clinicians must conduct examinations on a regular basis to maintain an acceptable skill level (Brazelton, 1984). Training, which requires a considerable time commitment, is generally conducted through workshops at medical centers specializing in neonatal and infant care and research. Administration of the scale takes from 15 minutes to 45 minutes.

The standard neurological examination is not as predictive of later neurological status of the infant as the BNBAS (Tronick &Brazelton, 1975). The BNBAS is associated to Bayley scores at 10 weeks (Sostek & Anders, 1977) and 9 months (Vaughn, Taraldson, Crichton & Egeland, 1980) and to predictions of the quality of maternal caregiving at 3 and 6 months (Vaughn, Taraldson, Crichton & Egeland, 1980).

Clinically, the scale has been successful as a teaching and learning tool when used with a new parent. When administered with the mother present, interaction between the infant and mother, one month later, was found to be more responsive and synchronized (Widmayer & Field, 1980; Worobey & Belsky, 1982). The BNBAS has been used as an outcome measure in exploring the effects on infants with prenatal drug exposure including cocaine (Chasnoff, Burns, Schnoll & Burns, 1985; Doberczak, Shanzer, Senie & Kandall, 1988), heroin (Chasnoff, Burns, Burns & Schnoll, 1986), and marijuana (Dreher, Nugent & Hudgins, 1994; Hayes, Dreher & Nugent, 1988).

The level of clinician training for reliability and the timing of the

administration are two inconsistencies noted in research studies. The original 28 neurobehavioral items have been used in research, but the additional 9 items for high risk or fragile neonates (Brazelton, 1984) have only been employed in a few published research studies of infants with PDE (Dreher, Nugent &Hudgins, 1994).

The BNBAS is limited to use with infants during the first 30 days of life. Other limitations are the extensive training required (about one week) for administration and interpretation, the average time required for testing, and the cost involved both for learning and administration in practice settings. The scale is not likely to be used in educational and clinical settings outside of large hospitals, due to the factors cited.

Hayes, Dreher and Nugent (1988) used the BNBAS to identify neurobehavioral effects of PDE from marijuana on neonates in rural Jamaica. The sample consisted of 24 exposed infants and 20 nonexposed infants. The assessments were conducted in the hospital at day 1 and day 3, and again at 1 month of age in the hospital maternity ward. The examiner was blind to the infants' group assignment and was trained appropriately to administer the BNBAS, including the supplementary items, and maintained a .90 reliability criterion. T-tests were used to compare performance of the infants on the BNBAS at day 3, which showed no significant difference on the 7 clusters or on the supplementary items. However, at 1 month of age, the marijuana exposed infants revealed significantly higher scores than the non marijuana exposed on the Autonomic and Reflex clusters of the BNBAS and higher scores (were less irritable) on the General Irritability item of the supplementary items. The marijuana exposed infants displayed better physiological stability at l month and required less examiner intervention to reach an organized state. The infants born to heavy marijuana users were reported to be more socially responsive, less

irritable, demonstrated less variability of tone and had better self regulation than the infants of non using mothers at 1 month of age.

This study was included because it used known group techniques for construct validity. T-test comparisons were computed between the two groups to determine whether a significant difference existed between day 1, day 3, and 30 days of age for neurobehavioral effects from prenatal marijuana exposure. Interrater reliability was .90. No training details were provided nor stability measure of the BNBAS for each age group tested (test-retest reliability).

Mullen Scale of Early Learning

The MSEL (Mullen, 1995) is a relatively new infant assessment measure and is based on a neurodevelopmental and intrasensory/intersensory learning model. The scale provides assessment in 5 domains of development with t-scores and age equivalent scores for each domain: gross motor, visual reception, fine motor, receptive language, and expressive language. A single composite score represents general intelligence. Each domain is evaluated individually with an age and t-score. T-scores falling between 40 and 60 are considered to be within the average range. The scale is appropriate to use with infants from birth to 36 months with an administration time of less than one hour. Average training time is 8 hours. The scales can be used individually, thereby allowing clinicians to assess strengths and weaknesses in particular domains and make appropriate recommendations for interventions.

The scale was standardized on 1,231 children 1 month to 37 months of age in 100 sites in the United States (U.S.). The sample approximated the population demographics (sex, race, parental occupation and urban-rural residence) of the U.S. census of 1989. Acceptable estimates are established for concurrent validity, test-retest reliability, interrater reliability, and internal consistency. Using a composite score of the four mental scales, the MSEL correlates .97 with the

Bayley MDI, indicating that the MSEL could be viewed as a measure of general intelligence comparable to the Bayley. However, there is some evidence that infants with PDE obtain lower scores on the MSEL than have been reported in previous research using the Bayley (Morrison, 1992). The few studies on long term cognitive development of PDE infants demonstrate that this population functions within the normal or low normal range and often the same as the comparison group. The comparison group is generally from the same demographic area and shares the same maternal characteristics (Chasnoff, Griffith, Freier& Murray, 1992; Griffith, Azuma & Chasnoff, 1994). The MSEL has been used with high risk infants such as those with prenatal drug exposure (Morrison & Villarreal, 1993).

The Mullen was used in two recent clinical studies (Morrison, 1992) of children with drug exposure, and has found that these children obtain lower t-scores than the norm. The Infant Development Service (KIDS) of the Easter Seal Program in San Rafael, California, found that 32 infants with PDE and a mean age of 15 months showed significant delays on the MSEL; 43% in gross motor, 42% in visual expression, 40% in visual reception, 38% in language reception, and 43% in language expression. The researchers used the criterion of a t-score of 32 or less (about one SD below the mean) and an age score of a month or more below chronological age and found the following delays; gross motor=13 (40%), visual reception = 14 (43%), visual expression =16 (50%), language reception = 19 (59%), and language expression = 13 (40%). Similar findings have been reported for 23 children with a mean age of 18 months at the Infant and Early Childhood Evaluation Clinic at Langley Porter Psychiatric Institute, with delays being registered in 8 (40%) for gross motor, 11 (52%) for visual reception, 12 (52%) for visual expression, 11 (52%) for language reception, and 8 (35%) for language expression (Morrison, 1992). Neither information on reliability of the assessment

measure nor clinician training is provided.

Certainly these studies provide evidence of the usefulness for assessment of separate domains of cognitive performance in order to provide necessary and appropriate interventions. The studies are limited by the small sample size and possible Type II error. Internal validity is threatened by the possible selection bias and nonblinded examinations with lack of control of any confounding effects.

Movement Assessment of Infants

The MAI (Chandler, Andrews & Swanson, 1980) was created out of the need for a consistent approach to the evaluation of motor function of infants birth through 12 months. The inventory consists of 65 test items and evaluates four areas of motor movement, which include muscle tone, primitive reflexes, automatic reactions, and volitional movement. Muscle tone items are rated on a 6 point scale while the other three categories are rated on a 4 point scale. Administration time is generally 30 to 40 minutes. The four risk areas are scored and summed to yield a total risk score at four months of age. The assessment of risk for motor dysfunction was provided using an a priori profile of normal 4 month old motor behavior.

Both reliability and validity studies have been reported on the normal profile. Predictive validity with a sample of 246 high risk infants found significant correlations of the MAI total risk score with developmental evaluations using the Bayley Scales of Infant Development at one and two years of age (Harris et al, 1984). Interrater reliability for the MAI total risk score at four months has been reported as 0.72 and 0.90 or above (Chandler, Andrews & Swanson, 1980; Harris, Haley, Tada & Swanson, 1984). Another study found the MAI to be twice as sensitive as the Bayley Motor Scale in detecting early indications of cerebral palsy (Harris, 1987). The MAI has been used in studies with infants prenatally

exposed to cocaine and polydrugs (Schneider, 1988; Schneider & Chasnoff, 1992) and to alcohol (Harris, Osborn, Weinberg, Loock & Junaid, 1993).

Schneider and Chasnoff (1992) compared 74 cocaine/polydrug exposed infants with 50 nondrug exposed infants at 4 months of age which were recruited from an ongoing comprehensive drug treatment program for pregnant and postpartum women. They were of various ethnic background, and from low income families. The polydrug group were identified by toxicology assay and the comparison group by maternal history and self report. There were no controls for confounding effects and nonblinded exams. Examiners were trained in the MAI and followed the procedure with checks on interrater reliability. Results indicated the mean total risk score for the cocaine/polydrug infants was higher than for the control group. The risk scores for the categories of muscle tone, primitive reflexes, and volitional movement were significantly poorer in the drug exposed group. The greatest differences between the two groups were in the muscle tone and primitive reflex categories, including increased tremors and extensor muscle tone. Construct validity was determined by the known group technique and differentiated risk, but may be limited due to non-blind examinations. Prior knowledge regarding group placement may influence examiner assessments. Possible selection bias is a threat to internal validity. Interrater reliability findings are not elaborated upon. The results only apply to 4-month-old infants with PDE from the same community of infants.

Summary of Assessment Measures

The Bayley, BNBAS, Mullens and the MAI measurement instruments provide useful and descriptive information (see Table 2.2). While these are excellent tools to determine the developmental level, motor, or cognitive performance of the infants or interactive behavior of the neonate, these tools often are not sensitive to the subtle behaviors exhibited by infants with PDE, such as passivity,

TEST	YEAR	AREAS OF ASSESSMENT	# OF ITEMS	AGES	N	CON-TROL	SITES	STRATA	VALIDITY	RELIABILITY	TRAINING/ ADMIN
1. BAYLEY	1964 Across USA	Mental Motor Behavior Summary scores for mental & motor abilities	MDI-163 PDI-81 IBR-30	2-30 mo. White Negro Puerto Rican Ruran & urban	1262	0	Clinic	Gender Race Parent ed. Age-census 1960	Content Correlations with Stanford Binet	Interrater 90% Test-retest Split-half coefficien adequate	7 days + 45 - 60m
2. BNBAS	1973 Re-evaluate 1984	Neurological, social, and interactive capacities	28B 20R 9 high-risk infant items	1-30 days (36-44 wks. gestation)	varies: small different studies	0	Hospital		Predictive concurrent face	Other researchers Interobserver	ten infants 3-5 days 15 min. +
3. MULLEN	1981-1989 > 100 sites in U.S.	Gross motor Visual reception Visual expression Language reception Language expression Measures developmen in 5 domains		1 month to 3€ months Black Hispanic-bilingual Asian Indian White Urban and rural	1231	0	100 sites: day care nursery home	Age Gender Race Parent occupation Residence U.S. census 1989	Concurrent MSEL Mentals & Bayley MS MSEL Composites & Bayley MS MSEL & Preschool Language Construct Criterion-rel ted-predictive	Test-retest 2 wks. .70-.99 Interscorer .78-1.0 Internal consistency alpha on 5 scales .95	8 hrs. 60m
4. MAI	1980	Muscle tone Primitive reflexes Automatic reactions Volitional movement	65	B-12 mo. Rural	35	0		4 mo. 12 mo.	Predictive with Bayley at 1 & 2 yrs	Interrater-4mo. (item; rater)	1-3 days 30 min to 1.5 hr.

hyperactivity, feeding difficulties, eye muscle imbalance, and imitating and maintaining eye contact.

For example, when the Bayley's composite score is used in research, or when an overall poor score is reported, the results do not pinpoint whether there is a specific area of impaired function, whether it reflects global or generalized functioning, or whether strong scores in one area are masking scores that reflect impaired functioning in another area. Thus the Bayley score alone may not be helpful in planning specific interventions in areas of need, and may not give information regarding the integrity of the self regulatory system or the integrity/ age appropriateness of attending behaviors.

All of these assessments are structured scales and can be administered in a hospital, home or office, and all are conducted in a one-to-one situation. The testing format allows the examiner to facilitate the infant's focus and state regulation during administration of test items. The Mullen does not give us information regarding the integrity of the infant's self-regulatory system, their typical ability to sustain self focus, or joint/mutual focus on activities in a typical environment. It also does not give us important information regarding visual reception and expression skills, social skills such as eye contact, visual monitoring, and visual attention to self-initiated tasks, all of which are involved in later learning success and would be important to know in developing appropriate infant specific intervention strategies. Chasnoff, Griffith, Freier, & Murray (1992) suggest that the examiner's intervention during testing with the BNBAS and Bayley may mask self-regulatory difficulties.

Traditional methods of infant assessment alone, such as the Bayley and BNBAS, may not be the best way to provide a complete picture of the infant's developmental status (Clark, Paulson & Conlin, 1993). The state of Michigan has enacted legislation entitling all disabled children to comprehensive special

education services from birth through age 26 since 1971. Their data indicate that a large percentage of young children with disabilities are not initially identified when early data are examined, so the opportunity for early intervention is missed. They report .7% of children are identified by age two, but that 7.5% are identified by age six, and 12.6% by age nine . One of the primary reasons they cite for the lack of early identification is that there are few valid instruments available for use with children birth to three years (Cicchetti & Wagner, 1990; Meisels, 1988, Meisels & Wasik, 1990). The traditional assessments currently being done are identifying only a small percent of the infants who are eventually identified with disabilities. There is a need for an assessment measure that provides information regarding behavioral and neurobiological systems which are not currently being evaluated with traditional assessments. This may increase the success of early identification for all infants who may be at risk but are currently undiagnosed.

Systems Theory as a Conceptual Model to Guide the Measurement of Neurobehavioral Status

Systems theory provides a useful framework for assessing the neurobehavioral status of the drug exposed infant, including the complex nature of the infant's biological and behavioral subsystems and the infant's external subsystems as part of a family and larger community. This theory can be discussed in a broader sense or narrowed down to specific internal and external subsystems.

An open system takes in and exchanges energy, matter and information within the entire system to create and nurture increased order and complexity. This is known as negentropy (von Bertalanffy, 1968). In an open functional system an infant learns to take in information and modulate, integrate, or inhibit (adapt) this input to maintain the system in a state of organization or homeostasis.

Turner (1991) proposed that the general systems theory (von Bertalanffy, 1968) be expanded to include the notion that cause and effect relationships occur within a complex system of interactional relationships. The whole system, as well as the subsystems, needs to maintain an organized state instead of a disorganized state for optimal growth and development to occur. The organized state embraces the notion of a balance between change and stability coexisting within and outside the infant's system (Wright & Leahey, 1984). The system depends on a circular feedback mechanism which is the systems response for intervention with stress-producing stimuli to maintain self organization (Mercer, 1989) and self regulation. The system is interconnected, and the functioning of one part needs to be viewed in the context of the interactions within the whole system. For example, if an infant is in a sitting position, begins to lose balance and falls over, a protective response of arms forward and hands out will occur. This is an automatic adaptive reflex or micro-feedback loop, which is a protective and adaptive response of the system. Another example of the micro-feedback loop as a system response for intervention is when an infant is placed in a backpack, which challenges the whole system due to the various atypical movements and increased visual stimulation experiences. These stress-producing experiences of moving and tipping from side to side and the accompanying sensation of being off-balance as well as the increased visual stimulation can be modulated and adapted by the whole system. These examples of a micro-feedback loop increases one's awareness of the infant's organization capacity to obtain developmental goals and adapt to a particular variable. See Figure 2.1 and Figure 2.2 for micro feedback loop described above.

The world view of the human infant is changing. The infant is not viewed as primarily passive or undifferentiated. The infant comes into the world with particular biological active propensities and with organized capacities for self-

Figure 2.1. Micro-feedback loop of an infant in sitting position losing balance.

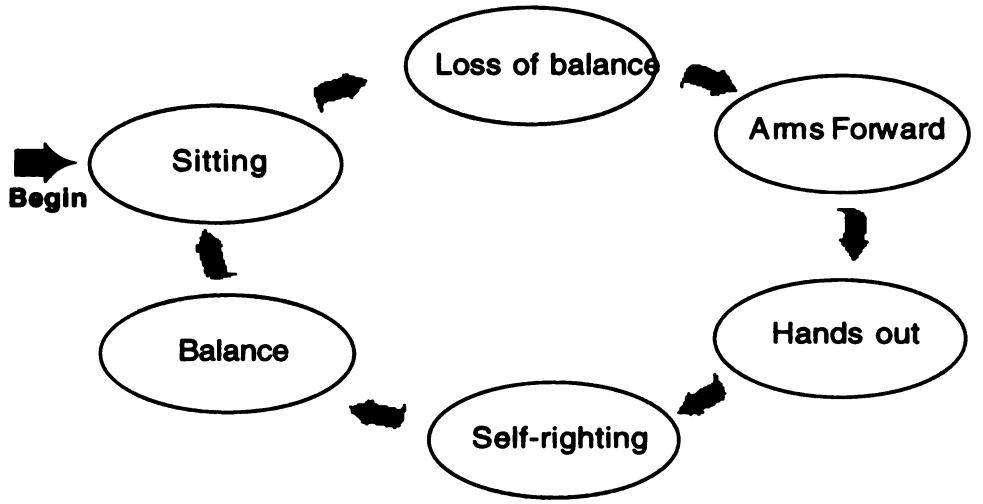
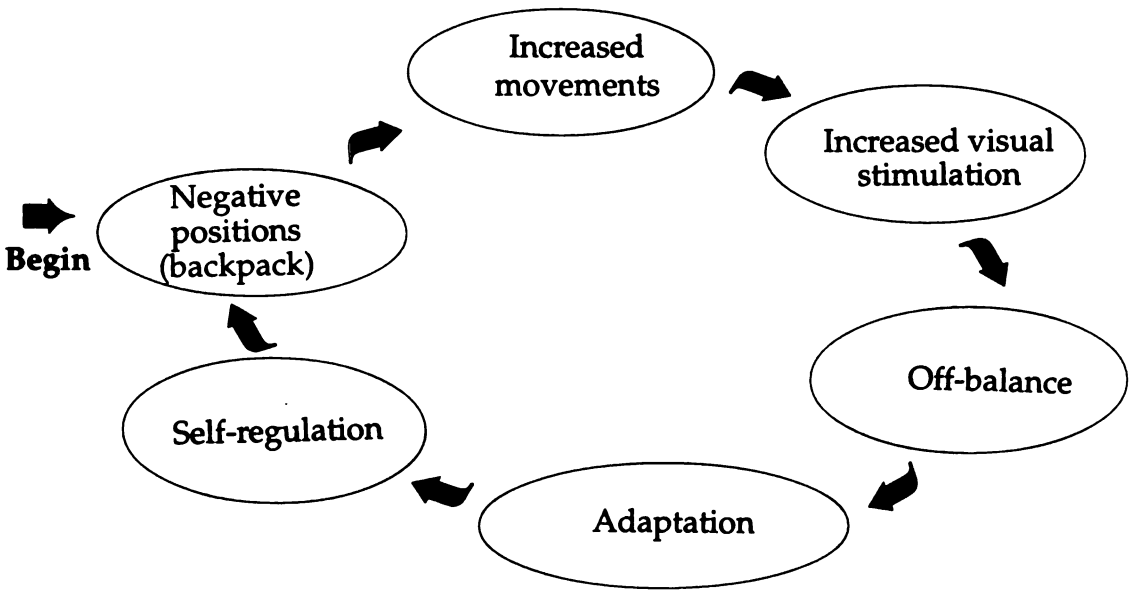


Figure 2.2. Micro-feedback loop of an infant adapting to placement in a backpack.



regulation of the system (Emde, 1989). The infant subsystem behaviors can be observed in sleep-wake cycles, self-comforting strategies, movement patterns, muscle tone, visual motor system functions, and interactional capabilities of the infant. These behaviors can include eye contact, soothing by the human touch, alertness to the human voice or music, initiating as well as sustaining and terminating human/object communication, and modulation of the motor system. If the infant with PDE's subsystem becomes disorganized, the infant cannot maintain homeostasis within the system.

If one infant subsystem is compromised or is unable to adapt, other subsystems and/or the whole system can also be compromised and in a state of instability and disorganization. The disorganized subsystem can influence other subsystems, both within and between subsystems, reducing optimal and integrated response to the environment. However, the overstimulated infant's system may allow the infant to sleep, which may permit time for the disorganized subsystem to recover from the instability. This micro-loop provides a protective response of sleep to maintain the infant system's integrity, stability and homeostasis.

Closed subsystems can occur within an open system. This can happen when an infant with PDE who is overstimulated stops all visual, auditory, or sensory response to the environment or, in effect, shuts down to protect the whole system.

If one fetal subsystem is compromised or is unable to adapt, other subsystems and/or the whole system can be compromised and in a state of disequilibrium. The subsystems influence one another, both within and between subsystems. The fetus exists in a complex and organized environment. The fetus is continually acting on as well as acted upon in this environment, and any invasion of drugs can affect the fetus. Negative effects to the fetus can occur with

episodes of maternal heroin withdrawal during pregnancy, which is reported to restrict fetal growth by decreasing uterine or placental blood flow (Naeye, 1965). Increases in muscle activation and increased metabolism and oxygen consumption occurs with severe maternal withdrawal, and this may also increase fetal activity and thus increase metabolic demands (Finnegan, 1976). These are examples of disorganization between subsystems and how subsystems influence each others' functioning.

Kopp (1982) suggests that modulation of physiological arousal in early infancy and organized responses to environmental stimuli during the first year of life depend mainly on constitutional factors, along with the caregiver's ability to respond to the infant's cues, to provide predictable routines, and to prevent overwhelming frustration. During the first year of life normal development is dependent upon both the infant's ability to modulate its reactions and affective and behavioral states along with the necessary caregiver support (Beeghly & Tronick, 1994). Infant self regulatory behaviors include physiologic mechanisms (both cardiac and respiratory systems), coping behaviors, (e.g., self comforting measures and withdrawal), attentional mechanisms, and cognitive and communicative abilities (Beeghly & Tronick, 1994). These behaviors are a reflection of the infant's ability to respond to external and internal ongoing events. Normal development is dependent upon the infant's capacity to control his or her affective and behavioral states and organization, and the caregiver's ability to facilitate the infant's self-regulatory actions (Beeghly & Tronick, 1994).

Kopp also proposes that early modulation of behavior and later self-regulatory abilities stems from both individual differences that have biological roots and from the quality of the caretaking environment. Prior to self regulation, the infant develops modulated states of arousal and organized patterns of functional reflexive movements followed by an ability to perform

voluntary motor movements, which have the capability of modulating attention and social exchanges as well as sensorimotor system (Kopp, 1982). Finally, the infant gains control by demonstration of initiating, modulating, maintaining or ceasing physical activities, communication, or emotions.

Tronick (1989) postulates that there are routine regulatory failures that occur during early social exchanges which result in disorganization of the infant. If these failures are of a short nature, then they are felt to be growth promoting and contribute to the infant's ability to self regulate, and are a part of normal infant development. If there is chronic regulatory failure, the infant becomes disorganized and unable to return to and maintain a steady state. This can compromise the infant and may play a part in long-term negative developmental consequences (Beeghly & Tronick, 1994).

Organized Infant

An organized infant is an infant who is capable of adapting to the extrauterine environment in terms of physiologic and behavioral responses to external events (D'Apolito, 1991). An organized infant is able to maintain the physiologic and behavioral systems in order to integrate these systems into smooth, purposeful movements and steady autonomic states during interactions with the environment. An organized infant can maintain a steady and stable physiological state with the ability to maintain smooth and continuous behavioral functioning. He or she is able to self comfort as well as to respond to caregiver efforts and to environmental strategies for comforting.

Disorganized Infant

The disorganized infant, such as the infant with PDE, may be unable to organize the system or to demonstrate and maintain neurobehavioral responses that are appropriate to make smooth transitions from one activity to another. Disorganized infants demonstrate greater depression of interactive behaviors, poor state organization (Chasnoff, Burns, Schnoll, & Burns, 1985) impairment of orientation, atypical motor and state regulation behaviors, and abnormal reflexes

at birth and in infancy (Chasnoff & Griffith, 1989; Lewis, Bennett & Schmeder, 1989). If disorganized behaviors or imbalances are not resolved, the system can further be compromised in both physiologic and behavioral functioning, which may contribute to compromised development and later poor school performance. Self regulatory behaviors can help to balance the system, along with the assistance and support from the caregiver and environment. Infant states of physiologic and behavioral organization and disorganization are presented in Table 2.3.

Researchers report neurobehavioral symptoms of infants with prenatal drug exposure such as tremors, jitteriness, irritability, hypertonicity, hyperactivity, high pitched and continuous crying (Chasnoff, Burns, Schnoll, & Burns, 1985; Chasnoff & Griffith, 1989; Doberczak, Shanzer, & Kandall, 1988; Lewis, 1991). On the BNBAS, several studies describe infants with PDE displaying compromised patterns of neurobehavioral organization such as impaired interactive abilities, poorer state organization, and habituation, as compared to a drug free control group (Chasnoff, Burns, Schnoll, & Burns, 1985; Chasnoff, Burns, & Burns, 1986; Richardson & Day, 1990). These behaviors and compromised patterns of neurobehavioral organization may be a direct result of PDE or they may be the result of indirect effects of PDE produced by other factors such as intrauterine growth retardation, maternal nutrition, and pre and postnatal factors. These studies suggest abnormal neurological symptoms and regulatory dysfunction in infants with PDE. Any one of these atypical neurobehavioral responses, if consistent and chronic, can disrupt the infant's capacity to self regulate behavioral and physiological states and disturb interaction with objects and people in the infant's environment.

Conceptual Model

The proposed conceptual model defines self regulation in terms of the integrity of the neurobehavioral responses and how they affect each other, both

Table 2.3. Organized and Disorganized Responses of the Infant

Organized responses

Physiological Symptoms

- Stability of heart and respiratory rates
- Consistency of skin color
- Tolerance of feedings

Behavioral Symptoms

- Smooth and synchronous body movements
- Smooth transitions between sleep and wake states.
- Ability to self-comfort, suck finger, position change, hand to face movements
- Ability to be comforted from the environment
- Ability to organize and shut out noxious stimuli by decreasing body movements or modulating from an awake to a sleep state.

Disorganized responses

Physiological Symptoms

- Fluctuations in heart and respiratory rate which may result in apnea, bradycardia, or tachypnea.
- Color changes
- Difficulty tolerating feedings, regurgitation
- Increased stools and change in consistancy
- Sweating, hiccoughs, yawning or sneezing

Behavioral Symptoms

- Changes in muscle tone, high, low, fluctuating, tone difference
- Frequency or rapidity of state changes, irritability/passivity, increased startles
- Difficult to comfort, limited ability to self comfort
- Tremors and jerky movement patterns
- Limited modulation of visual system

(Adapted from K. D'Apolito, 1991.)

within and between subsystems. The following subsystems provide a more complete picture of the infant with PDE and are important in understanding the infant's strengths and areas of risk or atypical responses. These subsystems include High Reactivity, Low Reactivity, Atypical Visual Functioning, Atypical ANS Response, Compromised System Regulation, Atypical Communication Patterns, and Atypical Play Response. An infant's subsystem is defined in terms of the neurobehavioral responses observed in infants with PDE and the ways in which the antecedent responses of self regulation might influence current and later development.

Infants with PDE often have difficulty within the High Reactivity and Low

Reactivity Subsystems. A disorganized nervous system can interfere with the infant's ability to regulate arousal states (Griffith, 1992). These difficulties may manifest by frequency or rapidity of state changes, passivity, irritability, or a tendency to become easily overstimulated and have difficulty interacting with their environment. How does the infant adapt and accommodate to disorganized arousal states or stress within the subsystem? Some infants have the ability to shut down and sleep, while others become more agitated by the stimuli and continue to cry, and still others may exhibit signs of extreme autonomic nervous system distress such as sneezing, yawning, and hiccupping. The extreme lack of self regulatory ability may interfere with caregiver/infant attachment and interaction which may create or contribute to an environment of physical abuse or neglect.

Lethargy, frequent increased startle response, tremor, and hyperactivity are examples of atypical neurobehavior subsystem responses of infants with PDE. The responses suggest an atypical quality to the behaviors, and are suggestive of central nervous system disorganization. Extreme lethargy, tremors and hyperactivity can be responsible for missed opportunities for developmental learning. Tremors can add a level of fatigue to the infant's system due to the extra effort he or she expends to control the movement, making motor tasks less pleasurable.

Difficult feeding is observed in infants with PDE. These infants may exhibit a variety of atypical oral motor behaviors, including uncoordinated suck-swallow pattern, or preemie-like suck pattern, inability to stabilize tongue in midline, and tongue tremors. They may also show other signs of biological symptoms such as regurgitation and loose stools. The abnormal oral motor behaviors may increase feeding time, and consequently require increased energy from the infant to accomplish the task. This dilemma may precipitate stress and frustration in the

infant and the mother.

Difficulties in the Atypical Visual Functioning subsystem might include difficulty tracking and initiating, gazed aversion, and maintaining eye contact. Gaze aversion or decreased eye contact with the environment can delay or impair development of visual attention skills, visual maturity, use of binocular vision skills and development of imitation skills, which can interfere with later learning and academic performance. Any of these deviations from the norm can effect social and communication skills, and may impair or delay visual maturity.

The atypical ANS response subsystem focuses on behaviors such as those observed in an infant during a stressful situation, and include sweating, frequent yawning, hiccupping, and sneezing. These behaviors are not subject to voluntary to control by the infant, but are necessary to help the subsystems mitigate stressful circumstances. If the stress-producing encounters are of short nature, they can be growth-promoting and contribute to the subsystem's overall ability to self regulate. If the infant's neurobehavioral responses continue and become chronic, the subsystem may not be able to maintain a steady state. Due to the infant subsystem's disorganization, negative developmental consequences may occur. This difficulty with imitation skills may place the infant with PDE at risk for specific attention difficulties in later school experiences.

Muscle tone subsystem variations noted in infants with PDE such as low or high muscle tone or tone differences in the extremities can interfere with acquisition and refinement of fine and gross motor activities. Hypertonicity can cause the infant to roll over at a few weeks of age, interfere with the ability to cuddle, delay pull to sit and control of arms to midline. These behaviors can make it difficult to complete a task or repeated attempts at the task to be successful. More energy is used to accomplish fine and gross motor tasks, thus some level of frustration is created (Lewis, Schmeder & Bennett, 1992).

Atypical responses in movement patterns are observed in the PDE infant. When the infant has limited or exaggerated movement patterns (High Reactivity and Low Reactivity items) such as low muscle tone, high muscle tone, or displays an intolerance difficult to cuddling due to their atypical movement patterns or muscle tone, the whole system is affected. If the infant's movement subsystem is reflecting difficulty with several responses, the subsystem might be completely out of balance and create difficulty for the infant to modulate and control the motor behaviors. If there were only one or two areas of imbalance, then the subsystem might be able to modulate and maintain appropriate performance, thereby self regulating the subsystem.

Differentiated cries, social laugh and smile, vocalization to the caregiver's response, and intonation are all part of the infant's communication subsystem. Any atypical behavior in this area can affect the infant and caregiver dyad, social interaction with the environment, and visual and auditory responses. These early language signs of communication by the infant may not affect current self regulation, but have an impact on the infant's internal and external environment, which affects later self regulation.

The atypical Play Response in this model is limited in infancy to imitating with objects and people, functional use of toys, initiation of play with objects and people, and distractibility and level of frustration. Being able to perform these tasks involves a level of control and attention by the infant. If the infant is unable to successfully control and modulate reflexive movements and neurobehavioral responses, or is easily distracted or frustrated, then the early play schemas may be delayed, or the quality of interaction may be compromised. These play behaviors are part of a repertoire of early play skills and involve both cognitive and communication abilities.

Summary

The development of self regulation in the infant involves attainment of modulation of functional states of behavior, development of the ability to perform voluntary motor movements as well as a broad repertoire of movements, and finally, the demonstration of emerging skills of initiation, modulation, and communication (Kopp, 1990). If attainment of self modulation and control of neurobehavioral responses is compromised for infants with PDE, then later self regulation may also be compromised, which may affect later cognitive performance, learning behavior and social stability. When infant system functioning is optimal, normal infant development is facilitated.

This conceptualization focuses on physiological and behavioral control for self regulation and looks at development through the integrity of the subsystems. Its usefulness lies in the explanation of how subsystems can interact and how these interactions can affect both within and between subsystems and the infant's whole system. The model conceptualizes the infant by viewing the functioning of the infant's individual subsystems and neurobehavioral responses as part of the infant's developing complex system.

Table 2.4 delineates the specific neurobehavioral characteristics within this conceptual model and their relationship to the specific subsystems of the model. The table also shows the capacity of existing infant assessments to measure these important characteristics. Clearly, a more comprehensive assessment is needed to assure that early identification of infants that demonstrate atypical neurobehaviors is made. This will facilitate identification, intervention, evaluation and follow-up in an efficient and timely manner. This conceptual model has served as the basis for development of the Lewis Neurobehavioral Assessment Scale (LNAS), the instrument to be tested within the study. The LNAS presents the complex neurobehavioral symptoms and interactions

exhibited by infants with prenatal drug exposure in their environment. When possible, the neurobehaviors and interactions are observed and documented in the infant's own environment, thus representing naturally occurring neurobehaviors and experiences.

Table 2.4. Cross-reference of Neurobehavioral Characteristics of Infants With PDE Cited in Literature and Included in Various Assessment Instruments

Characteristic	MSEL	BNBAS	MAI
HIGH REACTIVITY			
Irritibility		X	
Frequency or rapidity of state changes		X	
Difficult to comfort		X	
Frequent startle response			
Hyperactivity			
High muscle tone			
Tremors			
LOW REACTIVITY			
Passivity			
Dull alert state		X	
Lethargy			
Low muscle tone	X	X	X
Difficulty feeding			
ATYPICAL VISUAL FUNCTIONING			
Difficulty initiating eye contact	X		
Difficulty maintaining eye contact	X		
Gaze aversion			
Difficulty tracking	X		X
ANS ATYPICAL RESPONSE			
High-pitched cry			
Sweating			
Frequent yawning			
Hiccupping			
Sneezing			

Characteristic	MSEL	BNBAS	MAI
COMPROMISED SYSTEM REGULATION			
Jerky eye movement			
Unexplained fevers			
Increased respiration			
Nasal stuffiness			

Characteristic	MSEL	BNBAS	MAI
ATYPICAL COMMUNICATION PATTERNS			
Undifferentiated cries			
No social laugh	X		
No social smile	X		
Limited vocalizations	X		
Limited vocalization to caregiver's response	X		X
ATYPICAL PLAY RESPONSE			
Limited imitation with objects, people	X		
Limited functional use of toys			
Limited initiation of play			
Distractible			
Easily frustrated			

CHAPTER THREE

Methodology

The focus of this methodology chapter is to document the methods used to establish validity and reliability of the LNAS. Validity methods used for current testing included construct and predictive validity while reliability methods included interrater, test-retest and internal consistency. Prior validity and reliability testing is also addressed in this chapter, which focused on content and construct validity and interrater, test-retest reliability and internal consistency reliability.

A cross-sectional design using a convenience sample of 80 newborns was employed to determine the psychometric properties of the LNAS (see Appendix A for instrument). Forty infants with PDE and 40 non-drug exposed infants were assessed and compared during the first two weeks of life to determine validity and reliability of the assessment instrument. Of the 80 newborns assessed, 16 were assessed again at six months during a home visit. These six-month-old infants received the LNAS and the Mullen Scales of Early Learning (Mullen).

All newborns in the study had the revised LNAS, Nursing Child Assessment Feeding Scale (NCAFS) and the Parmelee Complications Scale administered at the birthing hospital or in their own homes when they were between 6 and 14 days of age. One week after the initial assessment, sixteen PDE infants had the LNAS administered again. Nurse clinicians who were conducting the assessments were blind as to which infants had prenatal drug exposure and which did not.

Sample

Research Settings

All infants in the study were referred to the investigator by professionals from one of four collaborating agencies — hospital, educational program, public health department, or pediatrician's office. The sites were chosen both for their composite cross

section of racial/ethnic infants and the availability of PDE infants. Both PDE and non-PDE infants were recruited from the same agencies.

Nature and Size of Sample

Sample size was based upon having adequate power to determine internal consistency of the LNAS. Nunnally (1978) states a minimum of 5 subjects per item should constitute any sample for instrument development. Testing of internal consistency within each subscale with a maximum number of seven items in each subscale required 35 infants per group. The sample included 40 infants with PDE and 40 infants without PDE.

Criteria For Sample Selection

Qualification for inclusion in the PDE group was either determined by a positive urine toxicology screen at birth for the mother and/or the newborn or by the mother's self-report of drug use during pregnancy. Drug exposure included one or more teratogenic drugs: amphetamines, barbiturates, benzodiazepines, cocaine, marijuana, methadone, opiates or PCP. Urine toxicology screens at birth only indicate drug exposure from 2 to 4 days prior to maternal use, making it difficult — if not impossible — to know what other drugs the fetus may have been exposed to during utero. The drugs included in this study were cited in the literature as drugs of choice by women who abuse drugs. No difference in infant neurobehavior symptoms was expected, based on drug exposure, as the literature consistently cites similar symptoms exhibited by infants with PDE across individual drug studies. (Chasnoff, Burns, Burns, Schnoll, 1986; Fried, 1985; Fried & Makin, 1987; Hans, 1989; Oro & Dixon, 1987; Tabor, Smith-Wallace & Yorekura, 1990). The drugs cited here were also included in routine hospital toxicology drug screening procedures at the sites chosen for inclusion in this study. A standardized way for assessing drug exposure across sites was important to assure reliable identification of a representative sample of drug

exposed infants.

Qualifications for inclusion in the comparison group were a negative urine toxicology screen at birth documented in medical records and/or a signed statement from the birth mother that she did not use drugs or alcohol during her pregnancy. This method of identifying PDE infants and non-PDE infants is consistent with other studies (Dixon & Bejar, 1989; Doberczak, Shanzer, Senie & Kandall, 1988; Eisen, Field, Banstra, Roberts & Morrow, 1991; Tabor, Smith-Wallace & Yonekura, 1990; Vega, et al, 1993).

Exclusion criteria were used in an attempt to achieve a more homogeneous sample and to decrease the likelihood of developmental risk associated with other factors that are known to affect infant developmental progress and outcome. Infants, both PDE and non-PDE, with the following conditions were excluded from the study:

1. Infant birth weight less than 1500 grams.
2. HIV serum positivity, congenital anomalies, Downs Syndrome, hearing deficit, or blindness.
3. Prenatal drug exposure to alcohol or cigarettes.

Human Subject Protection

After agency referral, the infant's caregiver was recruited by telephone by the researcher. The researcher discussed the purpose of the study, the voluntary nature of the participation, the fact that declining or accepting the invitation to participate would in no way affect the services the infant's caregiver might receive, the manner in which the infant was identified as a possible subject, the nature and length of the participation, the nature and length of the assessment, the scheduling requirements, and the risks and benefits, the confidentiality of the interview and answered any questions about the assessment process. The investigator discussed the reporting obligation if child abuse or family violence was

observed or disclosed during assessment. Consent forms as seen in Appendix B were obtained before any assessments were conducted.

The assessments used are accepted tools used in clinical practice. They did not present any unusual risk or harm to the infant. However, the testing was paced to reduce any possible stress, and the assessments did not have to be stopped due to the child experiencing undue distress or fatigue. If a developmental delay was observed, the caregiver was advised, and referral options were discussed. Seven infants were referred for early intervention services, two at birth, five at the six-month assessment.

To protect against risks to confidentiality, code numbers were assigned to each participant in the study. The nurse clinicians conducting testing were aware of names of the infants and their caregivers for assessment purposes only. All copies of assessments, background information, etc. were kept in a locked file drawer at one site, and computer files were accessible only to the researcher. After the assessments were completed, the researcher assigned a code number to the infant, thereby protecting the identity of participants. Individual participant identification was kept separate from the data, and a master list of participants was maintained by the researcher. Human subject approval was granted by the Committee on Human Research (CHR) at the University of California, San Francisco (UCSF), Project #96012454, on March 13, 1996. Renewal was approved on April 17, 1997.

The immediate benefits of participation for the infant in this study were the sharing of the infant's developmental performance with the caregiver and/or the referring agency. The sharing of information with the referring agency was only done with the caregiver's concurrence. This allowed for early identification of infant risk for treatment and follow-up services. A long-term benefit of the study is a clearer understanding of prenatal drug exposure on infant development and improved interventions for the future.

Data Collection Methods

Description of Instrument to be Tested

The LNAS is a clinical and research instrument designed to identify and

describe neurobehavioral characteristics of the PDE population from birth to 12 months of age. The instrument can be used alone or in combination with other measurements. The LNAS consists of two parts: Part A includes demographic information for descriptive purposes, Part B contains a frequency rating scale.

Part A of the LNAS is comprised of 15 demographic questions regarding the infants and can be completed by using data gathered from the caregiver, the clinician's assessment of the infant and medical records. These demographic data regarding the infant include: date of birth, age, anthropometry, gender, ethnic background, place of residence, number of foster home placements, hearing, and medical and drug diagnoses. This section of the tool provides important information about each infant's background and environment which could affect growth and development.

Part B of the LNAS consists of 35 items distributed across seven subscales which reflect the physical, motor, and communicative areas of development for the birth to 12 month population. Part B was the focus of testing in this research study. There are 35 items included in the following subscales: High Reactivity (7), Low Reactivity (5), Atypical Visual Functioning (4), Atypical Autonomic Nervous System (ANS) Response (5), Compromised System Regulation (4), Atypical Communication Patterns (6) , and Atypical Play Response (5). The first five subscales can be administered to infants from birth through 12 months of age. Atypical Communication Patterns and Atypical Play Response subscales are used if the infant is six months of age or older. Because this study focused on assessment of infants within the first two weeks of life, only the first five subscales were tested at this time. Each item is rated on a 5-point Likert rating scale, which measures the frequency with which these neurobehavioral characteristics appear, ranging from 1 (never), to 5 (almost always). Possible scores range from 35 to 175. A high score reflects a large number of high risk neurobehaviors and a more disorganized infant.

Previous Validity and Reliability Testing

Preliminary psychometric testing of the Lewis Protocol (LP) was conducted prior to this dissertation. The findings from that pilot study are presented in this section. After the completion of the pilot study, the name of the assessment instrument was changed from LP to LNAS.

Content validity. Fifty items were initially selected for Part B of the original LP because they represented particular aspects of infant functioning that were potentially susceptible to early intervention or treatment. Selection for both item content and definitions of neurobehavioral characteristics for the LP were derived from clinical experience, a review of the literature, and recommendations from a panel of experts. Two stages for determining content validity, as outlined by Lynn (1986) were used to validate the original study. The first stage, termed the developmental stage, was initially determined for the LP in three ways: First was by using the LP with infants during initial eligibility assessment for early intervention services. Second, a review of the literature was conducted to 1) describe known neurobehavioral characteristics of infants with PDE, 2) substantiate behaviors observed in these infants in clinical practice and 3) uncover formal screening assessments useful with this population.

Finally, a checklist was developed with item generation flowing from direct observations and a literature review. The checklist was continually modified and revised as the physical, motor, and social dimensions for infants with prenatal drug exposure became more apparent. The items were assembled into their first format in an attempt to make the instrument "clinician friendly". The LP has been used by the author and other clinicians for early intervention programming.

The second stage of content validity is termed the judgement quantification stage, and has two steps (Lynn, 1986). The first step involved the identification

of a specific number of experts who could judge that the LP items have content validity. The experts were selected on the basis of their knowledge and clinical experience in infant and early childhood development and their pioneering work with infants with PDE and their families. The expert panel consisted of three clinical nurse specialists, a developmental pediatrician, a developmental psychologist, and an occupational therapist, who was also a physical therapist and educational specialist. The experts were asked four questions pertaining to the LP and the subscale items. The questions were: Is the instrument clearly written? Does it measure what it is intended to measure? Do items need to be deleted or others added? Can the instrument be completed in a reasonable time period (Tornquist, 1986)? If the experts raised concerns regarding specific items on the LP, a discussion ensued with the primary author. These discussions led to the reworking of some items and the deletion of other items on the LP. Four items were identified as needing to be added to the LP; they were developed by the primary author, then reviewed by the experts prior to their inclusion in the protocol.

Reliability. Test-retest, interrater reliability, and internal consistency reliability were examined. To assess test-retest stability of the LP, the LP was administered to infants on one occasion and again four to seven days later (n=10). The correlation between assessments r=.98 (p=.004) provided a test-retest estimate that was statistically significant. This indicated a strong association between the two tests.

To assess interrater reliability of the LP, three clinicians independently assessed three infants with PDE and independently scored the protocol (n=9). The independent scorings achieved a minimum of 85% degree of agreement between raters.

Internal consistency of the LP was assessed by calculating Cronbach's alpha

coefficients for each of the subscales and for the total scale (n=103). The standardized alpha coefficients ranged in size from a=.44 to .68 for nine of the ten subscales and was a=.55 for the total LP. These alphas indicated problems with the existing subscales as well as the total assessment.

Construct validity. Forty of the 50 items were submitted to a principal components factor analysis with Varimax rotation (N=103). Ten items were not conducive to a principal component factor analysis due to the small sample size (N=40, 46), but were retained as they were age-related items. These ten items became subscales VI, Atypical Communication Response and VII, Atypical Play Response. This factor analysis led to a factor solution of seven factors with the eigenvalues displayed in Table 5. Many of the factors which emerged in the Varimax rotation were not the same as in the original subscales, thus providing further evidence that the tool needed to be revised. Thirteen items were dropped because the items did not load clearly on any factors.

The factor analysis of the 40 item tool yielded a seven factor solution with 27 items. Factor one was labeled High Reactivity (seven items). Factor two was labeled Atypical ANS Response (five items), Factor three was labeled Low Reactivity (five items), Factor four was labeled Compromised System Regulation (four items), Factor five was labeled Atypical Visual Functioning (three items), Factor six was labeled Gastrointestinal (two items), and Factor seven was labeled Tracking (one item). Only three of the factors which emerged in the Varimax rotation were almost identical to the original subscales, thus providing further evidence that the tool needed to be revised. Thirteen items were dropped, as the items did not load clearly on any factors. Factor six (Gastrointestinal) consisted of only two items with minimal commonality @ .657 and was dropped from the scale. Factor seven (tracking) could not stand alone but was thought to be very clinically significant, so was placed in the Atypical Visual Functioning subscale.

As modified, the LP includes 25 general items plus two age-specific subscales that contain five items each (i.e., VI Atypical Communications Patterns and VII Atypical Play Response) for a total of 35 items.

Cronbach's alpha coefficients were calculated again on the new subscales and there was a clear improvement in the internal consistency. These alpha coefficients are displayed in Table 3.1. The overall Cronbach's alpha coefficient for the 25 items (subscales I to V) was α=.81. The individual subscale Cronbach's alphas ranged from α=.64 for the System Regulation subscale to α=.83 for the High Reactivity subscale. With the exception of two subscales, Compromised System Regulation and Atypical Play Response, all alphas were above the .70 acceptable level for new instruments (Nunnally, 1978). The Compromised System Regulation subscale was determined to be clinically significant by the panel of experts, and thus essential for content validity, and was retained. The Cronbach's alpha for the two age-related subscales were Atypical Communication Patterns (α=.79, n=46) and Atypical Play Response (α=.63, n=40).

This preliminary psychometric evidence demonstrated initial support for construct validity and internal consistency of the LP. The revised LP subscales which emerged from the pilot study are presented in Table 3.2. The revised instrument was renamed the Lewis Neurobehavioral Assessment Scale (LNAS).

Study Procedures

Methods of Testing

Procedures in this study expanded previous pilot work on the LNAS. To determine further reliability of the LNAS, three methods were used: interrater, internal consistency, and test-retest reliability. Two types of validity were examined: construct and predictive validity.

Reliability Testing

Interrater reliability. Interrater reliability for the LNAS was determined by the degree to which two clinicians, during the same assessment time,

Table 3.1. Psychometric Properties of LNAS Subscales

LNAS Subscales	# of items	N	Eigenvalue	Cronbach's Alpha	M	SD
High Reactivity	7	103	5.37	0.83	10.2	6.2
Low Reactivity	5	103	2.7	0.75	2.8	3.8
Atypical Visual Functioning	3	103	1.07	0.81	3.3	3.7
ANS Atypical Response	5	103	3.6	0.79	4.1	4.2
Compromised System Regulation	4	103	2.23	0.64	1.3	2.4
Atypical Communication Pattern	5	46		0.79		
Atypical Play Response	5	40		0.63		

Table 3.2. LNAS Subscales

I. High Reactivity
   1. Irritibility
   2. Frequency or rapidity of state changes
   3. Difficult to comfort
   4. Frequent startle response
   5. Hyperactivity
   6. High muscle tone
   7. Tremors

II. Low Reactivity
   8. Passivity
   9. Dull alert state
   10. Lethargy
   11. Low muscle tone
   12. Difficult feeding

III. Atypical Visual Functioning
   13. Difficulty initiating eye contact
   14. Difficulty maintaining eye contact
   15. Gaze aversion
   16. Difficulty tracking

IV. Atypical ANS Response
   17. High-pitched cry
   18. Sweating
   19. Frequent yawning
   20. Hiccupping
   21. Sneezing

V. Compromised System Regulation
   22. Jerky eye movement
   23. Unexplained fevers
   24. Increased respirations
   25. Nasal stuffiness

VI. Atypical Communication Patterns (Administered at 4 months and older)
   26. Undifferentiated cries
   27. No social laugh
   28. No social smile
   29. Limited vocalizations
   30. Limited vocalization to caregiver's response

VII. Atypical Play Response (Administered at 6 months and older)
   31. Limited imitating with objects/ people
   32. Limited functional use of toys
   33. Limited initiation of play with objects/people
   34. Distractible
   35. Easily frustrated

independently scored the same ratings for the neurobehaviors being measured. Interrater reliability was established in the following manner; the two clinicians read the training material, met with the investigator, and then independently assessed and scored infants with PDE using the LNAS. After three practice sessions, the clinicians independently assessed and scored 38 infants. Correlations and paired t-tests were calculated to estimate interrater reliability between the trained clinicians on the LNAS subscale scores and total score.

Internal consistency. Internal consistency reliability is the degree to which the subparts of a scale are all measuring the same dimension (Polit & Hungler, 1995). Subscales six and seven were not tested in this study, because the behaviors were specific to 4 months and older infants. Internal consistency reliability was determined by computing Cronbach's alpha coefficient for each of the first five subscale scores and the sum score for the five subscales on the LNAS (N=80). Inter-scale correlations were calculated to investigate the relationship of the scales to one another. Inter-item correlations were then calculated on two subscales, Atypical ANS Response and Compromised System Regulation, to help understand the source of their low alpha coefficients.

Test-retest reliability. For short-term test-retest reliability, sixteen infants with PDE were assessed using the LNAS twice, approximately seven days apart, by the same trained clinician in the hospital or infant's home. Paired t-tests and Pearson correlation coefficients were computed to compare infants on total score and subscale scores between the first and second test administration. Using paired t-tests for test-retest reliability helped determine a significant difference between scores if one existed (Munro, Visintainer, & Page, 1986). To determine long-term stability of the LNAS, Pearson correlation coefficients were calculated between the LNAS at birth and the LNAS at six months of age (n=16) between the LNAS subscales and total score.

Validity Testing

Construct validity.

Construct validity of the LP was determined by the known group technique (Polit & Hungler, 1991). This technique was used to assess the LP's ability to discriminate infants with PDE from those without PDE. To evaluate the construct validity, the LP was administered by nurse clinicians to all infants with PDE and non-PDE infants in the birthing hospital or in the infant's home at one age point (N=80). T-tests for the LNAS subscale scores and total score were computed and analyzed between the two groups to determine whether a significant difference existed.

A second approach to construct validity was to determine the tool's ability to differentiate high and low risk infants. Two measures of risk were used. The first measure, the Parmalee Maternal Complications Scale (MCS), was administered to all infants with PDE and without PDE in the newborn period (N=80). Data for the Parmalee was acquired by a review of the maternal hospital obstetric record by the nurse clinician. Infants receiving scores above and below the median on the Parmalee were then compared via t-tests on their LNAS subscale scores and total score. Established validity and reliability for the Parmalee were reported by Francis, Self & Horowitz (1987). In addition, Field, Dempsey and Shuman (1983) showed its predictive validity with cognitive development at five years of age.

The second variable used to determine risk was infant birth weight. The 25th percentile for normal newborns as indicated by the National Center for Health Statistics was used as the high and low risk marker. Infants above the 25th percentile were coded low risk, and those below were coded high risk. T-tests were used to compare the two groups on the LNAS subscales and total score. Calculations were conducted on the total sample of both PDE and non-PDE infants (N=80) and on the PDE infants only (n=40).

Predictive Validity.

Predictive validity was determined by the degree to which the LNAS predicted an infant's Clarity of Cues and Responsiveness to the Parent during an infant feeding situation. The NCAFS measures parent-child interaction during feeding in the first year of life (Barnard, 1978). The subscales include: Sensitivity to Cues, Response to Distress, Social-Emotional Growth Fostering, Cognitive Growth Fostering, Clarity of Cues, and Responsiveness to Parent. Internal consistency has been established for both subscales scores and total score. Predictive validity has also been established. For instance, the NCAFS shows a significantly positive correlation with the Home Observation for Measurement of the Environment (HOME) Inventory at 8 and 12 months of age. Another study of abused and neglected premature infants showed lower scores in both Clarity of Cues and Responsiveness to Parent (Barnard, 1978).

It was predicted that if the infant with PDE scored high in the High and Low Reactivity areas of the LNAS, they would have lower scores on subscale V and VI on the NCAFS feeding scale (showing decreased Clarity of Cues and Responsiveness during a feeding situation). Predictive validity of the LNAS subscales was explored by correlating the LNAS subscale scores and total score with the corresponding NCAFS subscale scores using Pearson correlation coefficients (n=49).

A second approach in determining predictive validity was assessed by using the Mullen Scale of Early Learning (Mullen). The Mullen Scale of Early Learning is a measure of cognitive functioning for infants and preschool children from birth through 68 months. The Mullen Scale consists of one motor subscale and four cognitive subscales which include Gross Motor, Visual Receptive, Fine Motor, Receptive Language, and Expressive Language (Mullen, 1995). The Mullen Scale provides individual subscale scores and a composite score from the

four cognitive subscales. Internal consistency has been established for subscale scores (.75 to .83) and the composite score (.91). Test-retest for the Gross Motor Scale was .96 and the median for the cognitive scales were .84. The Mullen cognitive scale scores and composite score displayed a high correlation with the Bayley Mental Development Index, supporting construct validity. Predictive validity evidence was provided when a two-year study of children with learning disabilities or developmental delay was conducted on 4- and 5-year-old children (N=131). Significant correlations were found between the Mullen and the Metropolitan Readiness Test (Mullen 1995). The Mullen was administered to PDE and non-PDE infants at the 6 month home visit (n=15). It was predicted that infants showing high scores on the LNAS at birth would be negatively correlated with the Mullen scores at six months of age. The LNAS subscale scores and total scores were compared with the Mullen scores using Pearson correlations coefficients.

Training and Instrument Administration

Pediatric nurses who participated in the data collection process were trained in the administration of the assessment tools to be used. The nurses were knowledgeable regarding normal and atypical infant development and had experience handling infants with special needs. Only nurses trained by the investigator or the investigator's colleagues, as described below, participated in the data collection procedures.

A one-day seminar for the nurse clinicians was conducted by a clinical psychologist and a pediatric nurse practitioner consultant in the cognitive, physical, and motor development of the infant and young child. Young infants were observed with a parent during a structured assessment by the instructor. Observations of the infants were made and later, discussion with the instructor was conducted regarding the assessment of young infants. General administration

instructions, testing procedures, definition and interpretation of individual items and item scoring procedures were discussed for each assessment measure as well as general information about testing of infants and young children. Nurses administering the NCAFS had prior training and experience with that assessment measure, so no additional training was conducted for the NCAFS.

A training manual and standard training procedures were established for the LNAS. Nurses who collected data for the study participated in training and received a training manual in the use of the LNAS. This training consisted of a brief overview of the infant with PDE, introduction to the LNAS, general administration instructions, testing procedures, definition and interpretation of individual items, item scoring procedures, collection of demographic information and determination of chronological age. The Parmalee Complications Scale was discussed and reviewed for identifiers of perinatal and postnatal complications. All measures are included in Appendix C.

Some of Part B and all of Part A of the LNAS were administered by a trained nurse clinician to infants who were enrolled in the study. Another nurse clinician completed the NCAFS. After assessments were completed, the Parmalee Complications Scale and missing items from Part A of the LNAS were completed by review of medical charts of all infants at the birthing hospital. This allowed the nurse clinician collecting data to conduct the assessment blind of both the infants' drug exposure status, background data, and other test findings.

All of the items necessary for Part A and Part B of the LNAS were recorded from the clinician's assessment, observations of the infant, interview with the caregiver, or review of medical records. The NCAFS was completed after observation of a home visit interaction between the caregiver and infant during a feeding. Demographic information was collected from parent report and the hospital records.

Time. Completion of the four assessment measures took approximately one to one and one-half hours for PDE and non-PDE infants. With PDE infants only, the LNAS was administered a second time to 16 infants. This activity took an additional 20-30 minutes.

For infants six months of age, all tests were again administered, along with the Mullen. This activity took approximately one to one and one-half hours for each infant.

In all cases the LNAS was administered prior to any other testing and required 20-30 minutes. All assessments were paced to reduce infants' stress or discomfort. No assessments needed to be terminated due to infant discomfort or stress or caregiver-expressed stress.

CHAPTER FOUR

Results

The results of this research study are organized around three major sections. Presented first is the description of the sample including both PDE infants and the control group of non-PDE infants. Certain information about the caregiver is also presented. Second, there is a discussion of the results of Aim I regarding reliability testing of the LNAS. Third, results of Aim II addressing construct and predictive validity testing of the LNAS are presented.

Description of the Sample

The convenience sample consisted of 40 infants with PDE and a control group of 40 infants without PDE ranging in age from 6 to 14 days. All infants were referred by one of four agencies, hospital, public health department, pediatrician's office or educational site. The greatest number of infant referrals was made from the birthing hospital, which included 18 (45%) PDE infants and 25 (62.5%) non-PDE infants. Some infant assessments were administered in the birthing hospital, but most assessments were performed in the infant's home, which included 34 (85.0%) infants with PDE and 39 (97.5%) non-PDE infants. Five (12.5%) PDE infants were living in foster or foster adoptive homes, while all of the non-PDE were living with their natural parents. The gestational age ranged from 34 to 42 weeks for PDE infants and 35-42 weeks for non-PDE infants. The majority of infants were delivered vaginally — PDE 33 (82.5%); non-PDE 34 (85.0%). Descriptive characteristics of the PDE and non-PDE infants are presented in Tables 4.1 and 4.2. As shown in these two tables, the groups differed significantly in ethnicity, birth weight, and other physical characteristics at birth. The PDE group was more ethnically diverse and smaller in physical frame than non-PDE infants.

Table 4.1 Infant Demographic Characteristics

Variables	PDE Infants [a] Number (%)	Non-PDE Infants [a] Number (%)	Chi Square	p
Referral Agency			7.83	.050
Hospital	18 (45.0)	25 (52.5)		
Public Health Department	17 (42.5)	6 (15.0)		
Pediatrician	2 (05.0)	5 (12.5)		
Educational Site	3 (07.5)	4 (10.0)		
Assessment Site			3.91	.054
Home	34 (85.0)	39(97.5)		
Hospital	6 (07.5)	1 (01.3)		
Gender			.81	.369
Male	20 (50.0)	16 (40.0)		
Female	20 (50.0)	24 (60.0)		
Ethnicity			20.52	.000
African American	17 (42.5)	2 (05.0)		
Caucasian/white	16 (40.0)	29 (72.5)		
Hispanic American/ other Hispanic American	7 (17.5)	9 (22.5)		

Note. [a]n = 40 for each group.

Table 4.2 Infant Physical Characteristics

Variables	PDE Infants[a] M	SD	Range	Non-PDE Infants[a] M	SD	Range	t	p
Infant age (days)	11.9	2.6	6-14	12.3	1.9	7-14	.88	.381
Gestational age	39.0	1.9	34-42	39.5	1.6	35-42	1.39	.169
Birth weight	6.7	1.6	3.1-9.1	7.5	1.1	5.1-9.5	2.94	.004
Birth length	19.6	1.1	16.8-22.0	20.3	.94	17.8-22.0	2.99	.004
Birth Head Circumf.	13.2	.8	11.5-14.8	13.6	.63	12.0-15.0	2.62	.011
Apgars*								
1 min	7.9	1.8	1-10	8.4	9.2	5-10	1.14	.258
5 min	8.8	1.0	4-10	9.1	.4	8-10	1.61	.113

Note. [a]n = 40 for each group.
* Only 39 PDE infants had Apgar data.

Prenatal drug exposure was determined by birth mother's self-report and/or by a positive urine toxicology screen for the newborn and/or the mother. PDE infants were prenatally exposed to a variety of terratogenic drugs as demonstrated in Table 4.3. The number of drugs to which any newborn was exposed ranged from one drug to four drugs. Mothers admitted to prenatal use of 12 other drugs which were not detected in a urine toxicology screen performed at birth for that newborn or mother. Four mothers denied use of a particular drug which was later dectected by a urine toxicology screen. Polydrug exposure was documented by either maternal report or by a positive urine toxicology screen in 23 (57.5%) of the infants with PDE. Qualification for inclusion in the control

Table 4.3 Prenatal Drug Exposure

Drugs	Positive Toxicology Number (%)	Maternal Self Report Number (%)	Single Drug Use Number
Barbiturates	2(5.0)	1(2.5)	2
Cocaine	7(17.5)	7(17.5)	1
Heroin	4(10.0)	3(7.5)	2
Tetrahydrocannibis	11(27.5)	16(40.0)	6
Methadone	2(5.0)	2(5.0)	
Amphetamine	11(27.5)	18(45.0)	6
Phencyclidine (PCP)	1(2.5)	2(5.0)	
Benzodiazipines	1(2.5)	1(2.5)	
Alcohol (ETOH)	2(5.0)	16(40.0)	
Opiates	4(10.0)	2(5.0)	

Note. Numbers do not total 100% due to polydrug use of mothers.

group was a negative urine toxicology screen at birth for the newborn or the mother and/or a signed statement from the birth mother that she did not use drugs or alcohol during pregnancy. Infants were not included in the study if there was maternal use of cigarettes during pregnancy or if the mother had surgery during pregnancy, since the administration of analgesia and anethesia

was a confounding variable.

Newborns were diagnosed with a variety of prenatal and postnatal problems. Occurring most frequently were meconium staining, hyperbilirubinemia, tachypnea, small for gestional age (SGA), and intrauterine growth restriction (IUGR). However, only SGA and IUGR showed a significant difference between the PDE and non-PDE groups. Respiratory distress, as defined by meconium staining (27.5%), showed a trend toward significance and occurred more in infants with PDE. See Table 4.4 for Infant Medical Problems.

Table 4.4 Infant Medical Problems

Variables	PDE Infants [a] Number (%)	Non-PDE Infants [a] Number (%)	Chi Square	p
Intrauterine Growth Restriction	4(10.0)	0(0.0)	4.21	.04
Small for Gestational Age	6(15.0)	0(0.0)	6.48	.01
Anemia	2(5.0)	1(2.5)	1.01	.31
Cephalohematoma	2(5.0)	0(0.0)	2.05	.15
Congenital Hip	2(5.0)	0(0.0)	2.05	.15
Conjunctivitis	1(2.5)	0(0.0)	1.01	.31
Facial palsy	1(2.5)	0(0.0)	1.01	.31
Hepatitis C	2(5.0)	0(0.0)	2.05	.15
Hyperbilirubinemia	7(17.5)	6(15.0)	.09	.76
Hypoglycelmic	2(5.0)	2(5.0)	1.01	.31
Undescended testes	1(2.5)	0(0.0)	1.01	.31
Withdrawal	10(25.6)	0(0.0)	11.74	.00
Tachypnea	7(17.5)	3(7.5)	1.82	.18
Meconium	11(27.5)	5(12.5)	2.81	.09

Note. [a]n = 40 for each group.

The mean age at birth for mothers of both PDE and PDE infants was 27 years. In addition, as shown in Table 4.5, mothers of non-PDE infants had more prenatal care visits. PDE mothers were less likely to be married or living with someone, were nearly all unemployed, many were receiving government assistance (59.4%), and often were the sole financial support (39.4%) for their families.

Table 4.5 Maternal Characteristics

	Mothers of PDE Infants	Mothers of Non-PDE Infants		
Variables	**Number (%)**	**Number (%)**	**Chi Square**	**p**
Prenatal Care			21.80	.000
No	7 (17.5)	0(00.0)		
Yes	21 (52.5)	39 (97.5)		
Limited	9 (22.5)	1 (02.5)		
Delivery Method			.76	1.00
Cesarean	7 (17.5)	6 (15.0)		
Vaginal	33 (82.5)	34 (85.0)		
Marital Status[a]			16.04	.003
Single	21 (60.0)	11 (28.2)		
Married	11 (28.2)	26 (66.7)		
Divorced	4 (10.5)	2 (05.1)		
Widowed	2 (05.3)	0(00.0)		
Cohabiting w/ husband/partner	17 (51.5)	34 (85.0)	9.63	.002
Student	5 (15.17)	13 (32.5)	6.74	.081
Work status[a]			10.39	.001
Unemployed	30 (88.2)	21 (55.0)		
Employed	4 (11.8)	17 (45.0)		

Table 4.5 Maternal Characteristics, continued

Variables	Mothers of PDE Infants Number (%)	Mothers of Non-PDE Infants Number (%)	Chi Square	p
Government Assistance			8.69	.034
No	13 (40.6)	26 (65.0)		
Yes	19 (59.4)	13 (35.0)		
Sole Support			10.39	.001
No	20 (60.6)	36 (92.3)		
Yes	13 (39.4)	3 (07.7)		

Variables	Mothers of PDE Infants M	SD	Range	Mothers of Non-PDE Infants M	SD	Range
Age at birth	27	6.9	14-39	27	7.8	15-42
Years of education	11.9	2.7	8-24	12.7	2.9	6-19

Note. [a]n = 34 PDE for work status
35 PDE and non-PDE for marital status.
33 PDE, 39 non-PDE for years of age at birth and education.
32 PDE and non-PDE for government assistance.
33 PDE and 39 non-PDE for sole financial support.

Infant Performance on LNAS

The possible overall score to be obtained on the LNAS (5 subscales) for any one infant ranged from 25 to 125, a high score reflecting more atypical neurobehaviors or a more disorganized infant. Overall scores obtained from infants exposed to drugs in utero during this study ranged from 33 to 93, with a mean of 59 and a standard deviation of 15. For the control infants, scores ranged from 30 to 63, with a mean of 43 and a standard deviation of 10. The distribution of scores across infants was adequate to perform the statistical analyses requireed to meet the study aims. See Table 4.6 for subscale and total score characteristics.

Table 4.6 LNAS Subscale and Total Scale Scores for Sample

		PDE Infants[a]				Non-PDE Infants[a]			
Variable	**Scale Range**	**M**	**SD**	**Variance**	**Range**	**M**	**SD**	**Variance**	**Range**
I. High Reactivity	7-35	16.08	6.53	42.28	7-31	10.98	2.99	8.99	7-19
II. Low Reactivity	5-25	10.25	5.18	26.81	5-20	7.48	3.19	10.20	5-19
III. Atypical Visual Functioning	4-20	13.73	5.18	25.79	4-20	9.05	4.27	17.38	4-20
IV. Atypical ANS Response	5-25	12.87	3.16	10.01	5-20	10.67	2.10	4.43	5-16
V. Compromised System Regulation	4-20	6.30	2.69	7.24	4-15	5.05	1.47	2.15	4-9
Total Scale Score	25-125	59.23	14.93	223.82	33-93	43.23	9.64	92.98	30-63

Note. [a]n = 40 for each group.

Reliability Testing of the LNAS

The first aim of the research was to determine the reliability of the revised LNAS in a sample of ethnically diverse newborn infants with prenatal drug exposure. Three questions were addressed: l) Does the LNAS demonstrate interrater reliability? 2) Is the LNAS internally consistent? and 3) Does the LNAS have test-retest reliability?

Interrater Reliability

Interrater reliability testing was conducted with 38 infants, including PDE (n=27) and non-PDE (n=11) infants. The individual subscale correlations ranged from r = .95 to r = .98 (p = .000). The interrater reliability total score correlation was r = .98 (p= .000). In addition, both the subscale scores and total score indicated no statistically significant difference between the raters paired t-statistic scores on the LNAS. Table 4.7 displays the results for interrater reliability testing.

Table 4.7 Interrater Reliability

Subscales & Scale	Rater 1[a] m	Rater 2[a] m	r*	t**
I. High Reactivity	13.32	13.66	.95	-1.40
II. Low Reactivity	9.68	9.65	.97	.14
III. Atypical Visual Functioning	11.66	11.53	.98	.75
IV. Atypical ANS Response	11.71	11.84	.96	-1.15
V.Compromised System Regulation	5.29	5.37	.96	-1.00
Total Score	51.66	52.05	.98	.82

Note. [a]n = 38 for each group.
*all r's were significant at p<.000.
**no t statistic was significant.

Internal Consistency

Internal consistency reliability testing was conducted with all infants, both PDE and non-PDE (N = 80). As shown in Table 4.8 Cronbach's alpha coefficient estimates for individual subscale scores ranged from $\alpha = .53$ to $\alpha = .91$. The alpha for the 5 subscales scores together was $\alpha = .73$. Subscales for Atypical ANS Response and Compromised System Response demonstrated only moderate internal consistency. Cronbach alpha coefficient estimates were also calculated for individual subscale scores for PDE infants only (n=40) which showed the same two subscales having a lower internal consistency. These results are also displayed in Table 4.8.

Table 4.8 Cronbach Alpha Coefficients for LNAS

Subscales	Number Items	PDE & Non-PDE[a] Scale Alpha	PDE only[b] Alpha
I. High Reactivity	7	.87	.87
II. Low Reactivity	5	.83	.82
III. Atypical Visual Functioning	4	.91	.86
IV. Atypical ANS Response	5	.53	.49
V. Compromised System Regulation	4	.58	.61

Note. [a]N = 80. [b] n = 40.

Inter-scale correlations for all infants (N=80) were calculated to further investigate the relationship of scales to one another and are shown in Table 4.9.

Table 4.9 Inter-Scale Correlations Matrix for LNAS Subscales and Total Score for All Infants[a]

Variables	I	II	III	IV	V	Total Score
I. High Reactivity	1.00					
II. Low Reactivity	.16	1.00				
III. Atypical Visual Functioning	.45**	.57**	1.00			
IV. Atypical ANS Response	.57**	.13	.28**	1.00		
V. Compromised System Regulation	.75**	.15	.39**	.60**	1.00	
Total Score	.81**	.61**	.80**	.63**	.73*	1.00

Note. [a]N = 80 infants.
** Correlation significant at <.01, two-tailed.
* Correlation significant at <.05, two-tailed.

Subscale Low Reactivity was significantly associated with only one other subscale — Atypical Visual Functioning. All other interscale correlations were significant and ranged from moderate to strong associations, r =.39 to r=.81. In addition, all subscales were significantly correlated with the total scale score.

Inter-item correlations were also calculated for two subscales to help understand their lack of internal consistency. As shown in Table 4.10, for the Atypical

Table 4.10 Inter-Item Correlation Matrix for the LNAS Subscale Atypical ANS Response[a]

Variables	High-Pitched Cry	Sweating	Frequent Yawning	Hiccupping	Sneezing
High-Pitched Cry	1.00				
Sweating	.37**	1.00			
Frequent Yawning	.10	.29**	1.00		
Hiccupping	-.03	-.12	.30**	1.00	
Sneezing	.05	.21	.28**	.52**	1.00

Note. [a]N = 80.
**Correlations significant at <.01, two-tailed.

ANS Response subscale 5 items were weakly to moderately associated and significant (r=.28 to r=.52). Five items were not significantly related at all. High Pitched Cry was only related to one other item — Sweating. The Compromised System Regulation subscale items were weakly associated (r=.26 to r=.38) as shown in Table 4.11. However, Unexplained Fevers was only related to one other item — Nasal Stuffiness. Nasal Stuffiness showed significant relationships to all other items.

Table 4.11 Inter-Item Correlation Matrix for the LNAS Subscale Compromised System Regulation[a]

Variables	Jerky Eye Movement	Unexplained Fevers	Increased Respiration	Nasal Stuffiness
Jerky Eye Movement	1.00			
Unexplained Fevers	.21	1.00		
Increased Respiration	.26*	.11	1.00	
Nasal Stuffiness	.33 **	.29*	.38**	1.00

Note. [a]N = 80.
**Correlations significant at <.01, two-tailed.
*Correlations significant at <.05, two-tailed.

Test-Retest Reliability

Both the short and long term stability of the LNAS were examined. Short term test- retest reliability was conducted on a subgroup of PDE infants (n=l6) using a one-week interval. As shown in Table 4.12 Pearson correlations for subscales ranged from r=.78 to r=.92, all of which were statistically significant at p=.000. Subscale IV, Atypical ANS Response had the weakest correlation at r=.78. The LNAS total score correlation for test-retest reliability was r=.91 (p=.000). In addition, there was no statistically significant difference between the first and second LNAS infant assessments for any subscale or the total score as measured by t-tests.

Table 4.12 Test-Retest Results for the LNAS

Subscales & Scale	1st test[a] m	2nd test[a] m	t*	r**
I. High Reactivity	16.56	16.25	.419	.91
II. Low Reactivity	9.81	9.94	-.239	.92
III. Atypical Visual Functioning	14.00	13.50	.767	.89
IV. Atypical ANS Response	12.63	12.44	.282	.78
V.Compromised System Regulation	6.50	6.82	-1.000	.91
Total Score	59.50	58.94	.345	.91

Note. [a]n = 16 in each group.
*No t statistic was significant.
**All r's were significant at p<.000.

The long term stability of the measure was examined at 6 months of age (n=16). As shown in Table 4.13, all subscale scores and the total score at birth were significantly related to their respective scores at 6 months of age except for the subscale of Low Reactivity. Low Reactivity at birth showed no relationship to itself or any other LNAS score at 6 months. Other subscales showed stability ranging from r=.58 to r=.95 for Atypical ANS Response. The total score showed stability of r=.80 over the 6-month period.

Table 4.13 Pearson Correlations Between LNAS at Birth and LNAS at 6 Months[a]

Subscales & Scale	I. High Reactivity	II. Low Reactivity	III. Atypical Visual Functioning	IV. Atypical ANS Response	V. Compromised System Regulation	Total
I. High Reactivity	.72**	.29	.34	.84**	.58*	.68*
II. Low Reactivity	.24	.08	.12	.09	.42	.25
III. Atypical Visual Functioning	.75**	.38	.67**	.87**	.49	.78**
IV. Atypical ANS Response	.86**	.46	.58*	.95**	.74**	.88**
V. Compromised System Regulation	.63**	.15	.42	.45	.58*	.59*
Total Score	.82**	.33	.51*	.81**	.73**	.80**

Note. [a]n = 16 infants.
** Correlations significant at <.01, two-tailed.
* Correlations significant at <.05, two-tailed.

Validity Testing of the LNAS

The second research aim was to determine construct and predictive validity of the LNAS in a sample of ethnically diverse newborn infants with PDE. The following questions were addressed: 1) Does the LNAS differentiate infants with PDE from those without PDE? 2) Does the LNAS significantly differentiate low and high risk infants with PDE? 3) Does the LNAS predict an infant's clarity of cues and responsiveness to a parent/caregiver during an infant feeding situation? and 4) Does the LNAS predict early indicators of cognitive difficulty at 6 months of age?

Construct Validity

PDE versus non-PDE. Construct validity was assessed using the known group approach. First, the PDE infant group scores (n=40) were compared to

those of the non-PDE infants group scores (n=40). Results of t-tests used to examine the differences between the means of the two groups indicated that there were statistically significant differences between the mean scores of the two groups in all five subscales and the total score of the LNAS. These results indicate that the LNAS did differentiate infants with PDE from those without drug exposure in utero. The subscale Low Reactivity showed the most significant difference. Table 4.14 displays the difference between PDE and non-PDE groups.

Table 4.14 Differences Between PDE and Non-PDE Infants in LNAS Scores

Subscales & Scale	PDE[a] m	Non-PDE[a] m	t	p
I. High Reactivity	16.08	10.98	-4.50	.000
II. Low Reactivity	10.25	7.48	-2.89	.005
III. Atypical Visual Functioning	13.73	9.05	-4.56	.000
IV. Atypical ANS Response	12.87	10.67	-3.66	.000
V. Compromised System Regulation	6.30	5.05	-2.58	.010
Total Score	59.23	43.23	-5.75	.000

Note. [a]n = 40 infants in each group.

Low risk versus high risk. The second approach in determining construct validity was to determine whether the LNAS could differentiate high and low risk infants. Two measures were used as indicators of risk. The first risk measure was the Parmelee Maternal Complications Scale (MCS) . The median of the MCS score was used as the high and low risk marker. Infants above the median were coded high risk and those below the median coded low risk. Infants in the two groups were then compared via t-test on the LNAS subscale scores and total score. Two comparisons were performed, one with the total sample of PDE and non-PDE infants and a second comparison using the PDE group only. The median for the total sample was eight. Results demonstrated a statistically significant difference in all LNAS subscale scores and the total score between infants whose mothers were at high risk (n=36) versus low risk (n=44) due to maternal complications (see Table 4.15).

Table 4.15 Differences in LNAS Scores of All Infants at Low and High Risk From Maternal Complications

Subscales & Scale	High Risk[a] m	Low Risk[b] m	t	p
I. High Reactivity	15.47	11.80	-3.049	.003
II. Low Reactivity	10.58	7.50	-3.240	.002
III. Atypical Visual Functioning	13.58	9.37	-3.811	.000
IV. Atypical ANS Response	12.58	11.11	-2.260	.027
V. Compromised System Regulation	6.31	5.16	-2.340	.022
Total Score	58.53	44.87	-5.530	.000

Note. [a]n = 36 infants. [b]n = 44 infants.

When risk groups were compared within the PDE sample only, the median was 10. This analysis differentiated risk groups on the total scale score and on two of the subscale scores, Low Reactivity and Atypical Visual Functioning (see Table 4.16 for results).

Table 4.16 Differences in LNAS Scores of PDE Infants at Low and High Risk From Maternal Complications

Subscales & Scale	High Risk[a] m	Low Risk[b] m	t	p
I. High Reactivity	16.96	14.71	-1.13	.265
II. Low Reactivity	12.61	7.18	-3.98	.000
III. Atypical Visual Functioning	15.78	10.77	-3.48	.001
IV. Atypical ANS Response	13.09	12.59	-.52	.605
V. Compromised System Regulation	6.74	5.71	-1.29	.204
Total Score	65.17	50.94	-3.40	.002

Note. [a]n = 23 infants. [b]n = 17 infants.

Infant birth weight was the second variable used to determine risk. The National Center for Health Statistics (NCHS) 25th percentile for term infant's birth weight of 6.08 pounds was used as the high and low risk marker. The mean for the infants in this study was 6.92 pounds and the median was 7.09 pounds indicating heavier birth weights than the NCHS 25th percentile. Infants above the quartile were coded low risk and those below were coded high risk. Infants in the two groups were compared via t-tests on the LNAS subscales and total score. Calculations were performed on the total sample of both PDE and non-PDE groups (N=80) and on the PDE group only (n=40). The analysis for the total sample revealed statistically significant differences between the infants whose birth weight put them at risk (n=32) versus those with low risk birth weights (n=47) on all LNAS subscale scores and total score (see Table 4.17).

Table 4.17 Differences in LNAS Scores of all Infants at Low and High Risk From Birth Weight

Subscales & Scale	High Risk[a] m	Low Risk[b] m	t	p
I. High Reactivity	15.59	12.23	4.63	.000
II. Low Reactivity	10.06	7.81	3.10	.003
III. Atypical Visual Functioning	13.09	10.09	6.07	.000
IV. Atypical ANS Response	12.16	11.49	2.39	.019
V. Compromised System Regulation	6.34	5.17	2.99	.004
Total Score	66.78	46.58	5.48	.000

Note. [a]n = 32 infants. [b]n = 47 infants.

When the analysis was performed with PDE infants only, there were (n=16) infants in the high risk weight group and (n=24) infants in the low risk weight group. Results showed a statistically significant difference between the groups for the LNAS total score but for only 2 subscales: High Reactivity and Atypical Visual Functioning (see Table 4.18).

4.18 Differences in LNAS Scores of PDE Infants at Low and High Risk From Birth Weight

Subscales & Scale	High Risk[a] m	Low Risk[b] m	t	p
I. High Reactivity	19.69	13.75	2.92	.007
II. Low Reactivity	11.50	9.42	1.23	.229
III. Atypical Visual Functioning	16.88	11.63	4.05	.000
IV. Atypical ANS Response	13.38	12.54	.813	.422
V. Compromised System Regulation	7.13	5.75	1.62	.114
Total Score	68.56	53.08	3.62	.001

Note. [a]n = 16 infants. [b]n = 24 infants.

Predictive Validity

Infant interpersonal behavior. The first assessment of Predictive validity examined the hypothesis that an infant's score on the LNAS subscales of High Reactivity and Low Reactivity would be negatively related to the infant's Clarity of Cues and Responsiveness to Parent as measured by the Nursing Child Assessment Feeding Scale (NCAFS). Forty-nine infants were included in this analysis,

representing both PDE (n=21) and non-PDE (n=28) groups. As shown in Table 4.19, the LNAS subscales did not show a clear relationship to the infant's Clarity of Cues. The subscale of Low Reactivity was negatively and significantly related to Clarity of Cues. High Reactivity and Atypical Visual Functioning showed trends toward a relationship. However, all of the LNAS subscales and the total score were significantly related to the infant's Responsiveness to the Caregiver, with the Low Reactivity subscale presenting the strongest relationship of all.

Table 4.19 Pearson Correlations for the Relationship Between the LNAS and the Nursing Child Assessment Feeding Scales

Subscales & Scale	Clarity of Cues[a]	Responsiveness to Parent[a]
I. High Reactivity	-.22	-.29*
II. Low Reactivity	-.31*	-.53**
III. Atypical Visual Functioning	-.24	-.40**
IV. Atypical ANS Response	.04	-.36*
V. Compromised System Regulation	.07	-.38**
Total Score	-.08	-.52**

Note. [a]n = 49 for each group.
**Correlations significant at <.01, two-tailed.
*Correlations significant at <.05, two-tailed.

Cognitive development. Predictive validity was also explored by testing the hypothesis that the LNAS scores at birth would be correlated negatively with subscale scores of the Mullen Scales of Early Learning at 6 months of age. Data for 15 infants were available for this analysis including PDE and non-PDE infants. As shown in Table 4.20, Pearson correlation coefficients indicated the subscale of Atypical Visual Functioning was the strongest predictor of cognitive difficulties, with significant negative correlations to gross motor (-.61), visual receptive (-.72), fine motor (-.61) and the standard composite score for early learning (.-70). Low Reactivity was negatively correlated with visual receptive capacity at 6 months (-.62). Compromised System Regulation was negatively correlated with receptive language ability (-.54). The total LNAS score also showed significant correlations with visual reception, receptive language, and the standard composite Mullens score.

Table 4.20 Pearson Correlations for the Relationship Between the LNAS at Birth and Mullens Scores at 6 months[a]

Variables	Gross Motor t-score	Visual Receptive t-score	Fine Motor t-score	Receptive Language t-score	Standard Scale composite
I. Low Reactivity		-.62*			
III. Atypical Visual Functioning	-.61*	-.72**	-.61*		-.70**
V. Compromised System Regulation				-.54*	
Total Score		-.61*		-.53*	-.60*

Note. [a]n = 15 infants.
**Correlations significant at <.01, two-tailed.
*Correlations significant at <.05, two-tailed.

CHAPTER FIVE

Discussion

This chapter discussion includes four sections. Presented first is a discussion of the potential meaning and significance of the results of this study in relationship to the Research Aims and questions. Second, the strengths and limitations of the study are presented. Third, the implications for nurses are suggested and last, future directions for research are proposed.

Findings and Significance of the Research

The Profile of Drug Exposed Infants

This study consisted of 80 infants including 40 infants with PDE and a control group of 40 infants. The ethnicity of the PDE sample population consisted of African American (42.5%), Caucasian (40%) and Hispanic or other Hispanic American (17.5%). In the control group most of the women were Caucasian (72.5%) with only a small segment being African American (2%) and a larger number identified as Hispanic (22.5%). The control group's ethnicity was more congruent with a California state-wide study than the ethnic diversity of the PDE infants in this study. The largest number of infants exposed to drugs in California in 1992 were White/Non-Hispanic, followed by Hispanic and African American (Vega et. al., 1993). However, this study reflected the general PDE population reported in most of the research literature — that African American women were the largest ethnic group using drugs during pregnancy, followed by Caucasian women, and then Hispanic women. In this study, however, the number of Caucasian women who used drugs during pregnancy was almost equal to the number of African American women identified as drug users.

Among the study's pregnant women who used drugs during pregnancy, only 52.5% had complete prenatal care. This finding is higher than noted in other studies. The rest of the women had limited or no prenatal care in this study.

Limited prenatal care was defined as having four or fewer medical visits during pregnancy. Women who had complete prenatal care may reflect an outreach to support these women in their particular communities or women who are aware of the necessity of prenatal health care and/or have access and support to that care. In the control group almost all of the women had some level of prenatal care (97. 5%). Mothers of PDE infants were often unmarried and not living with a partner, almost all were unemployed, over half were receiving government financial assistance and frequently were the sole financial support of their families. Mothers in both the PDE and non-PDE groups were older in this study and had more years of education than reported in other studies. However, a large percentage of subjects in this study lived in rural areas and this may reflect a population of older women.

Many research studies provide evidence that infants with PDE are often born prior to 37 weeks gestation and have lower birth anthropometry measurements. In this study the majority of infants with PDE were birthed after 37 weeks gestation with a range of 34 to 42 weeks. This may be related to the longer term of prenatal care the drug using mothers received, or to other factors including maternal nutrition, and/or the length or type of drug exposure. The mean birth weight of infants with PDE in this study was 6.7 pounds, birth length was 19.6 inches and birth head circumference was 13.2 inches, all of which are at or below the 25th percentile according to normative data published by NCHS Growth Statistics (1994). Many factors may influence this finding, including maternal nutrition, socioeconomic status, the level of prenatal care, drug use, and gestational age.

The incidence of anemia, conjunctivitis, hyperbilirubinemia, hypoglycemia, and tachypnea were documented in both the PDE and non-PDE infants in this study. The incidence of Intrauterine Growth Restriction and SGA were docu-

mented in the PDE population only; however, all of the medical conditions are consistent with findings regarding the PDE population described in other studies. Congenital hip, facial palsy, and undescended testes were conditions found in some of the PDE infants in this study; these conditions are not found in the literature regarding infants with PDE. There appears to be a higher percentage of medical conditions in the PDE sample of this study than in the control group. This finding is consistent with other studies of PDE infants (MacGregor, et. al., 1987; Oro & Dixon, 1987).

Drug exposed infants and children are the fastest growing foster care population, and the children who are placed in a foster care environment are staying for a longer time period (U. S. Dept. of Human Services, 1992). In line with this national trend, this study had a 12.5% rate of PDE infants in foster care. Foster care could be a high risk factor for infants who experience a variety of foster home placements accompanied by inconsistent or inappropriate care, or it could be a positive factor for infants who are placed in a home with knowledgeable, caring, and nurturing caregivers.

The PDE population was exposed to a variety of drugs not unlike the infants reported in other studies; however, this study suggests a discrepancy between maternal self report of drug use and the positive drug toxicology screening performed after birth in the hospital. Relying only on positive drug toxicology reports does not always provide the evidence needed to identify the exposed infant and mother. Thirty percent of the women admitting to prenatal use of drugs during the clinician's home visit did not have positive urine toxicology screens. This disclosure might have been due in part to the clinicians going into the maternal home for the LNAS assessments, where the parent may have felt more control and trust, accompanied by a greater willingness to divulge drug use. These findings may have implications for the type and nature of infant and

maternal assessments done to identify PDE infants, and the development of guidelines and policies for identification of PDE infants.

Infants with PDE, in this study, scored higher than non-drug exposed infants on the LNAS total score and subscales scores, indicating that PDE infants displayed more atypical neurobehavioral abnormalities. Currently, there is no typical profile of infants and young children with in utero drug exposure, but studies describe particular neurobehavioral characteristics and concerns regarding motor development delays, poor social and play skills, and related attachment and separation issues. In this study, the major differences were in the areas of High Reactivity, Low Reactivity and Atypical Visual Functioning, suggesting that PDE infants have three potential areas of neurobehavioral problems: overreaction to their environment; under-reactivity to their environment; and/or difficulty in visual functioning and attention.

Reliability Findings of the LNAS

Interrater reliability. Interrater reliability testing between nurse clinicians trained to use the LNAS resulted in high reliability correlations for the subscales and total score. These results indicate that professionals trained in the use of the LNAS are assessing neurobehavioral characteristics in consistent ways.

Internal consistency. The Alpha coefficients of .90 for the overall score indicated excellent internal consistency for the total LNAS. The subscales of High Reactivity, Low Reactivity, and Atypical Visual Functioning all met or exceeded the criterion level of .80 which Nunnally (1978) states is necessary for mature psychosocial scales, pointing to very acceptable homogeneity of these subscales. However, subscales Atypical ANS Response and Compromised System Regulation demonstrated a low level of homogeneity at .53 and .58 respectively.

Inter-item correlations to further investigate the low alpha reliability of these

two subscales demonstrated a lack of relationship between some of the items. The items High Pitched Cry and Unexplained Fevers seemed to have little relationship to other items in their subscale. Findings clearly indicate a problem with these two subscales and suggest the need for refinement. However, their total elimination from the assessment tool seems premature, since they do appear to have clinical importance. In addition, both subscales were significantly correlated with the total LNAS score, suggesting that they do contribute to the overall profile of infant vulnerability.

Test-retest reliability. Both short term and long term stability of the LNAS were assessed. Strong Pearson correlations for one week stability indicated excellent test-retest reliability for the LNAS. Stability at six months was also impressive, despite the potential for developmental change in an infant's neurobehavioral profile. Scores for one of the subscales, Low Reactivity, changed significantly from birth to six months. These findings could indicate more variability in these particular neurobehaviors over time.

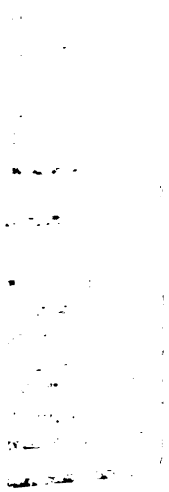
Validity Findings of the LNAS

Construct validity. Construct validity testing using the known group approach indicated that there was a statistically significant difference between PDE and non-PDE infants in this study as well as between high and low risk infants. When the total sample was used to compare high and low risk infants, all LNAS subscales indicated excellent ability to predict risk status. However, when only the PDE infants were used for the risk analysis, only the total score and the subscales of Atypical Visual Functioning and Low Reactivity showed a consistent ability to discriminate between high and low risk status. The subscale of Low Reactivity was a better predictor of risk when maternal complications were used as the index of risk. However, when low birth weight was the risk index, the subscale of High Reactivity was a better predictor of risk. The inability of all

subscales to discriminate between risk groups within the PDE sample could be related to the less adequate variance within overall scores of the PDE group, since they were more at risk to begin with. The smaller sample size also provided less power to identify potential differences that might actually exist in the larger population of PDE infants.

Predictive validity. Predictive validity was first assessed by examining the hypothesis that an infant's scores on the LNAS subscales of High Reactivity and Low Reactivity would be negatively related to an infant's clarity of cues and responsiveness to the parent. The infant's Clarity of Cues negatively correlated with the LNAS subscale Low Reactivity, but showed only a trend toward a relationship with the subscale High Reactivity. Since the reliability and construct validity of the High Reactivity Subscale were sound, the data could suggest that a newborn's under-reactivity to the environment is much more predictive than hyper-reactivity of the infant's decreased Clarity of Cues when interacting with a caregiver. The infant's Responsiveness to Parent was significantly correlated with all of the LNAS subscales and total score, supporting the validity of the LNAS as a predictor of infant interpersonal behavior. The data suggests that infants who exhibit neurobehavioral abnormalities and are disorganized behaviorally or physiologically cannot relate or respond as well to their caregiver or to events in their environment.

Predictive validity was also explored by testing the hypothesis that LNAS scores at birth would correlate negatively with indicators of early cognitive development at six months. This hypothesis was supported by the correlations of the total LNAS score and three LNAS subscale scores with infant scores on the Mullen Scales of Early Learning. The total LNAS score predicted problems with visual and language reception as well as the composite score for early learning difficulties. LNAS subscale Atypical Visual Functioning was the most predictive



of the subscales, with strong relationships to gross motor, visual receptive and fine motor problems as well as the composite score. The Low Reactivity subscale predicted visual receptive problems and the subscale Compromised System Regulation predicted receptive language problems. The data clearly indicates that infants who have atypical visual functioning at birth are the most at risk for later learning difficulties. They also support the validity of the Atypical Visual Function subscale as a strong predictor of later learning outcome. The predictive validity of the LNAS subscales of High Reactivity and Atypical ANS Response was not supported.

Strengths and Limitation of the Study

Strengths

This study focused on the refinement and testing of an assessment tool to identify neurobehavioral problems of infants exposed to drugs in utero. No typical profile of infants and young children with in utero drug exposure exists, but there are studies that describe particular neurobehavioral characteristics and concerns related to attachment and separation, motor development delays, poor social and quality of play skills. This study will add to the body of scientific data describing atypical neurobehaviors which correlate with prenatal drug exposure. The information can provide a logical framework for developing directed types of nursing interventions as well as providing a better understanding of the PDE population's specific areas of risk. This study provided further reliability and validity evidence for an assessment tool which is useful for collecting relevant data regarding the individual PDE infant's specific risk profile.

Home visiting was an important technique used in this study not only for gathering of information, but to evaluate the infant and the caregiver in their particular environment. The mother was able to share information in her home with the nurse clinician about prenatal drug use that was not detected by urine

toxicology screen or documented in any medical records. This is important information, as most statistics on maternal drug use and the testing of infants are based on the hospital urine toxicology screening, which may not be an accurate reflection of current or earlier drug use during pregnancy.

Limitations

The ethnic distribution between the PDE and non-PDE infants was a limitation of this study. Most of the control group were Caucasian, while the PDE group was almost equal in African American and Caucasian infants. Therefore, this information may not be generalizable to other populations of PDE infants.

Lack of control for other variables between birth and six-month testing was another limitation. The sample size was not large enough to control for gender, birth weight, ethnicity, socioeconomic class, or the infant's post natal experience or environment, all of which are known to produce differences in children. Brain research on animals demonstrates that early experience and the environment is critical to the "hard wiring" of the brain, thereby effecting development.

The inability to identify single drug effects was also a limitation. Polydrug use during pregnancy was common in this study as documented by positive toxicology screens and/or maternal admission. This is not an uncommon finding with the population studied. The toxic effects of the confounding substances on infant behavior and development have been documented repeatedly in the literature. However, the assessment measure was designed to be used with all infants with PDE regardless of the drug exposure.

A limitation to analyzing and interpreting the results of this study was the small sample size. Although there were seven subjects for each item of the tool and a minimum of 5 subjects are recommended for reliability studies (Nunnally, 1978), internal consistency of the LNAS subscales needs to be reexamined with a larger sample. In addition, the small sample size precluded use of factor analytic

techniques for testing construct validity. The small sample size used for both the NCAST and Mullen testing also limits the generalizing of the results. A larger sample size and a more diverse population needs to be examined.

Future Directions for Research

Interrater and test-retest reliability of the instrument look quite good. However, the study indicates problems with internal consistency of the subscales Atypical ANS Response and Compromised System Regulation. Based on the inter-item correlations within these two subscales, the elimination of two items may be warranted, as well as a reconsideration of the subscale structure of the tool. These refinements are further supported by the findings from construct validity testing. These same two subscales showed little ability to discriminate subtle differences in risk status within PDE infants.

Once the two problematic subscales are refined, further studies with a larger sample need to be pursued. Studies with larger samples will yield additional information about the value of specific items within the measure, will enable factor analytic approaches to examine the structure validity of the LNAS, and will allow for more adequate studies to correlate the LNAS with clinical developmental outcomes. Until these further studies are completed, the LNAS should be used with discretion and minimal reliance on the two problematic subscales. In contrast, the total LNAS score and three of its subscales can be used with substantial assurance of their validity and reliability.

Assessments could be done at different ages such as six months, 12 months and 24 months to see if the predictive value still holds. One might use the LNAS scores on the 12-month-old or under and see if those scores predict a three-year-old's performance on the Mullen.

In addition, future research needs to include testing of two other subscales which are administered to older infants starting at four months of age. These

two subscales need to be a part of any longitudinal studies that are initiated. If neurobehavioral differences continue to be observed, it would be useful to study how these differences affect various areas of learning and social development of the child.

This study has provided preliminary information that contributes to the understanding of infants with PDE. It is anticipated that this assessment measure will be used to further identify neurobehavioral characteristics of PDE. In addition, it can be used to provide knowledge upon which to build intervention strategies to enhance early organization and healthy development. These interventions can be evaluated by pre- and post-test comparison of LNAS scores.

It's not clear how biological and environmental factors may interact to influence the PDE infant during pregnancy or after birth. It would be useful to conduct this study in a broader geographical area and with a more diverse populations of pregnant women. A larger sample size would lend itself to a more complex multiple regression analyses such as path analysis. A path analysis would allow the researcher to determine the contribution of multiple factors to neurobehavioral status of the infant and to examine the interaction of neurobehavioral status with environmental variables in the process of development during the first year of life.

A larger sample size would also allow for development of research models to look at a variety of variables that may affect infant and child development. Animal research supports the idea that environmental variables can modify the overall size of the cerebral cortex, increase the number of synaptic and dendritic connections and modify the function of neurotransmitters in the central nervous system. This brain research lends credence to models of early intervention beginning at birth.

The results from the research indicate that some of the infants with PDE have

few or mild atypical neurobehaviors at birth while other infants are very disorganized. Longitudinal studies need to occur in order to determine if these differences persist over time, their potential etiology, and whether they interfere with social and behavioral development or learning outcomes.

Implications for Nursing Practice

This study will add to the body of knowledge and information about specific atypical neurobehaviors that the PDE population exhibit during the first year of life. This information will help clinicians, researchers and other professionals identify PDE infants and focus their observations to determine if and in what ways these early atypical neurobehaviors continue to manifest as the child grows and develops. These findings indicate the importance of looking at individual subscale scores, not just the total score, and documenting any atypical behaviors and difficulties the infant has in performing the task.

Once the assessment tool is refined, it can be used by a variety of professional nurses in the hospital, ambulatory, and home settings. The LNAS can be used by nurses with a minimal amount of training, be administered in a short time frame, and has a high level of interrater reliability.

The consistent significance of the Atypical Visual Function subscale as an indicator of risk may suggest its special importance in identifying early problems. It is currently unknown whether infants with PDE, like infants with cataracts, are not processing visual input adequately and, therefore, not stimulating the neurons in the occipital lobe which are necessary for brain development. Caregivers who do not have positive reactions or interactions with their infant and do not receive effective cues from the infant may not provide the types of infant stimulation needed for growth and development. This phenomenon can place the newborn at high risk for inappropriate care and interactions contributing to developmental problems. The LNAS information can be used for develop-

ment of better strategies to aid parents of PDE infants in support of their child's ongoing neurobehavioral adaptations. This may in turn promote stronger parent-child bonds and a healthier child.

The subscale scores of Atypical ANS Response and Compromised System Regulation have clinical importance. In a hospital setting, high scores indicate the need for nursing intervention, which is often treated with proper handling, to organize the infant and in some instances may involve the administration of medication. The subscale scores can serve as a basis for determination of the need for intervention.

Subtle atypical neurobehavioral findings documented by the LNAS can interfere with an infant's behavior organization. Documentation of these neurobehaviors after birth can lead to a more accurate identification of high risk infants. Nurses can use this early information to design and implement intervention strategies to help the infant to become more organized and help the caregiver understand their infant's behavioral cues. This early maternal-infant support can contribute to caregiver attachment and later appropriate social responses from the infant.

In this study there was no significant difference in the gestational age of the infants who are exposed to drugs in utero and those who were not exposed. However, drug exposure did have a statistically significant negative effect on birth weight, birth length and birth head circumference. Hospital screening for newborn drug exposure often occurs on infants below 37 weeks and on mothers with a history of no prenatal care, which may not be the best indicators for hospital toxicology screening policies. It may be more beneficial to screen infants who are SGA or have IUGR as determined by birth weight, length and head circumference.

Findings regarding the descriptive characteristics of the women who used

drugs in this study provide a basis for the development of nursing strategies that address multilevel parenting education and understanding about high risk infants and young children. Parents at all levels of the educational and socioeconomic spectrum need to be provided with educational approaches designed to meet their level of education and comprehension.

Home health and school nurses routinely do home visits with families. This study supports the contention that a home visit provides information about the child and family that may not be made discernible at health sites that are outside the home. Caregiver information and education can be provided during the home visit when parents are more relaxed and receptive to teaching.

Nurses can use the information in the study to consult and teach future and current parents about the effects of prenatal drug exposure on the infant. This same information can be shared with any professional working with the population in hospitals, schools, the community or home. The nurse, in many instances, works as part of a multidisciplinary team, and the information gathered from an LNAS assessment can be part of the team assessment.

Summary

This study provides evidence for the validity and reliability of the LNAS assessment tool in the early identification of neurobehavioral problems and ongoing assessment of PDE infants. The total LNAS score appears quite robust, with three of its subscales showing excellent reliability and validity. The subscale of Atypical Visual Functioning seems the strongest of the subscales, with High and Low Reactivity showing very acceptable findings as well. Data suggest that the subscales of Atypical ANS Response and Compromised System Regulation have problems with internal consistency and less value in discriminating risk within the already high risk group of PDE infants.

The findings, although limited by sample size, contribute valuable data re-

garding neurobehavioral characteristics of PDE infants at birth and their implications for later development. The instrument has multidisciplinary application and strengthens nursing's contribution to the care of high risk infant populations. Future studies must be conducted to refine two subscales of the instrument and determine its full utility within a larger population of PDE infants.

References

Almli, C.& Finger, S. (1984). Early brain damage. Orlando, FL: Academic Press.

Barnard, K. (1978). NCAST. Nursing child assessment scale training: Learning resource manual. Seattle, WA: NCAST Publications.

Bauchner, H., Zuckerman, B., McClain, M., Frank, D., Fried, L. & Kayne, H. (1988). Risk of sudden infant death syndrome among infants with in utero exposure to cocaine. Journal of Pediatrics, 113, 831-834.

Bayley, N. (1969). Manual for the Bayley Scales of Infant Development. New York: Psychological Corporation.

Bear, B. (1995). Drugs and the effects on health and development of the fetus, neonate, infant, and child. In K. Lewis (ed.) Infants and children with prenatal alcohol and drug exposure: A guide to identification and intervention (pp. 21-56). MN: Sunrise River Press.

Beeghly, M. & Tronick, E. (1994). Effects of prenatal exposure to cocaine in early infancy: Toxic effects on the process of mutual regulation. Infant Mental Health Journal, 15, (2), 158-175.

Bertacchi, G, (1987). Ready-Set-Grow, Cocaine, Heroin and Marijuana. Nursing Today, May/June, 14-25.

Billing, L., Eriksson, M., Jonsson, G., & Zetterstrom, R. (1994). The influence of environmental factors on behavioral problems in 8-year-old children exposed to amphetamine during fetal life. Child Abuse and Neglect, 18, 3-9.

Bingol, M., Fuchs, M., Halipas, N., Henrique, R., Pagan, M., & Diaz, V. (1986). Prune belly syndrome associated with maternal cocaine use. American Journal Of Human Genetics, 39, 147.

Brazelton, T.B. (1984). Neonatal Behavioral Assessment Scale. (Rev. Ed.) Clinics in Developmental Medicine, 88. Philadelphia: J. B. Lippincott.

Briggs, G., Freeman, R. & Yaffe, S. (1990). Drugs in pregnancy and lactation. Baltimore, MD: Williams & Wilkins.

Brown, E., & Zuckerman, B., (1991). The infant of the drug abusing mother. Pediatric Annals, 20, 555-563.

Chandler, L., Andrews, M., & Swanson, M. (1980). Movement assessment of infants: A manual. Rolling Bay, WA: Movement Assessment of Infants.

Chaney, N., Franke, J., & Wadington, W., (1988). Cocaine convulsions in a breast feeding baby. Journal Pediatrics, 112, 134-135.

Chasnoff, I. J. (1988). Drug use in pregnancy: Parameters of risk. The Pediatric Clinics of North America, 35, 1403-1412.

Chasnoff, I., Burns, K., & Burns, W. (1986). Prenatal drug exposure: Effects on neonatal and infant growth and development. Neurobehavior, Toxicology, and Teratology, 8, 357.

Chasnoff , I ., Burns, K., Burns, W., & Schnoll, S., (1986) . Prenatal drug exposure: Effects on neonatal and infant growth and development. Neurobehavioral Toxicology and Teratology, 8, 357-362.

Chasnoff, I., Bussey, M. & Savick, R, (1986). Perinatal cerebral infarction and maternal cocaine use. Journal Pediatrics, 108, 456-459.

Chasnoff, I. & Griffith, D. (1989). Cocaine: Clinical studies of pregnancy and the newborn. Annals of the New York Academy of Sciences, 562, 260-266.

Chasnoff, I., Griffith, D., Freier, C.& Murray, J. (1992). Cocaine/polydrug use in pregnancy: Two year follow–up. Pediatrics, 89 (2), 284–289.

Chasnoff, I., Griffith, D., MacGregor, S., Dirkes, K., Burns, K. A. (1989). Temporal patterns of cocaine use in pregnancy. Journal of American Medical Association, 261, 1741-1744.

Chasnoff, I., Hunt, C., Kletter, R., & Kaplan, D. (1989). Prenatal cocaine exposure is associated with respiratory pattern abnormalities. American Journal Diseases In Children, 989, (143) 538-587.

Chasnoff, I. J., Burns, K. A., & Burns, W. J. (1987). Cocaine use in pregnancy: Perinatal morbidity and mortality. Neurotoxicology and Teratology, 9, 291-293.

Chasnoff, I.J., Burns, W. J., Hatcher, R. P, & Burns, K.A., (1983). Phencyclidine: Effects on the fetus and neonate. Developmental Pharmacology Therapy, 6, 404-8.

Chasnoff, I. J., Burns, W. J., Schnoll, S. H., & Burns, K. A. (1985). Cocaine use in pregnancy. New England Journal of Medicine, 313, 666-669.

Cicchetti, D. & Wagner, S. (1990). Alternative assessment strategies for the evaluation of infants and toddlers. An organizational perspective. In S. J.

Meisels & J.P. Shonkoff (Eds.) Handbook of early childhood intervention. Cambridge: Cambridge University Press.

Clark, R., Paulson, A., Conlin, S. (1993). Assessment of developmental status and parent-infant relationships: The therapeutic process of evaluation. In C. Zeanah, Jr. (Ed.) Handbook of Infant Mental Health. New York: The Guilford Press.

Cobinik, R., Hood, R., & Chusid, E. (1959). The effect of maternal narcotic addiction on the newborn infant: Review of literature and report of 22 cases. Pediatrics 24, 288-304.

Committee on Drugs, (1994). The transfer of drugs and other chemicals into human milk. Pediatrics, 93(1),137-141.

Cummings, A. (1979). Transplacental disposition of phencyclidine in the pig. Xenobiotica, 9, 447-452.

Dalterio, S., Mayfield, D., Bartke, A. (1984). Early cannabinoid exposure influences neuroendocrine and reproductive functions in male mice: Prenatal exposure. Pharmacology Biochemical Behavior, 20, 107-1 14.

D'Apolito, K. (1991). What is an organized infant? Neonatal Network, 10 (1), 23-29.

Day, N., Richardson, G., Goldschmidt, L., Robles, N., Taylor, P., Stoffer, D., Cornelius, M. & Geva, D. (1994). Effect of prenatal marijuana exposure on the cognitive development of offspring at age three. Neurotoxicology and Teratology, 16 (920), 169-175.

de Ridder, M. (1994). Heroin : New facts about an old myth. Journal of Psychoactive Drugs, 26 (1), 65-68.

Desmond, M., & Wilson, G. (1975). Neonatal abstinence syndrome: Recognition and diagnosis. Addictive Disability, 1, 113-121.

Dixon, S . (1989). Effects of transplacental exposure to cocaine & methamphetamine on the neonate. Western Journal of Medicine, 150 (4), 436-442.

Dixon, S.D., & Bejar, R. (1989). Echoencephalographic findings in neonates associated with maternal cocaine and methamphetamine use: Incidence and clinical correlates. Journal of Pediatrics, 115, 770.

Doberczak, T., Shanzer, S., Senie, K., Kandall, S. (1988). Neonatal neurologic

and electroencephalographic effects of intrauterine cocaine exposure. Journal of Pediatrics. 113, (2), 354-359.

Dow-Edwards, D. (1995). Developmental toxicity of cocaine: Mechanisms of action. In Lewis, M. & Bendersky, M. (Eds.) Mothers, babies, and cocaine: The role of toxins in development (pp. 517). New Jersey: Lawrence Erlbaum Associates, Publishers.

Dreher, M., Nugent, K., & Hudgins, R. (1994). Prenatal marijuana exposure and neonatal outcomes in Jamaica: An ethnographic study. Pediatrics, 93 (2), 254-260.

Durand, D., Espinoza, A., & Nickerson, B. (1990). Association between prenatal cocaine exposure and sudden infant death syndrome. Journal of Pediatrics, 117, 909-911.

Eckerman, C., Lynne, A., & Gross, S. (1985). Different developmental courses for very low birthweight infants. Developmental Psychology, 21, 813-827.

Emde, R., (1989). The infant's relationship experience; Developmental and affective aspects. In A.J. Sameroff & R.M. Emde (Eds.) Relationship disturbances in early childhood (pp. 33-51). New York: Basic Books, Inc. Publishers.

Eriksson, M., Larsson, G., Winbladh, B., & Zetterstrom, R. (1978). The influence of amphetamine addiction on pregnancy and the newborn infant. Acta Paediatr Scand, 67, 95-99.

Eriksson, M., & Zetterstrom, R. (1981). The effect of amphetamine addiction on the fetus and child. Teratology, 24, 39A (abstra).

Ferrans, C., & Powers, M. (1992). Psychometric assessment of the quality of life index. Research in Nursing and Health, 15, 29-38.

Fico, T., & VanderWende, C. (1988). Phencyclidine during pregnancy: Fetal brain levels and neurobehavioral effects. Neurotoxicology and Teratology, 10, 349-354.

Finnegan, L. (1976). Clinical effects of pharmacological agents on pregnancy, the fetus and neonate. Annual New York Academy of Science, 281, 74-89.

Francis, P., Self, P., & Horowitz, F. (1989). The behavioral assessment of the neonate: An overview. In J. Osofsky (Ed.), Handbook of infant development (2nd Ed.). (pp. 723-779). New York: Wiley.

Fried, P. (1980). Marijuana use by pregnant women: Neurobehavioral effects in neonates. Drug & Alcohol Dependency, 6, 415-424.

Fried, P. (1985). Postnatal consequences of maternal marijuana use. National Institute on Drug Abuse Research Monograph, 59, Rockville MD, National Institute of Drug Abuse, 426-430.

Fried, P., & Makin, J. (1987). Neonatal behavioral correlates of prenatal exposure to marijuana, cigarettes and alcohol in a low risk population. Neurotoxicology and Teratology, 9, 1-7.

Fried, P.A. & Watkinson, B. (1990). Thirty-six and 48 month neurobehavioral follow-up of children prenatally exposed to marijuana, cigarettes, and alcohol. Developmental and Behavioral Pediatrics, 11, 49-58.

Fried, P., Watkinson, B., & Gray, R, (1992). A follow-up study of attentional behavior in 6 year old children exposed prenatally to marijuana, cigarettes, and alcohol. Neurotoxicology & Teratology, 14, 299-311.

Fried, P., Watkinson, B., & Willian, A. (1984). Marijuana use during pregnancy and decreased length of gestation. American Journal of Obstetry Gynecology, 150, 23.

Fries, M., Kuller, J., Norton, M., Yankowitz, J., Kobori, J., Good, W., Ferriero, D., Cox, V., Donlin, S., Golabi, M. (1993). Facial features of infants exposed prenatally to cocaine. Teratology, 48 (5), 413-420.

Galanter, M & Kleber, H. (1994). Textbook of substance abuse treatment. Washington, DC: American Psychiatric Press, Inc.

Gilbert, E.& Khoury, G. (1970). Dextroamphetamine and congenital cardiac malformations. Journal of Pediatrics, 76, 638.

Golden, N.L., Sokol, R. J., & Rubin. L.L. (1980). Angel dust: Possible effects on the fetus. Pediatrics, 65, 18-20.

Gomby, D. & Shiono, P. (1991). Estimating the number of substance exposed infants. Future of Children, I (I), 17-25.

Good, W., Ferriero, D., Golabi, M., Kobori, J. (1992). Abnormalities of the visual system in infants exposed to cocaine. Ophthalmology, 99 (3), 341-346.

Greenspan, S. (1991). Regulatory disorders I: Clinical perspectives. In M. Kilbey & K. Asghar (Eds.), Methodological issues in controlled studies on effects of prenatal drug exposure to drug abuse. NIDA Research Monograph, 114, 173-181.

Griffith, D. (1992). Prenatal exposure to cocaine and other drugs: Developmental and educational prognoses. Phi Delta Kappan, Sept, 30-34.

Griffith, D. (1995). Cognitive development of children prenatally exposed to alcohol, tobacco, and other drugs. In K. Lewis (ed.) Infants and children with prenatal alcohol and drug exposure: A guide to identification and intervention (pp.167-186), MN: Sunrise River Press.

Griffith, D., Azuma, S., Chasnoff , I ., (1994) . Three-year outcome of children exposed prenatally to drugs. J. Am. Academy Child Adolescent Psychiatry, 33 (1), 20-27.

Griffith, D. R. (1988). The effects of perinatal cocaine exposure on infant neuro-behavior and early maternal-infant interactions. In I. Chasnoff (Ed.) Drugs, alcohol, pregnancy, and parenting (pp.105-113). Hingham, MA: Kluwer Academic Publishers.

Gross, S., Oehler, J. & Eckerman, C. (1983). Head growth and development outcome in very low birthweight infants. Pediatrics, 71, 70-75.

Groves, P., Ryan, L., Linder, J. (1987). Amphetamine changes neostriatal morphology. In Friedman, D & Clouet, D. (Eds), The role of neuroplasticity in the response to drugs. National Institute on Drug Abuse (Research Monograph Series 78, 132-142). Rockville, MD: The Institute.

Hack, M., Breslan, N., Weissman, B., Aram, D., Klein, N., Borawski, E. (1991). Effects of very low birthweight and subnormal head size on cognitive abilities at school age. New England Journal of Medicine, 325, 231-237.

Hammer, R., Ricalde, A., & Seatriz, J. (1989). Effects of opiates on brain development. Neurotoxicology, 10, 475-484.

Hans, S., (1989). Developmental consequences of prenatal exposure to methadone. Annals New York Academy of Sciences, 562, 195-207.

Harris, S., Haley, S., Tada, W., Swanson, M. (1984). Reliability of observational measures of the Movement Assessment of Infants. Physical Therapy, 64, 471–475.

Harris, S., Swanson, M., Andrews, M., Sells, C., Robinson, N., Bennett, F., Chandler, L. (1984). Predictive validity of the movement assessment of infants. Developmental and Behavioral Pediatrics, 5, (6); 336–342.

Harris, S.R. (1987). Early detection of cerebral palsy: Sensitivity and specificity of two motor assessment tools. Journal of Perinatology, 7, 11-15

Harris, S.R., Osborn, J.A., Weinberg, J., Loock, C., Junaid, K. (1993). Effects of prenatal alcohol exposure on neuromotor and cognitive development during early childhood: A series of case reports. Physical Therapy, 73, 608-617.

Harry , G . J ., & Howard , J . (1992) . Phenylcyclidine : Experimental studies in animals and long-term developmental effects on humans. In T.B. Sonderegger (Ed.) Perinatal substance abuse. (pp.254-278). Baltimore, MD: The Johns Hopkins University Press.

Hatch, E.,& Bracken, M. (1986). Effect of marijuana use in pregnancy on fetal growth. American Journal of Epidemiology, 124, 986-993.

Hayes, J., Dreher, M. & Nugent, K. (1988) Neonatal outcomes with maternal marijuana use in Jamaican women. Pediatric Nursing, 14, (2), 107–110.

Hingson, R. Alpert, J., Day, N., Dooling, E., Kayne., H., Morelock, S., Oppenheimer, E., Rosett, H., Weiner, L. & Zuckerman, B. (1982). Effects of maternal drinking and marijuana use on fetal growth and development. Pediatrics, 7, 539-546.

Horowitz, F., & Brazelton, T. (1973). Research with the Brazelton neonatal scale. In T. B. Brazelton (Ed.), Neonatal Behavioral Assessment Scale. National Spastics Society Monograph. Philadelphia: Lippincott.

Householder, J., Hatcher, R., Burns, W., & Chasnoff, I. (1982) . Infants born to narcotic-addicted mothers. Psychological Bulletin, 92, 453-468.

Howard, J., Kropenski, V. & Tyler, R. (1986). The long term effects on neurodevelopment of infants exposed to PCP. In D.H. Coluet (Ed.) Phencyclidine: An update. National Institute on Drug Abuse (Research Monograph Series, 64, pp 623-630.) Rockville, M.D.: The Institute.

Hutchings, D. (1982). Methadone and heroin during pregnancy: a review of behavioral effects in human and animal offspring. Neurobehavioral Toxicology and Teratology, 4, 429-434.

International Network Parenthood - Drug Abuse (1996). Parenthood-drug abuse international conference proceedings. Brussels: Brussels Midrash.

Jacobs, B. L. (1985). Overview of the activity of the brain monoaminergic neurons across the sleep-wake cycle. In A. Wauquier, J. M. Monti, J. M., Gaillard, & M. Radilovacki (Eds.), Sleep-neurotransmitters and neuromodulators (pp. 1-14), New York: Raven Press.

Jacobson, J. & Jacobson, S. (1991). Assessment of teratogenic effects on cognitive and behavioral development in infancy and childhood. Research Monograph Series, 114. MD: National Institute on Drug Abuse.

Jarvis, M., & Schnoll, S. (1994). Methadone treatment during pregnancy. Journal of Psychoactive Drugs, 26 (2), 155-161.

Kaltenbach, K. & Finnegan, L. P. (1989). Prenatal narcotic exposure: Perinatal and developmental effects. Neurotoxicology, 10, 597-600.

Kammel, B., Gardner, J. & Freedland, R. (1996, April) Long-term cognitive and attentional effects of intrauterine cocaine exposure. Paper presented at the International Conference on Infant Studies, Providence, Rhode Island.

Kandall, D. & Gaines, J. (1991). Maternal substance use and subsequent sudden infant death syndrome (SIDS) in offspring. Neurotoxicology and Teratology, 13, 235-40.

Kandall, S., Albin, S., Gardner, L., Lee, K., Eidelman, A. & Lowinson, J. (1977). The narcotic dependent mother: Fetal and neonatal consequences. Early Human Development, 1, (2), 159-166.

Kandall, S. R., (1991). Perinatal effects of cocaine and amphetamine use during pregnancy. Bulletin of New York Academy Medicine, 67 (3), 240-255.

Kaufman, K.R., Petrucha, R.A., Pitts, F.N., & Kaufman, E.R. (1983). Phenocyclidine in umbilical cord blood: Preliminary data. American Journal Psychology, 140 (4), 450.

Kaufman, K.R., Petrucha, R.A., Pitts, F.N. & Weekes, J.D. (1983). PCP in amniotic fluid and breast milk: Case report. Journal of Clinical Psychology, 44 (7); 269.

Kopp, C. (1982). Antecedents of self-regulation: A developmental perspective. Developmental Psychology, 18, (2),199-214.

Kopp, C. , (1990). Risks in infancy: Appraising the research. Merrill-Palmer Quarterly, 36, (1),117-139.

Kron, R., Litt, M., & Phoenix, M. (1976). Neonatal narcotic abstinence: Effects of pharmacotherapeutic agents and maternal drug usage on nutritive sucking behavior. Journal of Pediatrics, 88, 637-648.

Lawton, T. A., (1992). Maternal cocaine addiction: Correlates and consequences. Unpublished doctoral dissertation., University of Michigan, Ann Arbor, MI.

Le Blanc, P.E., Parekh, A.J., Naso, B., Glass, L. (1987). Effects of intrauterine exposure to alkaloidal cocaine (crack) American Journal Disabilities Children 141, 937-938.

Lester, B. & Dreher, M. (1989). Effects of marijuana use during pregnancy on newborn cry. Child Development, 60, 765-771.

Lester, B. M. (1984). Data analysis and prediction. In T. B. Brazelton (Ed.), Neonatal Behavioral Assessment Scale (2nd ed.) Spastics International Medical Publications. London: Blackwell, Philadelphia: Lippincott.

Levine, J. (1971). Amphetamine ingestion with biliary atresia. Journal of Pediatrics, 79, 130-131.

Lewis, K. D. (1991). Behavioral characteristics of prenatal drug exposed infants using the Lewis Protocol. Unpublished thesis. University of San Francisco. San Francisco, CA.

Lewis, K., Schmeder, & Bennett, B. (1992). Maternal drug abuse and its effect on young children. Maternal Child Nursing, 17, (4), 198-203.

Lewis, K. D., Bennett, B., & Schmeder, N. (1989). The care of infants menaced by cocaine abuse. Maternal Child Nursing, 14, (4), 324-329.

Lifschitz, M., Wilson, G., Smith, E., Desmond, M. (1983). Fetal and postnatal growth of children born to narcotic-dependent women. Journal of Pediatrics, 102 (5), 686-691.

Lifschitz, M., Wilson, G., Smith, E., Desmond, M. (1985). Factors affecting head growth and intellectural function in children of drug addicts. Pediatrics, 75 (2), 269-274.

Linn, S., Schoenbaum, S., Monson, R., Rosner, R., Stubblefield, P., & Ryan, K. (1983). The association of marijuana use with outcome of pregnancy. American Journal of Public Health, 73, 1161-1164.

Lynch, E. (1995). Developmental assessment of infants and children who are at risk due to prenatal alcohol and drug exposure. In K. Lewis, Infants and children with prenatal alcohol and drug exposure: A guide to identification and intervention, (pp. 275-296). MN : Sunrise River Press.

Lynn, M. (1986). Determination and quantification of content validity. Nursing Research, 35(6), 382-385.

MacGregor, S., Keith, L.G., Chasnoff, I.J., Rosner, M., Chisum, G., Shaw, P., & Minogue, D. (1987). Cocaine use during pregnancy: Adverse perinatal outcome. American Journal of Obstetric Gynecology, 157, 686-690.

Martin, J. (1992). The effects of maternal use of tobacco products or amphetamines on offspring. In T. B. Sonderegger (Ed.) Perinatal Substance Abuse (pp. 279-304). Baltimore, MD: The Johns Hopkins University Press.

Mayes, L. (1994). Neurobiology of prenatal cocaine exposure effect on developing monoamine systems. Infant Mental Health Journal 15, (2) 121-133.

Mayes, L. & Bornstein, M. (1995). Developmental dilemmas for cocaine-abusing parents and their children. In Lewis, M & Bendersky, M. (Eds.) Mothers, babies and cocaine: The role of toxins in development (pp. 251-272). New Jersey: Lawrence Erlbaum Associates, Publishers.

Meisels, F., & Wasik, B. (1990). Who should be served? Identifying children in need of early intervention. In S. J. Meisels & J. P. Shonkoff (Eds). Handbook of early Childhood intervention (pp. 605-632). New York: Cambridge University Press.

Meisels, S. (1988). Developmental screening in early childhood: The interaction of research and social policy. In L. Breslow, J. Feilding & L. Lave (Eds.) Annual review of public health, Vol. 9 (pp. 527-550). Palo Alto, CA: Annual Review.

Mercer, R. T. (1989). Theoretical perspectives on the family. In C.L. Gilliss, B.L. Highley, B.M. Roberts & I. M. Martinson (Eds.). Towards a science of family nursing. (pp. 37-63). Menlo Park, Ca: Addison Wesley.

Middaugh, L.D. (1989). Prenatal amphetamine effects on behavior: Possible mediation by brain monoamines. In D.E. Hutchins (ed.) Prenatal Abuse of Licit and Illicit Drugs. (Annals of the New York Academy of Sciences, Vol 562, 308-318). New York: The New York Academy of Sciences.

Morrison, D. (1992). Issues in assessing the intelligence of prenatally drug exposed infants. Newsletter of the Clearinghouse for Drug Exposed Children, 3, (2), 1-3.

Morrison, D. & Villarreal, S. (1993). Cognitive performance of prenatally drug-exposed infants. Infant-Toddler Intervention, 3 (3), 220-221.

Mullen, E. (1989). Infant MSEL manual. Cranston, RI: T.O.T.A.L. Child Inc.

Mullen, E. (1995). Mullen scales of early learning. MN: American Guidance Service.

Munro, B., Visintainer M., & Page, E., (1986). Statistical Methods for Health Care Research. J.B. Lippincott Company: Philadelphia.

Naeye, R. (1965). Malnutrition, a probable cause of fetal growth retardation. Arch. Pathology, 79, 284-291.

Naeye, R., Blanc, W., Leblanc, W., & Khatamee, M. (1973). Fetal complications of maternal heroin addiction: Abnormal growth, infections, and episodes of stress. Journal of Pediatrics, 83 (6), 1055-1061

Neuspiel, D.R., & Hamel, S.C. (1991). Cocaine and infant behavior. Journal of Developmental and Behavioral Pediatrics, 12, 55-64.

Nicholas, J., & Schreiber, E. (1983). Phencyclidine exposure and the developing mouse: Behavioral teratological implications. Teratology 28, 319-326.

Nora, J.J., Vargo T., Nora A., Love K., McNamara, D.(1970) Dextroamphetamine: A possible environmental trigger in cardiovascular malformations. Lancet, 1, 1290-1291.

Novitt-Moreno, A. (1996). How your brain works, (pp. 133-137). Emeryville, CA.: Ziff-Davis Press.

Nunnally, J.C., (1978). Psychometric Theory. 2nd Ed., N.Y.: McGraw-Hill, Inc.

Oro, A.S., & Dixon, S.D. (1987). Perinatal cocaine and methamphetamine exposure: Maternal and neonatal correlates. Journal of Pediatrics, 11 (4), 571-578.

Perez-Reyes, M., Walls, M. (1982). Presence of tetrahydrocannabinol in human milk. New England Journal of Medicine, 307, 819.

Petrucha, R., Kaufman, K., & Pitts, F. (1982). Phencyclidine in pregnancy: A case report. Journal of Reproductive Medicine, 27, 301-303.

Phibbs, C., Bateman, D., & Schwartz, R. (1991). The neonatal costs of maternal cocaine use. Journal of the American Medical Association, 226 (II), 1521-1526.

Polit, D., & Hungler, B. (1991). Nursing Research: Principles and Methods (4th Ed.). New York: Lippincott Pub. Co.

Rahbar, F., Fomufod, A., White, D., & Westney, L, (1993). Impact of intrauterine exposure to Phencyclidine (PCP) and cocaine on neonates. Journal of the National Medical Association, 85 (5), 349-352

Ramer, C . (1974). The case history of an infant born to an amphetamine-addicted mother. Clinical Pediatric, 13, 596-597.

Ricaurte, G, Seiden, L. & Schuster, M. (1984). Further evidence that

amphetamines produce longlasting dopamine neurochemical deficits by destroying dopamine nerve fibers. Brain Residency, 303, 359-364.

Richardson, G., & Day, N. (1990). Maternal and neonatal effects of moderate cocaine use during pregnancy. Neurotoxicology and Teratology,13, 455-460.

Rodning, C., Beckwith, L, & Howard, J. (1989). Prenatal exposure to drugs: Behavioral distortions reflecting CNS impairment Neurotoxicology, 10, 629-634.

Sakellaridis, N., Mangoura, D., & Veradakis, A. (1986). Effects of opiates on the growth of neuron enriched cultures from chick embryonic brain. International Journal of Developmental Neuroscience, 4, 293-302.

Schneider, J. & Chasnoff, I. (1992). Motor assessment of cocaine/polydrug exposed infants at age 4 months. Neurotoxicology and Teratology, 14, 9 –101.

Schneider, J. W. (1988). Motor assessment and parent education beyond the newborn period. In I. J. Chasnoff, (Ed.). Drugs, alcohol, pregnancy and parenting. Hingham, MA: Kluwer Academic Publishers.

Smialek, J., Monforte, J., Aronow, R., Spitz, W. (1977). Methadone deaths in children: A continuing problem. JAMA, 238, 2516-2517.

Smith, A., Hui, F., & Crofford, H. (1977). Inhibition of growth in young mice treated with dimethadone. European Journal of Pharmacology, 43, 307-309.

Society for Neuroscience. (1996). Drugs and the brain. Brain Concepts: Drugs. Washington, D.C.

Sostek, A., & Anders, T. (1977). Relationships among the Brazelton Neonatal Scale, Bayley Infant Scales, and early temperament. Child Development, 48, 320–323.

Stafford, J., Rosen, T., Zaider, M., & Merriam, J. (1994). Prenatal cocaine exposure and the development of the human eye. Opthalmology, 101 (2), 301-308.

Steiner, E., Villien, T., Hailberg, M., Rani, A. (1984). Amphetamine secretion in breast milk. European Journal of Clinical Pharmacology, 27, 123-124.

Strauss, A.A., Modanlow, H.D., & Bosu, S.K. (1981) Neonatal manifestations of maternal phencyclidine (PCP) abuse. Pediatrics, 68 (4), 550.

Streissguth, A., Barr, H., Martin, D., Herman, C., (1980). Effects of maternal alcohol, nicotine, and caffeine use during pregnancy on infant mental and motor development at eight months. Alcoholism: Clinical and Experimental Research, 4, 152–164.

Tabor, B., Smith-Wallace, T., Yonekura, M., (1990). Perinatal outcome associated with PCP versus cocaine use. American Journal of Drug & Alcohol Abuse, 16 (3 & 4), 337-348.

Tennes, M., Avitable, N., Blackard, C., Boyles, C., Hassoin, B., Holmes, L. & Kreye, M. (1985). In Pinkert, T. M. (ed.) Current Research on the Consequences of Maternal Drug Abuse. (NIDA Research Monograph 59, pp. 48-60). Washington, DC: Department of Health and Human Services.

Tornquist, E.M., (1986). From proposal to publication. Menlo Park, CA: Addison-Wesley.

Tronick, E. (1989). Emotions and emotional communication in infants. American Psychology, 44,(2), 112-119.

Tronick, E. & Brazelton, T. (1975). Clinical uses of the Brazelton Neonatal Behavioral Assessment Scale. In B. Z. Friedlander & L. Rosenblum (Eds.), Exceptional Infant. New York: Brunner.

Turner, J. H. (1991). The structure of sociological theory. (5th ed.). Belmont, CA: Wadsworth.

U.S. Advisory Board on Child Abuse and Neglect (1990). Child abuse and neglect: Critical first steps in response to a national emergency. (Prepublication edition, June 1990, p. 20, Washington, D.C.: U.S. Advisory Board Child Abuse & Neglect.

U.S. Department of Health and Human Services (1990). Healthy people 2000. Washington, D.C.: Public Health Services.

Vaughn, E., Taraldson, B., Crichton, L., & Egeland, B. (1980). Relationships between neonatal behavioral organization and infant behavior in the first year of life. Infant Behavior and Development, 3, 47–66.

Vega, W.A., Noble, A., Kolady, B., Porter, P., Hwang, J., & Bale, A. (1993). Profile of alcohol and drug use during pregnancy in California, 1992. Sac. CA.: California Dep't of Alcohol and Drug Programs.

Volpe, J. (1992). Mechanisms of disease. New England Journal of Medicine, 327 (6), 399-407.

von Bertalanffy, L. (1968). General systems theory: Foundations, development, application. New York: Braziller.

Walters, D. E., & Carr, L.A. (1986). Changes in brain catecholamine mechan-

isms following perinatal exposure to marijuana. Pharmacoloy Biochemical Behavior, 25, 763-768.

Widmayer, S., & Field, T. (1980). Effects of Brazelton demonstrations on early interactions of preterm infants and their teenage mothers. Infant Behavior and Development, 3, 78-79.

Wilson, G., (1989). Clinical studies of infants & children exposed prenatally to heroin. Annals of the New York Academy of Sciences, 562, 183-194.

Wilson, G., Desmond, M., Verniaud W. (1973). Early development of infants of heroin-addicted mothers. American Journal Disability Child, 126, 457.

Woods, J., Plessinger, M., & Clark, K. (1987). Effect of cocaine on uterine blood flow and fetal oxygenation. Journal of the American Medical Association, 257, 957-961.

Worobey, J. & Belsky, J. (1982). Employing the Brazelton scale to influence mothering: An experimental comparison of three strategies. Developmental Psychology, 18, 736–743.

Wright, M. & Leahey, M., (1984). Nurses and families: A guide to family assessment and intervention Philadelphia: F.A. Davis Co.

Zuckerman, B., & Bresnahan, K. (1991).Developmental and behavioral consequences of prenatal drug and alcohol exposure. Pediatric Clinics of North America, 38 (6), 1387-1405.

Zuckerman, B., Frank, D., Hingston, R., Amaro, H., Levenson, S., Kayne, H., Parker, S., Vinci, R., Aboagye, K., Fried, L., Cabral, H., Timperi, R & Bauchner, H. (1989). Effects of maternal marijuana and cocaine use on fetal growth. New England Journal of Medicine, 320 (12), 762-768.

Zuckerman, B., & Brown, E. (1993). Maternal substance abuse and infant development. In C. Zeanah, Jr. (Ed.) Handbook of infant mental health (pp. 143-158). New York: The Guilford Press.

Appendix A

Lewis Neurobehavioral Assessment Scale

Behaviors of Prenatally Drug Exposed (PDE) Infants 0-12 months

Infant name/No._____ Date of Birth_____ Age_____

Today's date_____ Birth weight_____ Present weight_____ Birth head circ_____

Present head circ._____ Birth length_____ Present length_____ 1 Male 2 Female (circle #)

CODE:_____-_____ Gestation_____ 1-SGA 2-IUGR Clinician_____

Ethnicity (circle #) 1-Caucasian 2-African-American 3-Hispanic 4-Asian 5-other_____

Circle #: 1-Own home 2-Foster home 3 Hospital # Foster home placements_____ # Maternal placements_____

Diagnosis: Drug_____

Diagnosis: Medical_____

Prenatal care: Yes_____ No_____ Limited_____ Apgars: _____ _____

Scoring legend: 1 = never; 2 = rarely; 3 = occasionally; 4 = frequently; 5 = almost always

I. High Reactivity
1. Irritibility _____
2. Frequency/rapidity of state changes _____
3. Difficult to comfort _____
4. Frequent startle response _____
5. Hyperactivity _____
6. High muscle tone _____
7. Tremors _____

Subscale total _____

Comments_____

II. Low Reactivity
8. Passivity _____
9. Dull alert state _____
10. Lethargy _____
11. Low muscle tone _____
12. Difficult feeding _____

Subscale total _____

Comments_____

III. Atypical Visual Functioning
13. Difficulty initiating eye contact _____
14. Difficulty maintaining eye contact _____
15. Gaze aversion _____
16. Difficulty tracking _____

Subscale total _____

Comments_____

IV. Atypical ANS Response
*17. High pitched cry _____
*18. Sweating _____
*19. Frequent yawning _____
*20. Hiccupping _____
*21. Sneezing _____

Subscale total _____

Comments_____

V. Compromised System Regulation
22. Jerky eye movement _____
*23. Unexplained fevers _____
24. Increased respirations _____
25. Nasal stuffiness _____

Subscale total _____

Comments_____

VI. Atypical Communication Patterns
(Administered at 4 months and older)
26. Undifferentiated cries _____
27. No social laugh _____
28. No social smile _____
29. Limited vocalizations _____
30. Limited vocal. to caregiver's response _____

Subscale total _____

Comments_____

VII. Atypical Play Response
(Administered at 6 months and older)
31. Limited imitating /objects/people _____
32. Limited functional use of toys _____
33. Limited initiation of play _____
34. Distractible _____
35. Easily frustrated _____

Subscale total _____

Comments_____

Total score _____

**Items caregiver can confirm.*

Appendix A, continued

Definitions of Selected Items for the LNAS

I. High Reactivity

2. Frequency or rapidity of state changes — frequent changes from one state to another; changes from one state to another with no transition period. (Circle one or both states.)
5. Hyperactivity — almost constant motor activity. (Indicate areas or area observed.)
6. High muscle tone — hypertonic or excessive tone; increased resistance to being in the flexed position.
7. Tremors — fine or gross, involuntary, rhythmic shaking of the tongue, chin, arms, legs, or whole body. (Indicate which area.)

II. Low Reactivity

8. Passivity — unresponsive to environment, either internal or external, i.e., may awaken and not cry out or fuss, have lack of cueing behaviors.
9. Dull alert state — lack of affective response to caregiver or objects, lack of brightening, flat facial expression, unavailable and/or dull-looking.
10. Lethargy — sleeps or rests most of the time, abnormal drowsiness.
11. Low muscle tone — Hypotonic, limited control of head, trunk, or extremities, or feels heavy and difficult to control or hold.

III. Atypical Visual Functioning

15. Gaze aversion — avoiding eye contact; turning head and/or eyes away.
16. Difficulty tracking — difficulty visually following object upwards or downwards without pauses or jerkiness.

IV. Atypical Communication Patterns

26. Undifferentiated cries — does not have a repertoire of cries for different needs; i.e., hungry, tired, pain, wet, attention.
29. Limited vocalizations — limited frequency and variety of sounds such as vowels, consonants, and combinations of sounds.
30. Limited vocalization to caregivers response: Difficulty attending to and vocalizing in response to verbal initiation.

VII. Atypical Play Response

31. Limited imitation with/objects/people: limited ability to imitate behavior presented, i.e., shakes head, bangs at surface (6-7 mo.); raspberry (8 mo.); push car (9 mo.); kiss, hug, pat-a-cake (10 mo.); waves bye-bye (10-12 mo.).
32. Limited functional use of toys: Limited use of objects for the purpose for which they were designed, i.e.: smiles into mirror, lifts cup by handle (4-6 mo.); ringing bell (7 mo.); rolling ball, pulling string to get object, push car (10-11 mo.); spoon to mouth, puts telephone to ear or brush to hair (12 mo.).
33. Limited initiation of play: Limited initiation and persistence with play behaviors, i.e.: reaches and grasps object (5-6 mo.); bounces to indicate infant wants activity continued; banging at midline, uses a variety of sounds to get attention (7 mo.); repeats same sound or behavior (8 mo.); uses touch to gain attention for play or helping getting object (9 mo.); poking with index finger (10 mo.); combining objects in a container, attempts to put on coat to go out, peek-a-boo (11-12 mo.).
34. Distractible: Difficulty screening out non-relevant or distracting environmental sensory stimuli, especially sounds or images in order to concentrate on a particular task.
35. Easily frustrated: observable agitation or disorganization by the inability to persist with objects or tasks. Young infant may have uncoordinated suck/swallow and begins to cry, falls apart and can't get back to task. Older infant may display crying, agitation by kinetic movements due to difficulty with coordination, motor control and decreased ability to attend to meaningful tasks.

Appendix B

University of California, San Francisco
School of Nursing
Study Consent Form SR
Assessing Risk of Infants
Form I

A. Purpose and Background
Keeta Lewis, a doctoral student at USCF, School Of Nursing, and Dr. Sandra Weiss, her advisor, are doing a study about testing the behavior and development of babies. My baby and I have been asked to participate in the research study.

B. Procedures
If I agree to be in the study, the following will happen:
1. Information about my baby's birth history and health will be gathered from medical records.
2. My baby will be tested to see how my baby responds to a caregiver's voice, moves about, reacts to people in his or her environment.
These activities will take about 45 minutes at a location which is best for me — in the birth hospital, in my home, or at a local clinic.
3. My baby and I may be asked to repeat one of the tests a second time two weeks later. This will take about 45 minutes.

C. Risks and Discomforts
Our participation in the study will in no way change the care we may be getting at a clinic, hospital, or physician's office. The interview may seem boring. I may decline to answer any questions or stop my involvement in the study at any time.

Confidentiality: Participation in research may involve a loss of privacy. Study records will be kept as confidential as is possible. Only identification numbers will appear on all written records and these will be kept in a locked file. If the results of this study are published in professional journals, our names will not be used. Only members of the research team will see the data. At the end of the research, all identifying records and information will be destroyed.

D. Benefits
There will be no direct benefit to my baby or me from participating in the study. I may enjoy answering the questions about my baby. Our participation will help nurses and other professionals understand how they can help babies as they develop.

E. Costs
There will be no cost to me as a result of taking part in this study.

Appendix B, continued

F. Payment
I will not receive any money for participating in this study.

G. Questions
I have talked to_____about this study and have had my questions answered. If I have any further questions, I may contact Keeta Lewis at 707-255-4626. I can also contact the Committee on Human Research, which is concerned with protection of volunteers in research projects. I may reach the Committee Office between 8:00 a.m. and 5:00 p.m., Monday to Friday, by calling 415-476-1814 or by writing to the Committee on Human Research, Suite II, Laurel Heights Campus, Box 0616, University of California, San Francisco, Ca. 94143-0616.

Consent
Participation in this research is voluntary. I am free to decline to be in this study or to withdraw from it at any time, with no effects on my baby's health care or my own. I will be given a copy of this consent form to keep.

_____
Date Subject's Caregiver Signature

_____
Date Person Obtaining Consent

Appendix B, continued

University of California, San Francisco
School of Nursing
Study Consent Form SR
Assessing Risk of Infants
Form II

A. Purpose and Background
Keeta Lewis, a doctoral student at USCF, School Of Nursing, and Dr. Sandra Weiss, her advisor, are doing a study about testing the behavior and development of babies. My baby and I have been asked to participate in the research study.

B. Procedures
If I agree to be in the study, the following will happen:
1. Information about my baby's birth history and health will be gathered from medical records or assessments.
2. My baby will be tested to see how my baby does things such as: responds to a caregiver's voice, moves about, communicates with people, and plays with toys and objects.
3. I will be asked, through an interview, about how my baby communicates with people, and how my baby is growing and adapting in our home.
These activities will take about 1 1/2 to 2 hours at a location which is best for me — either in my home or at a local clinic.
4. My baby and I may be asked to repeat one of the tests a second time two weeks later. This will take about 45 minutes.

C. Risks and Discomforts
Our participation in the study will in no way change the care we may be getting at a clinic, hospital, or physician's office. The interview may seem boring. I may decline to answer any questions or stop my involvement in the study at any time.

Confidentiality: Participation in research may involve a loss of privacy. Study records will be kept as confidential as is possible. Only identification numbers will appear on all written records and these will be kept in a locked file. If the results of this study are published in professional journals, our names will not be used. Only members of the research team will see the data. At the end of the research, all identifying records and information will be destroyed.

D. Benefits
There will be no direct benefit to my baby or me from participating in the study. I may enjoy answering the questions about my baby. Our participation will help nurses and other professionals understand how they can help babies as they develop.

Appendix B, continued

E. Costs
There will be no cost to me as a result of taking part in this study.

F. Payment
I will not receive any money for participating in this study.

G. Questions
I have talked to_____about this study and have had my questions answered. If I have any further questions, I may contact Keeta Lewis at 707-255-4626. I can also contact the Committee on Human Research, which is concerned with protection of volunteers in research projects. I may reach the Committee Office between 8:00 a.m. and 5:00 p.m., Monday to Friday, by calling 415-476-1814 or by writing to the Committee on Human Research, Suite ll, Laurel Heights Campus, Box 0616, University of California, San Francisco, Ca. 94143-0616.

Consent
Participation in this research is voluntary. I am free to decline to be in this study or to withdraw from it at any time, with no effects on my baby's health care or my own. I will be given a copy of this consent form to keep.

_____

Date Subject's Caregiver Signature

_____

Date Person Obtaining Consent

Appendix C

USUAL FEEDING TIME (CIRCLE)
YES NO

PERSON OBSERVED IN INTERACTION (CIRCLE)
MOTHER FATHER OTHER

MAJOR CAREGIVER (CIRCLE)
YES NO

TYPE OF FEEDING (CIRCLE)
BREAST BOTTLE SOLID

LENGTH OF FEEDING (CIRCLE)
10 OR LESS 10-20 20-30 30 OR MORE

SETTING (CIRCLE)
HOME CLINIC OTHER

UNIVERSITY OF WASHINGTON
SCHOOL OF NURSING
NURSING CHILD ASSESSMENT TRAINING

FEEDING SCALE
(BIRTH TO ONE YEAR)

RECORDER'S NAME_____

DATE_____

CHILD'S FIRST NAME_____

CHILD'S AGE (IN MONTHS)_____

CHILD'S SEX_____

MOTHER'S AGE_____

PARITY_____

MOTHER'S EDUCATION (CIRCLE)
6 YRS. OR LESS 7-8-9-10-11-12-13-14-15-16-17-18-19-20 +

MARITAL STATUS (CIRCLE)
MARRIED NOT MARRIED

MOTHER'S AGE (AT BIRTH OF CHILD)_____

	YES	NO
I. SENSITIVITY TO CUES		
1. PARENT POSITIONS CHILD SO THAT CHILD IS SAFE BUT CAN MOVE HIS ARMS.		
2. PARENT POSITIONS CHILD SO THAT THE CHILD'S HEAD IS HIGHER THAN HIPS.		
3. PARENT POSITIONS CHILD SO THAT TRUNK-TO-TRUNK CONTACT IS MAINTAINED DURING MORE THAN HALF OF THE BREAST OR BOTTLE FEEDING (50%+).		
4. PARENT POSITIONS CHILD SO THAT EYE-TO-EYE CONTACT IS POSSIBLE.		
5. PARENT'S FACE IS AT LEAST 7-9 INCHES OR MORE FROM THE CHILD'S FACE DURING FEEDING EXCEPT WHEN KISSING, CARESSING, HUGGING OR BURPING THE CHILD.		
6. PARENT SMILES, VERBALIZES, OR MAKES EYE CONTACT WITH CHILD WHEN CHILD IS IN OPEN-FACE-GAZE POSITION.		
7. PARENT COMMENTS VERBALLY ON CHILD'S HUNGER CUES PRIOR TO FEEDING.		
8. PARENT COMMENTS VERBALLY ON CHILD'S SATIATION CUES BEFORE TERMINATING FEEDING.		
9. PARENT VARIES THE INTENSITY OF VERBAL STIMULATION DURING FEEDING.		
10. PARENT VARIES INTENSITY OF ROCKING OR MOVING THE CHILD DURING THE FEEDING.		
11. PARENT VARIES THE INTENSITY OF TOUCH DURING THE FEEDING.		
12. PARENT ALLOWS PAUSES IN FEEDING WHEN THE CHILD INDICATES BY CRY FACE, HALT HAND, BACK ARCHING, PULLING AWAY, PUSHING FOOD AWAY, TRAY POUNDING, TURNING HEAD, SHAKING HEAD NO OR SAYING "NO" OR FALLING ASLEEP OR WHEN CHILD IS IN PAUSE PHASE OF THE BURST-PAUSE SEQUENCE OF SUCKING (75% OF THE TIME).		
13. PARENT SLOWS PACE OF FEEDING OR PAUSES WHEN CHILD AVERTS GAZE, PLACES HAND-TO-EAR, HAND-TO-MOUTH, HAND-BEHIND-HEAD, HAND-BACK-OF-NECK, HANDS OVER STOMACH, YAWNS, RUBS EYE OR DISPLAYS FIDGET MOVEMENT (75% OF THE TIME).		
14. PARENT TERMINATES THE FEEDING WHEN THE CHILD TURNS HEAD, FALLS ASLEEP, COMPRESSES LIPS, PUSHES FOOD AWAY, SHAKES HEAD "NO" OR SAYS "NO," ONCE OR MORE OR AFTER OTHER METHODS (REPOSITIONING, BURPING, OR WAITING) HAVE PROVED UNSUCCESSFUL.		
*15. PARENT DOES NOT INTERRUPT CHILD'S SUCKING OR CHEWING BY REMOVING THE NIPPLE, JIGGLING THE NIPPLE, OR OFFERING THE CHILD MORE OR OTHER KINDS OF FOOD WHILE CHILD IS EATING.		
*16. PARENT DOES NOT OFFER FOOD WHEN THE CHILD LOOKS AWAY, LOOKS BOWL, TURNS AWAY OR TURNS AROUND.		
SUBSCALE TOTAL (NO. OF YES ANSWERS)		
II. RESPONSE TO DISTRESS (INDICATE IN BOX WHETHER OCCURRED OR NOT IF NO DISTRESS, MARK EACH BOX "YES".) IF CHILD SHOWS DISTRESS DURING THE FEEDING DOES THE PARENT:		
17. STOP OR START FEEDING IN RESPONSE TO THE CHILD'S DISTRESS.		
18. CHANGE THE CHILD'S POSITION IN RESPONSE TO CHILD'S DISTRESS.		
19. MAKE POSITIVE OR SYMPATHETIC VERBALIZATION IN RESPONSE TO CHILD'S DISTRESS.		
20. CHANGES VOICE VOLUME TO SOFTER OR HIGHER PITCH IN RESPONSE TO CHILD'S DISTRESS.		
21. MAKES SOOTHING NON-VERBAL EFFORTS IN RESPONSE TO CHILD'S DISTRESS.		
22. DIVERTS CHILD'S ATTENTION BY PLAYING GAMES, INTRODUCING A TOY, OR MAKING FACES IN RESPONSE TO CHILD'S DISTRESS.		
23. PARENT DOES NOT MAKE NEGATIVE VERBAL RESPONSE IN RESPONSE TO CHILD'S DISTRESS.		
24. PARENT DOES NOT MAKE NEGATIVE COMMENTS TO HOME VISITOR ABOUT CHILD IN RESPONSE TO CHILD'S DISTRESS.		
25. PARENT DOES NOT YELL AT THE CHILD IN RESPONSE TO HIS DISTRESS.		
26. PARENT DOES NOT USE ABRUPT MOVEMENTS OR ROUGH HANDLING IN RESPONSE TO CHILD'S DISTRESS.		
27. PARENT DOES NOT SLAP, HIT, OR SPANK CHILD IN RESPONSE TO DISTRESS.		
SUBSCALE TOTAL (NO. OF YES ANSWERS)		
III. SOCIAL-EMOTIONAL GROWTH FOSTERING		
28. PARENT PAYS MORE ATTENTION TO CHILD DURING FEEDING THAN TO OTHER PEOPLE OR THINGS IN ENVIRONMENT.		
29. PARENT IS IN EN FACE POSITION FOR MORE THAN HALF OF THE FEEDING (50%+).		
30. PARENT SUCCEEDS IN MAKING EYE CONTACT WITH CHILD ONCE DURING FEEDING.		
31. PARENT'S FACIAL EXPRESSION CHANGES AT LEAST TWICE DURING FEEDING.		
32. PARENT ENGAGES IN SOCIAL FORMS OF INTERACTION (PLAYS GAMES WITH CHILD) AT LEAST ONCE DURING THE FEEDING.		
33. PARENT USES POSITIVE STATEMENTS IN TALKING TO CHILD DURING THE FEEDING.		
34. PARENT PRAISES CHILD OR SOME QUALITY OF THE CHILD'S BEHAVIOR DURING THE FEEDING.		
35. PARENT HUMS, CROONS, SINGS OR CHANGES THE PITCH OF HISPER VOICE DURING THE FEEDING.		
36. PARENT LAUGHS OR SMILES DURING THE FEEDING.		
37. PARENT USES GENTLE FORMS OF TOUCHING DURING THE FEEDING.		
38. PARENT SMILES, VERBALIZES OR TOUCHES CHILD WITHIN 5 SECONDS OF CHILD SMILING OR VOCALIZING AT PARENT.		
39 PARENT DOES NOT COMPRESS LIPS, GRIMACE, OR FROWN WHEN MAKING EYE CONTACT WITH CHILD.		
40. PARENT DOES NOT SLAP, HIT, SHAKE, OR GRAB CHILD OR CHILD'S EXTREMITIES DURING THE FEEDING.		
41. PARENT DOES NOT MAKE NEGATIVE OR UNCOMPLIMENTARY REMARKS TO THE CHILD OR HOME VISITOR ABOUT THE CHILD OR CHILD'S BEHAVIOR		
SUBSCALE TOTAL (NO. OF YES ANSWERS)		
IV. COGNITIVE GROWTH FOSTERING		
42. PARENT PROVIDES CHILD WITH OBJECTS, FINGER FOODS, TOYS, AND/OR UTENSILS		
43. PARENT ENCOURAGES AND/OR ALLOWS THE CHILD TO EXPLORE THE BREAST, BOTTLE, FOOD, CUP, BOWL OR THE PARENT DURING FEEDING.		
44. PARENT TALKS TO THE CHILD USING TWO WORDS AT LEAST THREE TIMES DURING THE FEEDING.		
45. PARENT VERBALLY DESCRIBES SOME ASPECT OF THE FOOD OR FEEDING SITUATION TO CHILD DURING FEEDING.		
46. PARENT TALKS TO CHILD ABOUT THINGS OTHER THAN FOOD, EATING, OR THINGS RELATED TO THE FEEDING.		
47. PARENT USES STATEMENTS THAT DESCRIBE, ASK QUESTIONS OR EXPLAIN CONSEQUENCES OF BEHAVIOR MORE THAN COMMANDS IN TALKING TO THE CHILD.		
48. PARENT VERBALIZES TO CHILD WITHIN FIVE SECONDS AFTER CHILD HAS VOCALIZED.		
49. PARENT VERBALIZES TO CHILD WITHIN FIVE SECONDS AFTER CHILD'S MOVEMENT OF ARMS, LEGS, HANDS, HEAD, TRUNK.		
50. PARENT DOES NOT TALK BABY TALK.		

Appendix C, Continued

	YES	NO
I. CLARITY OF CUES		
S1. CHILD SIGNALS READINESS TO EAT.		
S2. CHILD DISPLAYS A BUILD-UP OF TENSION AT THE BEGINNING OF FEEDING.		
S3. CHILD DEMONSTRATES A DECREASE IN TENSION WITHIN A FEW MINUTES AFTER FEEDING HAS BEGUN.		
S4. CHILD HAS PERIODS OF ALERTNESS DURING THE FEEDING.		
S5. CHILD DISPLAYS AT LEAST TWO DIFFERENT EMOTIONS DURING THE FEEDING.		
S6. CHILD HAS PERIODS OF ACTIVITY AND INACTIVITY DURING THE FEEDING.		
S7. CHILD'S MOVEMENTS ARE SMOOTH AND COORDINATED DURING THE FEEDING.		
S8. CHILD'S ARM AND LEG MOVEMENTS ARE GENERALLY DIRECTED TOWARD PARENT DURING FEEDING (NOT DIFFUSE).		
S9. CHILD MAKES CONTACT WITH PARENT'S FACE OR EYES AT LEAST ONCE DURING FEEDING.		
S10. CHILD VOCALIZES DURING FEEDING.		
S11. CHILD SMILES OR LAUGHS DURING FEEDING.		
S12. CHILD AVERTS GAZE, LOOKS DOWN OR TURNS AWAY DURING FEEDING.		
S13. CHILD ACTIVELY REJECTS FOOD OFFERED.		
S14. CHILD DEMONSTRATES SATISFACTION AT END OF FEEDING THROUGH SLEEP, FACIAL EXPRESSIONS, DECREASED MUSCLE TONE, ARMS EXTENDED ALONG SIDE, VOCALIZATIONS OR CHANGE IN ACTIVITY LEVEL OR MOOD.		
S15. CHILD DOES NOT HAVE MORE THAN TWO RAPID STATE CHANGES DURING FEEDING.		
SUBSCALE TOTAL (NO. OF YES ANSWERS)		
RESPONSIVENESS TO PARENT		
S16. CHILD RESPONDS TO FEEDING ATTEMPTS BY PARENT DURING FEEDING.		
S17. CHILD RESPONDS TO GAMES, SOCIAL PLAY OR SOCIAL CUES OF PARENT DURING FEEDING.		
S18. CHILD LOOKS IN THE DIRECTION OF THE PARENT'S FACE AFTER PARENT HAS ATTEMPTED TO ALERT THE CHILD VERBALLY OR NON-VERBALLY DURING FEEDING.		
S19. CHILD VOCALIZES TO PARENT DURING FEEDING.		
S20. CHILD VOCALIZES OR SMILES WITHIN 5 SECONDS OF PARENT'S VOCALIZATION.		
S21. CHILD SMILES AT PARENT DURING FEEDING.		
S22. CHILD EXPLORES PARENT OR REACHES OUT TO TOUCH PARENT DURING FEEDING.		
S23. CHILD SHOWS A CHANGE IN LEVEL OF MOTOR ACTIVITY WITHIN 6 SECONDS OF BEING HANDLED OR REPOSITIONED BY PARENT.		
S24. CHILD SHOWS POTENT DISENGAGEMENT CUES DURING LAST HALF OF FEEDING.		
S25. CHILD SHOWS POTENT DISENGAGEMENT CUES WITHIN 5 SECONDS AFTER PARENT MOVES CLOSER THAN 7 TO 8 INCHES FROM CHILD'S FACE.		
S26. CHILD DOES NOT TURN AWAY OR AVERT GAZE FROM PARENT DURING FIRST HALF OF FEEDING.		
SUBSCALE TOTAL (NO. OF YES ANSWERS)		

ENTER TOTALS FOR EACH CATEGORY:	
SENSITIVITY TO CUES	
RESPONSE TO DISTRESS	
SOCIAL-EMOTIONAL GROWTH FOSTERING	
COGNITIVE GROWTH FOSTERING	
CLARITY OF CUES	
RESPONSIVENESS TO PARENT	
TOTAL (NO. OF YES ANSWERS)	

HOME VISIT QUESTIONS:

1. WOULD YOU SAY THIS WAS A TYPICAL FEEDING?
   A. YES B. NO
   IF NO, WHY NOT?

2. WERE YOU UNCOMFORTABLE DURING ANY PART OF THE FEEDING DUE TO MY PRESENCE?
   A. YES B. NO
   IF YES, WHY?

3. DO YOU HAVE ANY CONCERNS ABOUT THE FEEDING OR YOUR CHILD'S EATING?
   A. YES NO
   IF YES, SPECIFY.

4. OBSERVER'S COMMENTS:

NURSING CHILD ASSESSMENT SATELLITE TRAINING
UNIVERSITY OF WASHINGTON
SCHOOL OF NURSING, WJ-10
SEATTLE, WASHINGTON 98195
USA
(206) 543-8528

Appendix C, Continued

MATERNAL PRENATAL COMPLICATIONS SCALE

ID Code _____ Rater _____ Date _____

Review the mother's chart material to obtain the following information about the mother's prenatal complications. Please check those items present in the mother's prenatal history, specifying additional information when indicated.

1. ___ Baby's gestational age: less than 37 wk
2. ___ Baby's birth weight: less than 2500 g
3. ___ Mother's marital status: other than married
4. ___ Maternal age: under 18 or over 30
5. ___ Previous abortions: 1 or more
6. ___ Previous stillbirths: 1 or more
7. ___ Prolonged unwanted sterility
8. ___ Time since last pregnancy: less than 12 mo
9. ___ Parity: greater than 7
10. ___ Pelvis disproportion
11. ___ Blood group incompatability
12. ___ Maternal chronic disease

    Specify _____

13. ___ Maternal drug use

    ___ Alcohol use

    ___ Other drug use

    Specify _____

    ___ Toxicology screen on blood/urine (circle type)

    Results _____

14. ___ No prenatal care during first half of pregnancy
15. ___ Bleeding during pregnancy
16. ___ Infections or acute medical problems during pregnancy

    Specify _____

Appendix C, Continued

17. ___ Medications given during pregnancy

    Specify _____

18. ___ Blood pressure during pregnancy: greater than 140/90

19. ___ Albuminuria

20. ___ Hyperemesis

21. ___ Hemoglobin at delivery: less than 12 gm

22. ___ Multiple birth

23. ___ Membranes ruptured prior to delivery

24. ___ Delivery not spontaneous. Example: augmented with Pitocin

25. ___ Forceps used; vacuum extraction (circle one)

26. ___ Duration, first stage: less than 3, greater than 20 hr

27. ___ Duration, second stage: less than 10, greater than 120 min

28. ___ Onset of labor induced, not spontaneous

29. ___ Intrapartum drugs used (including analgesics)

30. ___ Amniotic fluid not clear

31. ___ Fetal presentation not vertex

32. ___ Intrapartum heart rate: less than 100, greater than 160 per min

33. ___ Nuchal or knotted cord

34. ___ Cord prolapse

35. ___ Placental infarction

36. ___ Placenta previa or abrupta

37. ___ Onset of newborn respiration not within 6 min

38. ___ Resuscitation of infant required

39. ___ Apgar score, 1 min: less than 7

40. ___ Apgar score, 5 min: less than 7

Appendix C, Continued

INFANT POSTNATAL COMPLICATIONS SCALE

ID Code _____ Rater _____ Date _____

Review the chart material to obtain the following information about the baby's postnatal complications. Please check those items present in the baby's postnatal course during the first month of life.

1. ___ Respiratory distress
   - ___ Asphyxia
   - ___ Required resuscitation
   - ___ Received supplemental oxygen
2. ___ Ventilatory assistance
3. ___ Infection
4. ___ Noninfectious illness (anomaly, hemorrhage) or injury

   Specify _____
5. ___ Metabolic abnormality
6. ___ Convulsion
7. ___ Hyperbilirubinemia
8. ___ Temperature disturbance
9. ___ No feeding within 48 hours of birth
10. ___ Surgery

Appendix C, Continued

Eileen M. Mullen

RECORD FORM

Child's Name _____

ID _____ Phone Number _____

Nickname _____ ❑ Boy ❑ Girl

Address _____

_____

Child's Primary Language _____

Mother's Name _____

Father's Name _____

Examiner _____

School _____

No. Weeks Gestation (G.A.) _____ Birth Weight _____

Apgars 1 min. _____ 5 min. _____

Hospital _____

	Year	Month	Day
Testing Date			
Birth Date			
Chronological Age			
Adjusted Age (Children under two years: See Chapter 3 in manual.)			

Does the child have a known uncorrected vision problem? ❑ No ❑ Yes

Does the child have a known uncorrected hearing problem? ❑ No ❑ Yes

Personal or physical characteristics that may affect the child's test results

_____

_____

Is the child on any medication? ❑ No ❑ Yes (please specify)

_____

Referred by _____

Reason for Referral _____

Additional Information/Comments _____

_____

_____

_____

_____

AGS®

Appendix C, Continued

Scale 2. Visual Reception

Item — Score

1. Fixates on and tracks triangle (S) 2 1 0
   ① fixates ② tracks
2. Tracks schematic face 90 degrees (S) 1 0
3. Tracks moving bull's-eye 180 degrees (PPr) 1 0
4. Localizes alternating red ball and schematic face (PPr) .. 1 0
5. Stares at own hand (S) 1 0
6. Localizes bull's-eye near and far (SSit) 1 0
7. Looks for dropped spoon (A/V) (SSit) 1 0
8. Pulls cord to obtain disc (SSit) 1 0
9. Looks for ring hidden under washcloth (Sit) 2 1 0
   ① partially hidden ② fully hidden
10. Turns cup right-side up 1 0
11. Makes object association 1 0 (1)
    ___ brush ___ spoon ___ cup ___ ball
12. Looks for car under two washcloths 1 0
13. Shows interest in book as hinge 1 0
14. Attends to picture (A/V) 1 0
15. Looks for toy covered, then displaced 1 0
16. Discriminates forms on formboard 4 3 2 1 0
    ① ● ② ●■ ③ ●■▲ ④ ●■▲✦

17A. Matches objects with naming (A/V) (19 months or younger)

OR

17B. Matches objects without naming (20 months or older)
___ shoes ___ cars ___ keys ___ sticks
① one object with naming 3 2 1 0
② two objects without naming
③ three objects without naming

18. Nests nesting cups 2 1 0
    ① nests three cups
    ② nests four cups
19. Sorts spoons and blocks by category ..... 1 0 (2 each)
20. Matches by shape ..... 1 0 (2 each)
    ___ circles ___ squares ___ triangles
21. Matches pictures 1 0 (2)
    ___ shoe ___ cup ___ plane
22. Matches by size, color ..... 1 0 (2 each)
    ___ large red circles ___ small red circles
    ___ large yellow circles ___ small yellow circles
23. Memory for one picture 1 0
24. Spatial details I 1 0
25. Spatial details II 2 1 0 (4) (3)
    flower ___ 1 ___ 2 ___ 3 ___ 4
26. Memory for objects 1 0 (2)
    ___ key ___ ball ___ car
27. Discriminates spatial position 1 0 (4)
    form ___ 1 ___ 2 ___ 3 ___ 4
28. Matches letters 1 0 (4)
    ___ L ___ C ___ N ___ B ___ H ___ P
29. Discriminates left/right 4 3 2 1 0 (4) (3) (2) (1)
    ___ bunny ___ hammer ___ child ___ wagon
30. Matches letters, words 4 3 2 1 0 (6) (5) (4) (2)
    ___ B ___ t ___ d ___ n ___ rt
    ___ be ___ bat ___ coat ___ will
31. Memory for three pictures 1 0
    ___ key ___ book ___ chair
32. Spatial details III 2 1 0 (4) (3)
    dog ___ 1 ___ 2 ___ 3 ___ 4 ___ 5
33. Memory for form 2 1 0 (3) (2)
    form ___ 1 ___ 2 ___ 3 ___ 4

Visual Reception Raw Score ☐

Scale 3. Fine Motor

Item — Score

1. Arms flexed/hands fisted (S) 1 0
2. Holds ring reflexively (S) 1 0
3. Brings fist to mouth (P) 1 0
4. Bilateral orientation in midline (S) 1 0
5. Grasp reflex integrated (S) 1 0
6. Grasps peg (ulnar palmar) (PPr or SSit) 1 0
7. Reaches for and grasps block (radial palmar grasp) (SSit) 1 0
8. Transfers, bangs, drops (SSit) 1 0 (2)
9. Refined grasp/thumb opposition (Sit) 1 0
10. Uses pincer grasp (Sit) 2 1 0
    ① partial pincer ② refined pincer
11. Bangs in midline, horizontal movement (Sit) 1 0
12. Takes blocks out, puts blocks in 3 2 1 0
    Task 1: 1 block ① in or ① out
    Task 2: 4 blocks ② in or ② out
    Task 3: 7 to 8 blocks ③ in
13. Uses two hands together 1 0
14. Turns pages in a book 2 1 0
    ① several at a time ② one at a time
15. Imitates crayon lines ..... 3 2 1 0 (item tested)
    Task 1: ① any direction
    ② vertical line
    Task 2: ① horizontal line
16. Puts pennies in slot, horizontal and vertical ..... 2 1 0 (item tested)
    Task 1: ① 3 pennies/horizontal
    Task 2: ① 3 pennies/vertical
17. Stacks blocks vertically 3 2 1 0
    ① 3-5 blocks ② 6-8 blocks
    ③ 9 or more blocks
18. Imitates four-block train 2 1 0
    ① train ② train with driver
19. Unscrews, screws nut and bolt 1 0
20. Strings beads 1 0 (3)
21. Imitates four-block tower 1 0
22. Copies circle, circle and line ..... 2 1 0 (item tested)
    Task 1: ① circle
    Task 2: ① circle and line
23. Draws in path 2 1 0
    ___ Example ___ Figure 1 ___ Figure 2 ___ Figure 3 (3) (2)
24. Cuts with scissors 2 1 0
    ① 1-inch cut ② 2-inch cut
25. Folds paper three times 1 0
26. Imitates drawings ..... 3 2 1 0 (item tested)
    Task 1: ① circle in circle
    Task 2: ① square
    Task 3: ① left diagonal
27. Touches fingers I 1 0
28. Touches fingers II 1 0
29. Folds paper twice to form square 1 0
30. Copies shapes and letters ..... 5 4 3 2 1 0 (item tested)
    Task 1: ① cross
    Task 2: ① square
    Task 3: ① LED
    Task 4: ① triangle
    Task 5: ① X

Fine Motor Raw Score ☐

Appendix C, Continued

Scale 4. Receptive Language

Item — Score

1. Reacts reflexively to loud noise (S) 1 0
2. Alerts to sound (S) 1 0
3. Responds to voice and face by smiling (A/V) (S) 1 0
4. Coordinates listening and turning (PPr) 1 0
5. Responds to voice and face by vocalizing (A/V) (PPr or SSat) 1 0
6. Coordinates listening and looking (SSit) 1 0
7. Enjoys self/mirror interaction (A/V) (SSit) 1 0
8. Attends to words and movement (A/V) (SSit or Sit) 1 0
9. Recognizes familiar names, words 1 0
10. Recognizes own name 1 0
11. Understands inhibitory words 1 0
12. Understands simple verbal input 1 0
13. Understands gesture and commands (A/V) 1 0
14. Identifies objects (A/V) 1 0
15. Gives toy on verbal request 1 0
16. Comprehends questions I 1 0 (1)
    ___ chair ___ door
17. Follows directions 1 0 (2)
    ___ block ___ car
18. Recognizes body parts (A/V) 3 2 1 0
    ___ eyes ___ nose ___ mouth
    ___ ears ___ hands ___ feet ___ hair
    ① 1 to 3 body parts
    ② 4 or 5 body parts
    ③ 6 or 7 body parts
19. Comprehends questions II (A/V) 1 0 (1)
    ___ cat ___ cup ___ car
20. Follows related commands 1 0 (1)
    ___ ball ___ box
21. Identifies pictures (A/V) 1 0 (2)
    ___ car ___ ball ___ shoe ___ doll
22. Auditory spatial awareness 4 3 2 1 0
    ___in ___under ___behind ___in front of ___beside
    ① 1 position ② 2 positions
    ③ 3 positions ④ 4 or 5 positions
23. Comprehends action words (A/V) 2 1 0 (2-3) (1)
    ___eating ___ sleeping ___washing
24. Identifies object function (A/V) 1 0 (3)
    ___ car ___ scissors ___ spoon ___ chair
25. Follows two unrelated commands 1 0 (1)
    ___ set 1 ___ set 2
26. Size concepts (A/V) 1 0 (3)
    ___ trial 1 ___ trial 2 ___ trial 3 ___ trial 4
27. Identifies colors (A/V) 1 0 (4)
    ___ red ___ green ___ yellow ___ blue
    ___ orange ___ black ___ brown ___ purple
28. Length concepts (A/V) 1 0 (3)
    ___ trial 1 ___ trial 2 ___ trial 3 ___ trial 4
29. Comparative concepts (A/V) 4 3 2 1 0 (6) (5) (4) (3)
    ___ same ___ not same ___ most
    ___ least ___ first ___ last ___ second
    ___ middle ___ left of the tree ___ nearest
30. General knowledge (see flap) 5 4 3 2 1 0 (10) (9) (8) (7) (6)
31. Follows three unrelated commands 1 0
32. Has concept of six, eight ..... 2 1 0 (each task)
    Task 1 ① 6 blocks
    Task 2 ① 8 blocks
33. Identifies letters (A/V) 2 1 0 (14) (12)
    ___T ___C ___L ___O ___D ___N ___S
    ___R ___B ___G ___M ___H ___X ___P

Receptive Language Raw Score ____

Scale 5. Expressive Language

Item — Score

1. Sucking, swallowing, chewing movements 1 0
2. Vocalizes (S) 1 0
3. Smiles and makes happy sounds (S) 1 0
4. Coos, chuckles, or laughs 1 0 (2)
5. Makes vocalizations (such as *eh, eh, m*) 1 0
6. Plays with sounds (such as *o, u, eh-goo*) 1 0
7. Voluntary babbling (such as "*ba, ba, ba*") 1 0
8. Produces three consonant sounds (such as *p, d, k, g, m*) 1 0
9. Vocalizes two-syllable sounds (such as "*dada*" or "*baba*") 1 0
10. Plays gesture/language game 1 0
11. Says first words 3 2 1 0
    ① says 1 word
    ② says 2 to 7 words
    ③ says 8 words
12. Jabbers with inflection 1 0
13. Combines jargon/gestures 1 0
14. Combines words/gestures 1 0
15. Names objects 3 2 1 0
    ___ ball ___ book ___ car
    ___ cup ___ key ___ knife
    ① names 1 - 3 objects
    ② names 4 - 5 objects
    ③ names 6 objects
16. Labels picture 1 0 (1)
    ___ ball ___ dog ___ baby
17. Uses two-word phrase 1 0
18. Picture vocabulary (see flap) 5 4 3 2 1 0
    ① names 5-10 pictures
    ② names 11-14 pictures
    ③ names 15-16 pictures
    ④ names 17 pictures
    ⑤ names 18 pictures
19. Uses pronouns 1 0
20. Counts to two, three, twelve 3 2 1 0
    ① counts to 2
    ② counts to 3
    ③ counts to 12
21. Repeats two numbers 1 0 (1)
    ___ 6 - 2
    ___ 4 - 7
22. Uses three- to four-word sentences 1 0
23. Answers questions (see flap) 2 1 0
    ① answers two questions
    ② answers three questions
24. Verbal analogies (see flap) 5 4 3 2 1 0 (8) (7) (6) (5) (4)
25. Repeats sentences I 1 0 (2)
    ___ sentence 1 ___ sentence 2
26. Oral vocabulary (see flap) 4 3 2 1 0 (9) (8) (7) (6)
27. Practical reasoning (see flap) 4 3 2 1 0 (7) (6) (5) (4)
28. Repeats sentences II 2 1 0 (2) (1)
    ___ sentence 1 ___ sentence 2 ___ sentence 3

Expressive Language Raw Score ____

Appendix C, Continued

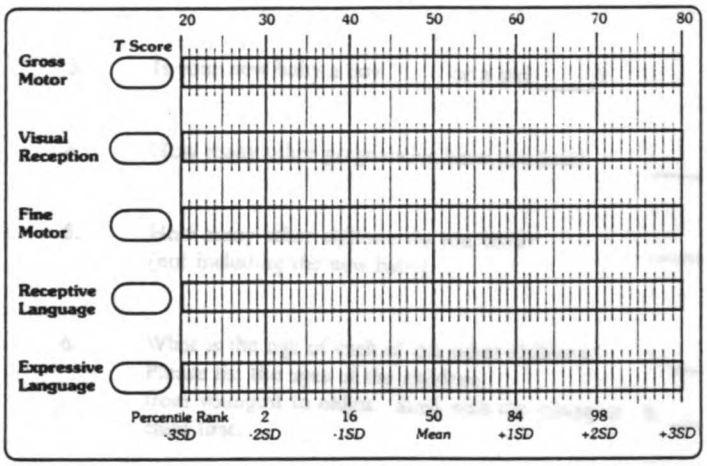
Score Summary

Scale	Raw Score	*T* Score M=50, SD=10 (Table C.1)	Band of Error __% Confidence (Table C.1)	Percentile Rank (Table C.2)	Descriptive Category (Table C.2)	Age Equivalent (Transfer from chart)
Gross Motor			±			
Visual Reception			±			
Fine Motor			±			
Receptive Language			±			
Expressive Language			±			

Cognitive *T* Score Sum

Early Learning Composite (Optional)	Standard Score M=100, SD=15 (Table C.3)	Band of Error __% Confidence (Table C.3)	Percentile Rank (Table C.3)	Descriptive Category (Table C.3)
		±		

Scale *T* Score Profile



Observations _____

For additional forms, call or write AGS, 4201 Woodland Road, Circle Pines, MN 55014-1796; toll-free 1-800-328-2560. In Canada, 1-800-263-3558. Ask for item #11152 (25 per package).

Age Equivalents

Stage	Age Equivalent	Gross Motor	Visual Reception	Fine Motor	Receptive Language	Expressive Language
	70	—	—	—	—	49-50
	69	—	50	—	48	—
	68	—	—	49	—	—
	67	—	—	—	—	48
	66	—	49	—	—	—
	65	—	—	48	47	—
	64	—	—	—	—	—
	63	—	—	—	—	47
	62	—	—	47	46	—
	61	—	—	—	—	—
	60	—	48	—	—	46
	59	—	—	46	45	—
	58	—	—	—	—	45
	57	—	47	45	44	—
	56	—	—	—	—	—
	55	—	—	44	43	44
	54	—	46	—	—	—
	53	—	—	43	42	43
	52	—	45	—	—	—
	51	—	—	42	41	42
	50	—	44	—	—	41
	49	—	—	41	40	—
	48	—	43	—	—	40
	47	—	—	40	39	—
	46	—	42	—	38	39
	45	—	41	39	—	38
	44	—	—	38	37	—
	43	—	40	—	—	37
	42	—	—	37	36	36
	41	—	39	—	35	—
	40	—	38	36	—	35
	39	—	37	35	34	34
	38	—	—	—	—	—
	37	—	36	34	33	33
8	36	—	35	33	32	32
8	35	—	—	—	—	31
	34	—	34	32	31	—
	33	32-36	33	31	30	30
	32	31	—	—	—	29
	31	—	32	30	29	28
7	30	30	31	29	28	—
7	29	—	30	—	—	27
	28	29	—	28	27	26
	27	28	29	27	26	25
	26	—	28	26	—	24
	25	27	27	—	25	—
	24	—	26	25	24	23
6	23	26	25	24	23	22
6	22	25	—	23	22	21
	21	24	24	22	—	20
	20	23	23	21	21	19
	19	—	22	—	20	—
	18	22	21	20	19	18
5	17	21	20	19	18	17
5	16	20	19	18	17	16
	15	19	18	17	16	15
	14	17-18	17	16	15	14
4	13	16	16	15	14	13
4	12	15	15	14	—	12
	11	14	14	13	13	—
	10	13	13	12	12	11
3	9	12	12	11	11	10
3	8	11	11	10	10	9
	7	10	10	9	9	8
	6	9	8-9	8	8	7
2	5	8	7	7	7	6
2	4	7	6	6	6	5
	3	5-6	5	5	5	4
1	2	4	4	4	4	3
1	1	0-3	0-3	0-3	0-3	0-2

Appendix C, Continued

DEMOGRAPHIC QUESTIONNAIRE

ID Code _____ Date _____

We would like some additional information about your background. Please complete the following items. If you have any questions about this questionnaire, please feel free to talk to the Research Assistant.

1. **What is your age?** _____

2. **What is your marital status?**

   1. [ ] single, never married
   2. [ ] married
   3. [ ] divorced or separated
   4. [ ] widowed

3. Is your new baby a boy ____ or a girl ____?

4. How many other pregnancies have you had? _____

5. How many other children do you have? (not including the new baby) _____

6. What is the age of each of the other children? Please list the ages of the children from youngest to oldest. Start with the youngest child first.

   a. ___
   b. ___
   c. ___
   d. ___
   e. ___

7. Do you have a place to live? ___ Yes ___ No

   If yes, what is the total number of people living in your household (including yourself)? _____

8. Does your family receive money from any government agency to help with housing costs? ___ Yes ___ No

Appendix C, Continued

9. How does your family pay for health care?

1. [ ] private insurance or HMO
2. [ ] MediCare, MediCal or other government program
3. [ ] family pays all costs
4. [ ] other _____

10. Do you live with a husband, boyfriend or partner? ___ Yes ___ No

11. How much emotional support and help do you get from your husband, boyfriend or partner? (Choose one)

1	2	3	4
None at all	A little bit	Quite a bit	Very, very much

12. How much emotional support and help do you get from your parents?

1	2	3	4
None at all	A little bit	Quite a bit	Very, very much

13. How much emotional support and help do you get from your friends?

1	2	3	4
None at all	A little bit	Quite a bit	Very, very much

14. Are you employed? ___ Yes ___ No

15. If yes, what kind of work do you do?

a. main job _____ ___ full-time
___ part-time

b. second job _____ ___ full-time
(if you have one)
___ part-time

Do you have any other sources of income? ___ Yes ___ No

Please explain _____

16. Are you the only financial support for your family? ___ Yes ___ No

Appendix C, Continued

17. Was your family income $12,000 or less in 1991? ___ Yes ___ No

18. Was your family income enough to cover the family's needs? (please check one):

1. [ ] not enough income for family needs
2. [ ] barely enough income for family needs
3. [ ] adequate income, but no extra to spend
4. [ ] adequate income and some extra to spend
5. [ ] more than adequate income

19. Does your family receive money from any government agency to help with living expenses? ___ Yes ___ No

20. How much formal education have you had?
Exact number of years, counting from 1st grade? _____

Please check the highest level of school that you have completed:

1. [ ] no formal education
2. [ ] less than 6th grade
3. [ ] completed 6th grade
4. [ ] completed some high school
5. [ ] graduated from high school
6. [ ] completed some college
7. [ ] completed baccalaureate degree
8. [ ] completed graduate or professional degree

21. What is your cultural heritage or background? _____

22. If you had to select from the following choices, how would you describe yourself?

1. [ ] Afro-American or Black
2. [ ] Caucasian or White (non-Hispanic)
3. [ ] Japanese-American or Japanese
4. [ ] Chinese-American or Chinese
5. [ ] Philipino-American or Philipino
6. [ ] Southeast Asian-American or Southeast Asian
7. [ ] other Asian-American or other Asian
8. [ ] Mexican-American or Mexican
9. [ ] Central American
10. [ ] South American
11. [ ] other Hispanic-American or Hispanic
12. [ ] Native American
13. [ ] other _____

23. In what country were you born? _____

Appendix C, Continued

24. If you have a husband or partner, where was this person born? _____

25. In what country was your mother born? _____

26. In what country was your father born? _____

27. What is the main language you speak? _____

What other languages do you speak? _____

28. How many years have you been in the U. S.? _____

29. How would you describe the amount of stress you feel in the following aspects of your life? Please circle one answer for each aspect.

a. stress from my work situation

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

b. stress from money worries or lack of money

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

c. stress from my parents

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

d. stress from discrimination or cultural adjustment

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

e. stress from being a parent

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

Appendix C, Continued

f. stress from my husband or partner

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

g. stress from having this baby

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

h. stress from nervousness or other mental problems

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

i. stress from other things (please explain below)

1	2	3	4
no stress	a little stress	quite a bit of stress	very, very much stress

kinds of stress: _____

_____

_____