The Problem of Prenatal Cocaine Exposure

A Rush to Judgment

VALID concern about the high rate of cocaine use among pregnant women has resulted in an apparent rush to judgment about the extent and permanency of specific effects of intrauterine cocaine exposure on newborns. Predictions of an adverse developmental outcome for these children are being made despite a lack of supportive scientific evidence. Whatever the true outcome, we are concerned that premature conclusions about the severity and universality of cocaine effects are in themselves potentially harmful to children.

Although the prevalence of cocaine/crack use is declining (in 1990 an estimated 6.6 million individuals reported use in the preceding year compared with 12 million in 1988), certain groups continue to use the drug at high or increasing rates.[1,2] Also, more women of childbearing age seem to be affected by cocaine use compared with previous drug epidemics.[3] At present no reliable national estimates of the extent or patterns of cocaine use during pregnancy exist. Depending on the methods and the populations studied, prevalence estimates from individual centers range from 3% to 50%.[4-6] The higher prevalence estimates are most often reported from centers serving poor, inner city mothers.

Reports in the scientific literature of adverse effects in infants born to cocaine-using mothers certainly raise legitimate concern for the well-being of these children.[6-11] Our review of the current literature on the subject[12,13] indicates that available evidence from the newborn period is far too slim and fragmented to allow any clear predictions about the effects of intrauterine exposure to cocaine on the course and outcome of child growth and development. Most studies involve only relatively small numbers of subjects and either do not control or incompletely control for confounding variables such as other drugs and/or biological and sociodemographic cofactors known to contribute to poor outcomes in such children.[14-19] Findings about neurobehavioral effects in the newborn period have been inconsistent or contradictory.[12,13] Significantly, no prospective study of unique long-term consequences of intrauterine cocaine, non-opiate exposure has been published in the peer-review literature.

Because of its urgent and potentially catastrophic nature, intrauterine cocaine exposure has attracted attention from the lay media (*New York Times*. May 19, 1990:1; May 25, 1990:1; August 19, 1990:CN 6). Infants exposed to cocaine in utero are often represented as severely or even irrevocably brain damaged—to the point that they may never function normally in society. On this account, a very large group of children is in danger of being "written off." Moreover, a social sentiment has arisen that the loss of these children is entirely attributable to the prenatal effects of cocaine (a permanent biological factor). Such a conviction works toward exempting society from having to face other possible explanations of the children's plight—explanations such as poverty, community violence, inadequate education, and diminishing employment opportunities that require deeper understanding of wider social values.

Selected Scientific Issues

The scientific literature regarding prenatal cocaine exposure is plagued with a number of methodologic problems that raise at least five critical issues.

First, study populations have not been well defined and are not generalizable to the larger population of women using cocaine during their pregnancy. Generalizing from poorly defined or highly selective samples is risky. For example, one early report involving a small, uncontrolled convenience sample of infants exposed prenatally indicated that 10 of 66 (150/1000) had died of sudden infant death syndrome.[20] This result was disseminated despite a published, systematic investigation that found only a small increase of sudden infant death syndrome among infants prenatally exposed to cocaine.[21] Two subsequent epidemiologic studies[22,23] suggested a moderately elevated risk of sudden infant death syndrome (8.5/1000), but an association has not been firmly established.[24,25] Reports of developmental and behavioral problems among cocaine-exposed children of preschool age attending special education programs may suffer from sample bias similar to the initial sudden infant death syndrome report.

A second methodologic issue is the identification of women using cocaine during pregnancy. Reliance on self-reporting underidentifies users.[26] In one study, 24% of women who used cocaine would not have been identified had not urine assays been conducted.[6] Urine assays at the birth of the child also provide limited information because positive results identify only women who have used the drug within 1 to 4 days of the test. Urine assays performed on mothers post partum identify only 25% of cocaine users, and these are most likely to be

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the heavier users.[27] The misclassification of cocaine users threatens the validity of developmental findings either by falsely selecting for heavy users or by falsely including users with nonusers.

A third methodologic issue is identifying the timing, quantity, and duration of prenatal exposure as well as other prenatal influences associated with cocaine. Is an infant exposed to cocaine early in the first trimester at equal risk with an infant whose mother continues to use the drug throughout pregnancy?[28] Frequency, amount, and timing of cocaine use by pregnant women vary. In one of the few prospective studies conducted, one third of women who reported using cocaine used it less than once per month and 50% used it at least weekly.[29] Only one study has evaluated outcomes in newborns based on different patterns of maternal use.[30] Determining the nature of exposure to cocaine is further complicated by the tendency of cocaine users to use other drugs (eg, alcohol, heroin, marijuana, tobacco), to practice other poor health behaviors (eg, inadequate nutrition and prenatal care[6]), and to experience an increased incidence of sexually transmitted diseases[6] (such as syphilis). The specific effects of cocaine on fetal development may be additive to, or synergistic with, these other factors. For example, in one study, infants of mothers who had a urine assay positive for cocaine during pregnancy were 407 g lighter at birth compared with infants of mothers who did not use cocaine. However, only 25% of the weight decrement could be attributed directly to cocaine. The remainder was attributable to the effect of cigarettes and marijuana, other drugs, and poor nutrition.[6]

A fourth methodologic issue is how to disentangle the unique effects of intrauterine cocaine exposure from the confounding negative effects of cocaine use on the child's family and community and on parenting behaviors. Cocaine intensifies already well-recognized environmental hazards for mothers and their infants—poverty, violence, abandonment, homelessness, multiple short-term foster placements, and inadequate or abusive parenting.[12,31] These can be significant factors in and of themselves inasmuch as styles of parenting occupy an important place in determining the child's early cognitive, social, and emotional development.[32] Additionally, poor environments put children at risk for other potentially neurotoxic exposure such as lead poisoning. Differentiating the relative importance of the multiple poor health behaviors prenatally and environmental factors associated with parental drug use postnatally from neurophysiologic manifestations specific to the actions of prenatal cocaine exposure is difficult.

The fifth methodologic issue concerns identifying the specific functions in children that are likely to be compromised by cocaine. General measures of developmental outcome may not provide critical information on how cocaine harms children or precisely which functions may be affected, so that appropriate intervention or remediation can be designed. To find proper measures requires us to ask what is currently known about neurological and behavioral effects specific to cocaine. Cocaine inhibits the reuptake and metabolism at the neural junction of the major monoamine transmitters that play an important role in central control of basic processes including autonomic function, behavioral state regulation, and response to sensory stimuli.[33] By altering this neurotransmitter activity in the developing nervous system, chronic exposure to cocaine in utero may adversely affect these functions. One study suggests possible difficulties in infants' ability to regulate states of arousal (ie, the ability to modulate reactivity to both internal and external stimuli), which may lead to behaviors such as impulsivity and mood instability.[7]

Further, along with reported smaller head circumference in exposed infants,[6] findings of structural changes to the brain, such as echodensities and echolucencies primarily in the frontal lobe and basal ganglia,[34] suggest that prenatal exposure to a combination of amphetamines and cocaine may also result in specific morphologic alterations in brain structure. Each of these single study findings, all of which need replication, suggests that it may be most productive to examine different aspects of autonomic nervous system regulation such as attentional states, different types of information processing, or the capacity to modulate anxiety.

Social and Political Issues

These unresolved scientific considerations and recommendations are closely tied to a significant social-political issue. Why is there today such an urgency to label prenatally cocaine-exposed children as irremediably damaged (*Rolling Stone*. October 18, 1990:68)? What are society's attitudes toward and responsibility for these disadvantaged children? These problems are not explicitly methodologic ones although they are predicated on empirical findings. Moreover, in themselves, they carry significant medical and psychosocial risks for the children.

First, labels have a way of becoming self-fulfilling. Minimally, expectations for such children are lowered.[35] The attribution of irremediable damage makes it more difficult to find services for these children, and such services may be geared to caretake rather than to challenge children's capacities or to remediate effectively. Even more damaging is the difficulty finding adequate homes for such children since potential foster or adoptive parents are often concerned about assuming the care of cocaine-exposed children because of their perceived impairments (*New York Times*. May 19, 1990:1; September 21, 1990:1).

Second, labels also carry with them a risk for biasing and undermining clinical decisions and scientific publications. A 1989 report in *The Lancet* indicates that abstracts regarding the impact of prenatal cocaine use were more likely to be accepted for presentation at the annual meeting of the Society for Pediatric Research if they reported positive results (ie, evidence of impairment) than if they failed to show such results even though the rejected papers with negative findings tended to be methodologically more rigorous.[36] Clinical decisions are also affected by underlying attitudes. Another study shows that, given equivalent extent of use of illegal drugs by pregnant women, physicians and clinics are more likely to report to law-enforcement agencies black women or women on welfare than white or middle-class women.[4] Prejudice exists and it can well bias interpretation of the data, particularly when observers are not blinded to study group assignment.

Labeling and isolating infants and young children because of their prenatal experience are irrational and inhumane actions. Condemning these children with labels of permanent handicap and failure is premature and may lead us to overlook what we have long known about the remediating effects of early intervention.[37] Studies of preterm or ill newborns fail to support biologic determinism.[38-40] Environments contribute significantly to the outcome of infants with biologic vul-

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nerabilities at birth.[41] Even among infants exposed to narcotics prenatally, the home environment, and not the amount of narcotics, seems to be the more important predictor of developmental outcome.[42]

Nothing in this commentary is meant to underestimate the potential impact of the use of cocaine by pregnant women or to suggest other than that the most important, and most effective, way of dealing with this problem would be to prevent it. However, our history of success in prohibiting the use of illegal or even legal toxic substances does not inspire the hope that preventing cocaine use will occur quickly. Even were we to solve the problem of cocaine use in pregnancy in the next few years, thousands of children will still be born after intrauterine exposure to cocaine. While this commentary is intended to highlight the limitations of information currently available, the children can and need to be helped through early intervention strategies about which much is already known.

While we await research findings regarding discrete neurodevelopmental and/or learning problems that may require specific therapeutic interventions and/or educational strategies, we have the responsibility to provide comprehensive services for these infants and their mothers. For the infants, such services include adequate nutrition, health care, and early developmental intervention programs, and for the mothers, drug treatment, health care, and family support assistance. The availability of multiple services is surely critical, but the coordination of service delivery is just as important. Consistent with the recommendation of the National Commission on Infant Mortality and with our own experience, [12] the provision of services in a model of "one-stop shopping" may be most effective, because keeping multiple appointments in different sites is difficult for all mothers with young babies, and especially those using drugs. Providing key services such as pediatric health care, drug treatment, child development, and family planning in one location with one appointment system and the same staff may facilitate compliance with these services and improvement of the health and well-being of both mothers and children.

Good science is needed to make sound clinical and public policy decisions.[43] Separating specific developmental impairments attributable to prenatal cocaine exposure from the effects of inadequate caretaking is difficult but necessary to develop policy and appropriate interventions. For policymakers, social agencies, and health and educational institutions, we recommend a suspension of judgment about the developmental outcome of cocaine-exposed babies until solid scientific data are available. Whatever the damage from prenatal exposure to cocaine may prove to be, outcome will not be improved by an attitude that assumes that exposed children cannot be helped or that they are different from other children.

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