Neurobiology of Birth Weight Influence on Blood Pressure Values and Variability in Children and Adolescents

To the Editor:
Lurbe et al1 find an independent and significant inverse relationship for birth weight and 24-hour systolic blood pressure in 630 healthy children, ages 4 to 18 years (P<0.001). Likewise, birth weight was independently and inversely correlated with 24-hour systolic blood pressure variability (P<0.03).

Neurobiological features are suggested by a report that dopamine is a potent inhibitor of growth hormone and by studies linking anxiety and vasospasm with dopamine abnormalities lateralized to the right hemisphere. This hypothesis is supported by optimal response organization and working memory at intermediate dopamine tone in a mediofrontostriatal activation system, and deactivation of the right hemisphere, a state marker of depression, that promotes dominance of the left hemisphere associated with cardiac dysrhythmia and vasoconstriction.2,3

These findings should prompt clarification of the relationship of low birth weight to blood pressure1 by neurochemical and pharmacotherapeutic investigation of ancient neuromodulatory systems in complex evolving brains.3–5

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Response
We appreciate the interest Dr Friedman has shown in our research on the impact of birth weight on the values and/or characteristics of blood pressure during childhood and adolescence, which was published in the September 20011 issue. An inverse relationship between birth weight and blood pressure levels has been demonstrated not only by our group,2 but also by others.3

The mechanistic link between birth weight and blood pressure (BP), however, remains elusive. Increased fetal exposure to maternal glucocorticoids, which exert organizational effects or imprinting patterns of response in vascular structures and cerebral tissue that persist throughout life, has been implicated.4 Glucocorticoids in maternal circulation are normally prevented from gaining access to the fetus by a placental enzyme, 11-β-hydroxysteroid dehydrogenase, which catalyzes the rapidly metabolized cortisol and corticosterone into inactive products. Studies conducted in rats demonstrated a low activity level of this enzyme in newborn animals that have a large placenta but a low birth weight.5 Furthermore, the administration of low doses of dexamethasone to pregnant rats led to persistently raised blood pressure in their offspring.6 Whether or not an increase in glucocorticoids exposure may modify dopamine metabolism in intrauterine life has not been explored until now. Neurobiological research can be one of the ways to a better understanding of this complex process.

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References
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