Fetal hypertension and abnormal fetal cardiac morphology

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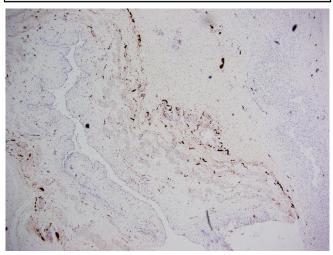
TO THE EDITORS: Dr Youssef and colleagues describe abnormal fetal cardiac morphology in preeclamps d fetal growth restriction in a large series of pregnancies in Portugal. Our finding of hyperplasia of the tunica media of the umbilical arteries in the proximal umbilical cord offers supportive evidence for their observations consistent with Barkers hypothesis of the fetal origins of adult disease.²

We define "fetal hypertension" as hyperplasia of the tunica media of the extrafetal segments of the umbilical arteries, associated with injuries to vasomotor nerves of the intrafetal segment of the umbilical artery (Figure). The innervation of umbilical arteries has been a contentious matter for more than 150 years, with some colleagues confident that the

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FIGURE

The urachus and intrafetal segments of right and left umbilical arteries



Cross section of the intrafetal segments of the right and left umbilical arteries as they converge on the urachus (U) at the umbilicus to form the umbilical cord. The courses of the two umbilical arteries are indicated by two groups of injured (brown), perivascular nerves which we propose are injured by high, intravascular pressures in the syndrome of "fetal hypertension".

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umbilical artery has vasomotor nerves, and many more convinced that it does not!^{3,4} If the fetus has "hypertension," then the intrafetal segments of the umbilical arteries show wide-ranging injuries to vasomotor nerves that may extend into the proximal umbilical cord (Figure).

This particular fetus had marked cardiac abnormalities at 24+1 weeks that may be attributable to sustained maternal hypertension (180/130 mm Hg) including tricuspid valve incompetence, pulmonary stenosis, and abnormal aortic valve development. Histology showed widespread aberrant reinnervation throughout the upper thorax including the esophagus, heart, and lungs.

Dr Youssef et al find little difference between babies with preeclampsia (PE) and fetal growth restriction (FGR) and those with FGR. We propose that the direct neural connections between the uterus and kidneys have been avulsed, either by persistent physical efforts during defecation or excessive traction to the cervix.⁵ If the neural connections are "lost," then there will be FGR but no PE; if they are intact, then it will produce FGR with "classic" PE. Clearly, both groups of babies will need careful evaluation at birth including assessment of their renal function.

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The authors report no conflict of interest.

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