Comment on: Preventing preeclampsia with aspirin: does dose or timing matter?

TO THE EDITORS: Tong et al1 state that possible biological mechanism of action of aspirin in the prevention of preeclampsia are that it: (1) “facilitates early placentation embedding, a process that is in fact poorly understood but is likely to be complete by 16 weeks’ gestation”; and/or (2) also increases prostacyclin (vasodilator); and/or (3) may decrease endothelial (blood vessel) dysfunction.1 Tong et al1 do not mention the effect of aspirin on platelets. By the inhibition of prostaglandin synthetase, aspirin blocks the production of thromboxane, the mediator of platelet activity. Thus aspirin renders the platelets nonfunctional and hence maintains circulation in small diseased blood vessels.

As far as placentation is concerned: “the spiral arteries after being breached by the non-villous trophoblast, undergo extensive adaptations and structural alterations in order to provide the intervillous space of the growing placenta with an adequate amount of maternal blood.”2 This process they termed “physiological change.”

“From 15 or 16 weeks’ gestation, there seems to be a wave of intra-arterial trophoblast migration beyond the deciduomymometrial junction into the true myometrial segments of the spiral arteries.”3

We used color Doppler ultrasound to describe the changes in the uteroplacental circulation in the midtrimester of nulliparous pregnancy observing and quantitating the development of a low-resistance circulation.4 We found that the indices of flow fell from 14-18 weeks and fell even further from 18-24 weeks.

Failure of this process of physiological change in the spiral arteries of the placental bed is found in cases of preeclampsia. Thus the earliest known feature of preeclampsia, the failure of physiological change in the spiral arterioles, is probably an event predominantly of the second trimester.

These spiral arterioles, where physiological change has not occurred, can then undergo atheromatous-like disease termed “acute atherosis.”5 This results in a further restriction of blood flow and even complete vascular occlusion.2

Our uterine artery Doppler observations of patients at high risk of preeclampsia at 12 weeks’ gestation6 were that both patients treated with aspirin and those not treated showed a reduction in uteroplacental resistance toward the expected median at 24 weeks’ gestation. Also there was no significant difference between the observational and aspirin-treated cohorts. These observations would tend to indicate that aspirin does not have an effect on the failure of the normal physiological change of the spiral arterioles. Hence any effect on the uteroplacental circulation is more likely to be in the prevention of acute atherosis probably via its action on platelets.

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REFERENCES

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REPLY

We thank McMaster-Fay and Hyett1 for their comment on our editorial2 that discussed 2 meta-analyses3,4. Their comment was not directed at our interpretation of these important studies but rather, at our brief preamble where we...