Better understanding needed of physiology of sustaining life in utero

Zulfi qar Bhutta and colleagues pose the question of what we can do to reduce stillbirth rates. Improving our understanding of the physiological processes behind sustaining life in utero might be useful to aid appropriate care provision.

Even healthy babies are compromised in utero. Being an end organ of the mother, the fetus’s oxygen concentrations are exceedingly low, as shown by the high haemoglobin concentrations, the presence of fetal haemoglobin, and the special preferential flow to the myocardium and brain. Realising the precarious state of existence of all fetuses is a first step in recognition of the root cause of the problem.

The health of the placenta is crucial to the life of the fetus. Delivery before the placenta undergoes senescence at 42 weeks in normal babies and before 38 weeks in diabetic women might prevent a substantial number of deaths or morbidity. Focus on placental function should be the basis of better care for the fetus.

Acute compromise in placental function as in abruptio placentae, rupture of the uterus, and maternal haemorrhage will cause immediate fetal compromise if not death. This is consistent with the inverse correlation between the availability of timely caesarean section and incidence of stillbirth. Timely intervention can reduce a high proportion of the damage currently seen.

Stillbirths, challenged babies, and healthy babies form a spectrum. They are the product of the nature of care we put into addressing their physiological needs in utero. Improving this knowledge might have a substantial effect on the kind of care we deliver to this under-recognised risk group.

I declare that I have no conflicts of interest.

YooKuen Chan
chanya@ummc.edu.my
Department of Anesthesiology and Critical Care, Faculty of Medicine, University of Malaya, 50603 Kuala Lumpur, Malaysia

Genetic factors in stillbirths

Given the dearth of attention to stillbirths, The Lancet’s Series has properly addressed avoidable factors to reduce the incidence. Little discussion has been given to genetic factors, which have major ramifications for families and societies.

About 15–25% of stillbirths are caused mainly by genetic factors. In recognition, professional organisations such as the American College of Obstetricians and Gynecologists recommend and provide guidelines for genetic testing of stillborn babies. Up to 20% of stillborn babies with malformations have chromosomal abnormalities, as do 8–13% of those without overt malformations.

To identify these chromosomal abnormalities, tissue should ideally be obtained before delivery by amniocentesis or chorionic villus sampling. Cell cultures initiated directly from tissues of delivered stillborn babies are rarely successful.

Stillbirth can also be caused by single gene mutations. A diagnostic pitfall here is that even well known disorders (eg, trisomy 21) are difficult to recognise morphologically owing to maceration. All stillborn babies should ideally be examined by a geneticist, and undergo autopsy for diagnosis that could alter management in future pregnancies. If declined, whole-body imaging by ultrasonography, MRI, or plain radiography can still be useful. Tissue should be preserved for molecular studies. Complex (polygenic) genetic factors generate variations that predispose to stillbirth, one example being the genetic nature of altered birthweight. As gene sequencing becomes more available, mutations having fetal death as their only manifestation will be identified. Genes that perturb normal placental function are especially attractive targets.

Attacking avoidable factors to reduce stillbirths remains the priority. However, identification of stillbirths with major genetic causes is increasingly worthy of medical attention and dedicated resources.

We declare that we have no conflicts of interest.

Joe Leigh Simpson, Juan M Acuna
simpsonj@fiu.edu
Department of Human and Molecular Genetics, Florida International University Herbert Wertheim College of Medicine, Miami, FL 33199, USA