did normative data become available on this measure, once
the automation of computer measurement had been perfected
for the EEG. Subsequent EEG studies were able to confirm the
maturational delay reported by these authors in regard to brain
development. 1

Though not many empirical data were presented in the
paper with regard to social and emotional functioning, the
authors did report on their impressions. It is noticeable
that in both their own later publications and the work of
other researchers confirmation of disturbed emotional and
psychological factors, other than intellectual functioning,
was found among individuals with a history of early
undernutrition.

This paper is an example of pioneering research in the field
of undernutrition and its effects on growth and behaviour.
Reviewing it does however allow for speculation on issues that
the authors may, given the state-of-the-art research today, wish
the current generation of researchers would address. Three
examples come to mind. The relatively recent identification
of several points where spurts in growth as well as ‘pruning’
take place in the developmental trajectory of children raises
the question of repeated vulnerability once the all-important
period of growth reported on by Stoch and Smythe has been
weathered. What would improved techniques of monitoring
of brain function reveal in a repeat of this classic study?
Functional assessments of both metabolic and EEG indicators
of the brain in action are now possible, allowing great
resolution in both measurement and identification of maturing
function in specific areas of the brain. What of a closer
monitoring of social and emotional development over the early
lifespan? Stoch and Smythe challenge us!

Reference

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Observations on the origin of congenital intestinal atresia

Lewis Spitz

The article by Louw and Barnard entitled ‘Congenital intestinal
atresia – observations on its origin’ published in The Lancet in
1995 was a landmark paper that elucidated the pathogenesis
of intestinal atresia and radically altered the surgical treatment
of the condition.

As a result of a review of cases of intestinal atresia at
Great Ormond Street Hospital, Louw postulated that at least
some atresias might have been due to interference with the
blood supply to that portion of the fetal gut. This study was

Louw and Barnard proposed that ‘strangulation of foetal
bowel may end in disappearance of the infarcted portion, with,
at most, a complicating meconium peritonitis’. This sequence
of events was possible only because of the sterile environment
of the fetal intestine in utero.

Barnard embarked on a series of experiments involving
interfering with the blood supply to a segment of bowel in
the fetal pup. Barnard stated triumphantly in this article that
‘after many disappointments due to anaesthetic and technical
difficulties, death of the foetus, premature labour and

cannibalism, success has now been achieved in two animals’.
This was a remarkable achievement at the time and a testament
to Barnard’s persistence and technical skills.

The outcome of the experiments together with the clinical
findings at surgery supported the theory of a ‘vascular
accident’ as the cause of intestinal atresia. From a practical
point of view, the authors made the assumption that if the
vascular origin of atresia was accepted, it was likely that the
blood supply to portions of the bowel adjacent to the atretic
segment would be compromised, not sufficiently to cause
necrosis but sufficiently to cause a functional problem with
resultant defective peristalsis.

Their recommendation was that the blind bulbous end
of the proximal intestine should always be resected before
an anastomosis is performed. The immediate result of this
policy was a reduction in the mortality for intestinal atresia at
Great Ormond Street Hospital from 69% to 33%. The advice
was rapidly adopted universally and became standard in
the management of intestinal atresia. A truly remarkable
achievement.

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