of the report, have now added new information to this previously undescribed condition.

Immunocytochemistry using either paraffin sections or cryostat sections of formalin-fixed tissue, and highly specific antibodies to insulin (dilution 1:9000), glucagon (dilution 1:5000), somatostatin (dilution 1:1000), and vasoactive intestinal polypeptide (dilution 1:1000) showed only very few, weakly stained somatostatin cells. Electron microscopical examination of pancreatic tissue which had been taken almost agonally and immediately fixed in 2·5% glutaraldehyde in 0·1 mol/l phosphate buffer at 4°C confirmed that autolysis at necropsy was minimal. The exocrine, acinar cells appeared normal. Rare, often solitary endocrine cells could be identified by their typical granule structures. Most of them were the D-(somatostatin) cells (Figure (a) and α-(glucagon) cells (b and c), although occasional pp cells (d) and cells with an unidentified polypeptide hormone (labelled d in a) could also be seen. Only very rarely was a B-cell with insulin granules (Figure d) observed. None of these cells was associated, as in a normal pancreas, with large groups of 'islets'. Thus, all 4 well-recognised endocrine cell types were identified, although in extremely reduced numbers.

We have been unable to trace any previous or subsequent descriptions of similar infants with congenital absence of pancreatic β-cells.

Reference

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Plasma prostacyclin from birth to adolescence

Sir,

The finding that plasma concentrations of 6-keto-prostaglandin $F_{1\alpha}$ (6-keto-$PGF_{1\alpha}$), one of several degradation products of prostacyclin (PGI_2), fall from a high value at birth to a lower, steady range in the first week is of interest.[1] Urinary excretion of 6-keto-$PGF_{1\alpha}$ shows a similar pattern[2] and fetal plasma concentrations are markedly raised at mid-trimester declining towards term.[3] However, it is presumptive to assume that these findings reflect physiological changes in plasma PGI_2 in the fetus and newborn. Firstly there is no universal agreement that PGI_2 acts as a circulating hormone.[4 5] Furthermore, neonatal plasma demonstrates a diminished ability to support the generation of PGI_2-like activity from endothelium *in vitro*,[6] and there is an argument that the normal bleeding time of the neonate is the result of a balanced reduction of platelet pro-aggregatory and endothelial anti-aggregatory (possible PGI_2) effect. In the fetus or newborn it is unacceptable to infer from the observations on 6-keto-$PGF_{1\alpha}$ concentration that plasma PGI_2 levels or vascular PGI_2 effects are greater than at other ages without knowledge of the distribution, catabolism, and excretion of the measured metabolite. Unfortunately this information is lacking and a more cautious interpretation, such as that offered by other investigators,[2] is appropriate.

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Dr Kääpa and co-workers comment:

We thank Dr Taylor and Dr Lote for their interest in our work.[1] We agree that since submitting our paper it has been increasingly evident that the concentration of prostacyclin (PGI_2) in the circulation is lower than is needed to affect platelet aggregation *in vitro*.[2–5] Thus the concentration of 6-keto-prostaglandin $F_{1\alpha}$ (6-keto-$PGF_{1\alpha}$), the main degradation product of PGI_2 in human plasma,[6] does not necessarily reflect the changes of PGI_2 in the circulation. Nevertheless, plasma and urinary 6-keto-$PGF_{1\alpha}$ could well reflect the physiologically important production of PGI_2 in the body.

We believe that our main finding on the increased PGI_2 production during the neonate period is valid.[1] The fact that the urinary excretion of 6-keto-$PGF_{1\alpha}$, as measured by radioimmunoassay[7] and gas chromatography-mass spectrometry,[8] is increased during the first days of life strongly supports our view and clearly shows that the increased plasma 6-keto-PGF_1 [1] is not a result