Exstrophy of the Bladder: Its Variations and Causation

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Abstract

Exstrophy of the urinary bladder is a very exotic malformation essentially limited to humans. This type of deformity presents a full panorama from very mild cases of epispadias to full cloacal exstrophy. Unlike many malformations, it does not correspond to a normal stage of embryonic development, and on that account does not appear to be a simple arrest of development. The least degrees of this anatomic progression (such as balanitic epispadias) are very rare, as also are the extreme deviations (such as cloacal extrophy). The central and rather advanced form (classical exstrophy) is by far the most frequent in the panorama of this anomalous complex. Largely from these observations, an embryogenic theory was developed that a wedge-like effect of a persistent cloacal membrane could be the fundamental cause. The most common denominator is the musculoskeletal deformity, the separation of the symphyses and recti muscles. This theory was tested by actually inserting a wedge in the embryo of the chicken; thus for the first time extrophy was produced experimentally (indeed in a species in which exstrophy is not known to occur spontaneously). This theory and its experimental proof would indicate that there are no structures missing in the classical exstrophy and, therefore, functional closure of exstrophy should be feasible. However, clinical experience has long shown that urinary control is only rarely obtained; or, if such is obtained, the upper urinary tracts rapidly become damaged. Reimplantation of the ureter at the time of closure has been found to prevent reflux and thus protect the upper urinary tracts in most instances. A study of the bony deformity of the pelvis indicates that accurate approximation of the tissues might be better obtained if the bony structures were divided at more suitable places at the time of the closure. Such a study has been begun, partly based on models made from x-rays.