INTRODUCTION

Sperm motility is generally recognized as the most important indicator of seminal quality (Mac Leod and Gold , 1935). Abnormal motility as an isolated disorder may exist in 24% of subfertile patients. In these patients acquired or congenital (genetic) lesions of flagellar function are likely (Lipshultz, 1985). The effect of morphological alternations on fertility has been reported by many authors (Sherins, 1977 and Roger, 1983). There is clear invers correlation between progressive motility and incidence of sperm locomotor system anomalies (Marina et al, 1989), and this indicates that structural alterations are the most important causes of low spermatic motility.

The molarity of these alterations cannot be diagnosed by the ordinary microscope (Marina et al, 1989), that is why transmission electron microscopy has been used extensively to study the ultrastructure of spermatozoa under normal and pathological conditions. Such investigations helped much, in diagnosis of many andrological disorders, by finding special sperm abnormalities undetectable optically.

Electron microscopy has expanded our knowledge of pathological conditions in certain cases of male infertility. In particular, in cases of infertility associated with immotile spermatozoa, several distinct abnormalities in fine structure have been found that could not have been resolved by light microscopy (Afzelius et al ., 1976). These include lack of dynein arms on the microtubule doublets of the axoneme [comprising Kartagener's syndrome or immotile cilia syndrome] (Pedersen and Rebbe, 1975), lack of radial spokes (Sturgess et al., 1979), the deletion of the central microtubules (Bacetti et al., 1979) and transposition of the number 1 microtubule doublet (Sturgess et al., 1980).