

Mainzer). The spleen is often enlarged and contains a pigment resembling that found in malaria and similar to that existing in the enteric tract of the worms (Hutchison). A marked involvement of the cervix and vagina may occur in women and may finally lead to a cicatricial constriction of the vaginal canal, following a preceding thickening and wrinkling of the vaginal mucosa with the appearance of a widespread verrucosis, complicated by abscess and scar formation (Hutchison). A marked eosinophilia is found in the blood. While the serum of infected persons gives, during the later stages, a positive complement fixation test with the liver extract from infected snails (Fairley), there does not develop any appreciable degree of immunity against the infectious agent, even in the presence of a prolonged or repeated parasitic infestation.

VII. PATHOLOGY

The cystoscopic examination performed during an early stage of a vesical schistosomiasis shows an inflamed and hyperplastic vesical mucosa, with dilated submucosal veins. This change is present in the entire bladder, but especially marked in the trigonal and paratrigoal region and in the posterior wall of the bladder as well as the posterior portion of the urethra. The prostate gland is swollen and indurated. When a massive infestation of the bladder wall with parasitic ova exists, there occurs a diffuse thickening of the mucosa without the formation of localized polypous mucosal projections. These rather plump, localized, inflammatory, mucosal and submucosal reactions, which make their first appearance at a relatively early stage, consist of a vascular inflammatory granulation tissue and are most frequently located and most numerous in the lower segment and posterior wall of the bladder. Coincidental with the extrusion of the parasitic eggs through the vesical mucosa, parts of this tissue may slough and the defects may be covered by mineral precipitations (phosphates) from the urine. Scaled off fragments of these mineral crusts may form together with necrotic eggs and clotted blood, the nucleus of calculi which grow by the superficial deposition of oxalate and uric acid crystals. Leukoplakias are frequently found during this stage in the vesical mucosa. During the further course of the disease, the vesical lumen shrinks and the walls become increasingly rigid. Secondary bacterial infections contribute to and aggravate the inflammatory reactions in the bladder wall, causing the development of abscesses and fistulas. The spreading of these processes upon the posterior urethra may cause a clogging of the urethral lumen, due to mucosal swelling and thickening and the lodging of larger particles of necrotic matter expelled from the bladder into the urethra. Similar inflammatory changes develop in the ureters and renal pelvis when the infection spreads in a retrograde direction.

The pathologic vesical lesions described furnish the soil from which papillomas and cancers may develop in the bladder and also occasionally in the