regeneration of flat and syncytial-like tubular epithelium with marked distortion of the tubules, and associated abundant irregular overgrowth of connective tissue. Arteriosclerosis of kidney arteries usually prominent; infarct formation occasional (includes in general the old secondary contracted kidney and Jores Form 2, Frey's Form 2, Volhard's Form 2 and probably cases of Form 3. Nephropathia chronica inflammatoria of Aschoff).

b. Focal and patchy: Characterized early by a hyaline swelling and fatty degeneration of intima and endothelial cells of the small renal vessels and capillaries, associated with cellular interstitial and periglomerular cell foci and focal parenchymatous degeneration. All of these acquire gradually more momentum, but the involvement of the small vessels remains most conspicuous. It leads to obliteration of their lumen, thereby adding quantitative to the qualitative disturbance. Thus results collapse of affected parts, the extent of which necessarily varies, so that at times the superficial appearance of the kidney may be only very finely granular. The extent of new connective tissue formation is also correspondingly irregular, usually most pronounced around Bowman's capsule. The tubules are relatively well preserved until late in the disease, and epithelial desquamation is insignificant. The disease is usually accompanied by arteriosclerosis of the kidney arteries. (Includes in general, Jores' Type 1, Frey's Type 1, Subdivision b, and Volhard's Type 1, and possibly cases of Type 3. Nephropathia chronica degenerative sive circulatoria of Aschoff. The old genuine contracted red kidney or primary interstitial nephritis.)

The difference between a and b lies in the fact that in a, a relatively strong or declinningly strong irritative influence has affected the kidney very diffusely, perhaps as the result of a previous severe degenerative and exudative nephritis; in b, a much less severe but persistent irritant, which is never sufficiently strong to produce severe diffuse degeneration and exudation, gradually involves the kidney in disease. It naturally affects the walls of the smaller blood-vessels and the glomeruli prominently first, on account of their exposure by virtue of anatomical structure to irritants and blood-pressure. By almost simultaneous extension of its irritating influence it attacks patches of parenchyma and the neighboring periglomerular parts which respond by focal cell accumulation. Their appearance is then manifest, as we have seen, before any quantitative interference by vascular obliteration is possible. In all forms of contracted kidney heart hypertrophy is the rule.

Finally, one may group as independent non-inflammatory affections the senile atrophy and the true arteriosclerotic atrophy of the Ziegler type. The latter is plainly characterized by patchy loss of substance due to elastic thickening and gradual obliteration of arteries alone. It does