individuals affected are in general of middle age (average age, 45.6 years) suggesting the presence of preparatory changes in the affected area at the time of the burn. Treves and Pack stated that the average age of individuals with acute burn scar cancers was 52 years at the time of the burn.

Chronic burn scar cancers have a latency period of 32.5 years (average), according to Treves and Pack, and of 23 to 59 years, according to Ullmann. Roffo and Gandolfo gave the following distribution of their cases upon the various decades of latency period: 1-10 years, 8 cases; 11-20 years, 4 cases; 21-30 years, 11 cases; 30-35 years, 3 cases (average, 18 years). In eleven cases collected from the literature the average latency period was 31 years (range, 9 to 51 years: 1-10 years, 1 case; 11-20 years, 3 cases; 21-30 years, 1 case; 31-40 years, 2 cases; 41-50 years, 3 cases; 51-60 years, 1 case). The average age of the individual composing this series was 14 years at the time of the thermic injury, while it was 20 years in the series observed by Treves and Pack (against 45.6 and 52 years, respectively, in two acute series analyzed). The first symptoms of a malignant development (ulceration) in the scar were observed within 6 to 41 years after the accident (average, 22 years), while the primary ulcerative thermic defect never healed completely before cancer ensued after 18 to 51 years (average, 40 years) in three cases.

Causative Mechanism. The causative mechanism active in the production of acute and chronic burn scar cancer is unknown. Some investigators favor the conception that the vascular and nutritive disturbances present in the scar tissue, especially in the large extensive scars often found after burns, play an etiological role, as scar tissue is poorly vascularized and therefore insufficiently nourished, causing it to break down easily and to ulcerate. In burn scars, as in any other scars, there exists an abnormal relation between the epithelium and the connective tissue, which is said to predispose to malignant transformations. Such changes may be stimulated, according to these investigators, by the fact that a scar has no elasticity and reacts therefore more strongly upon a trauma than normal tissue. The presence of folds and nooks in the surface of the scar favor the retention of irritating material, while the configuration of the scar aggravates the normal friction exerted by clothing upon the epidermal lining. All these factors produce, in the opinion of Treves and Pack, a favorable soil for a cancerous change. Whereas these investigators seem to favor the theory of Virchow (chronic irritation as cause of cancerigenesis), Roffo and Gandolfo contended that burn scar carcinomas exemplified the theory of Ribbert, as these neoplasms appeared to originate from misplaced epithelial cell groups. In as much as more or less extensive burns are relatively common, while malignant sequelae arising from them are comparatively rare, it is evident that these concepts of carcinogenesis in burn scars do not offer any plausible explanation.

Treves and Pack proposed, on the other hand, that in the production of acute burn scar cancer tissue, toxins are released by the autolysis and heterolysis of the burn eschar, which may act as direct cancerigenic agents. This conception gains in probability when consideration is given to the fact that during the