minase activity was relatively low in the large granules and was completely absent from the microsome or small granule fraction. Subsequent to these observations it was found that vitamin $B_6$ is an integral part of this enzyme system.[13] Thus the data presented above support those of Claude since the small granules contain no transaminase activity and very little vitamin $B_6$ while the parts of the liver cell that contain transaminase activity are particularly rich in vitamin $B_6$. No information is available on the presence or distribution in these tissues of other enzymes which contain vitamin $B_6$.

The hepatic carcinogen 4-dimethylaminoazobenzene reduced the level of riboflavin[1,2] and vitamin $B_6$ in the large granules and supernatant fluid, but in both cases the reduction of protein in the large granules paralleled the decrease in vitamin content. This is probably the result of a reduction of the number of large granules in the liver cells.[3]

*Summary.* The intracellular distribution of vitamin $B_6$ in rat liver tumors induced by 4-dimethylaminoazobenzene and in the livers of rats and mice before and after administration of the azo dye was determined after differential centrifugation of the tissue homogenates in hypertonic sucrose solution.

In all cases vitamin $B_6$ was concentrated in the washed large granules or mitochondria (28 to 45% of total) and in the supernatant fluids (45 to 64% of total). The washed nuclear fractions contained 3 to 11% of the total vitamin $B_6$ present. The unwashed small granules or microsomes contained 4 to 8% of the total vitamin $B_6$; in the case of normal rat liver one washing reduced the level to about 1½%.

The ingestion of the azo dye reduced the levels of the vitamin in the large granule and supernatant fluid fractions of the livers in each species. The decrease in protein content of the large granules paralleled the decrease in vitamin $B_6$ content. The large granule and supernatant fluid fractions of the liver tumors contained even less vitamin $B_6$ than was found in the corresponding fractions from the livers of rats fed the dye and the ratio of the vitamin to protein was also much lower.

[13] Wynne, A. M., in *Ann. Rev. of Biochem.*, 1946, **15**, 58.

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## 17262. Healing of Tuberculous Pulmonary Cavities by Means of Skin Grafts.

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Giant tuberculous pulmonary cavities continue to present one of the most complex problems in the surgical treatment of pulmonary tuberculosis. Various types of collapse therapy have resulted in a high percentage of failures. Intracavitary drainage in tension or blocked cavities, combined with collapse therapy, has greatly increased the number of good results.

Intracavitary drainage without collapse therapy provides relief from toxicity but is of little value in cavity closure. In advanced pulmonary disease with multiple cavitation, collapse therapy is contraindicated.

We wish to present a somewhat different point of view. Let us consider that a tuberculous pulmonary cavity is a chronic lung abscess: The lung surrounding a chronic nonspecific lung abscess shows little disease. When such an abscess is drained, the surrounding lung tissues fill in the defect occupied by the abscess. In a tuberculous lung abscess, the surrounding lung tissues are usually diseased and cannot expand to fill in the space occupied by the abscess cavity. Drainage is of cleansing value in pulmonary tuberculous cavities, as in other types of pulmonary abscesses, but cavity obliteration does not occur without col-

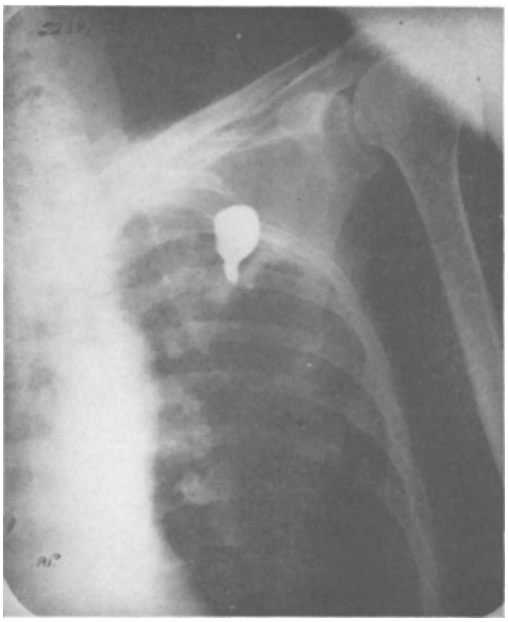

FIG. 1.
Five months following cavernostomy and repeated skin grafts the lipiodol is lying on the skin lining the former cavity which is continuous with the skin of the chest wall and does not enter the bronchial tree.

lapse therapy.

At the Grace Dart Home Hospital large cavities have been treated by intracavitary drainage. The residual cavity space has been opened into by cavernostomy, carried out through the sinus tract of the drainage tube. The exposed cavity walls and floor have been covered with split-thickness skin grafts or pinch grafts. Gradually the boundaries of the cavity are lined with skin which grows out to meet the skin on the surface of the chest wall. The cavity thus becomes obliterated, leaving a defect in the chest wall.

The first case selected was a 52-year-old male with far-advanced bilateral pulmonary

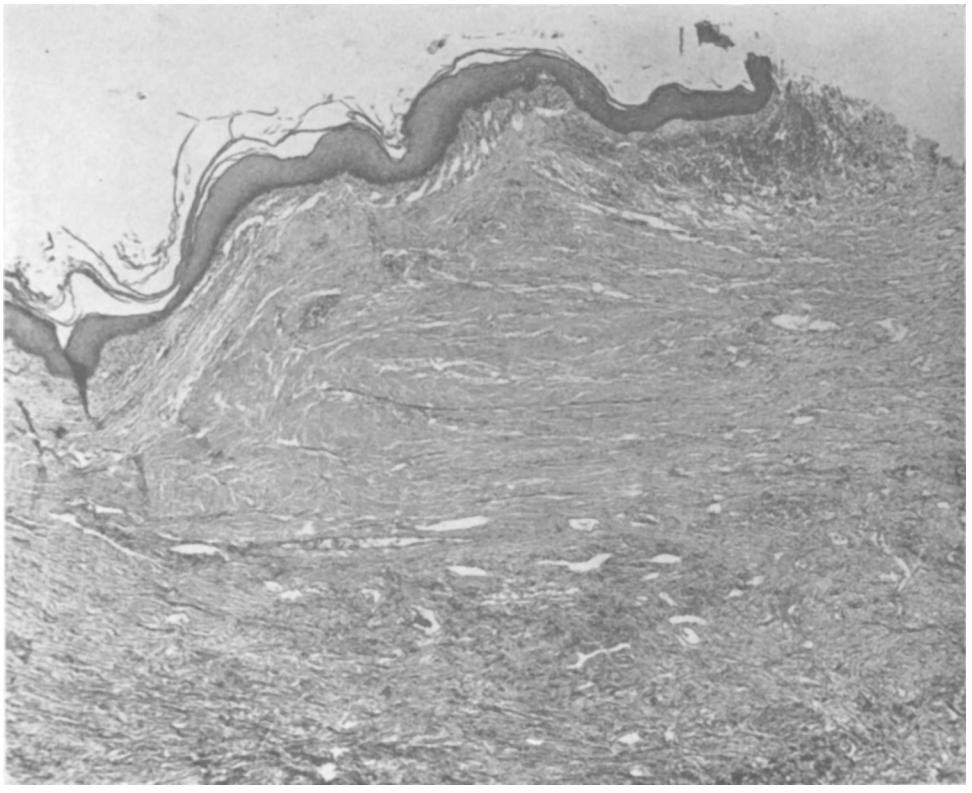

FIG. 2.
Section taken through wall of skin grafted cavity. Note lung covered by a layer of skin with proliferation of fibrous tissue and compressed pulmonary alveoli.

disease with giant bilateral apical cavities. On February 4, 1948, an anterior stage thoracoplasty was done on the left side. On May 28, 1948, intracavitary drainage was established in the left apical cavity. The poor general condition of the patient prevented further collapse therapy. On September 22, 1948, the left apical cavity was entered into through the drainage tract. Cavernostomy was done, and the depths of the cavity were lined with a split-thickness skin graft. This was repeated on October 20, 1948, November 17, 1948 and on February 9, 1949; on the last occasion pinch grafts were used. In all instances mentioned, at least a 75% "take" of the graft was obtained. The cavity on the left side diminished and gradually was covered with skin; the bronchial openings, which at first were large, gradually closed. At autopsy, May 8, 1949, there was no evidence of an open bronchus leading to the skin surface. The surface skin on the chest wall showed a depression about 1½″ in diameter which entered into the lung. The cavity seemed to be completely covered by skin and was entirely exteriorized. The cavity on the right side remained unchanged. Two other cases have been treated. In both skin grafting has been successful and the cavities seem to be healing.

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## 17263. Effect of Alloxan in Rabbits with Temporary Occlusion of the Arteries to the Pancreas.*

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Numerous investigations have shown that the injection of alloxan into various animals causes destruction of the pancreatic islets of Langerhans with the production of diabetes. After the injection of a diabetogenic dose of alloxan intravenously into a rabbit hyperglycemia appears which reaches its peak within 2 hours. This in turn is followed in 2 to 9 hours by profound hypoglycemia frequently with convulsions. Finally within 24 to 48 hours diabetes develops and although most agree that the diabetes results from the destruction of the islets of Langerhans, the initial hyperglycemic phase and especially the hypoglycemic phase have caused much speculation and disagreement.

Although diabetes usually is considered to result from an action of the drug on the pancreatic islets, Jimenez-Diaz[1] reported that clamping the renal blood vessels immediately before the injection of alloxan prevents the development of diabetes. Others[2] have not been able to confirm this.

In the present investigation alloxan was injected into rabbits with the blood supply to the pancreas occluded temporarily in order (1) to confirm that alloxan diabetes is pancreatic in origin, and (2) to determine if the hypoglycemia is dependent upon alloxan producing histological changes in the islets of Langerhans.

*Materials and Methods.* Male albino rabbits weighing 2.0 to 2.7 kg were used and each was fasted for 16 to 18 hours before an experiment. Anesthesia was obtained by 18 to 20 mg/kg of 5% sodium pentobarbital intravenously supplemented by ether given by open mask as required. A transverse incision was made in the left upper quadrant of the abdomen extending quite far laterally. The celiac axis and the superior mesenteric artery were exposed and clamped close to the abdominal

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1 Jimenez-Diaz, G., Grande-Covian, F., and DeOya, J. C., *Nature*, 1946, **158**, 589.

2 Martinez, C., Gitter, S., and Covian, M. R., *Rev. Soc. argent de biol.*, 1947, **223**, 81.