pulp of the spleen. The method used by Payr in transplanting thyroid tissue was followed. In four animals (2 dogs and 2 cats) pancreatic cells were demonstrable in the spleen. The interval between the operation and the death of the animal varied from 18 hours to 13 days. In five animals (all dogs) no pancreatic remains were found. Autolysis was rapid. In one experiment all the pancreatic cells had disappeared 21 hours after the transplantation. The bit of tissue found 13 days after the transplant measured only 1 mm. by 0.1 mm. It consisted of normal appearing acini surrounded by connective tissue. No islands of Langerhans were demonstrable.

In one dog a large pancreatic graft was placed in the spleen with the blood supply preserved by means of a mesenteric stalk. Three weeks later the original blood supply was cut off and all the pancreatic tissue except the graft extirpated. Diabetes did not develop, but the tolerance for glucose fell within a few weeks to a low point. At the autopsy 187 days after the second operation a large abscess was found in the lower part of the spleen. Projecting into the spleen from the wall of the abscess was a cone-shaped mass of fibrous tissue. In this were the remains of the pancreatic transplant measuring less than 1 cm. in length. The pancreatic tissue consisted of acini separated by connective tissue. In some of the cells, masses of zymogen granules were present. These were no demonstrable islands of Langerhans. This experiment proves that pancreatic tissue implanted in the spleen and separated from its original vascular and nervous connections can live and functionate for months.

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The production of reversed cardiac mechanism in the dog.

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In a series of experiments, which were published with Kessel and Mason,[1] on the excised perfused hearts of dogs, we were able to show that "excision of the sinus node results in an immedate

[1] *Heart*, June 15, 1912, iii, 311.

cessation of the contractions of the whole heart," that "after excision of the sinus node and the ensuing stoppage, the rate of the whole heart falls and does not again reach the original rate," and that "the function of the secondary pacemaker devolves on no special portion of the heart." The results of experiments performed by others and differing from ours were mentioned at that time. I have continued the experiments on dogs, anesthetized with ether, the hearts of which were left *in situ* and normally nourished. After sufficient controls were registered electrocardiographically, the sinus node was clamped with a specially constructed T clamp, 5 cm. long and 1 mm. wide. In the perfused hearts the rate fell 10 to 70 beats, usually 30 to 50 beats, after the node was excised. In the present series the fall in rate was 10 to 90, usually about 50. Stoppage, which is a usual phenomenon in excised hearts, occurred also in these, but very rarely. The site of the secondary pacemaker varied in these, as it did in the perfused hearts. Photographs of three hearts showing the areas of the auricular surfaces which had been included in the jaws of the clamp, and a series of curves from each of the corresponding experiments and of one in addition were demonstrated. The relation of the disturbed function of the heart to the area clamped will be investigated histologically and reported in detail later. The curves indicate that in each of these cases, there was a dislocation of the pacemaking function from the site responsible for it, and that, after clamping had taken place, this function devolved upon the ventricles, the junctional tissues or another portion of the auricle. In one of the experiments, first the ventricles (more probably the junctional tissues) and later the auricles set the pace. One of the experiments yielded curves which bore a close resemblance to those obtained from a patient by Williams and James,[1] in which the wave representing auricular contraction was found between the R and T waves, and was inverted. It is demonstrated by the experiments that after clamping an area along the sulcus terminalis, a site other than the normal pacemaker assumes the pacemaking function and that a "reversal of the cardiac mechanism" takes place.

[1] PROC. SOC. EXP. BIOL. AND MED., Oct. 16, 1912, X, 13.