and typical changes of the rat hypophysis regularly follow thyroidectomy and hyperthyroidism. Our homoplastic thyroid grafts maintained the hypophysis of the recipient in a normal condition. In the one thyroidectomized rat in which the graft failed to take, the typical thyroidectomy changes occurred in the anterior hypophysis. One rat, furthermore, showed hyperthyroid changes in the hypophysis, and an examination of the thyroid graft revealed an exceptionally large gland in a hyperactive state.

Those rats which were not sacrificed at one month for cytological examination have now reached an age of from 3 to 6 months. Their appearance and reactions are completely normal. They show no indications of any thyroid disturbances. They will be under observation in experiments now in progress, at the end of which a final report on the entire thyroid transplant series will be made.

From these series of auto- and homo-thyroid grafts, it is obvious that in our Long-Evans colony of rats the thyroid of an infantile animal may be grafted into a thyroidectomized rat of any age with the expectancy of 100% viable and functional grafts, which persist indefinitely.

The next stages in the experiment will be attempts to transplant the infantile rat thyroid into rats of an unrelated strain and if these attempts are successful, heterografts will then be attempted.

## 8581 C

## Multi-plane Chest Electrocardiography.

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Recent investigations in the field of electrocardiography have been directed towards methods for the more accurate detection and localization of myocardial lesions. The 3 conventional leads frequently fail to detect alterations consequent to a lesion and they have recently been supplemented by chest and precordial leads introduced by Wolferth and Wood,<sup>1, 2, 3, 4, 5</sup> Hoffman and Delong,<sup>6</sup>

<sup>1</sup> Wolferth, C. C., and Wood, F. C., Am. J. M. Sc., 1932, 183, 30.

<sup>2</sup> Wolferth, C. C., and Wood, F. C., M. Clin. North America, 1932, 16, 161.

<sup>3</sup> Wood, F. C., and Wolferth, C. C., J. Clin. Invest., 1932, 11, 815.

<sup>4</sup> Wood, F. C., and Wolferth, C. C., Arch. Int. Med., 1933, 51, 771.

<sup>&</sup>lt;sup>5</sup> Wood, F. C., Bellet, S., McMillan, T. M., and Wolferth, C. C., Arch. Int. Med., 1933, 52, 752.

<sup>6</sup> Hoffman, A. M., and Delong, E., Arch. Int. Med., 1933, 51, 947.

Wilson and his co-workers<sup>7, 8, 9, 10</sup> and others. However cases of acute coronary occlusion which fail to produce electrocardiographic alterations even in these leads are not an uncommon occurrence. In this communication I will discuss a method of chest lead application which I previously reported with the study of a series of normal cases.<sup>11</sup> My method entails the use of long linear electrodes placed parallel to and beyond the borders of the heart anteriorly and posteriorly. Various combinations of these electrodes are used in the hope of utilizing other recording planes.

The right and left arm electrode wires of the conventional leads are connected to the chest electrodes in such a manner that the current take-off is kept in the same relationship to the current direction within the heart as in lead I of the 3 standard leads. The right arm electrode wire is therefore always connected to the chest electrode which lies in closest relation to the tail of the arrow while the left arm electrode wire is connected to the chest electrode which lies in closest relation to the head of the arrow representing the heart action current direction. The size of the heart and the position of the cardiac borders are determined. The chest electrodes are placed about 2 or 3 cm, beyond the estimated position of the borders of the heart.

Flexible electrodes about 1/2-inch wide and 4 or more inches in length, depending on the size of the heart, are used. Coils of copper wire or suitable German silver plates are used. The electrodes should be covered with gauze soaked in warm saline solution or electrode jelly may be used. The electrodes are held firmly in place by an assistant whose hands are insulated with rubber gloves, or by a spring clamp or rubber bands.

TABLE I.		
Leads	Right Arm Electrode Applied to Position	
m R'R	R'	R
$\mathbf{L}'\mathbf{L}$	$\mathbf{L}'$	${f L}$
S'I	S'	I
R'L	$\mathbf{R'}$	${f L}$
L'R	$\mathbf{L}'$	${f R}$
hoRL	R	L

<sup>7</sup> Wilson, F. N., Am. Heart J., 1929-30, 5, 599.

<sup>8</sup> Wilson, F. N., Macleod, A. G., and Barker, P. S., Am. Heart J., 1931, 7, 207.

<sup>9</sup> Wilson, F. N., MacLeod, A. G., and Barker, P. S., Am. Heart J., 1932, 7, 313.

<sup>10</sup> Wilson, F. N., MacLeod, A. G., Barker, P. S., Johnston, F. D., and Klostermeyer, L. L., Heart, 1933, 16, 155.

<sup>11</sup> Weinstein, J., Ann. Int. Med., 1934, 7, 1503.

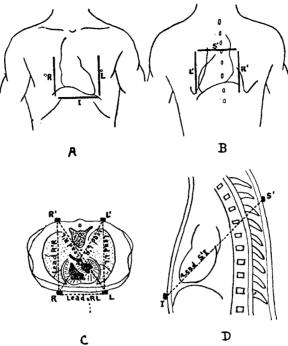


Fig. 1.

Illustration of the positions for the application of the electrodes. Their length and location are dependent on the size of the heart.

A. On the anterior chest wall: R is a vertical line beyond the right border of the heart extending from about the level of the right chondrosternal junction downward. L is a vertical line beyond the left border of the heart at the same level as R. I is a horizontal line beyond the inferior border of the heart extending from the lower end of R to the lower end of L.

B. On the posterior chest wall: Positions R' and L' are determined by the extension of positions R and L directly posteriorly to the posterior chest wall. S' is a horizontal line extending from the upper end of R' to the upper end of L'.

C and D. Cross section and lateral views of the chest illustrating the positions for the application of the electrodes and the combinations used for the multiplane chest leads.

The electrodes are applied to the positions on the chest wall and connected in the various combinations as illustrated in Figure 1.

A study of 100 normal cases gave strikingly uniform tracings in all the multi-plane chest leads. Fig. 2 illustrates a normal case taken from this group. The standard leads, lead IV and the multi-plane chest leads are shown. The time element remains the same in all the leads. The conformation of the waves in the multi-plane chest leads resemble in most respects the waves in the standard leads. The T wave is upright, the only exceptions being in leads R'R and L'R where normally the T wave may be upright, diphasic or even inverted depending on the normal physiologic rotation of the heart as with an elevation of the diaphragm. The QRS com-

plex may vary in direction but a prominent Q wave is not present normally in any of the multi-plane chest leads. The RT transition is under all circumstances normally on the iso-electric line.

The following cases of acute coronary occlusion, although showing comparatively normal tracings in the 3 standard leads and on lead IV, presented definite alterations in the multi-plane chest leads.

Case 97 (Fig. 3), a female 41 years of age, gave a history of an essential hypertension of many years duration. For the past year she had suffered with palpitation, dyspnoea and precordial pain on exertion requiring rest for relief. Ten days before admission to the hospital, the patient suffered a severe attack of precordial pain lasting 20 hours and requiring repeated hypodermic injections of morphine. She ran a subsequent temperature up to 102°F. She had a rapid red cell sedimentation rate and a leukocytosis of 15,800 with 84% polymorphonuclear leukocytes. Physical examination revealed a blood pressure 168 systolic and 110 diastolic, slight cyanosis of lips. The heart was enlarged to the left, the aorta percussed widened. The heart sounds were of fair quality, A<sub>2</sub> was accentuated, no murmurs were heard. The liver was not enlarged and there were no dependent edemas.

The standard leads and lead IV showed relatively normal tracings. The multi-plane chest leads showed an elevation of the RT segments and coronary type T waves in leads L'L, S'I and R'L, lead RL showed an elevation of the RT segment with an inversion of the T wave, leads L'L, S'I, R'L and RL showed prominent Q waves.

The patient died 7 days after this examination. Post mortem examination of the heart revealed sclerosis of the coronary arteries and an occlusion of an anomalous branch of the left coronary artery with a myomalacia of the postero-lateral wall of the left ventricle.

Case 143 (Fig. 4), a male 44 years of age, who gave a history of a hypertension of many years duration and a story of an acute coronary closure one year ago, 3 days before this examination suffered a similar attack. Physical examination revealed a slight cyanosis of the lips but no evidence of venous stasis. The heart was percussed markedly out to the left but the apex impulse was felt well within the nipple line. The radial pulses were weak. The liver was not enlarged and there was no peripheral edema. The blood pressure was 120 systolic and 84 diastolic. The temperature was 101°F. The white blood count was 18,500 with 90% polymorphonuclear leukocytes. The sedimentation rate was rapid.

The 3 standard leads and lead IV were comparatively normal with the exception of a slight slurring of the main deflections and the suggestion of a low voltage. The multi-plane chest leads revealed 3 mm. elevation of the RT segments and coronary type T waves in leads L'L, R'L and RL, leads R'R and L'R showed slight alterations in the RT transition, lead S'I remained normal.

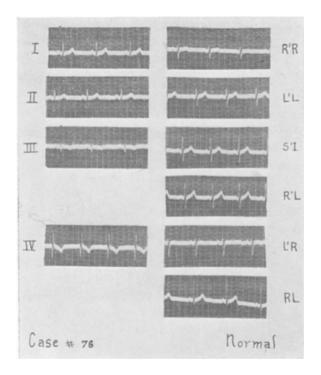


Fig. 2.

The patient died 4 days after the onset of this attack. Post mortem examination of the heart revealed a sclerosis of the left main coronary artery. There was an eurysmal dilatation involving the postero-lateral wall of the left ventricle extending down but not involving the apex. There was an area of myomalacia involving this area and the adjacent musculature extending to and involving the posterior part of the septum.

In the application of this method in experimentally produced myocardial lesions in cats, <sup>12</sup> a review of the records obtained in 25 experiments showed RT segment alterations in the standard leads in

<sup>12</sup> Abramson, D. I., and Weinstein, J., Am. J. Physiol., 1935, 3, 1382.

only about 50% of the experiments, and lead IV showed similar changes in 66% of the series. There were instances when the standard leads showed changes while lead IV remained normal and vice versa. But two or more of the multi-plane chest leads demonstrated the lesion in all the experiments. It was further observed that when the lesion was produced on the left ventricle, anterior or posterior surface, there resulted an RT elevation in leads R'L and RL while lead L'R remained normal. When the lesion was produced on the right ventricle, anterior or posterior surface, there resulted an RT segment elevation in lead L'R and a depression in lead RL while lead R'L remained normal.

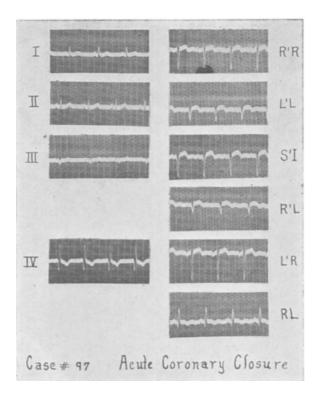


Fig. 3.

While the question of localization cannot as a rule be applied clinically because the lesion most often is not confined to one ventricle or one surface, however, the 2 clinical cases here presented conform to this classification on localization.

The multi-plane chest lead method was used in a series of 62

cases where the clinical picture suggested a coronary occlusion although the 3 standard leads and lead IV remained normal. While 12 cases of these series showed definite changes in the multi-plane chest leads, the 2 cases here discussed were the only ones where post mortem examinations were possible.

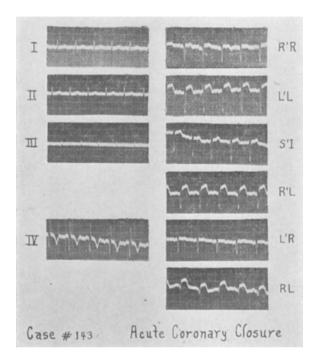


Fig. 4.

Summary. A system of multi-plane chest leads is presented. In this method long electrodes, placed parallel to and beyond the borders of the heart anteriorly and posteriorly, are connected in various combinations. Two cases of acute coronary occlusion with autopsy investigations are presented. In both cases, while the 3 standard leads and lead IV showed relatively normal tracings, the changes in the multi-plane chest leads indicated profound myocardial damage.