ysis 3. All samples were taken in the morning before breakfast. Every subject examined had previously been on an ordinary mixed diet and approximately one-third of the epileptics were on phenobarbital therapy. Samples were obtained in several instances within one hour after a seizure. The shortest time interval between the lumbar puncture and a subsequent *grand mal* attack was 1.2 hours.

Figure 1 gives the results. The mean values for the pH (7.327) and the CO_2 content (60.0 vol. %) of the epileptic patients are practically identical with those found for the non-epileptic control group (pH 7.328, CO_2 60.6 vol. %). In a few non-epileptic cases in which the comparison was made, fluid taken from the lateral ventricles did not differ significantly, as regards these factors, from that obtained simultaneously by lumbar puncture. In but one epileptic and one non-epileptic was the pH reading below 7.30. This could readily be accounted for in the case of the former as a result of a severe convulsion which occurred a few minutes before the sample was taken. The pH was 7.25 and the CO_2 content 66.5 volumes percent, showing the low pH to be due in part to an accumulation of CO_2. The only low pH value (7.28) for the control group was found in the case of a mongolian idiot, who struggled violently before the lumbar puncture could be made. The maximum pH for the epileptics was 7.39, which occurred 4 times only and that for the non-epileptics 7.37, a difference well within the range of error in measurement. No values were found which would indicate the slightest tendency toward an "alkaline drift" in the spinal fluid of the epileptics. In contrast with the findings of Geyelin, Bigwood and Wheatley[10] for blood, the range of variation in pH values for spinal fluid was remarkably narrow in our series.

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The Electrocardiogram in Coronary Thrombosis.

FRANK N. WILSON, PAUL S. BARKER, A. GARRARD MACLEOD AND L. L. KLOSTERMYER.

From the Department of Internal Medicine, University of Michigan Medical School.

The tentative conclusions below are based upon a study of the electrocardiograms in 56 cases of coronary thrombosis, in 17 of which a post-mortem examination of the heart was made.

The electrocardiogram is of great value in the diagnosis of this condition, particularly when it is possible to obtain a series of curves.

The most important feature of the abnormal *T*-deflections (described by Smith,[1] Pardee[2] and others) that occur in cardiac infarction is the characteristic and progressive change in form which they undergo. These changes in the *T*-deflection are usually accompanied by changes in the form of the initial deflections *(QRS)* which have not been fully described although the large *Q*-waves that frequently occur in lead III have received considerable attention (W. J. Wilson,[3] Parkinson and Bedford,[4] Levine and Brown,[5] Pardee[6]). The curves may be divided into 2 groups:

(a) In typical curves of the first group the initial ventricular deflections of lead I are usually of small or medium amplitude, and there is a broad and conspicuous *Q*-deflection in this lead. In leads II and III the first initial deflection is upward and is followed by an S-wave, often of large amplitude. These changes in *QRS* occur early and commonly outlast the *T*-wave changes that accompany them. In the early stages of cardiac infarction there is, in curves belonging to this group, an elevation of the *S-T* segment in lead I and a depression of this segment in lead III. This partial fusion of *R* and *T* in the first lead and of *S* and *T* in the third often gives rise to curves which resemble in general outline the monophasic responses yielded by injured heart muscle, as Clarke and Smith[7] have pointed out. In the later stages there is a progressive inversion of *T* of lead I; the final portion of *T* is the first to become inverted and the inversion progresses backward toward *QRS* so that *T* finally becomes a deep V-shaped depression. Changes of the opposite type occur in lead III in which *T* is converted into a deflection which resembles an inverted V in form. In a small number of cases in which the standard leads yielded curves of this type serial precordial leads[8] were also employed. The electrocardiograms so obtained are unlike those that we have obtained by a similar method of leading in any other condition. The chief initial deflection of these curves, which is upward and of large amplitude, is not preceded by a downward deflection as is ordinarily the case. The *T*-deflection of these special

1 Smith, F. M., *Arch. Int. Med.*, 1918, **22**, 8.
2 Pardee, H. E. B., *Arch. Int. Med.*, 1920, **26**, 244.
3 Wilson, W. J., *Ann. Clin. Med.*, 1926-27, **5**, 238.
4 Parkinson, John, and Bedford, D. Evan, *Heart*, 1927-29, **14**, 195.
5 Levine, S. A., and Brown, C. L., *Medicine*, 1929, **8**, 245.
6 Pardee, H. E. B., *Arch. Int. Med.*, 1930, **46**, 470.
7 Clarke, N. E., and Smith, F. J., *J. Lab. and Clin. Med.*, 1926, **11**, 1071.
8 Wilson, F. N., Macleod, A. G., and Barker, P. S., *Am. Heart J.*, 1932, **7**, 305.

leads is strongly positive when T of standard lead I is sharply negative. When T of lead I is not inverted, however, the T-deflections of precordial leads may be negative.

(b) In typical curves of the second group large Q-waves occur in lead III and usually in lead II as well; in lead III Q is often the largest of the initial deflections. As a rule no Q wave is present in lead I. The T-wave changes that occur in lead I are similar to those that occur in lead III in the first group and vice versa. In the late stages of cardiac thrombosis T is sharply inverted in leads II and III. In the cases in which precordial leads have been taken the curves obtained have been less distinctive than those described in the preceding paragraph. The first initial deflection has always been downward and it has usually been of large amplitude, particularly in those leads in which the exploring electrode was placed near the cardiac apex. The T-waves of the special leads were strongly inverted in cases in which T was sharply inverted in lead III; when there was an elevation of the S-T segment in lead III this occurred in the precordial leads also.

In the autopsied cases curves of the type described under (a) have been associated, with one possible exception, with infarcts located on the anterior or antero-lateral surface of the left ventricle. Curves of the type described under (b) have been associated with infarcts located on the posterior wall of the left ventricle. These findings are in agreement with those of Barnes and Whitten.[9] Many of the patients studied displayed curves which could be easily classified as belonging to one group or the other although they did not show all of the features described as typical of the group to which they obviously belonged. In other instances the electrocardiogram was definitely abnormal but showed no peculiarities that would suggest cardiac infarction.

Changes in heart rhythm, particularly the occurrence of partial or complete A-V block, paroxysmal tachycardia of ventricular origin, and the sudden development of intraventricular block, or of very small ventricular complexes are also common in coronary thrombosis and are of diagnostic value when the other data available are suggestive.

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[9] Barnes, A. R., and Whitten, M. B., *Am. Heart J.*, 1929, **5**, 142.