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K. M. ALLY, M.B.B.S., and W. B. GREENOUGH, III, M. D.
Pakistan-SEATO Cholera Research Laboratory, Dacca, East Pakistan

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Pakistan-SEATO Cholera Research Laboratory, Dacca, East Pakistan.

In the patient stricken with cholera massive loss of watery stool results in such severe dehydration that circulatory collapse often results within a few hours of onset. At the same time profound electrolyte depletion and acidosis develop. The effects of these changes on the electrocardiogram would appear to be of considerable interest, but few carefully collected observations have appeared in the literature to date. Striking serial electro-cardiographical changes were observed in cholera patients studied before and after electrolyte and fluid replacement therapy at the Pakistan SEATO Cholera Research Laboratory in 1962 and 1963. Therefore a prospective study was undertaken in which electro-cardiograms (ECG's) were obtained from patients with shock due to cholera and noncholera diarrhea, as well as a variety of hypotensive states resulting from nondiarrheal illnesses.

MATERIALS AND METHODS.

Electrocardigrams were obtained from 30 consecutive patients entering the Cholera Research Laboratory in shock due to acute diarrheal illnesses between February and June 1964. Bacteriological diagnosis of infection with **V. cholerae** was established and supported by rises in agglutinating antibody titer in 19 cases. In two patients noncholera vibrios were obtained on stool cultures; in one of these an antibody rise against the homologous noncholera vibrio organism was demonstrated. In the remaining nine no pathogens were obtained on repeated stool cultures and rises in agglutina-

ting titer against V. **cholerae** did not occur. None of these patients had any previous history of cardiac or pulmonary disease. In 25 out of the 30 cases adequate serial ECG records for analysis were obtained. Initial tracings were taken before any therapy. The methods used in the treatment of cholera and other dehydrating diarrheal illnesses have been described previously (1).

In addition ECGS were obtained at Dacca Medical College Hospital and Mitford Medical College Hospital on 20 patients in shock due to a variety of causes (listed in Table I under Haemorrhage and Miscellaneous Group). Tracings were taken as early as possible and whenever possible blood samples were obtained for estimations of plasma protein concentration, hematocrit, and serum electrolytes.

The tracings were done with a Cambridge Trans-Scribe ECG recording instrument. All patients were observed personally by one of us. The definition of shock employed in the study was a systolic blood pressure of 50 mm. Hg. or less by palpation over the radial and /or brachial arteries.

Electrocardiographic tracings collected by the Nutrition Survey of Pakistan on 171 East Pakistani healthy subjects were kindly made available to us by Dr. Kamaluddin Ahmed and were reviewed with special reference to the height of P waves.

RESULTS.

The electrocardiographic findings in severe dehydrating diarrheal disease may be divided into two phases: (1) the phase of collapse before any treatment has been given, and (2) the phase of replacement when fluid and electrolyte losses are being restored. During the second phase the composition and the amount of fluid given alter the ECG results and must be exactly specified to allow any interpretation of the observations. We will consider here only the changes seen during shock and immediately after recovery from shock. Most of the patients received a solution containing 5 gm NaCl,4 gm $NaHCO_3$, and 1 gm KCl in a liter of distilled water to correct both their dehydration and electrolyte deficiences (1). The changes seen during vascular collapse are illustrated in figure 1.

There were three major departures from normal (Figure 1) :

1) gaint P waves having a peculiar sharp and peaked character.
2) absolute or relative shift of the electrical axis towards the right.
3) elevation of the T waves.

Identical changes were seen in shock due both to cholera and to diarrhea caused by other organisms (table II). A detailed description of the clinical, biochemical, and bacteriological features of these cases has been reported (2).

An attempt was made to correlate the height of the P wave with :

1) the degree of dehydration (as measured by the difference between acute and convalescent plasma protein concentration) and

2) the degree of acidosis (as reflected by the initial CO_2 content of plasma).

Scattergraphs of these comparisons indicate no significant correlation between the electrocardiographic P wave changes and either hemoconcentration or acidosis, the coefficients of correlation for plasma protein and CO_2 being 0.346 and 0.009 respectively. Furthermore, during the phase of rehydration, often after the administration of only a few ccs of rapidly infused fluid, whether it be saline, or a solution containing lactate or bicarbonate and potassium, the P wave change disappeared, as did the T wave elevations.

The change in axis towards normal was not quite as rapid as the disappearance of gaint P waves, and required more complete rehydration. Significant ST-segment depression was present in four of the patients with diarrheal disease, and also tended to disappear slowly during rehydration.

Similar electrocardiographic patterns were encountered infrequently in cases of shock due to haemorrhage and other causes (table II). The P wave elevation was never as remarkable as in the diarrheal group. The high T wave seen in the diarrheal group was not seen in any of this group. ST-segment depressions were present in a minority of the patients in both groups. Unfortunately no axis shift observations could be made as serial ECGs were not available. All these cases had received variable amounts of intravenous infusions during the preceding 24 hour period and earlier. The ECG most closely approaching the change seen in acute diarrhea may be seen in figure 2, which was obtained from a case of eclampsia with sudden fatal vascular collapse during the post-abortion period. This would appear to be a case of acute Cor-Pulmonale, possibly related to pulmonary embolism.

The amplitude of P waves in electrocardiograms of 171 normal East Pakistani subjects (collected by the Nutrition Survey of East Pakistan) ranged from 1.1 to 1.7 mm.

DISCUSSION.

The electrocardiographic changes during vascular collapse due to cholera and other diarrheal diseases are of particular interest since there are few other clinical situations in which physiologic alterations engendered by the profuse loss of water and electrolytes over such a brief period of time can be observed. Thus basic information concerning electrophysiology of the heart might be available due to this remarkable experiment imposed by nature. The findings in the present study confirm and extend those reported by Bien and Tung in cholera patients observed in Peiping in 1932 (3).

It would appear that several major derangements present in cholera might have electrocardiographic correlates. One of these is myocardial anoxia due to low blood flow in the coronary sys-

tem. It is now known that extraordinarily low cardiac outputs are present in cases of only moderate severity (4). Although the oxygenation of blood remains normal reduced flow may have resulted in ischemia. The changes observed in the ST-segments of some tracings were quite typical of ischemia and are perhaps best attributed to this proces.

The changes in the P and T waves and the electrical axis are more complex and at this time cannot be attributed with assurance to any single physiologic change. Similar changes in P waves have been reported in pulmonary hypertension, in alkalosis produced by the intravenous administration of sodium bicarbonate or sodium hydroxide, after the administration of adrenaline, and in cerebrovascular accidents (5, 6, 7).

It is possible at the present time to say the ECG changes in shock due to diarrheal disease are not a consequence of pulmonary hypertension*, hemoconcentration, or acidosis. We regard them as probably due to change in position of the heart associated with an extraordinarily low atrial and ventricular volume and consequent low cardiac output. We would therefore expect that similar changes would be seen in situations where similarly low cardiac volumes and outputs are encountered, such as terminal hemorrhagic shock, vena caval occlusion, severe dehydration due to sweating or urinary losses as in diabetes or the diuresis after acute tubular necrosis. Careful electrocardiographic studies of such clinical situations would provide an opportunity to test this hypothesis.

* Hemodynamic studies during cardiac catheterization by Harvey et al (4) indicated that the presence of giant P waves could not be related to changes in the pulmonary arterial pressure.

SUMMARY.

Thirty cases in vascular collapse due to cholera and non-cholera diarrhea were analysed as to their pre-and post-hydration ECGs, Right axis deviation, relative or absolute, giant P waves, and elevated T waves occurred with remarkable frequency in these patients. The changes did not correlate with hemoconcentration, acidosis, or pulmonary pressure and may have been positional due to extraordinarily low volume obtaining in the atria and ventricles consequent upon massive intravascular volume deficit. These ECG changes were only rarely encountered in patients with vascular collapse due to causes other than severe dehydration.

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TABLE I.

Admission Diagnosis of Cases in Shock of Non-Diarrheal Etiology.

HAEMORRHAGE — 12		MISCELLANEOUS — 8	
Gun shot injury	1	Infection :	
Gastro-intestinal bleeding	1	Meningitis	1
Vaginal bleeding :		Enteric fever	1
abortion	3	Pneumonia	1
retained placenta	3	Extensive superficial burns	1
late pregnancy	3	Post-operative collapse following appendicectomy with sepsis	1
dead foetus	1	Intestinal obstruction	1
		Eclampsia with pulmonary embolism	1
		Debilitated condition due to carcinoma of the stomach	1

TABLE II.

The Electrocardiogram in Shock.

	Total cases	P wave 3 mm or more	Axis shift of 20° or more	T vave 8 mm or more	S-T segment depression >0.9mm	Plasma Proteing conc. 10 gm% or more	Co_2 15 mE/L or less
True Cholera	19	15/19	16/17	8/14	4/19	16/19	11/16
Non-Cholera diarrhea *	11	8/11	5/9	4/9	0/11	10/11	9/11
Shock due to miscellaneous causes	20	1/20	**	0/20	4/20	0/20	0/20

* "Non-vibrio Cholera" and cases with non-cholera vibrio on stool culture (3).

** Serial ECGs not available.

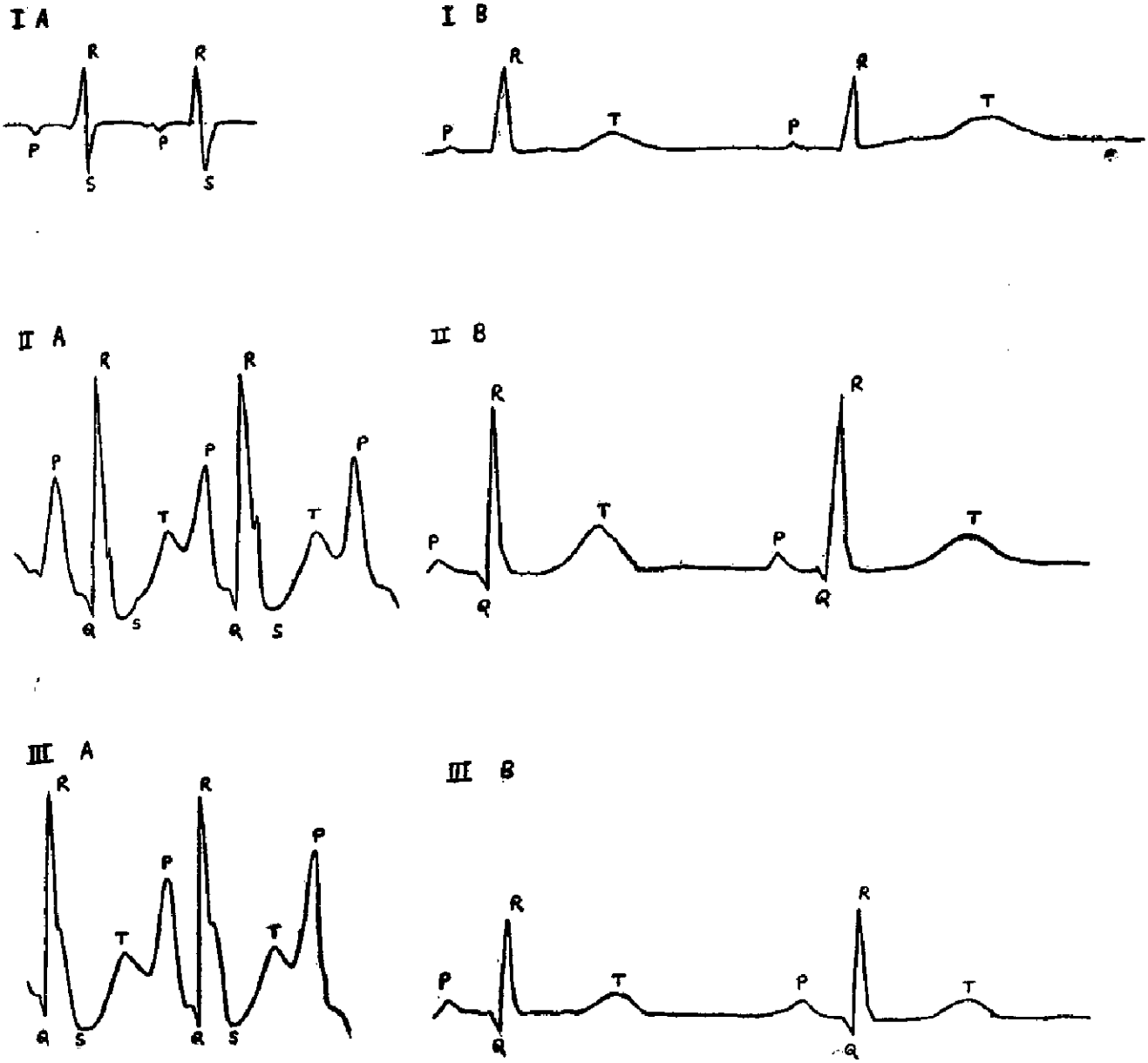

FIGURE 1.

Leads A I, II and III show the Electrocardiographic leads during shock
Leads B I, II and III show the Electrocardiographic leads during convalescence.

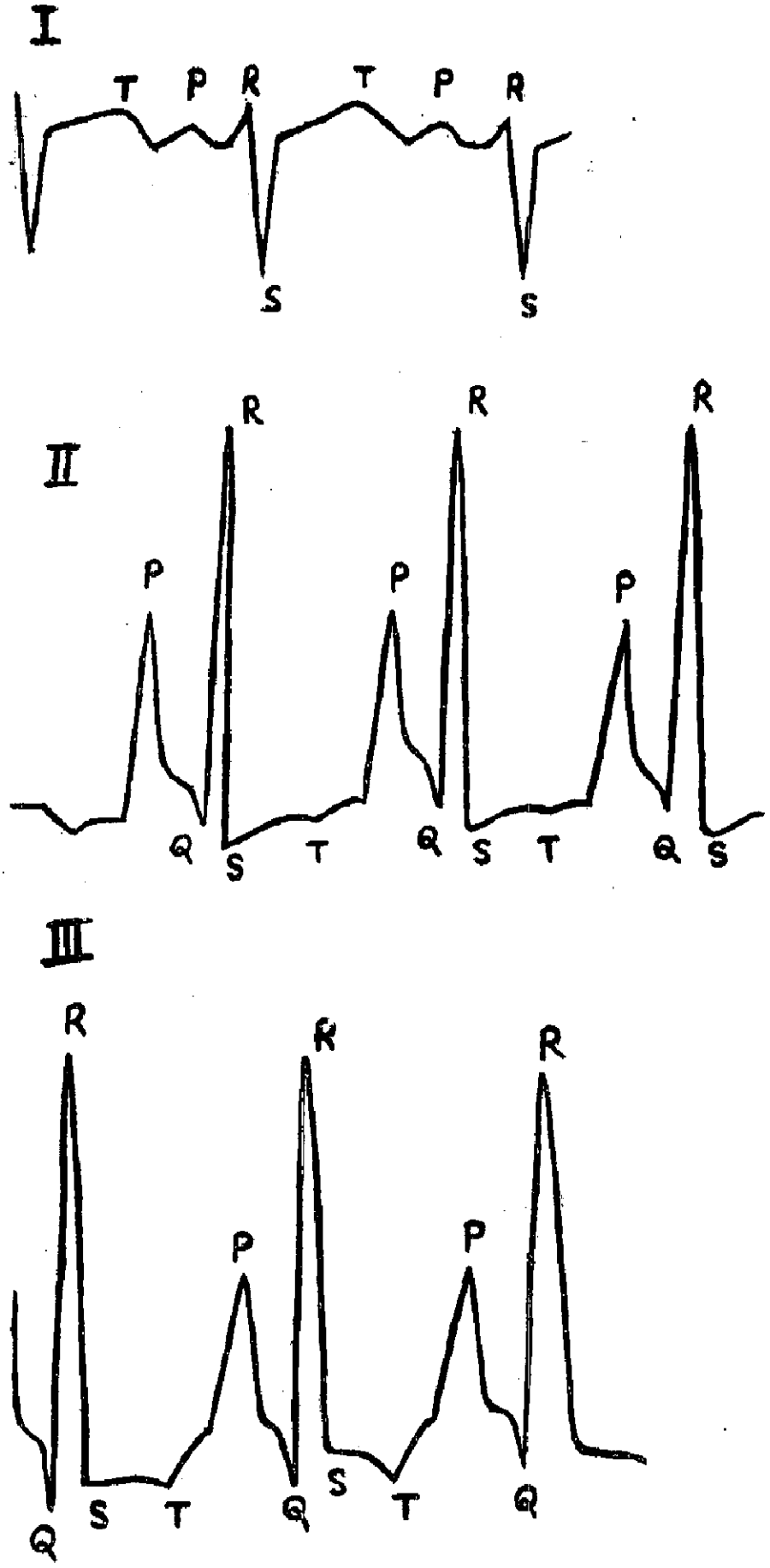

FIGURE 2.

Leads I, II and III show the Electrocardiographic lead in a case of shock during post-abortive period in Eclampsia.

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