FIG. 1.—Diagram to illustrate the formation of an ultrasonic trace.

FIG. 2.—"A" mode presentation of ultrasonic scan from anterior chest wall and anterior myocardium in a case of pericardial effusion. The upper trace is taken in a different phase of the cardiac cycle from the lower.

FIG. 3.—"B" mode presentation of same case as in Fig. 2 showing a bare area which increases in width during systole.

FIG. 4.—Normal echogram showing a bare area between the reflections from the anterior chest wall and from the myocardium. The bare area is of constant width.

FIG. 5.—Echogram made with the transducer over the apex of the heart showing an increase in bare area width during diastole and the anterior myocardium touching the chest wall in systole.

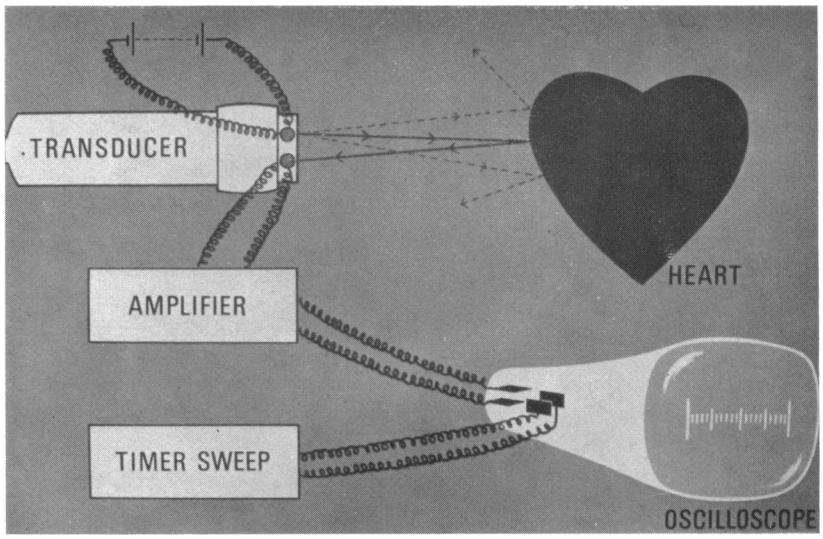

FIG. 1

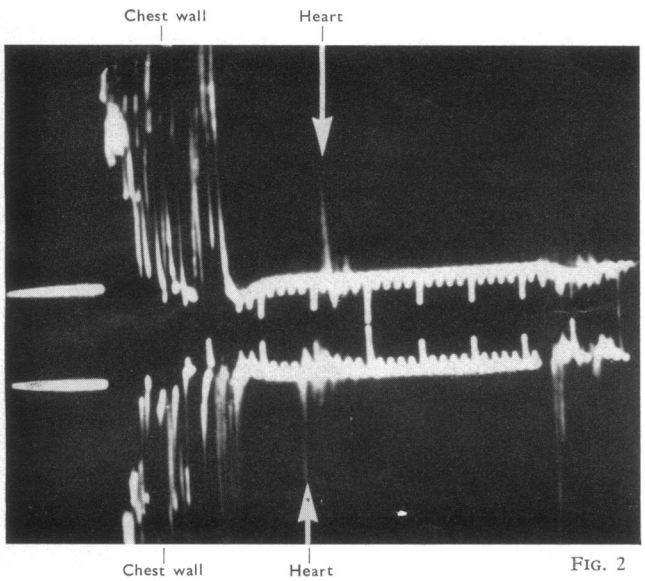

FIG. 2

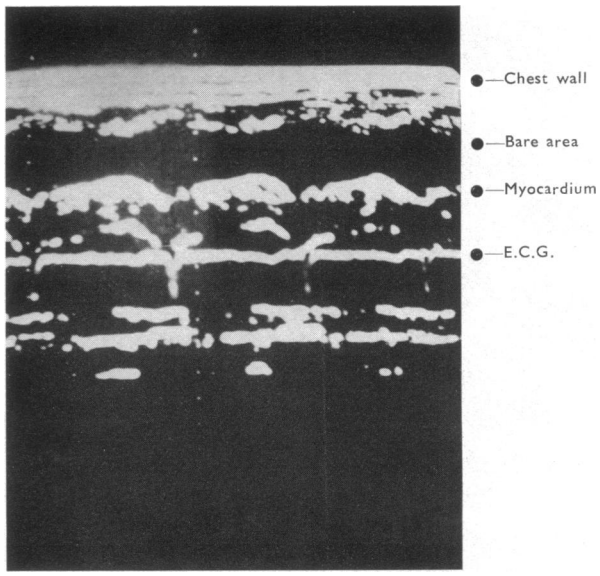

FIG. 3

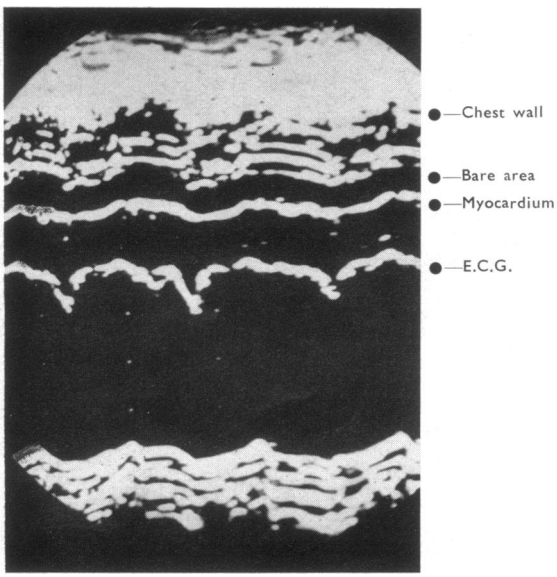

FIG. 4

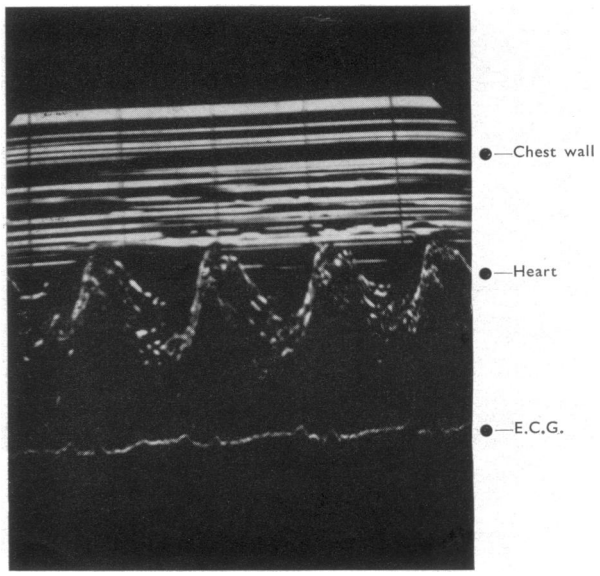

FIG. 5

Preliminary Communications

Diagnosis of Pericardial Effusion by Ultrasound

[WITH SPECIAL PLATE FACING PAGE 341]

Brit. med. J., 1968, **3**, 356–357

Summary: A reliable method of determining the presence of a pericardial effusion has been evolved. Ultrasonic waves reflected from the posterior part of the anterior chest wall and from the anterior surface of the anterior myocardium and pericardium produce echoes separated from each other by a bare area. This bare area varies in width and the variation has the same periodicity as the cardiac cycle.

INTRODUCTION

Pericardial effusion is often difficult to distinguish from cardiac dilatation in heart failure. The classical clinical signs—a paradoxical pulse, raised jugular venous pressure with a steep "y" descent, and a positive Kussmaul sign—may each be absent in effusion and present in heart failure. The most reliable clinical differentiation is made from auscultation. While a third heart sound is rarely heard in effusion, it is usually audible in heart failure, but exceptions occur even to this rule.

The shape and size of the heart on plain *x*-ray film are little better for the diagnosis of pericardial effusion (Besterman and Thomas, 1953). Only right atrial angiocardiography introduced by Williams and Steinberg (1949) is really helpful. However, this involves passing a cardiac catheter. *X*-ray examination after intravenous carbon dioxide described by Paul *et al.* (1957), though simpler, gives both false-negatives and false-positives. The gamma-ray scintillation camera has been used more recently (Rosenthall, 1966), but its value is not yet proved and the equipment is expensive and not generally available.

Ultrasound can be used to make an accurate diagnosis of pericardial fluid at the bedside and without disturbing the patient, so that paracentesis can be employed as a therapeutic measure and needling the patient as a diagnostic trial can be avoided.

METHOD

When an electrical current is applied to a transducer of barium titanate ultrasonic waves are generated. Such waves are reflected back to their source when they strike surfaces perpendicular to the direction of the wave. If the transducer is located on the precordium such surfaces include the anterior chest wall and the pericardium. When the reflected waves arrive back at the transducer a secondary electric current is produced. If both electrical impulses are fed through a suitable amplifier they can be made to deflect the beam of an oscilloscope (Special Plate, Fig. 1). This will produce an appearance such as is seen in Fig. 2 (Special Plate). The distance between the initial signal and the reflected signal is proportional to the depth of the reflecting surface.

The impulse from the Eskoline 20 ultrasonoscope machine made by Smith Kline Instruments, which has been used for all the observations in this paper, uses an ultrasound frequency of 2.25 megaHertz (MHz) with pulses of one microsecond duration and a pulse repetition rate of 1,000 per second. It is thus possible to obtain what is in effect a continuous recording of a moving surface. By modulating the amplitude of the oscilloscope deflections until they are reduced to dots, and by applying a time sweep to the base-line of the oscilloscope, movement can be demonstrated more satisfactorily than by the "A" mode display illustrated in Fig. 2 (Special Plate). Dots 1 cm. apart are flashed on to the oscilloscope every half-second and a synchronous electrocardiogram is displayed. This enables accurate timing to be performed. A permanent record is obtained by photographing the oscilloscope with the shutter on "bulb." A polaroid camera is convenient for this purpose. This "B" mode display of a patient with a pericardial effusion is illustrated in Fig. 3 (Special Plate).

The conglomeration of echoes immediately below the transducer are those reflected from the anterior chest wall. Below this there is frequently an area devoid of any echoes. Below this again there is an echo which originates from the anterior myocardium and undulates with a periodicity identical with that of the electrocardiogram. Many normal subjects do not show a "bare area," but when they do it is of constant width (Special Plate, Fig. 4). In patients with pericardial effusion this bare area is always present, and varies in width phasically with the cardiac cycle. It is thus possible to differentiate between a pericardial effusion and a large heart shadow from other causes.

RESULTS

Twenty-seven patients with radiological cardiomegaly have been studied, the undulating bare area between the anterior chest wall and the anterior myocardium being used as the criterion for pericardial effusion. Nine of these patients were found to have effusions. Of these nine the diagnosis was confirmed by paracentesis in five, after right-sided angiography in two of them. In the remaining four the diagnosis was supported by the rapid alteration in heart size radiologically in the absence of cardiac failure, together with the clinical signs of a pericardial effusion. In the remaining 18 patients the subsequent clinical course confirmed that the cardiomegaly was not due to fluid in the pericardium. In four of them paracentesis was attempted unsuccessfully.

DISCUSSION

Ultrasound was first shown to be of value in diagnosing pericardial effusions by Edler (1955). Feigenbaum *et al.* (1965) introduced saline into the pericardial sacs of seven animals and demonstrated that the echo previously received from the posterior heart–lung interface changed from a single impulse to a double one. Moss and Bruhn (1966), using this criterion of a double echo from the posterior heart on an A scan, obtained positive results in seven out of seven patients with pericardial effusions but had one false-positive. Soulen *et al.* (1966) described the pulsation of the anterior of the two shadows received from the back of the heart and non-pulsation of the posterior echo in pericardial effusion. Feigenbaum *et al.* (1966b) described the use of the B mode presentation for demonstrating the varying width of the area from which no echoes were received behind the heart and pericardial effusions. They had three false-positives but no false-negatives in a series of 54 cases, 29 of which had effusions. Feigenbaum *et al.* (1966a), studying cardiac movement in pericardial effusions, used both the anterior and posterior cardiac surfaces without commenting on the anterior bare area. In tamponade they noted a very reduced cardiac movement, and the tracing which they reproduce shows very little variability in the width of the bare area.

In our experience accurate results are obtained only when the anterior myocardial echoes were considered exclusively. In earlier studies in which we considered the posterior heart surface we, like previous workers, obtained both false-negatives and false-positives. It is clear that with a patient lying on his back the heart will tend to gravitate dorsally. In systole it will usually tend to move away from the anterior chest wall. Normally the negative pressure in the pericardial sac, the parietal layer of which is adherent to the chest wall, prevents any significant movement. When there is fluid in the pericardial space it ebbs and flows with each heart beat and so allows the myocardium to move relative to the anterior chest wall. If the recording is made directly over the apex of the left ventricle the forward systolic movement which can be palpated as the apex beat will reduce the width of the bare area in systole (Special Plate, Fig. 5). Recordings over other parts of the heart will sometimes diminish the bare area in diastole.

Because the rotatory movement of the heart makes it theoretically possible to record over an area of myocardium which is immobile, tracings should be made from at least two positions on the chest before denying the presence of an effusion. Gravity will tend to minimize the distance between the back of the myocardium and the lung–pericardial interface, which is apt to alter in position with respiration and so make recording difficult and inaccurate, especially in the presence of lung disease.

Most body tissues contain interfaces which reflect ultrasound. Fluid, however, is homogeneous and so gives rise to no echo. Hence the bare area in patients with pericardial effusion.

Fat is nearly homogeneous, too, and produces little or no echo. This probably accounts for the bare area which is seen in many normal subjects, as the heart is usually surrounded by a layer of fat. When fluid is present as well, this merely adds width to the bare area. This fat layer makes it necessary to demonstrate variations in width of the bare area—not merely the presence of a " double shadow "—in order to diagnose pericardial effusion. This fat is probably the main source of the errors described by Feigenbaum *et al.* (1966a) and Moss and Bruhn (1966).

The width of the bare area will tend to be greater in patients with a large pericardial effusion, but, because there is a variable amount of fat which cannot be determined until after the effusion has gone, it is impossible to estimate with any accuracy the amount of fluid present. Further, the tenseness of the effusion will alter its apparent amount. If the parietal pericardium is lax the fluid will tend to bulge out sideways and so reduce the thickness of the anterior layer of fluid.

Use of only the anterior surface of the heart allows the trace obtained with the Eskoline 20 to be spread out so that the area under consideration can be seen more clearly and the variation in width to be shown more easily. Though we have not examined a patient with tamponade (a condition which should be diagnosed clinically and not require ultrasonics for its recognition), it is likely that even with very reduced myocardial movement the bare area will, when magnified, continue to show variation in width. All clinically significant amounts of fluid can be detected, but it is not impossible that small amounts may be present without being revealed.

This method of diagnosing pericardial effusion is completely atraumatic, involving as it does merely placing a flat transducer on the chest. The Eskoline 20 is portable and can be brought to the bedside, and it can be transported in the boot of an ordinary car. The examination can therefore be done with the minimum disturbance to a patient who may be seriously ill and unfit to stand more vigorous manipulative investigations.

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Medical Memoranda

Neonatal Meningitis Caused by Edwardsiella tarda

Brit. med. J., 1968, **3**, 357–358

This report is that on a newborn baby who had fatal septicaemia and meningitis caused by the organism *Edwardsiella tarda* of Ewing *et al.* (1965). We believe that this is the second report of meningitis caused by that organism, the first being by Sonnenwirth and Kallus (1968).

CASE REPORT

The patient was a female infant born at another hospital on 25 November 1966. The delivery was at full term and was normal. She was first seen in this hospital on 5 December. The mother complained of the infant's inability to suck properly, loss of weight, and fever of two days' duration. There was one sibling alive and well. On examination the temperature was 101° F. (38.3° C.), she had Cheyne–Stokes respiration, there was no neck stiffness, and the cardiovascular system was normal. A diagnosis of septicaemia was made and meningitis was queried. She was then admitted to hospital for treatment.

On the day of admission a blood culture was taken and lumbar puncture performed. The C.S.F. contained 9,625 white blood cells per cu. mm. with 90% polymorphs. Culture of the C.S.F. showed a pure moderate growth of a Gram-negative bacillus subsequently identified as *E. tarda*. The blood culture also yielded a growth of an identical organism. The haematological findings were as follows: Hb 11.1 g./100 ml.; white blood cells 26,000/cu. mm. (57% neutrophils, 30% lymphocytes, 9% monocytes, 2% metamyelocytes, 2% myelocytes); P.C.V. 46%; M.C.H.C. 34%. The red blood cells were normal.

Treatment was started on the day of admission with penicillin 0.25 mega unit, chloramphenicol 50 mg., and sulphadiazine 0.25 g. by six-hourly intravenous injections. After two days of unsettled