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.

This work was supported by a grant to one of us (R. B. P.) from the British Heart Foundation.

R. B. PRIDIE, M.B., F.F.R.
T. A. TURNBULL, M.R.C.S., D.A.
Radiodiagnostic Department, the London Hospital, London E.1.

REFERENCES

Besterman, E. M. M., and Thomas, G. T. (1953). *Brit. Heart J.*, **15**, 113.
Edler, I. (1955). *Acta med. scand.*, Suppl. No. 308, p. 32.
Feigenbaum, H., Waldhausen, J. A., and Hyde, L. P. (1965). *J. Amer. med. Ass.*, **191**, 711.
Feigenbaum, H., Zaky, A., and Grabhorn, L. L. (1966a). *Circulation*, **34**, 611.
Feigenbaum, H., Zaky, A., and Waldhausen, J. A. (1966b). *Ann. intern. Med.*, **65**, 443.
Moss, A. J., and Bruhn, F. (1966). *New Engl. J. Med.*, **274**, 380.
Paul, R. E., Durant, T. M., Oppenheimer, M. J., and Stauffer, H. M. (1957). *Amer. J. Roentgenol.*, **78**, 224.
Rosenthall, L. (1966). *Radiology*, **86**, 634.
Soulen, R. L., Lapayowker, M. S., and Gimenez, J. L. (1966). *Radiology*, **86**, 1047.
Williams, R. G., and Steinberg, I. (1949). *Amer. J. Roentgenol.*, **61**, 41.

# *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).

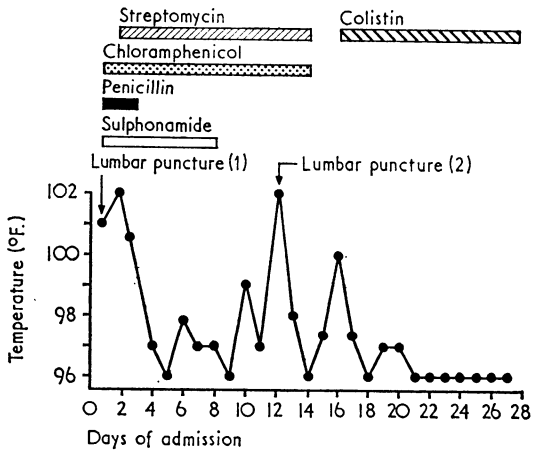
### CASE REPORT

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

pyrexia, 50 mg. of streptomycin six-hourly was added (see Chart). In spite of massive antibiotic therapy the infant's condition was still poor on the fifth day of admission. The temperature came down to 96° F. (35.6° C.) and remained subnormal for five days. On the 12th day she had a pyrexia of 102° F. (38.9° C.), and vomited at all feeds. A pericardial rub was heard on auscultation. The electrocardiograph at this time showed flattened T waves on all leads. The chest *x*-ray picture was normal. The liver was enlarged. A presumptive diagnosis of galactosaemia was suggested but the urine showed no abnormality. A second lumbar puncture was performed and the C.S.F. contained 1,700 W.B.C.s (mainly lymphocytes) and 3,720 R.B.C.s. No organisms were seen or cultured. The temperature fell again to 96° F. (35.6° C.) and therapy was resumed by giving 100,000 units of colistin sulphomethate sodium intramuscularly every six hours. The temperature fell to subnormal values and remained so until death two weeks later.




Temperature and treatment chart.

In the middle of the third week of admission a cellulitis of the nasolabial folds and cancrum of the left nose were noticed. There was bleeding from the nostrils, which was controlled with vitamin K injections. Oliguria and deterioration in the general condition occurred and death took place near the end of the fourth week of admission.

At necropsy the brain was soft, with a collection of 50 ml. of mucopurulent fluid over the surface. Thick pus was present on the base of the brain. The pathologist's diagnoses were bronchopneumonia and meningitis with hydrocephalus.

## Bacteriology

The blood culture was carried out according to our routine procedure, 3–5 ml. of blood being inoculated into two Castaneda (1947) bottles—one containing thioglycollate broth and the other bile peptone medium without the addition of tellurite (Cruikshank, 1965). Both bottles were incubated in air at 37° C. After 48 hours' incubation they showed pure growths of a Gram-negative bacillus with the following characteristics. The bacilli were short and of medium width. On blood agar plates incubated at 37° C. aerobically, the organism gave rise to smooth semitransparent colonies with entire edge and measuring about 1–2 mm. in 24 hours. On MacConkey's agar the colonies were minute after 24 hours' incubation at 37° C., but they increased in size to 1–2 mm. in 48 hours. The organism was catalase-positive, oxidase-negative (Kovacs, 1956), and it attacked glucose and maltose by fermentation (not oxidation—Hugh and Leifson, 1953), with production of acid and gas. Fermentation tests for lactose, mannite, salicin, dulcitol, adonitol, sorbitol, Rhamnose, xylose, arabinose, and inulin were negative. Nitrates were reduced to nitrites. The methyl red test was positive. Indole was produced from peptone water cultures. No growth occurred in potassium cyanide and Koser's (1923) citrate. Malonate and phenylpyruvic acid tests were negative. Tests for lysine and ornithine decarboxylases (Møller, 1955 ; Edwards and Ewing, 1962) were negative. Voges–Proskauer test (Barritt, 1936) was negative. Urea was not broken down. $H_2S$ was produced overnight in TSI agar and the organism was motile. The organism was identified as *E. tarda*.

Culture of the first C.S.F. specimen was done by inoculating blood agar and chocolate agar plates. Incubation was carried out at 37° C. ; the chocolate plate incubated in air containing 3% $CO_2$, the blood agar plate in air without $CO_2$. Both plates yielded pure growths of *E. tarda*.

Antibiotic sensitivity tests were carried out with Oxoid multodisc 969E. The discs contained the following antibiotics: penicillin 1.5 units ; streptomycin 10 μg. ; tetracycline 10 μg. ; chloramphenicol 10 μg. The organism was resistant to penicillin but sensitive to streptomycin, tetracycline, and chloramphenicol.

## Comment

*Edwardsiella* is a new genus of Enterobacteriaceae based on a new species, *Edwardsiella tarda* (Ewing *et al.*, 1965). This organism is the same as that previously called the "Bartholomew group" (King and Adler, 1964), the "Asakusa group" (Sakazaki, 1965), and "Bacterium 1483–59" (Ewing *et al.*, 1965). Its distribution in pathological material is not yet fully known. Out of the 37 strains reported by Ewing *et al.* (1965) 34 were from human beings, of whom 18 had no history of infection, five had diarrhoea, and one strain was isolated from the blood and one from urine. Of the 256 isolates studied by Sakazaki (1965) all but seven were from snakes, five from faeces of patients who had gastroenteritis, and two from seals. The present report shows that the bacterium is also capable of causing meningitis. The portal of entry in this particular case is not certain. It is possible that the organism was derived from the mother, probably infected from a meal of snake flesh, as some people do eat snakes in this part of the world. The lack of improvement on the intensive antibiotic therapy at the beginning of the illness may well have been due to incompatibility or antagonism between the antibiotics used (Barber and Garrod, 1963).

We thank Dr. S. P. Lapage, of Colindale, for confirming the identity of the organism, and Dr. C. Sinnette for permission to publish this paper.

O. A. Okubadejo, M.D.
K. O. Alausa, B.Sc.

Department of Medical Microbiology,
University College Hospital,
Ibadan, Nigeria.

## References

Barber, M., and Garrod, L. P. (1963). *Antibiotic and Chemotherapy.* Edinburgh and London.
Barritt, M. M. (1936). *J. Path. Bact.*, **42**, 441.
Castaneda, M. R. (1947). *Proc. Soc. exp. Biol. (N.Y.)*, **64**, 114.
Cruikshank, R. (editor) (1965). *Medical Microbiology*, 11th ed. Edinburgh and London.
Edwards, P. R., and Ewing, W. H. (1962). *Identification of Enterobacteriaceae*, 2nd ed., p. 242. Minneapolis.
Ewing, W. H., McWhorter, A. C., Escobar, M. R., and Lubin, A. H. (1965). *Int. Bull. bact. Nomencl.*, **15**, 33.
Hugh, R., and Leifson E. (1953). *J. Bact.*, **66**, 24.
King, B. M., and Adler, D. L. (1964). *Amer. J. clin. Path.*, **41**, 230.
Koser, S. A. (1923). *J. Bact.*, **8**, 493.
Kovacs, N. (1956). *Nature (Lond.)*, **178**, 703.
Møller, V. (1955). *Acta path. microbiol. scand.*, **36**, 158.
Sakazaki, R. (1965). *Int. Bull. bact. Nomencl.*, **15**, 45.
Sonnenwirth, A. C., and Kallus, B. A. (1968). *Amer. J. clin. Path.*, **49**, 92.