infected with the Ogawa type of organism. The death rate was also higher in that type. Mutations of the serological types are little-understood phenomena. These were not abolished by chemotherapy.

Conclusion

Sulphaguanidine, formosulphathiazole, and formosulphacetamide given orally did not reduce the mortality rate in cholera patients, and results in these cases compared unfavourably with those in cases where no chemotherapy was given; other treatment remained the same. No special vibriocidal effect of these compounds was apparent *in vivo*, the *V. cholerae* disappearing from the stools practically at the same rate, both in those who had chemotherapy and in those who had none. In assessing the value of the vibriocidal effect of any drug in this disease *in vivo*, it is important to note that the *V. cholerae* disappears from the human stool automatically at a fairly quick rate. This disease characteristically comes on and passes off like a storm, and once it is fully developed the most important part of treatment is undoubtedly to replace quickly the fluid and salt lost and to prevent the development of the irreversible condition of circulatory collapse. This peripheral circulatory failure is an important cause of persistent anuria occurring as a result of failure of adequate glomerular filtration pressure, which ultimately leads to complete renal failure. Chemotherapeutic drugs, to be of any real value in cholera, must have a quicker action than any of the sulphonamides tested in this series, and also should not be toxic.

My thanks are due to Mr. P. N. Sen Gupta, of Bengal Immunity Research Institute, for bacteriological work; to Dr. A. N. Bose, of the same institute, for estimating the blood sulphonamides; and to the Director of the Institute, Dr. U. P. Basu, for supplying free of cost the sulphacetamide used and for allowing the investigations to be carried out there. Thanks are also due to Messrs. Ciba Pharma Ltd. for supply free of cost the formosulphathiazole, in the form of "formocibazole." I am grateful to my house-physicians and nursing staff, but for whose untiring co-operation during an epidemic this work would not have been possible.

REFERENCES

Bhatnagar, S. S., *et al.* (1948). *British Medical Journal*, **1**, 719.
Bose, A. N. (1949). *Ann. Biochem. exp. Med.*, **9**, 201.
Carruthers, L. B. (1942). *Trans. R. Soc. trop. Med. Hyg.*, **36**, 89
Chopra, R. N., *et al.* (1941). *Indian med. Gaz.*, **76**, 712.
Lahiri, S. C. (1945). *J. Indian med. Ass.*, **14**, 113.
—— (1948). *Indian med. Gaz.*, **83**, 24.
—— (1949). *J. Indian med. Ass.*, **18**, 155.
—— and Dutta, S. K. (1949). Ibid., **19**, 1.

The Tavistock Clinic, as an out-patient hospital dealing with psychological problems of individuals and families, has been absorbed in the National Health Service, but a branch of it known as the Tavistock Institute of Human Relations has been incorporated as a separate social research organization concerned with the psychology of relations between human beings in domestic, industrial, and civic groups. It is governed by a council of eight, four of whom are medical, and the council is responsible to an association of 50 members elected on a basis of professional activity or interest in applied social research. In a small brochure recently issued from 2, Beaumont Street, W.1, an account is given of four years' work. About 70 projects of different kinds have been undertaken, some of them for government departments or local authorities, others for industrial concerns or voluntary organizations interested in community and family affairs. To name a few: a study of some aspects of resettlement in the British zone of Germany; the design of a medical statistics service in a local health authority; the job analysis of nursing auxiliary roles in a hospital group; the development of a new type of family case-work agency; the assessment of safety education films for children.

THE INCIDENCE AND PREVENTION OF TETANUS AMONG CIVILIANS

BY

E. T. CONYBEARE, O.B.E., M.D., F.R.C.P.
Senior Medical Officer, Ministry of Health

AND

W. P. D. LOGAN, M.D., B.Sc., D.P.H.
Medical Statistician, General Register Office

Tetanus is not a notifiable disease in England and Wales, and the only useful indication of its general incidence is that obtainable from the mortality statistics of the General Register Office.

Recent Trend of Mortality

Table I gives the numbers of registered deaths and the death rates from 1931 to 1948, and the latter are illustrated in Fig. 1. (See footnote to Table I regarding exclusion of non-civilians.) The male death rate has recently shown a definite tendency to decline, and the rates during 1946–8 were about half of those recorded between 1931 and 1940. Female rates, on the other hand, remained fairly steady, declining only very slightly during the period.

TABLE I.—*Deaths From Tetanus and Death Rates Per Million Living by Sex. England and Wales, 1931–48*

Year	Males		Females	
	No. of Deaths	Rate per Million Living	No. of Deaths	Rate per Million Living
1931	92	4·8	25	1·2
1932	93	4·8	21	1·0
1933	74	3·8	28	1·3
1934	85	4·4	26	1·2
1935	98	5·0	28	1·3
1936	86	4·4	29	1·4
1937	85	4·3	22	1·0
1938	72	3·6	17	0·8
1939	62	3·1	23	1·1
1940	91	5·0	18	0·8
1941	79	4·6	20	0·9
1942	66	3·9	29	1·4
1943	61	3·7	24	1·1
1944	55	3·4	33	1·5
1945	55	3·4	24	1·1
1946	41	2·2	22	1·0
1947	49	2·5	22	1·0
1948	48	2·4	21	0·9

Note.—This and subsequent tables exclude non-civilian males on and after September 3, 1939, and non-civilian females on and after June 1, 1941.

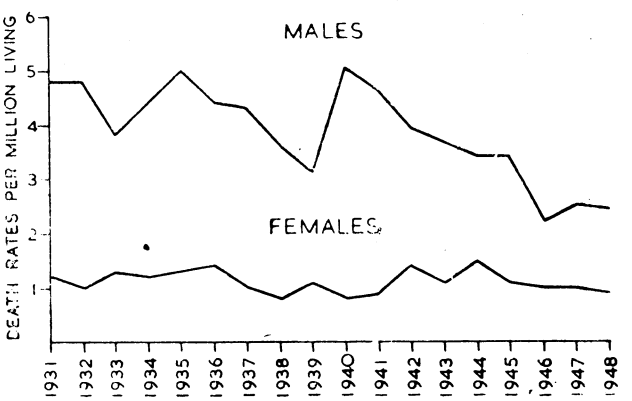

FIG. 1.—Annual death rates from tetanus per million living at all ages. England and Wales, 1931–48.

Sex and Age Incidence in 1928–37 and 1938–47

Table II compares deaths and mean annual death rates by sex and age during the two decennia 1928–37 and 1938–47, these death rates being illustrated in Fig. 2. Only in boys aged between 1 and 10 were the death rates higher in the second of the two decennia. In each sex the mortality was highest during infancy and childhood, lowest during the middle years of life, rising again after 50, especially among males. The death rates among males at every age in both decennia were much higher than the corresponding female rates; in 1938–47 they ranged from 1.7 times the female rate under 1 year of age to 11 times at 75 years and over.

TABLE II.—*Deaths From Tetanus and Mean Annual Death Rates Per Million Living by Sex and Age. England and Wales, in Each of the Two Decennia 1928–37 and 1938–47*

Age Group	Males				Females			
	1928–37		1938–47		1928–37		1938–47	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate
0– ..	25	8·3	23	6·9	12	4·1	13	4·1
1– ..	39	3·3	44	3·7	23	2·0	21	1·8
5– ..	86	5·3	93	6·5	30	1·9	25	1·8
10– ..	143	8·6	76	5·2	31	1·9	14	1·0
15– ..	77	4·7	41	3·0	13	0·8	10	0·6
20– ..	75	4·4	15	1·8	15	0·9	14	0·9
25– ..	66	4·0	17	1·6	17	1·0	13	0·8
30– ..	56	3·7	21	1·6	22	1·3	14	0·8
35– ..	43	3·2	32	2·2	17	1·1	20	1·2
40– ..	51	4·1	40	2·9	20	1·4	11	0·7
45– ..	39	3·3	40	3·2	15	1·1	6	0·4
50– ..	57	5·1	28	2·4	13	1·0	15	1·1
55– ..	55	5·5	32	3·0	18	1·6	14	1·1
60– ..	62	7·7	51	5·5	16	1·7	16	1·4
65– ..	32	5·3	37	5·0	11	1·5	17	1·9
70– ..	28	7·1	26	5·0	7	1·4	6	0·9
75+ ..	15	4·5	21	4·5	5	0·9	3	0·4
Total ..	949	4·9	637	3·6	285	1·4	232	1·1

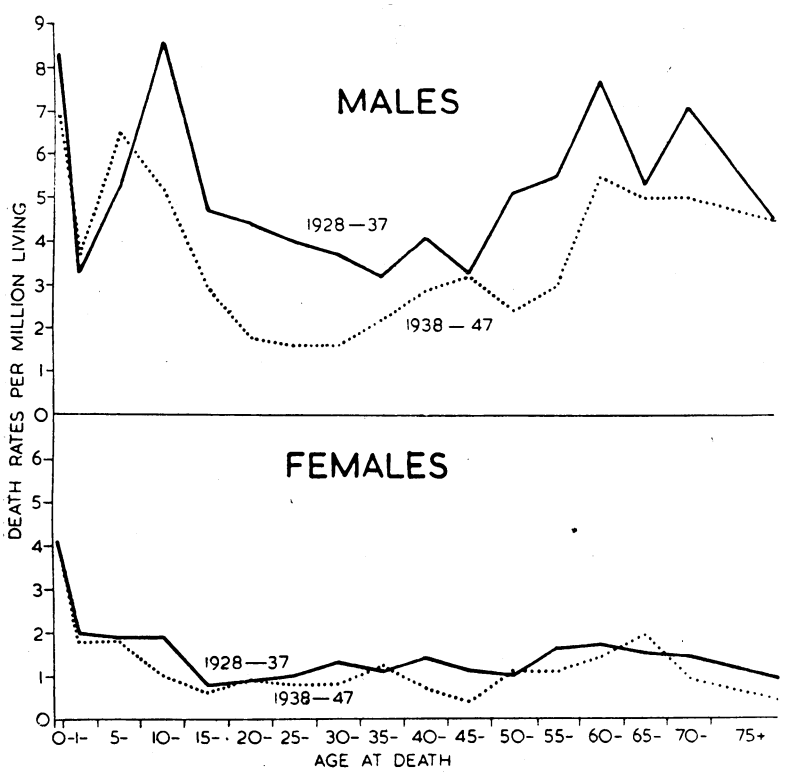

FIG. 2.—Mean annual death rates from tetanus per million living. Males and females by age. England and Wales, 1928–37 and 1938–47.

Mortality Among Infants.—Table III shows the deaths of 36 (23 males, 13 females) infants between 1938 and 1947 by sex and age in months—29 were under 1 month.

TABLE III.—*Deaths From Tetanus Under 1 Year, by Sex and Age in Months, England and Wales, 1938–47*

Year	Sex	Months												Total
		–1	1	2	3	4	5	6	7	8	9	10	11	
1938	M	—	—	—	—	—	—	—	—	—	—	—	—	—
	F	—	—	—	—	—	—	—	—	—	—	—	—	—
1939	M	1	—	—	—	—	—	—	—	—	—	—	—	1
	F	2	—	—	—	—	—	—	—	—	—	—	—	2
1940	M	3	1	—	—	—	—	—	—	—	—	—	—	4
	F	1	—	—	—	—	—	—	—	—	—	—	—	1
1941	M	—	—	—	—	—	—	—	—	—	—	—	—	—
	F	—	—	—	—	—	—	1	—	—	—	—	—	1
1942	M	1	—	—	—	—	—	—	—	—	—	—	—	1
	F	2	1	—	—	1	—	—	—	—	—	—	—	4
1943	M	1	—	—	—	—	—	—	—	—	—	—	—	1
	F	—	—	—	—	—	—	—	—	—	—	—	—	—
1944	M	5	—	—	—	—	—	—	—	—	—	1	—	6
	F	3	—	—	—	—	—	—	—	—	—	—	—	3
1945	M	3	—	—	—	—	—	—	—	—	—	—	—	3
	F	1	—	—	—	—	—	—	—	—	—	—	—	1
1946	M	2	—	1	—	—	—	—	—	—	—	—	—	3
	F	1	—	—	—	—	—	—	—	—	—	—	—	1
1947	M	3	—	—	—	—	—	—	—	—	—	—	1	4
	F	—	—	—	—	—	—	—	—	—	—	—	—	—
Total	M	19	1	1	—	—	—	—	—	—	—	1	1	23
	F	10	1	—	—	1	—	1	—	—	—	—	—	13

Infantile tetanus is, therefore, almost entirely a disease of the neonatal period, and even at this age, as at other ages, affects males much more than females.

Fatality.—In the absence of comprehensive morbidity statistics it is difficult to estimate the fatality (case mortality) rate of tetanus in the civilian population. Except in infancy, and if treatment is prompt, it is possible that about 40–50% of cases will recover (Press, 1948). Before antitoxic serum became available for treatment the recovery rate was only about 15% (Bruce, 1920).

Geographical Incidence

Table IV gives numbers of deaths and death rates in the geographical regions of England and Wales during 1938–48, distinguishing the sexes during the periods 1938–41 and 1947–8. It has been noted in France that the death rates from tetanus have for many years been consistently higher in two or three of the departments into which the country is divided for purposes of civil administration. A similar tendency is revealed by the regional analysis in Table IV, which indicates that between 1938 and 1948 the highest rates in England and Wales were consistently recorded in the East and South-west regions, the difference between the rates in these regions and the others being more than would be likely to have arisen by chance.

A possible explanation for the high rates in the East and South-west regions is that a large proportion of the popu-

lation in those areas live in rural districts and are engaged in agriculture. Horses and other farm animals undoubtedly excrete *Clostridium tetani*, and a wound suffered by a person living in a country area is more likely to be contaminated by dirt or soil infected with tetanus bacilli than a wound in a town dweller. This explanation is supported by Table V, which compares the death rates from tetanus in 1946–8 among residents of Greater London, county boroughs, other urban districts, and rural districts. In each sex the death rates in the rural districts were about three times as high as in Greater London and the large towns, and more than half as high again as in the smaller towns.

TABLE IV.—*Deaths From Tetanus and Mean Annual Death Rates Per Million Living in Geographical Regions. England and Wales, 1938–48*

	1938–41				1947–8				1938–48	
	Males		Females		Males		Females		Persons	
	No	Rate	No.	Rate	No	Rate	No.	Rate	No.	Rate
England and Wales	310	4·1	78	0·9	97	2·4	43	1·0	938	2·1
Greater London	41	2·9	6	0·4	10	1·3	4	0·5	111	1·4
Remainder of South-east	63	5·9	14	1·1	22	3·8	12	1·8	199	3·1
North I	10	2·5	4	0·9	6	2·8	2	0·9	43	1·9
North II	15	6·2	2	0·7	4	3·2	2	1·5	47	3·4
North III	13	2·0	5	0·7	5	1·5	1	0·3	38	1·0
North IV	29	2·6	14	1·1	9	1·6	4	0·6	87	1·3
Midland I	39	4·3	6	0·6	17	3·4	2	0·4	104	1·9
Midland II	23	4·9	5	0·9	4	1·6	5	1·8	59	2·1
East	29	8·3	6	1·5	10	5·4	6	3·0	103	5·1
South-west	32	8·0	10	2·1	7	3·4	5	2·1	92	3·8
Wales I	13	3·7	5	1·3	1	0·6	—	—	36	1·8
Wales II	3	2·2	1	0·6	2	3·0	—	—	19	2·4

Note.—For the constitution of the geographical regions see the Registrar-General's Statistical Review for any recent year.

TABLE V.—*Deaths From Tetanus and Mean Annual Death Rates Per Million Living. Density Aggregates of England and Wales, 1946–8*

	Males		Females		Persons	
	No.	Rate	No.	Rate	No.	Rate
England and Wales	138	6·9	65	2·9	203	4·8
Greater London	16	4·2	6	1·4	22	2·7
County boroughs	26	4·3	14	2·1	40	3·1
Other urban districts	47	7·5	27	3·8	74	5·5
Rural districts	49	13·0	18	4·5	67	8·6

Occupational Mortality.—It seems probable, in view of the relatively high mortality of tetanus in rural districts, that persons engaged in farming and related occupations are exposed to the greatest risk. This, however, cannot at present be confirmed from the national mortality statistics, because the relationship between tetanus and occupation has not so far been studied in the Registrar-General's Occupational Mortality Supplements.

Prevention

Two forms of artificial immunization can be used in the prevention of tetanus. Each has been tested on a large scale in one of the two world wars.

Passive Immunization.—That an injection of antitoxic serum given soon after the time of wounding would prevent tetanus was clearly demonstrated in the 1914–18 war. In September, 1914, the tetanus rate among wounded British soldiers was 9 per 1,000. Routine injection of antitetanus serum was started in October, 1914, and by the end of the year the rate had fallen to 1.4 per 1,000 (Bruce, 1920).

Active Immunization.—Proof of the efficacy of this form of prophylaxis against tetanus also rests on military experience. A formolized toxoid for the production of active human immunity against tetanus was first produced in France about 1931. Active immunization of the French Army with this toxoid was begun in 1936, and by 1939 600,000 French soldiers had received two injections of toxoid at approximately a year's interval, which was then thought sufficient to give a solid immunity against tetanus. Unfortunately, owing to the French collapse in 1940, it proved impossible to preserve the records of this pioneer experiment of military medicine and to determine its results (Ramon, 1950).

Active immunization against tetanus with a formolized toxoid was begun in the British Army in 1939 and continued throughout the war. British wounded were, however, also given injections of antitoxin as soon after the injury as possible. This combined active and passive immunization was extremely successful, the number of cases of tetanus being reduced to insignificant proportions, the incidence among British troops being no more than 1/25 what it was in the 1914–18 war (Boyd, 1946).

The armed Forces of the United States during the last war relied entirely upon active immunization to prevent tetanus. At the time of wounding, or as soon after as possible, a boosting dose of toxoid was given, and not, as in the British Army, an injection of antitoxin. The results of this active immunization used alone proved to be as good as those of combined active and passive immunization used by the British, and the number of cases of tetanus among the American armed Forces was negligible (Long and Sartwell, 1947).

Experience in the British and U.S. Armies during the last war showed, moreover, that tetanus toxoid, unlike some other antigens, could be given subcutaneously without producing a local reaction and that it rarely caused general reactions. Active immunization with tetanus toxoid is therefore a practically trouble-free procedure.

Only one of the two available methods of artificial immunization against tetanus has hitherto been used in this country on any considerable scale among civilians.

Passive Immunization of Civilians

The injection of antitoxic serum for the prevention of tetanus became established as a routine in civilian hospitals and also to a certain extent in civilian general practice all over the world before the end of the 1914–18 war. It is still the only prophylaxis against tetanus in general use among civilians in this country.

The mortality data given above show, however, that this procedure is either not completely effective or is not applied widely enough among civilians. Moreover, civil and military experience shows that in susceptible persons tetanus antitoxin, like other antisera, is occasionally a cause of severe hypersensitivity reactions, some of which, unfortunately, prove fatal. More often antitoxin causes the less severe but temporarily disabling sensitivity reaction comprising an irritating skin rash and joint effusions known as "serum sickness." In view of the frequency of these complications many doctors, before deciding whether or not to give prophylactic injections of antitoxic serum, attempt to discriminate between wounds which may be more or less likely to cause tetanus.

It seems, however, to be the specific contamination which determines subsequent tetanus rather than the extent or depth of the wound, and tetanus has

occasionally been reported after trivial abrasions of a sort which would not be likely to receive medical or any other treatment. In general medical practice it must in fact often be extremely difficult to balance the risk of tetanus arising from a trivial wound against the risk of a serious hypersensitivity reaction to antitoxin. In hospital practice such decisions hardly ever have to be made, because only wounds of considerable severity are ordinarily presented for treatment, and for these antitoxin is quite rightly given as a routine.

Active Immunization of Civilians

Military experience clearly suggests that civilian tetanus might be almost completely prevented by means of active immunization with toxoid. Widespread adoption of this relatively trouble-free type of inoculation against tetanus would, moreover, free the doctor in general practice from the onus of making the difficult decisions which he now often has to make about the prophylactic use of tetanus antiserum in dealing with trivial injuries. It should be remembered, however, that the administration of routine pre-injury active immunization instead of, as at present, an *ad hoc* post-injury passive immunization against tetanus is likely for obvious reasons to be much more difficult among civilians than among soldiers.

The present incidence and mortality of tetanus are so low that in order to bring them to the vanishing-point—and anything less could hardly be regarded as worth while—it would be necessary to initiate and maintain active immunity in the whole population. Further, in order to prevent tetanus during the first month of life, when, as has been shown above, the risk is particularly great, special measures to ensure that the active immunity of expectant mothers was raised to the highest possible level during pregnancy would need to be taken.

The method of active immunization against tetanus now advocated (Parish, 1948) is to inject three doses of toxoid. The interval between the first and second injections is six weeks, and the third injection is given after a further interval of six to twelve months. To give such a series of injections to every man, woman, and child in the country would clearly be an expensive undertaking and extremely difficult to organize, especially on a voluntary basis, which is the only one that can be seriously considered.

Failing universal active immunization against tetanus, which can hardly be regarded as a practical proposition for health departments or for the medical profession at the present time, there remains to be considered the active immunization of population groups selected by age, place of residence, or occupation.

The active immunization of a selected age group of the population has been attempted in France, where since 1940 it has been obligatory by law for tetanus toxoid to be given at the same time as immunization against diphtheria—at present, before the age of 18 months (Ramon, 1950). No figures are available, however, to show the extent to which this obligatory active immunization against tetanus is actually done, and the published rates do not yet show that tetanus has substantially declined as a cause of death among French children in the last few years.

Serious consideration has also been given in the United States to the active immunization with toxoid of selected population groups. In one review of tetanus in America (Press, 1948) it was noted that nearly one-half the reported cases of the disease followed injuries which were so trivial as to go untreated or undetected, and it was estimated that if the passive form of tetanus prophylaxis at present advocated—that is, immunization with antitoxin after injury—were to be conscientiously performed nearly one-third of the total population would at some time during life receive an injection of antitoxin. The high incidence of sensitization to serum proteins likely to result might be regarded as an important argument in favour of active immunization as an alternative form of prophylaxis, at least in children and in those adults whose occupation exposes them to more than the average risk of accident. In another review (Long, 1948), after an analysis of data showing that in America children from 5 to 14 years run the greatest risk of tetanus, the conclusion reached was that, although active immunization with toxoid is warranted for children and for those with special occupational hazards, it cannot at present be advocated for the United States population as a whole.

Active immunization of the whole population against tetanus has recently been advocated as a long-term policy in Denmark, civilians working with earth—that is, farmers, gardeners, and navvies—together with patients having chronic ulcers, being regarded as special groups deserving immediate attention (Lassen, 1949).

In this country, in view of the relatively high death rates from tetanus among children from 5 to 14 years of age, it might at present be reasonable to offer parents active immunization against tetanus when they are being asked to agree to the "boosting" inoculation against diphtheria at the time of entry to school—that is, at about 5 years of age. This ought not to present any insuperable administrative difficulty, and the active immunization of children of this age group might be specially urged in rural districts where the local population seems to run a greater risk from tetanus than elsewhere.

Until further data are available it is impossible to consider whether active immunization against tetanus should be advocated as a protection against special risks in certain occupations.

Summary and Conclusions

A review of the mortality statistics of tetanus among civilians in England and Wales during 1927–48 shows that during this period mortality in each sex was highest during infancy and childhood, lowest during the middle years of life, and rose again after the age of 50, especially among males.

In 1946–8 there was a considerable decline in the mortality of males from tetanus, and a much smaller decline among females.

Tetanus male death rates at all ages are much higher than the corresponding female rates.

Fatal infantile tetanus was, and is, almost entirely a disease of the neonatal period.

Between 1938 and 1948 the highest death rates from tetanus were consistently recorded in the East and South-west regions of the country. It is unlikely that these higher rates were due to chance.

Death rates from tetanus were higher among the population of rural districts than in London or in the large and small towns.

The outstanding success of active immunization against tetanus in the military experience of the last war is held to justify a careful consideration of the application of this form of prophylaxis among the civilian population in peacetime.

Active immunization of the whole civilian population against tetanus in peacetime is rejected as impracticable, but it is suggested that the active immunization of children at about the age of entry to school would be both practicable and justified, especially in rural districts.

REFERENCES

Boyd, J. S. K. (1946). *Lancet*, **1**, 113.
Bruce, D. (1920). *J. Hyg., Camb.*, **19**, 1.
Lassen, H. C. A. (1949). *Acta med. scand.*, **136**, Suppl. 234, p. 214.
Long, A. P. (1948). *Amer. J. publ. Hlth*, **38**, 485.
—— and Sartwell, P. E. (1947). *Bull. U.S. Army med. Dept.*, **7**, 371.
Parish, H. J. (1948). *Bacterial and Virus Diseases*, p. 48. Livingstone, Edinburgh.
Press, E. (1948). *New Engl. J. Med.*, **239**, 50.
Ramon, G. (1950). *Le Principe des Anatoxines et ses Applications*. Masson, Paris.

SPONTANEOUS INTRAPERITONEAL RUPTURE OF THE BLADDER IN THE PUERPERIUM

BY

A. L. DEACON, F.R.C.S.Ed., M.R.C.O.G.
Assistant Surgeon, Birmingham Women's and Maternity Hospitals

In most cases in which the bladder is ruptured, trauma due to a penetrating body from without or to a bony fragment from within is responsible, and the rupture is usually extraperitoneal. Most instances of intraperitoneal rupture are due to the concussion of a blow at a time when the bladder is distended, and it is commonly the alcoholic who is the victim of this disaster. Spontaneous rupture occurs with relative rarity, and Stone (1931) could find only 40 cases in the literature. He found it to be due to disease of the bladder wall, inflammatory or malignant, or to obstruction at the bladder neck or urethra, leading to gross over-distension of the bladder. Paralytic lesions form a small percentage of this type. This spontaneous type of rupture may be extraperitoneal, though it is more often intraperitoneal, the former being associated with diseases of the bladder wall, whilst the latter most frequently occurs with an obstructive lesion.

In Stone's review the precipitating factor immediately responsible for the catastrophe was invariably a sudden increase in intra-abdominal pressure, due to such causes as attempted micturition, straining at stool, and sudden lifting of a heavy weight. On five occasions the distended bladder ruptured during labour and once during the manual expression of the placenta on the fifth day of the puerperium. Rupture has been reported by Sarkar (1944) and Mussgnug (1947) in association with retention of urine due to an incarcerated gravid uterus. Claye (1941) describes a fatal case of rupture of the bladder wall on the tenth day of the puerperium due to a neglected distension with overflow, though in his case the tears were not complete and there was no extravasation of urine.

The mortality rate in this catastrophe has always been very high, and Stone's investigations showed that before 1900 the rates for intraperitoneal and extraperitoneal spontaneous rupture were 92.3% and 50% respectively. After 1900 the rates were 45.4% and *nil* respectively.

The clinical picture varies considerably, and the extraperitoneal type, with its less acute symptoms, often does not reach the consultant until there are signs of extravasation of urine, with its concomitant infection. In the intraperitoneal type, however, after a period of retention of urine, a sudden increase in intra-abdominal pressure is followed by severe abdominal pain and collapse. The picture of "acute abdomen" with rigidity follows, though shifting dullness may also be elicited in many cases.

In most of the earlier cases treatment was unsatisfactory, as it was not realized that post-operative drainage of the bladder was essential. Since this has been carried out the results have steadily improved, and Stone maintains that repair of the rent in the bladder is unnecessary, so long as the peritoneal cavity is drained and a suprapubic cystotomy carried out.

Diagnosis of spontaneous rupture of the bladder is difficult unless there is a history of retention of urine, with an associated obstructive lesion. In the following case there was no suspicion of the true diagnosis until the peritoneal cavity was searched at laparotomy.

Case History

A married woman aged 36 had a normal delivery of her second child on September 25, 1950, after a labour lasting seven hours. Her doctor had to express the placenta, and she had a second-degree perineal tear, which was sutured. Her first child had been stillborn two and a half years before, following a severe pre-eclamptic toxaemia. During the puerperium the patient complained of abdominal discomfort, which was thought to be due to after-pains; but she was otherwise well, and her chart showed no rise of temperature. On the morning of the fifth day after delivery the midwife suggested that the time had come for her to spend a few hours a day lying on her abdomen lest her uterus should become retroverted. This injunction was obediently followed, and immediately the patient felt a severe pain in the upper abdomen and collapsed. She vomited once shortly afterwards.

I first saw her in consultation with the doctor a few hours later, when the above history was obtained. At this time her temperature was 97° F. (36.1° C.), the pulse rate was 106 and of fairly good volume, while she was sweating profusely. Her colour was good, however, and the diagnosis of pulmonary embolus, as suspected by the doctor, seemed improbable. The pain was localized to the upper abdomen, and on inspection the abdomen appeared generally distended, though this was most marked below the umbilicus. Palpation revealed a cystic swelling which filled the whole of the lower abdomen and rose to about two fingerbreadths above the level of the umbilicus. This was tender, but not as markedly so as the epigastrium and hypochondria. There was no rigidity, but the muscles had been stretched by the pregnancy and had not yet regained their tone, and the most outstanding feature was the pain felt when the pressure of the hand was released. The swelling in the abdomen was dull to percussion. A pelvic examination was not then carried out as it was obvious that the patient would have to be moved into hospital. A ruptured ovarian cyst was provisionally diagnosed, and arrangements were made for her transfer.

On arrival at hospital nearly two hours later, after an ambulance journey of 16 miles, she was immediately catheterized, 26 oz. (740 ml.) of rather dark-coloured urine being obtained. When she was seen immediately afterwards the clinical picture had changed. She looked more shocked than when first seen, with a cyanotic tinge about the lips, though the pulse rate was about the same, and the B.P. was 110/70. On examination, however, instead of the cystic lower abdominal swelling, there was now a