chance it seems possible that vasodilatation was more likely to occur in an individual with a small blood volume.

The cases with a venous-pressure rise either had no clinical evidence of peripheral vasodilatation or only developed signs and symptoms of this after the rise was already considerable. The peripheral vasodilatation was not accompanied by the fall in venous pressure described by Murphy.

Venous Pressure During the Transfusion of Cases of Pulmonary Disease

These 13 pulmonary cases, transfused at rates varying from 17 to 40 c.cm. a minute, were comparable to 16 of the first group of 30 cases, in which the rates varied from 16 to 45 c.cm. a minute. In the latter group 13 of the 16 cases showed a significant rise of venous pressure, whereas only 6 of the 13 patients with pulmonary disease showed an increase of similar extent. Venous-pressure rises were apparently less frequent in the group suffering from pulmonary disease, but the number of cases examined was too small to warrant drawing conclusions. In this group, too, the venous pressure increased with the amount given. There was a slight decrease during the interval when the intermediate vital capacity was being recorded.

Glaser and McMichael (1940) found a significant increase in vital capacity following venesection. They calculated lung volume in each instance and converted the changes into equivalent changes in blood volume. From these figures they deduced that 47% of the blood withdrawn came from the pulmonary vascular bed. Conversely, during a rapid transfusion one might expect that an appreciable proportion of the increased blood volume would be accommodated by the lungs. Altschule and Gilligan recorded the vital capacity in 4 of their cases, but they could find no change. In the present investigation vital-capacity changes were studied in patients with pulmonary disease. Such cases were chosen because in subjects with an already reduced vital capacity any further-reduction due to the accommodation of the transfused blood in the lungs would be more readily appreciated. In point of fact, a reduction was seen in every case except one, but in most instances the reduction was such that it could account for only a small proportion of the transfused fluid.

If it is accepted that reduction of vital capacity is due to pulmonary congestion, then the question whether this is brought about by heart failure from overloading the circulation must be considered. In such cases a systemic venous-pressure rise would be expected. However, the only cases (3 and 7) that showed a well-marked reduction in vital capacity and an increase in venous pressure had no signs or symptoms suggestive of cardiac embarrassment. Furthermore, the 2 cases (12 and 13) that did develop respiratory signs and had the greatest percentage reduction in vital capacity had no increase in venous pressure; and the 4 cases (1, 2, 5, and 6) that had the greatest rise in venous pressure had only slight reduction in vital capacity. In this series there appears to be no direct relation between venous pressure and vital capacity. The changes in vital capacity therefore cannot be ascribed to cardiac failure. The accommodation of the blood is perhaps best explained by alteration in the capacity of various parts of the circulatory system. The signs and symptoms of peripheral vasodilatation seen indicate that the capacity of the subpapillary venous plexus was being increased. The rise in venous pressure and the reduction of vital capacity show that the larger veins and the lung capillary bed respectively were accommodating the extra fluid.

Since this paper was written Sharpey-Shafer and Wallace (1942) have published observations on the rapid transfusion mainly of serum and saline (300 c.cm. to 2,100 c.cm.) to subjects without cardiovascular disease. Their results indicate that the venous pressure is raised when there is considerable retention of injected fluid. Dilatation of peripheral vessels was noted, and an engorgement of the pulmonary vessels was shown by vital-capacity and radiographic records.

Conclusions

The venesection of normal subjects is accompanied by a fall in venous pressure. During the following 30 minutes there is a return towards the original venous pressure, although this level is not regained in this period.

During the transfusion of blood to unselected hospital patients there is in the majority of cases a rise of venous pressure which is approximately proportional to the amount given, and occurs more frequently at the faster rates. Signs and symptoms suggestive of peripheral vasodilatation were encountered in 3 of the 43 cases transfused. In these cases vasodilatation occurred after a rise in venous pressure and was not followed by a fall.

After transfusion, patients suffering from pulmonary disease showed signs of pulmonary congestion, as revealed by a reduction in vital capacity. This decrease was not related to the venous-pressure changes.

We should like to thank Dr. W. D. Nicol and the staffs of the medical and thoracic units of the Emergency Hospital and Mr. C. F. Swinton of the Mayday Hospital for access to cases. We are indebted to Dr. O. M. Solandt for help and encouragement.

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A WATER-BORNE AND MILK-BORNE OUT-BREAK OF PARATYPHOID B INFECTION IN WORCESTERSHIRE

POTENTIAL DANGERS OF RAM-WATER SUPPLIES

BY

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This small but none the less interesting paratyphoid B outbreak occurred from June to Aug., 1941, in one of the rural districts of Worcestershire where I was acting medical officer of health. It comprised a total of 17 primary and secondary cases, notified over a period of two months, and is of particular interest owing to the extreme rarity of epidemics of paratyphoid B fever recorded in which the organism has been recovered from water and which, in that respect, may be regarded as water-borne. Since the water that was implicated was not actually a drinking water, and since the milk supply was apparently contaminated secondarily from this water supply, I prefer to designate this outbreak milk-borne and water-borne rather than water-borne.

It will suffice to state that cases of paratyphoid B infection had recently occurred in adjacent areas and that the alleged source of infection had been traced to cream buns. While every patient in the present outbreak denied consumption of cream buns during the incubation period of their attack, an indirect and subtle connexion probably existed between these two epidemics.

History of Onset of Outbreak and Events Leading up to the Investigation of Farm

My suspicions were aroused on the discovery that there was a common milk supply to the first two cases of paratyphoid B reported (Table, Cases 1 and 2). On July 2, accompanied by Mr. Owen, county sanitary officer, I visited the farm which supplied milk to both these cases in order to ascertain, in particular, whether there was any sickness among the milk-handlers. The machine-milker, who had had some slight symptoms for a week or two, was on July 4 proved to have a mild attack of paratyphoid B fever (Case 5). Incidentally, his son, who had been admitted to hospital on June 26 with a history of three weeks' diarrhoea, was later diagnosed as a case of paratyphoid (Case 6). This important information proved the latter could not have derived his infection from milk contaminated by his father. A search for missed cases, carriers, and other sources of infection was made.

This bottling establishment also received milk from six local producers, and, while paratyphoid B organisms were absent in all milk samples submitted for examination, they were present

in the ram-water used for dairying purposes. This ram-water was therefore the *missing link* we were seeking; for it was proved that the small son of the machine-milker contracted his infection from actually drinking the ram-water as it flowed in the gully outside the dairy.

The machine-milker had ample opportunity of contracting paratyphoid from (1) his little son at home, (2) the tap-water in the cowshed, and/or (3) drinking the raw milk. At the same time in the process of hand-stripping he had plenty of opportunities of contaminating the milk himself. The milk could also have been contaminated by the ram-water delivered

Common Milk Supply.—Some of the paratyphoid B organisms, originally discharged from the depot in the sewage effluent, evidently completed their circular tour in the suspected milk supply, and a total of 5 cases of paratyphoid B fever (Cases 3, 4, 7, 13, 17) were notified among its employees. The fact that no raw milk was served over the counter at the canteens accounts for the negligible number of cases at the depot. The canteen cook (Case 3) was one of the few persons with access to raw milk. Milk was available only in tea; however, the actual procedure of making tea varied somewhat at the several canteens.

Table showing Details of Cases

Case	Sex	Age	Paratyphoid B Widal Result "H"	Faeces	Urine	Clinical History	Remarks
1	F	6	+	—	+	"Cold" for 5 days. June 21: Fever, lassitude. 22nd: Diarrhoea. 29th: Rash, abdomen tumid, spleen +, liver +. "Typhoid state" for 2 weeks	Milk stated by parents always boiled previously. All family ate ice-cream twice a week
2	M	27	+	—	+	Indefinite. Jaundice since April 14. Stated illness began May 6. Fever June 13. Admitted hospital 20th. Epistaxis 28th. July 1: Tumidity, spleen +, liver +, "typhoid state" for one week. Jaundiced until about July 7	Date of onset very indefinite. At work until fever began. Very fond of raw milk
3	F	43	+	—	+	Poorly for about a week at work. June 24: Headache. 27th: Fever started; backache, headache, spleen +, liver +. No rash or tumidity	Canteen cook at depot. Handled uncooked food until evening June 27. Drank glass of raw milk daily
4	F	19	+	+	—	Sudden onset. Giddiness, pains in stomach and back. Frequent epistaxes June 21 to 28. July 1: Rash, tumidity, spleen +, liver +, constipation	Last worked at depot June 8. On holiday until July 1. No raw milk except in tea
5	M	26	+	—	—	Indefinite (1-2 weeks). Indigestion and backache. No diarrhoea. July 3: Headache; no rash or tumidity, spleen +, liver +	Machine-milker to bottling establishment. Father of Case 6. Fond of raw milk
6	M	2	+	—	—	Indefinite. Diarrhoea for 3 weeks. Admitted as suspected bronchopneumonia, diagnosed peritonitis until + Widal ("H" & "O") result July 5. Tumidity +, rash +, respirations rapid and shallow, with cough	Son of Case 5. History of having actually drunk ram-water. Drank raw milk
7	M	30	+	+	—	June 24: Epistaxis. 25th: Headache and lassitude. July 3: Diarrhoea and fever several days. 11th: Stiff neck, spleen +, liver +, rash +	No raw milk at depot except in tea; this had previously been added in urn
8	F	24	+	—	—	Sudden onset of intense headache, fever, and vomiting. July 6 Two rigors. 13th: Rash +	Stated she always boiled milk before drinking
9	M	1½	Nil	+	—	Diarrhoea since July 14. Fever since 18th. 22nd: Rash +, tumidity +	Admitted on + faeces (collected July 21, reported 23rd). Reputed to have last drunk raw milk from suspected source on June 18. Evidence inconclusive
10	F	18	+	+	—	July 22: Fever, headache, and aches in legs. 23rd: Rash +	Admitted as suspected case, being mother of Case 9. Blood culture — (collected July 23). Faeces + (received July 26). Leucocytes 11,550 (Aug. 7)
11	M	21	+	N.I.	Nil	July 22: Headache, nausea, diarrhoea. 28th: Rash +, tumidity +, spleen +	Admitted on + blood culture (collected July 23, reported 28th). Died from haemorrhage Aug. 5. Uncls of Case 9
12	F	60	+	+	—	July 24: Sudden onset; headache, diarrhoea, vomiting. 25th: Collapsed with sudden acute abdominal pain and diarrhoea. 28th: Rash +, tumidity +	Blood culture — (collected July 25). Admitted on + faeces (collected 25th, reported 28th). Drank one glass of raw milk daily
13	F	21	+	+	—	Headache and pains in legs for 2 weeks; diarrhoea. July 26: Rash +. 28th: Epistaxis. 29th: Spleen +, liver +	No raw milk at depot except in tea. Last at work on July 21
14	M	55	+	+	—	Headache and febricula since July 22. Diarrhoea since 24th. Rash +. Aug. 1: Spleen +, liver +	Blood culture — (collected July 23). Grandfather of Case 9
15	F	10	+	+	—	Headache, fever, diarrhoea. Aug. 1: Spleen +, liver +	Blood culture — (collected July 23). Aunt of Case 9
16	F	11/12	+	+	—	Aug. 2: Fever for four days. 6th: Diarrhoea began. 8th: Fever, diarrhoea with mucus until 10th. Drowsy, tumidity +, green stools; rash +	All well-water boiled and all milk reported sterilized for child. Grandparents (living in same cottage) take milk from suspected farm; but child stated to drink milk from another source
17	M	58	+	—	—	Aug. 17: Sudden onset. Fever, diarrhoea, and vomiting 2 days; tumidity +. 28th: Admitted on relatively high paratyphoid B agglutinin titre	No raw milk at depot except in tea from urn. History T.A.B. inoculation (1917). Widal on Aug. 29 showed rising titre paratyphoid B agglutinins from 1/500 to 1/1,000 "H" in two days. [Typhoid agglutinin remained constant (1/125 "H")]

from the cowshed tap. The milk was supplied from this farm in bottles to the private customers and in bulk to a big depot in the district.

Events Leading to Investigation of Depot

Canteen Cook suffering from Paratyphoid.—On July 3 (i.e., the day before the Widal result of the machine-milker) Mr. Owen and I visited the depot to investigate a case of paratyphoid (Case 3) reported at one of the canteens. The patient was a cook who had been ill at work for about a week. It was evident that a salad-borne epidemic among the depot workers on that particular site was probable, and, in addition, a milk-borne epidemic among the farmer's private and depot customers was imminent.

Source of the Contaminated Ram-water.—One of the sources of the ram-water—two miles up-stream—was traced to the outfall of a sewage effluent from the depot.

Course of Streams Contaminated by Sewage Effluents

A complete survey was made of farms and dwellings situated along the course of streams receiving effluents from the depot sites. We first explored the course of the brook from which the bottling establishment derived its ram-supply. This procedure entailed a visit to every farm in order to ascertain certain facts. The following five questions were asked (the answers apply only to the bottling establishment):

1. *Whether any Case of Sickness had Occurred Recently.*—One case of sickness—i.e., the machine-milker (Case 5), who was an immediate contact of Case 6—which may be regarded as definitely water-borne in origin. The type of sanitary accommodation at the milker's home is a privy, and the bucket contents are disposed of by burial in the garden.

2. *Access of Milch Cows to Brook containing Effluent, on Account of Possible Source of Contaminated Udders.*—At the

time of the outbreak all 12 milch cows had access to the contaminated brook water.

3. *Use, if any, made of Brook Water.*—The ram-water supply from the brook was used in particular for washing the machine-milker's hands before hand-stripping, and each udder and teats before milking, and for supplying the cooler and washing out the cowsheds. In addition it was used at the farmhouse for washing up the plates and dishes.

4. *Drainage therefrom—as any Contributing Source of Pollution to the Brook above the Ram-intake.*—The sanitary accommodation, used exclusively by the farm household, consists of a W.C. (supplied by ram-water) that discharges into a septic tank. The point of entry of the drainage from the farm is definitely below the point of intake of ram-water.

5. *Any Special Action taken.*—Immediate action taken was: (a) sterilization of the milk (from July 4 to 22); (b) removal of cows from access to brook; (c) use of well-water only; (d) cutting off the ram (following the bacteriological report on ram-water sample—July 12). Remote action taken was: (a) after a period of 18 days sterilization of the milk was no longer enforced; (b) after a period of 9 days the farmer was permitted to use chlorinated ram-water for washing out the empty cowsheds and for the cooler; (c) to continue employing well-water for cleansing the udders and teats and for washing the milker's hands and the bottles.

Commentary on Case Histories

Two cases (16 and 17) are worthy of special mention. Case 16 (see Table) gives an interesting history of combined Sonne dysentery and paratyphoid B infection. The source of infection remains a mystery. Case 17 gave a history of absence from the depot for several days during the incubation period, so the probability is that the source of infection was not derived from the depot; in any event, milk may definitely be ruled out. It is reasonable to suppose that the remaining 15 cases were associated in some way with spread of infection from the ram-water and/or the milker who contaminated the milk, or by secondary infection from another case (e.g., Cases 10, 11, 14, and 15 secondary to Case 9). Case 9, in my opinion, was attributable to raw milk drunk on July 2.

Whether the machine-milker (Case 5) was infected by ram-water from the cowshed tap or by direct contact with his son (Case 6) during his illness at home it is impossible to prove. As the onset of illness in Case 6 was estimated to be about June 5, it is safe to assume that the ram-water had been contaminated with paratyphoid B organisms some days previously and that the incubation period was exceptionally short (Warren, 1941).

Record of Laboratory Findings in Connexion with Cases

Isolation of Paratyphoid B Organisms by Culture from Faeces and Urine.—In studying the bacteriological results obtained from samples of faeces and urine submitted to the laboratory (see Table), it will be noted that there were 3 cases reported with urine + only; 9 cases with faeces + only; and 4 cases with faeces — and urine —. The first 3 cases recorded suffice to show how essential it is for specimens of urine to be sent to the laboratory; this is particularly important in the case of men, who micturate more frequently than women and who have correspondingly fewer facilities for washing their hands after the act. On the other hand, of course, women urinary carriers with unclean habits may be a greater menace to public health on account of their food-handling occupations.

Agglutination Tests.—With regard to the determination of the paratyphoid B Widal results, positive titres were obtained up to dilution 1/250 "H," except in two instances: Case 15 was admitted to hospital on obtaining an end-point of 1/125 "H," as the child was obviously a secondary case due to infection from Case 9. In Case 16 there was not sufficient blood to proceed further with the dilutions; but there is no doubt about the diagnosis, for paratyphoid B bacilli were isolated from the faeces.

Blood Cultures.—Blood cultures were carried out on 4 patients in one family, the primary case of which—Case 9—was admitted to hospital on the recovery of paratyphoid B organisms from the stools. Only one positive paratyphoid B culture was obtained: this patient subsequently died.

Special Bacteriological Investigation

As a matter of exceptional interest, Mr. Monk submitted several cultures of paratyphoid B bacilli (through the medium of the Ministry of Health Regional Office) to Dr. A. Felix for phage typing. All these cultures, which were isolated during the outbreak, were tested against Vi phage preparations and found to react in an identical manner. The cultures were isolated from two patients, from the sewage effluent at the depot, and from the ram-water at the farm; and the result indicated that they all belonged to the same phage type.

Period of Survival of the Bacilli.—Using the dye method, it was estimated that the paratyphoid B organisms travelled through two miles of open stream for about 6 hours 7 minutes, and then through slightly less than 1/3 mile of piping for 34 minutes; the total distance from the sewage outfall at the depot to the tank at the bottling establishment taking 6½ hours. These times and distances have been included in order to show the conditions through which *B. paratyphosus* B survived.

The Effluent Dilution Factor.—From observation alone it was evident that the effluent from the depot constituted a substantial fraction of the volume of the brook—even by the time it reached the point of ram-intake, which was situated two miles below the outfall. So I decided to estimate this dilution figure experimentally.

Determination of the Dilution Factor.—First of all it must be understood that the dilution of the effluent by streamlets within the precincts of the depot—over a distance of about 1/4 mile—may be regarded as negligible from a practical point of view. The degree of dilution undergone by the effluent at a point in the stream below the last tributary and above the position of the ram-intake was estimated. A dye was used for timing the speed of the stream. The true dilution factor was less than 1 in 4; or, putting the result still more dramatically, the effluent was diluted by slightly less than three times its volume of stream water along its course to the ram. Under weather conditions existing at the time of this experiment the dilution at the point of ram-intake was probably greater than just before the actual outbreak. I am indebted to Mr. W. Pembleton, the district sanitary inspector, for assistance in carrying out this investigation.

Revelations and Recommendations

The epidemic illustrates two important facts:

1. *Potential Dangers of Hydraulic Ram-water Supplies under Wartime Conditions.*—Sewage effluents must find some outlet for discharge, and, in rural districts, brooks may be regarded as their natural course. The potential risk run by persons using hydraulic ram-water for domestic purposes must therefore be apparent, even though it may not be very significant. However, when an individual happens to be a food-handler, the health of a community may be endangered. Therefore, every farmer making use of a ram-water supply for cleansing the cowsheds and dairy must regard this water as potentially dangerous—for brook water is liable at any time to pollution. Ram-water really consists of a mixture of samples collected from a brook over a continuous period. Any sample taken direct from a contaminated brook may contain no harmful organisms, whereas a specimen of ram-water from the same brook is more likely to do so, for it is an accumulation of an infinite number of samples. This fact is pointed out as it is not generally realized that ram-water is even more unsafe than brook water *per se*. All users of ram-water should therefore take warning of the dangers of ram-supplies—especially under wartime conditions.

2. *The Importance of a Primary Planning Co-operation.*—During wartime large factories and works of every description are springing up in rural districts all over the country. They are built so rapidly and so secretly that the powers-that-be seldom consult the local authority on matters regarding the discharge of their sewage effluents. Co-operation between any particular branch of the Services concerned and the sanitary authority is essential, and, in order that the local knowledge of the latter shall be employed to the mutual advantage of both parties, it must start at an early stage in the planning of the sewage works and before the actual building has begun. This planning co-operation is of particular importance in rural districts, where there is danger of the effluents contaminating brooks used for dairying purposes; for the local sanitary

authority is the one and only body possessing that local knowledge of the district so essential before the plans are made. In the interest of general public health it is vitally important that powers over which the civilian authorities have no control should not discharge their sewage effluents into streams in dairying localities without this primary planning co-operation with the local sanitary authority.

Conclusions

The outbreak was due to a combination of factors, the chief predisposing causes being: (1) a hot spell and drought for the previous six weeks; (2) the recent incidence of paratyphoid fever in surrounding districts; (3) the inadequate dilution of the effluent from one of the sewage works at the depot; and (4) the comparative proximity of sewage outfall to ram-intake.

I am very grateful to Dr. Wyndham Parker, county medical officer, for his valued advice on all matters and for kind permission to publish this article. Thanks are due to the local sanitary staff and to Mr. R. W. T. Owen, county sanitary officer, for his indispensable support in carrying out the investigations. I am indebted to all the medical superintendents of isolation hospitals and the district medical officers of health who were good enough to supply me with clinical data, and to the local general practitioners for taking such prompt action to establish an early diagnosis in suspected cases. Grateful acknowledgment is made to Mr. H. E. Monk, county analyst and bacteriologist, and to his staff, for their enthusiastic co-operation. I also wish to express my appreciation of Prof. J. F. D. Shrewsbury's kindly help and criticism.

FATAL PHOSPHORUS POISONING FROM AN EXPLOSIVE BULLET

BY

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It is well recognized that certain types of incendiary bomb may cause local damage to skin or a wound from phosphorus contained in them, but, so far as I am aware, there is no appreciable risk of poisoning from absorption in these cases. The proportion of phosphorus present is, I understand, quite low—only about 4%. The risk of absorbing a lethal amount of phosphorus is apparently very much greater in penetrating wounds caused by a certain new type of explosive bullet. In the case recorded below there seems to be no reasonable doubt that death was due to phosphorus. Similar cases may have occurred without recognition of the cause of death, and I feel that it is important that the medical profession should be on the alert for them.

Case Report

A Canadian airman navigator aged 24 was wounded in the early hours of Sept. 14 in action over enemy territory. His aeroplane landed at a near-by aerodrome, where he was given morphine and his wound was dressed with sulphanilamide. He arrived at the Norfolk and Norwich Hospital at 9 a.m. There was a ragged entrance wound on the outer aspect of the left thigh, 2 in. long. His abdomen was rigid and tender, with dullness over the lower half. He was suffering from extreme shock. Pulse 144; B.P. 65/0. Three pints of serum were given intravenously and his B.P. gradually rose to 120/60. A radiograph showed a metallic foreign body in the centre of the abdomen, a punch fracture of the wing of the left ilium, and also some small metallic fragments in the thigh above the entrance wound and below the fracture.

Operation was begun at 11.30 a.m. The abdomen was opened and over 2 pints of blood mopped up. A ragged portion of a distorted bullet was found embedded in the upper part of the mesentery. This was extracted, and large torn branches of the superior mesenteric vessels were ligated. A transverse tear in a loop of jejunum was sutured and invaginated. There were no other intra-abdominal injuries, but some scattered fragments of bone were present in the subperitoneal layer of the abdominal wall on the left side. The abdominal incision was closed without drainage. The entrance wound in the left thigh was then enlarged up to the site of the penetrating fracture in the ilium, the edges of the skin and damaged muscle were excised, and some loose debris wiped away with gauze. While doing this it was noticed with astonishment by myself and my assistant that the wound emitted a visible vapour. This vapour was then found to possess the smell characteristic of phosphorus. The wound was impregnated with sulphanilamide powder and lightly packed with gauze. My first impression on smelling the vapour was that it was due to a tracer bullet, but examination shortly afterwards in a dark room failed to demonstrate any phosphorescence. During the operation a blood transfusion of the same group (O) as that of the patient was started, and it was continued until he had received 3 pints. The blood

pressure at the end of the operation was only 84/40, but by 8 p.m. it had risen to 134/60.

The following day the patient's general condition was fair, with some vomiting. On the 16th he had improved, and his general condition was satisfactory: no vomiting; abdomen soft; no gas in the wound or spread of infection. On the 17th he was not so well, and was slightly restless. Abdomen soft; no vomiting. The wound in the thigh was unhealthy. On the 18th he was obviously deteriorating and very ill—restless and semi-comatose. There was a lemon tint in the skin. A blood count showed 3,000,000 red cells with haemoglobin only 38%. A pint of blood was given. No urine had been passed for 24 hours, and a catheter withdrew less than 2 oz. The blood transfusion was followed by a continuous drip of saline and dextrose. The abdominal wound broke down and was strapped.

On the 19th the patient was comatose. In spite of the many pints of fluid given intravenously the anuria continued, not more than 1½ oz. of urine being withdrawn by catheter. Examination of the blood showed that the haemoglobin had risen to 58%. The blood urea was 453 mg. per 100 c.cm. His pulse remained comparatively slow—92 to 100 during the day—and was of quite good volume. He died at 1.50 a.m. on the 20th—that is, 6 days after being wounded.

Report on the Necropsy, by Dr. G. P. C. Claridge, Hon. Pathologist to the Norfolk and Norwich Hospital

At the post-mortem examination a gaping wound was found in the left thigh from which there was a track up into the tissues of the left flank passing through the iliac bone. The edges of this wound showed some necrosis, but not more than one often sees in these war wounds. The peritoneal cavity was clean and the stitching of the intestine sound. The liver was not enlarged, but on section had a homogeneous appearance and the cut surface was of a dull yellow colour. Apart from some slight swelling of the kidneys there was nothing of note in the other abdominal and thoracic organs.

Microscopical sections of the liver show extensive necrosis with fatty degeneration; the lobules cannot be identified, while the liver cells, which can still be recognized as cells, have a hazy outline with absent or shadowy nuclei which take the eosin stain, and a granular and highly vacuolated cytoplasm. Sections of the kidneys show the changes of catarrhal nephritis, the cells of the convoluted tubules being enlarged, granular, and in many cases necrotic. These changes in the kidneys are in themselves insufficient to account for the anuria. The gross destructive lesion of the liver is typical of acute phosphorus poisoning.

Comments

The rapid deterioration in the patient's condition after making good progress for the first three days, with the local condition in the abdomen and thigh satisfactory, was most puzzling at the time. The anuria and uraemia led me to surmise that it might be caused by some incompatibility of blood, although there were no symptoms to suggest that during the transfusions. The microscopical appearance of the kidneys makes it unlikely that incompatibility had any part in the fatal result.

The condition of the liver is not readily explained by anything other than phosphorus poisoning. If that explanation is correct, the important point arises as to what could be done to eradicate or counteract the poison in similar cases in the future. A more extensive excision of the damaged area than usual is indicated, and the application of a chemical antidote. I am ignorant as to whether copper sulphate would be of value. I believe that its use in the treatment of phosphorus burns of the skin is mainly as a colour index of the exact position of the phosphorus, and that the phosphorus has to be scraped off and further copper sulphate applied, to discover if there is any more to remove.

Apart from the evidence of necrosis of the liver and the presence of the vapour in the wound, with its distinctive smell, I have recently collected further and more convincing proof that this case was one of phosphorus poisoning. Lieut. K. M. Wheeler, R.E., has, with much kindness, great enthusiasm, and some risk to himself, done some research work for me. He has matched the distorted remains of the bullet with some identical bullets from a batch found on a wrecked German plane. His investigation proved it to be an explosive bullet—containing a charge of high explosive and also 3¼ gr. of phosphorus—which is double the minimum fatal dose recorded (Sydney Smith, *Forensic Medicine*, 4th ed., p. 472). A replica of the striker of the detonator in the bullet was found in the portion of bullet removed from the patient; and