and fibrous healing is proceeding apace in the depths, and when 5 or even 10 local applications at intervals have been necessary to obtain a measure of bacteriostasis, which even then may be only temporary.

And as, according to the example cited, it is easier to treat to a successful conclusion the pneumonia from the outset than the complicating lung abscess later, so in cases of infected wounds involving bone it is prudent to employ full chemotherapeutic measures at the time of full surgical treatment rather than later, when sequestra and sinuses may have formed in association with established infection. Full surgical procedure is of first importance, but we would emphasize that in cases of the kind considered here the pre- and post-operative administration of a sulphonamide, preferably sulphathiazole, is equally essential, and should be supplemented by local application, especially when many fascial planes have been disturbed or there is much tissue damage. For minimum pre-operative medication, 6 g. of sulphathiazole within 4 hours yields a blood concentration of 4 mg. per 100 c.cm., so that intramuscular and intravenous routes are not always necessary to supply the immediately available sulphonamide requisite in the blood and tissue fluids.

There is little doubt that sulphathiazole should replace sulphapyridine, both orally and locally, in the treatment of such cases of staphylococcal infection, for this drug is better tolerated and has a greater bacteriostatic action. Further, adequate oral administration of sulphathiazole should be continued as necessary, so that essential tissue-fluid concentrations are present when multiplication of organisms occurs or is likely to occur, thus ensuring that bacteriostasis is sufficient to permit the several body-defence mechanisms to operate unhindered or in conditions as favourable as possible.

Comment

While the treatment of infected compound fractures in wounds is essentially surgical, the role of the sulphonamides must not be forgotten or ignored. Sulphathiazole particularly is a valuable supplement. Given orally and locally at the time of major surgical measures, it provides the desired maximum bacteriostatic action when and where the diminished remaining foci of infection are most open to successful attack.

Since staphylococcal osteomyelitis is likely to be a not uncommon feature of many severe wounds sustained in this war by civilians and Service personnel alike, it behoves those concerned with early treatment to ensure that surgical treatment is adequately supplemented by chemotherapy so as to prevent the establishment of infection in bone, the treatment of which is notoriously difficult.

Summary

Sulphathiazole is more efficacious than sulphapyridine in the treatment of wounds with compound fractures that have become infected with *Staph. pyogenes*. The necessity for pre- and post-operative chemotherapy with this sulphonamide is stressed, as is also the combination of local with oral administration, especially at the time of radical surgical treatment. Sulphathiazole applied locally unsupported by simultaneous oral administration is of little value in the treatment of established infections of the kind discussed. Four cases are described illustrating these observations.

Strains of *Staph. pyogenes* recovered from our cases showed diminution of the α -haemolysin production but no alteration in coagulase in association with a local environment containing sulphathiazole. No sulphonamide-resistant staphylococci were encountered.

Our thanks are due to the Medical Director-General of the Navy and to Surg. Rear-Admiral H. E. R. Stephens, R.N., for permission to record these cases. We are indebted to Messrs. May and Baker Ltd., who kindly supplied the sulphathiazole used in the treatment of our early cases; and to Sick Berth Chief Petty Officers F. C. Prince and A. R. Woodman for technical assistance.

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J. Lusser (*Ann. Pediatr.*, 1942, 152, 226) records his observations on 1,000 cases of scarlet fever at the children's clinic at Basle during the period 1930-40, 368 of which had relapses. 127 of the latter had recurrent eruptions. The second or third attacks usually had a mild course. The ages of the patients ranged from 1½ to 15 years; the incidence was highest between 4 and 15, while young children were less frequently attacked.

VENOUS PRESSURE DURING VENESECTION AND BLOOD TRANSFUSION*

BY

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AND

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In man few observations have been made on the changes in venous pressure during the withdrawal and administration of blood, although the effects during the intravenous injection of crystalloid solutions have been investigated (Altschule and Gilligan, 1938; Murphy, 1941). In the present paper observations of venous-pressure changes have been made upon healthy individuals during venesection, and upon unselected hospital patients during the administration of blood. As an extension of this work similar observations were made during the transfusion of a number of patients suffering from pulmonary disease: in this group the vital capacity was measured before, during, and after transfusion. In all cases preliminary and final arterial blood pressures were taken, the pulse and respiratory rates noted at frequent intervals, and any change in the clinical condition of the subject recorded.

The venous pressure was estimated by the direct method, using a modification of the original Moritz and von Tabora (1910) technique. A stand was made with a backboard, to which the vertical arm of the manometer was clamped. The crosspiece rested on a horizontal ledge attached to the backboard. The backboard-and-ledge system was easily adjustable to different heights. In place of the reservoir a 30-c.cm. syringe, also resting on the horizontal ledge, was fitted to the crosspiece. By means of this syringe the manometer (7 mm. bore) was filled with 3% sodium citrate solution, and thereafter was flushed with this solution when required. The reference point taken was that recommended by Lyons *et al.* (1938)—an arbitrary point, with the subject supine, 10 cm. above the posterior surface of the thorax. The arm used for the venous-pressure estimation was abducted to an angle of 45 degrees. Where necessary, to prevent venous spasm a hot-water bottle was placed underneath the arm. Warm citrate solution was used throughout. An intradermal injection of local anaesthetic was given before all venepunctures.

An antecubital vein was chosen. A 16 s.w.g. intravenous needle was then inserted and connected with the manometer. If the needle was well in position the fluid in the manometer fell rapidly at first but later more slowly until equilibrium of pressure between the fluid in the manometer and the blood in the vein was reached. At this point there were minor respiratory fluctuations, either spontaneous or easily induced by deep breathing and occasionally pulse transmissions. Approximately 1 to 5 minutes were allowed to elapse before proceeding further, to ensure a steady base line of venous pressure. Throughout the experiment a rapid return of the manometer reading to the original level, after the apparatus had been flushed with fresh citrate, was regarded as the best criterion of patency of the system. In 4 cases venous spasm resulted in unsatisfactory readings and these records had to be abandoned.

Venesection

To minimize anxiety in the subject only donors previously venesected were chosen. In all cases complete rest for 10 to 15 minutes in the supine position was imposed, to allow the blood pressure and pulse rate to become stable. The venous pressure was then determined. Venesection with a 10 s.w.g. intravenous needle was performed on the opposite arm. A sphygmomanometer cuff maintained at a pressure of 60 to 70 mm. Hg facilitated the flow of blood. The average time taken to collect 430 c.cm. was 4 minutes. The venous pressure and pulse rate were recorded after 100 c.cm., 200 c.cm., 300 c.cm., 400 c.cm., and 430 c.cm. of blood had been with-

* A report to the Medical Research Council from the S.W. London Blood Supply Depot.

drawn. In most cases the venous-pressure readings were continued at 5-minute intervals for 30 minutes after venesection.

In 19 of the 20 cases the initial venous pressure varied between 60 and 145 mm. H₂O. In one very obese woman the pressure was 167 mm. H₂O (Table I). In every case there

TABLE I.—Effect of Venesection on Venous Pressure, Blood Pressure, and Pulse Rate

Case	Venous Pressure (mm. H ₂ O)			Blood Pressure		Pulse Rate	
	Before Venesection	After Venesection	30 min. Later	Before Venesection	After Venesection	Before Venesection	After Venesection
1	115	35	44 (15 min.)	120/85	—	80	78
2	97	43	46 "	95/60	—	68	70
3	115	52	90 "	145/90	140/90	80	80
4	110	55	90	140/90	140/90	80	80
5	167	110	112	140/85	130/80	72	72
6	110	50	—	154/104	154/100	76	76
7	125	75	95	130/75	112/95	88	92
8	130	80	90	148/85	145/85	100	96
9	80	30	31	105/65	105/65	75	84
10	130	115	80	136/84	—	112	112
11	94	48	80	105/60	105/70	90	92
12	148	105	105	130/90	—	70	70
13	79	43	57	115/70	112/70	84	78
14	130	95	98	148/106	140/102	76	76
15	60	25	—	110/76	108/76	96	96
16	100	78	85	170/110	160/110	100	100
17	72	55	55	125/80	110/70	76	76
18	68	58	55 (15 min.)	118/75	—	88	94
19	145	110	109 "	155/95	135/75	100	76
20	75	30	53 "	115/80	115/60	88	56

was a fall in the venous pressure during bleeding, varying from 13 to 80 mm. H₂O, with an average of 43 mm. In most cases the venous pressure showed a fall after 100 to 200 c.cm. of blood had been withdrawn, and it continued to fall until the end of the venesection. After venesection the venous pressure rose gradually, but in none of the cases did it return to the original value within 30 minutes. The changes in the blood pressure and pulse rate were variable and not related to the constant fall in venous pressure.

Transfusion

Observations were made on 30 unselected hospital patients (19 males, 11 females) suffering from secondary anaemias. In many cases the slight degree of anaemia would not have justified transfusion in the ordinary way. The chest measurements and approximate height and weight of all patients were recorded. They were allowed one pillow. This had no noticeable effect on the venous-pressure level. The preliminary rest period of 15 minutes was imposed and the venous pressure determined. The opposite arm was used for the transfusion, which was administered by gravity with the Medical Research Council giving-apparatus. Warmed compatible blood was given to all cases. The amounts varied from 500 c.cm. to 1,000 c.cm. In 4 cases the venous-pressure system blocked towards the end of the transfusion. The venous pressure was noted so far as was possible after every 50 c.cm. of blood had been transfused. In 5 cases the readings were continued at 5-minute intervals for 10 minutes after the end of transfusion and in a further 5 cases for 20 to 30 minutes. A rise of 20 mm. or less has been considered insignificant.

Venous Pressure and Amount Given.—Of the 30 patients, 19 showed a rise in venous pressure greater than 20 mm. H₂O, 10 no rise, and 1 a fall. In 13 cases the rise was between 20 and 50 mm. and in 6 cases between 50 and 85 mm. H₂O. In each of the 19 patients who showed a rise in venous pressure this increase was approximately proportional to the amount given (see Graph).

Venous Pressure and Transfusion Rate.—14 cases were transfused at rates varying from 7 to 15 c.cm. a minute, the average being 12 c.cm.: 8 showed no change in venous pressure, 4 a rise between 20 and 50 mm., and 2 a rise between 50 and 80 mm. H₂O. 12 cases were transfused at rates of 16 to 30 c.cm. a minute, the average being 20 c.cm.: 3 showed no change in venous pressure, 6 a rise between 20 and 50 mm., and 3 a rise between 50 and 80 mm. H₂O. 4 cases were transfused at rates of 30 to 45 c.cm. a minute, the average being 37 c.cm. All 4 showed a rise: in 3 the increase was between 20 and 50 mm. and in 1 between 50 and 80 mm. H₂O. There was apparently no constant relation between the rate of transfusion and the rise of venous pressure, although

proportionally more cases showed a rise at the faster rates. Signs and symptoms suggestive of vasodilatation were encountered in 3 cases. In one of these the interpretation of the signs was complicated by the onset of allergic manifestations.

Graph of the venous-pressure changes during the transfusion of blood to 30 unselected hospital patients.

Case 1.—Gastric ulcer in a spinster aged 33, weight 7 st. 12 lb., height 5 ft. 4 in.; Hb 66%. The patient was given 540 c.cm. of blood at an average rate of 22 c.cm. a minute. The venous pressure at the end of transfusion showed a rise of 36 mm. H₂O. The blood pressure was 110/65 before and after transfusion. After 250 c.cm. had been given, flushing of the face and arms was observed and at the same time the patient complained of hotness of the face. This was followed successively by complaint of a throbbing sensation in the head and tingling of the lips. The flushing of the face and arms was present for several hours after the completion of transfusion. There was no rise in temperature.

Case 2.—A married woman aged 42; diagnosis—carcinoma of the rectum: weight 8 st. 4 lb., height 5 ft. 5 in.; Hb 78%. The patient was transfused with 950 c.cm. of blood at a rate of 16 c.cm. a minute. The venous pressure showed a rise of 64 mm. H₂O. The blood pressure before transfusion was 135/90 and immediately after transfusion 100/55. When 850 c.cm. had been given she complained of throbbing in the ears, followed in three minutes by palpitations, a feeling of fullness and hotness, and a generalized pumping sensation. After the onset of the symptoms, flushing of the face and both arms was noticed, and this lasted for 10 minutes after the completion of transfusion. There were no further signs or symptoms.

Case 3.—A married woman aged 61; a convalescent air-raid casualty: weight 7½ st., height 5 ft. 3 in.; Hb 66%. The patient was transfused with 990 c.cm. of blood at a rate of 15 c.cm. a minute. The venous-pressure rise was 38 mm. H₂O. After 450 c.cm. the patient complained of generalized hotness. After 600 c.cm. oedema of the right eyelid was observed, followed by flushing of the face. After a further 250 c.cm. the left eyelid also became oedematous, the flushing still being present. All signs had disappeared within 2 hours.

These 3 patients were transfused at rates varying from 15 to 22 c.cm. a minute (average 18 c.cm.). The rise in venous pressure varied from 36 to 64 mm. H₂O (average 46 mm.). In Cases 1 and 2 the venous pressure had risen significantly before the onset of signs or symptoms of vasodilatation, and remained steady in Case 1 and continued to rise in Case 2 until the end of transfusion. In Case 3 the venous pressure blocked just after the vasodilatation became evident.

The 10 cases in which recordings were continued after the end of transfusion showed a gradual fall in venous pressure, but in no case was there a return to the basal level within the next 10 to 30 minutes. In 26 of the patients blood-pressure readings were taken before and after transfusion. The changes after transfusion were variable and apparently not related to the frequent rise in venous pressure. Only minor variations in pulse rate were recorded, and these were inconstant.

Transfusion of Cases of Pulmonary Disease

Examination was made of 13 such cases—3 females, 10 males. The patients were allowed two flat pillows instead of one, as most of them found the supine position irksome. This position was maintained for at least 15 minutes before the vital capacity was recorded. The vital capacity was measured again after 500 c.cm. of blood had been given and at the end of transfusion (after 1,000 c.cm.). A Kendrick recording spirometer was used. In patients with rib resection the arm opposite the resected ribs was used for the venous-pressure estimation.

The transfusion rates given below are calculated from the actual time during which the 1,000 c.cm. of blood was being administered. However, it must be pointed out that after the first 500 c.cm. had been administered there was a time interval varying from 6 to 15 minutes (average 8 minutes), during which the vital capacity was being recorded and no blood transfused. The transfusion rates varied from 17 to 40 c.cm. a minute (average rate 23 c.cm.). The amount given was 800 to 1,000 c.cm. One case had 500 c.cm. only.

Vital Capacity.—With one exception every patient showed a decrease in vital capacity after receiving 1,000 c.cm. (1 case 500 c.cm.) of blood. The percentage reduction bore no constant relation to the original vital capacity. Clinically, 3 patients

TABLE II.—Effect of Transfusion of 1,000 c.cm. of Blood on Vital Capacity and Venous Pressure

Case	Sex, Age, Diagnosis	Rate (c.cm. per min.)	Vital Capacity (c.cm.)		Venous-pressure Change during Transf. (mm. H ₂ O)	Transfusion Signs or Symptoms
			Before Transfusion	Reduction after Transf.		
1	M. 43 Empyema	33	2,450	150	+45	Headache
2	M. 44 Lung abscess	40	2,250	150	+77	Nil
3	M. 25 Pneumectomy	20	1,900	350	+21	"
4	M. 52 Carc. bronchus	23	1,750	400	— 1	Nil. (Only 540 c.cm. blood given)
5	M. 59 Empyema	28	1,700	150	+81	Nil
6	M. 39 Pneumectomy	22	1,450	150	+66	"
7	M. 50 Carc. bronchus	19	1,300	300	+33	"
8	F. 55 Empyema; rib resection	18	1,300	100	+ 6	Rigor 40 min. after transfusion
9	M. 21 Empyema; rib resection	20	1,250	200	—35	Urticaria, headache
10	M. 38 Pneumectomy	25	1,250	300	+14	Urticaria
11	M. 21 Pneumectomy	20	1,150	+50	—20	"
12	F. 45 Empyema; rib resection	17	1,100	300	— 5	Rigor 30 min. after transf. Dry cough during following night
13	F. 32 Pneumectomy	18	750	200	— 5	Dyspnoea during following night

*showed an increase of above 4 respirations a minute during the transfusion. One of these complained of a dry cough and another of dyspnoea during the night following the transfusion; the third patient, however, had no symptoms (Cases 12, 13, and 10, respectively; Table II). To exclude

the possibility that the recumbent attitude was responsible for the reduction in vital capacity one normal subject and Case 10 of the pulmonary disease series assumed the posture for one hour without being transfused. There was no change in vital capacity in the normal subject, and only a 50 c.cm. drop in the other.

Venous Pressure.—Of the 13 cases of pulmonary disease 6 showed during transfusion a rise in venous pressure greater than 20 mm. H₂O, 2 a fall, and 5 no appreciable change. Again, in the 6 cases showing this rise the increase was approximately proportional to the amount given. None of these 13 patients developed signs or symptoms suggestive of vasodilatation.

Discussion

Normal Venous Pressure.—Lyons *et al.* (1938) consider that the normal venous pressure lies between 50 and 150 mm. H₂O. The same reference point being used, the cases in this investigation showed a similar range of venous pressure; though in one obese woman it was 167 mm. H₂O. According to Kroetz (1922) a raised venous pressure is common in obese subjects.

Venous Pressure During and After Venesection.—The constant and often striking fall in venous pressure during venesection (Table I) was, under the conditions of this investigation, unrelated to the arterial blood pressure or pulse rate. The subjects were a selected group in that all had previously undergone venesection. None had an established vasomotor collapse ("faint"), and so no record of the venous pressure during such an attack was obtained.

Venous Pressure During and After Blood Transfusion

Altschule and Gilligan (1938) in their study of the dynamics of the circulation used saline as a transfusion fluid and recorded the venous pressure at intervals. They found that it rose in the majority of cases transfused at rates above 20 c.cm. a minute, and this rise was proportional to the amount of fluid given. The venous pressure fell rapidly immediately after the end of these transfusions and reached the original level within 20 minutes. A rise in pulse pressure was commonly seen during transfusion. In all their cases receiving large-volume (1,000 to 1,500 c.cm.) transfusions a widespread flush was observed. The rise in venous pressure they interpret as being due to a failure of the peripheral vascular bed to dilate rapidly enough to accommodate the increased blood volume. Murphy (1941), who also used saline as a transfusion fluid, noted a rise in venous pressure during the first half-hour of transfusion. This was followed by a fall attributed by him to vasodilatation. There is no mention of this having been observed clinically, nor is it clear at what intervals the venous-pressure readings were recorded.

The present investigation, in which blood was used as a transfusion fluid and the venous pressure was recorded at frequent intervals, gave results similar to those of Altschule and Gilligan. As stated, 19 of the 30 cases had a rise of venous pressure greater than 20 mm. H₂O. This rise was approximately proportional to the amount of fluid given. The rate of transfusion played a less important part, but on the whole the faster rates showed greater venous-pressure rises. No correlation could be drawn between the venous-pressure rise and the age, sex, haemoglobin values, or chest measurements of the recipients.

In contrast to the findings of Altschule and Gilligan, the fall in venous pressure after the completion of the transfusion was slow. Moreover, in none of the 10 cases which were observed for the subsequent 10 to 30 minutes was there a decrease to the pre-transfusion level. This is probably due to the fact that blood was used as the transfusion fluid, and as a result the increase in blood volume was maintained for a much longer period than after the infusion of crystalloid solutions.

Symptoms and signs suggestive of vasodilatation were encountered in 2 patients who received 1,000 c.cm. of blood and 1 who received 500 c.cm. The rate of transfusion was greater than 15 c.cm. a minute and the rise of venous pressure greater than 30 mm. H₂O. However, other patients transfused at similar and faster rates and showing similar rises in venous pressure experienced no symptoms. All 3 patients were females of small stature. Although not beyond the limits of

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