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ANAESTHESIA FOR AORTIC RECONSTRUCTION*

BY

HARRY L. THORNTON, F.F.A.R.C.S., D.A.

Consultant Anaesthetist, St. Mary's Hospital, London, W.2

Reconstruction of the thoracic and abdominal aorta calls for an anaesthetic technique appropriate to other operations in the region of these structures. There are, however, certain features peculiar to this branch of surgery which demand some special thought from the anaesthetist. Apart from the technical problems involved in maintaining (often for many hours) an even state of anaesthesia, combined with the profound degree of muscular relaxation necessitated by the surgical approach, there are other, and vital, matters concerning blood and respiratory chemistry, fluid and blood replacement, and the control of the haemodynamic disturbances of the surgical procedure. The nature of the operation may necessitate interruption of the circulation to vital organs for long periods, and in such cases the operation can be conducted safely only with the aid of induced hypothermia; this technique will itself impose some problems of anaesthetic management.

The anaesthetic techniques described below have been followed in 71 of the cases of aortic occlusion or aneurysm undergoing operation by members of the surgical unit of St. Mary's Hospital, London, under the direction of Professor Charles Rob. The anaesthetic procedure was conducted by three members of the hospital consultant staff, and their assistants. The 71 cases concerned are briefly summarized in Table I.

*Abstracted from a paper read before the Section of Anaesthetics at the Annual Meeting of the British Medical Association, Brighton, 1956.

TABLE I

	Requiring Hypothermia	Not Requiring Hypothermia
Aneurysm	5	37
Thrombosis	6	22
Coarctation	1	0
Total	12	59

TABLE II.—Fatal Cases

Requiring Hypothermia	Not Requiring Hypothermia
Aneurysm:	Aneurysm:
Haemorrhage	Haemorrhage
Suprarenal failure	Myocardial infarction
Myocardial infarction	Paralytic ileus and pulmonary sepsis
Thrombosis	Undecided
Coarctation	Haemorrhage
Total	Coronary thrombosis
3	9

The operative mortality (deaths occurring in the first 24 hours of the operative period) was 12 out of 71 (16.9%) (Table II).

Anaesthesia as a contributory cause of death could be implicated for certain in one case (pulmonary sepsis following aspiration of vomitus during induction of anaesthesia for reopening abdomen 12 hours after operation for aortic aneurysm. Ileus not diagnosed pre-operatively). One other case (listed as "undecided") was a possible "anaesthetic death." In the light of later experience it was thought that respiratory acidosis might have been a contributory factor (see last two cases in second column).

Type of Patient

Although, in the main, the patients likely to be encountered in surgery of the aorta may be described as being "in the prime of life," they are by no means in the prime of physical condition. With some few exceptions (such as the rare traumatic case, and some cases of aortic coarctation) they have reached middle or late middle age, and are suffering from severe arterial disease. Atherosclerosis is not likely to be confined to the portion of the aorta requiring surgery; coronary, cerebral, and renal vessels are likely to share in the general disease. Often there is hypertension, which may be of severe degree. There may have been past episodes of cardiac infarction or cerebral thrombosis. Such patients tend to be "old beyond their years," physically flabby, and often obese.

This is our somewhat unpromising material, and to subject such patients to major surgery, with the attendant hazards of blood loss and traumatic shock, under general anaesthesia (possibly maintained for as long as 12 hours), without the most careful pre-operative assessment and preparation, would be to court almost certain disaster. Only in the case of true emergency (such as leading aneurysm or haemorrhage from other causes) is it justifiable to omit the full pre-operative routine which is described below. The period of assessment and preparation may precede the operation by 7 to 10 days.

Pre-operative Investigations

Aortography.—In all cases the site of the lesion is determined by an aortogram, usually performed under local analgesia, assisted by intravenous pethidine. Only in exceptionally difficult cases, or with unusually apprehensive patients, will a general anaesthetic be necessary; since complete apnoea at the time of the exposure and the prone position are demanded, this will require tracheal intubation

and controlled respiration. The choice of the subsequent anaesthetic technique will depend on the site of the lesion as revealed by the aortogram. Should the lesion be estimated to lie 2 cm. or less below the level of the renal arteries, clamping of the aorta above this level and interruption of the renal circulation must be envisaged. In lesions of the thoracic aorta the proximal clamp will occlude some part of the blood supply to the spinal cord via the spinal branches of the posterior intercostal arteries. Such cases will call for induced hypothermia.

In addition to a complete physical examination the following routine investigations are carried out on all cases: complete blood count; estimation of haemoglobin, serum proteins, blood sugar, and blood urea; and a complete picture of the plasma electrolytes. Renal and respiratory function tests are made. Wherever electrolyte imbalance is found, every endeavour is made to restore normality, and not until stability seems assured is surgery contemplated. Certain cases require additional investigation and preparation. Of particular importance are those showing respiratory insufficiency; an important example of this group is that of chronic emphysema, which most anaesthetists will recognize as a troublesome problem in major surgery. In all such cases the blood oxygen content, pH, and plasma carbon dioxide content are determined, and operation is deferred until physiotherapy, together with antibiotic treatment of residual infection, has resulted in maximal improvement in the patient's vital capacity. The problem of respiratory acidosis is of great importance in the management of anaesthesia for aortic surgery—and, indeed, for most other types of thoracic and abdominal surgery—and is referred to below.

On the night before operation water diuresis is induced by the slow intravenous infusion of 1–2 litres of 5% glucose in water. This is believed to give some measure of protection from subsequent renal failure, particularly when surgical interruption of the renal circulation is anticipated (Wynn, unpublished observation).

It will be seen that cases for aortic reconstruction (or disobliteration) fall into two main groups: (1) those with lesions situated more than 2 cm. below the renal arteries, and (2) those with lesions at, above, or close to the renal arteries or involving the thoracic aorta—that is, cases not requiring induced hypothermia (group 1), and those in which induced hypothermia is obligatory (group 2). For convenience these two groups are considered separately with regard to management, the simpler (non-hypothermic) group being taken first.

Anaesthetic Management of Group 1 Cases

In contrast with those of the second group, group 1 cases will suffer little disturbance of normal physiology, except that at certain stages in the operation severe variations in blood pressure are to be expected and considerable loss of blood may be inevitable. As in all abdominal surgery, muscular relaxation of a high degree is demanded, and, since the operation is likely to be lengthy, the anaesthetic agents must be of minimal toxicity. Our choice at St. Mary's Hospital is nitrous oxide and oxygen, preceded by thiopentone, and supplemented by pethidine. Curare is used to provide the necessary relaxation. Endotracheal intubation is invariably employed, and respiration is controlled manually throughout the operation, with carbon-dioxide absorption on the "to-and-fro" principle. We are careful to avoid small gas-flows, with their potential danger of underventilation.

The administration of parenteral fluids, both in replacement therapy and as vehicles for drugs required in the course of operation, has received some special attention from the team in our metabolic unit. This matter is discussed below. For group 1 cases we prefer to use an isotonic dextrose-saline solution (dextrose 4.3%, saline 0.18%), which is usually administered through a "polythene" intravenous cannula. The solution can be exchanged for whole blood when necessary. An intra-arterial blood transfusion can be set up at very short notice. Since the lower limbs

are not available for this purpose (on account of surgical occlusion of the aorta), a transfusion set, kept externally sterile, is retained for the use of the surgeon as need arises. We do not favour the use of the radial or other proximal limb arteries for transfusion purposes, because of the risk of spasm and ischaemia. In most cases the aorta, proximal to the clamps, is the vessel of choice. The necessary transfusion pressure is obtained by suspending the blood-bottle from the theatre gallery at a height of about 10 ft. (3 metres), an extension of the transfusion tube allowing for this distance. Blood pressure and pulse rate are recorded at 10-minute intervals, and electrocardiographic tracings are made when required.

Recently we have come to appreciate the great importance of maintaining a normal blood pH, and, in order to detect and check developing respiratory acidosis, estimations of arterial blood pH and total carbon dioxide are made at frequent intervals; or whenever there is clinical suspicion of rising blood pCO₂ (rising systolic and diastolic blood pressure, sweating, tachycardia). Further estimations are made at the conclusion of operation, when spontaneous respiration is re-established.

Blood Replacement.—So far as is possible, blood replacement is adjusted to blood loss. The suction-bottle is calibrated in millilitres, and all swabs and packs are weighed before and after use on a sterile balance, blood loss being calculated on a weight-volume coefficient. Since no wet packs are used, an estimate accurate to $\pm 10\%$ is possible. The most critical period of the operation is when the distal aortic clamp is released. Close co-operation between surgeon and anaesthetist ensures that the latter has ample warning and can take the necessary action to avert a dangerous fall in blood pressure. Provided that the intravenous blood

FIG. 1.—Pneumatic tourniquets control flow of blood to legs on release of clamps.

transfusion is capable of a rapid increase in rate of flow (500 ml. in five minutes) at least five minutes before release of the clamp, it is seldom necessary to transfuse intra-arterially. To prevent too rapid a loss of blood into the lower limbs we use pneumatic tourniquets, which can be gradually deflated, and, if necessary, reinflated to check dangerous falls in blood pressure (Fig. 1). Unless unusually severe leakage at the anastomosis occurs on removal of the clamps, blood pressure should be controlled and stabilized within 5–10 minutes.

Anaesthetic Management of Group 2 Cases

In the surgical unit of St. Mary's Hospital we have found the method of *surface-cooling* both simple and effective. We aim at reducing the body temperature to between 26 and 27° C. (78.8 and 80.6° F.), and require about one and a half hours to achieve this degree of hypothermia. The procedure is as follows.

One hour prior to cooling—that is, two and a half hours before the planned time of operation—the patient is premedicated with pethidine, 100 mg., scopolamine, 1/150 gr.

(0.43 mg.), and chlorpromazine, 25–50 mg. intramuscularly. My own practice is to give chlorpromazine, 25 mg., some days before operation, and to observe the effect. Should tachycardia or marked hypotension result I omit the chlorpromazine from the premedication entirely. If, on the

FIG. 2.—Set-up of anaesthetic and other apparatus during induction of hypothermia. Note ice-bags. *Left foreground:* Control for cutting in thermocouple leads for direct temperature readings. *Left background:* Electrocardiogram. (The apparatus attached to right lower leg is for measuring calf blood flow.) *Right foreground:* Anaesthetic apparatus and sphygmomanometer

other hand, the drug is well tolerated, I give the larger or the smaller dose, according to the patient's fitness and physique. We have not been impressed with the "lytic cocktail" or other methods of so-called "chemical hibernation"; indeed, we feel that such techniques may add further hazards to the operation, tending to aggravate hypotension and to produce excessive thermo-lability.

Induction of anaesthesia is with thiopentone, and tracheal intubation is performed with the aid of a short-acting muscle-relaxant (succinylcholine).

Maintenance of anaesthesia is by nitrous oxide and oxygen with manually controlled respiration. Increments of pethidine are added intravenously as required. Before cooling is started, tubocurarine chloride, 20–30 mg., is administered. This not only facilitates the control of respiration in a light plane of anaesthesia, but is essential in preventing shivering, with the attendant risk of ventricular fibrillation.

Cooling (conducted on the operating table) is begun by placing ice-bags in close contact with the patient's exposed neck, thorax, abdomen, flanks, thighs, and legs, covered overall by a wet cotton sheet, from which evaporation is assisted by electric fans. The theatre temperature is kept as low as possible (40–60° F.; 3.3–15.5° C.). Body temperature is constantly recorded by thermocouples placed in the upper and lower oesophagus and rectum. Pulse, blood pressure, and electrocardiographic tracings are taken at short intervals, the latter being particularly valuable in giving warning of ventricular arrhythmias (Fig. 2).

Since Wynn (1954) has shown that glucose ceases to be metabolized at a body temperature below 31° C. (87.8° F.), the only parenteral infusion used, apart from blood, is half normal saline, to which is added such drugs as are required in the course of operation. All active cooling is stopped when the body temperature has reached about 28° C. (82.4° F.), after which, in a cool theatre, it will continue to fall until it stabilizes at between 26 and 27.5° C. (78.8 and 81.5° F.). Too rapid a fall in temperature can be checked

by removing some of the ice-bags, stopping the fans, and recovering exposed parts of the body with blankets. Operation is started as soon as the required basic temperature has been reached. At this stage it is usually necessary to give a further small dose of curare and/or pethidine.

Provided that the cooling process has proceeded smoothly, the patient, although presenting a somewhat alarmingly lifeless appearance, should by this time have suffered little or no fall in blood pressure. The pulse rate will have fallen considerably (50–60 a minute).

Cases of this group require especially careful handling at the time when the distal aortic clamp is released, for, although vasoconstriction is active as a result of the hypothermia, reflex hyperaemia will still occur in the lower limbs, in response to the restored circulation (Fig. 3). Unless this is adequately controlled by the tourniquets a hypotensive crisis will be precipitated, and this is likely to prove more difficult to treat than in the non-hypothermic cases. Infusion of nor-adrenaline (1/500,000) will be of great help in such cases, provided that there is not the additional complication of uncompensated blood loss.

A danger associated with massive blood transfusion is inherent in the quantity of sodium citrate which may be infused. Like glucose, the citrate ions of sodium citrate are not metabolized in the hypothermic state, and may reach a concentration high enough to be toxic to the myocardium (Bunker *et al.*, 1955). Furthermore, stored blood has a high potassium-ion concentration which can cause cardiac arrest. In all cases it is advisable that only freshly donated blood

be used and that an adequate supply be available at the time of operation. (Aneurysm cases may require as much as 3–4 litres; thrombosis cases, somewhat less.) If the quantity of citrated blood is necessarily heavy (above 4 litres) we advise the addition of calcium gluconate, 0.5 g. to each half-litre.

Post-operative Points

At the conclusion of the operation the anaesthetist must be satisfied upon the following points: (1) blood loss has been restored (verified gravimetrically and volumetrically);

(2) blood pressure is stable at a safe level (not less than 100 mm. Hg systolic, with or without noradrenaline); (3) the neuromuscular block has been fully reversed by neostigmine; (4) central respiratory depression has been abolished (this may require nalorphine); (5) the blood pH and pCO₂ are normal and stable; and (6) rewarming of the hypothermic patient is proceeding at the rate of 1–1.5° C. an hour, but no faster.

FIG. 3.—Reflex hyperaemia of lower limbs following release of aortic clamp.

These last two points deserve some further amplification, and the fifth point—that is, the matter of blood pH—will be taken first, since this applies to both hypothermic and non-hypothermic cases, and is closely related to the problems

of persistent neuromuscular block and central respiratory depression. In a proportion of cases, Wynn (1956, personal communication) and the metabolic team of the surgical unit at St. Mary's Hospital have found that there is a tendency to recurrence of a neuromuscular block which has been previously reversed by neostigmine at some time during the recovery period. It is possible that in the hypothermic patient curare may persist in some depot inaccessible to the action of neostigmine; or, in those cases where the renal circulation has been interrupted, there could be delayed excretion of curare. Undoubtedly there are other factors as yet not fully understood. Scurr (1954) has already drawn attention to the apparent synergism between carbon dioxide and the neuromuscular blocking agents, and it would seem that in certain circumstances a vicious circle could become established (Fig. 4).

To break this circle the full respiratory efficiency needs to be restored, and treatment must be at the appropriate level. Thus, if reversal of central respiratory depression plus reversal of neuromuscular block (nalorphine plus neostigmine) fails to restore the *status quo*, artificial ventilation aimed at elimination of carbon dioxide must be instituted without delay, and must be continued until the pH and $p\text{CO}_2$ return to normal and show no tendency to further change. Reintubation of the patient and connexion to a respiratory pump for an hour or more has sometimes proved necessary. Respiratory acidosis must never be allowed to develop. The longer treatment is delayed the more difficult will it be to reverse the process.

FIG. 4.—Vicious circle of carbon dioxide retention.

Illustrative Case

A somewhat obese plethoric man aged 44 had a recent history of claudication of buttocks and legs. Aortogram (under general anaesthesia) showed occlusion of abdominal aorta immediately below renal arteries, with well-marked collateral circulation. Operation was begun at 9 a.m. under induced hypothermic and thiopentone-nitrous-oxide-oxygen-pethidine-curare anaesthesia with manually controlled respiration. Duration of anaesthesia was about eight hours. Surgical procedure (disobliteration and graft) required clamping of aorta above renal vessels for about two hours. Operation was completed at 6 p.m.

Post-operative Course.—Patient regained consciousness and spoke rationally about 30 minutes after return to the ward. Although respiration was spontaneous on removal of endotracheal tube, there was marked "tracheal tug," which was not abolished by neostigmine, 2.5 mg., repeated after 20 minutes. Oxygen was administered by means of a plastic facepiece at the rate of approximately 8 litres a minute, a good colour being maintained. Rewarming proceeded normally; no shivering. Three hours later (9 p.m.) respiration became shallow and soon ceased, although the colour up to this time (with oxygen) had seemed satisfactory. Patient appeared deeply unconscious. Artificial respiration with oxygen by means of a bag and facepiece (later replaced by cuffed tube) was immediately begun. Blood samples were obtained for pH, HCO_3^- , and $p\text{CO}_2$, which revealed very gross respiratory acidosis (see Fig. 5).

Four hours later (1 a.m.) the patient still appeared deeply comatose; pH was now rising, and HCO_3^- and $p\text{CO}_2$ falling slightly. At this stage a Waters CO_2 absorber was placed in circuit. Much heat was generated, requiring change of canister after 20 minutes. Artificial respiration continued until 6 a.m. (a further five hours), when consciousness and spontaneous respiration returned. Blood samples now showed an almost complete return to normal, except the pH, which remained a little low (7.35) for about a further hour. Thereafter the patient made an uninterrupted recovery.

Comment.—An interesting feature of this case was revealed some time later. The patient, who was himself a

medical man, was able to recall in detail many of the events which took place when he appeared to be deeply comatose. This would seem to be a case of respiratory acidosis simulating complete neuromuscular block (Scurr, 1954).

Rewarming of the hypothermic patient requires the most careful vigilance on the part of medical and nursing staff. If

FIG. 5.—Case of severe carbon dioxide retention.

rewarming proceeds too rapidly, vasodilatation will result in a severe fall in blood pressure; if too slowly, shivering and shock may be produced. We believe that a safe average rate of rewarming lies between 1 and 1.5° C. an hour. Heat-loss is prevented by blanketing in preference to the application of outside sources of heat, such as electric cradles. Should the body temperature rise too rapidly, and result in a fall in blood pressure, it may be necessary to reapply tourniquets to the limb from time to time, whilst removing some or all of the blankets.

Conclusion

It should be observed that although the operative death rate in cases of aortic reconstruction might appear to be high in comparison with that encountered in the general run of major abdominal and thoraco-abdominal surgery, due appreciation must be given to the extreme severity of the operations performed on patients almost invariably in the "poor-risk" group. As in all comparatively new branches of surgery, improvement in this respect may be expected to follow future improvements in technique, both surgical and anaesthetic. A decrease in the incidence of operative shock and blood loss is already beginning to reflect a steady diminution in total operating-time, whilst experience has taught us to avoid many of the earlier hazards of anaesthesia.

The paramount importance of team-work in the management of these cases must be stressed. Without the close co-operation of the many special departments (electrocardiographic, metabolic, biochemical, to mention only a few of those whose services we are fortunate in having at our disposal) the anaesthetic and operative risk in major aortic surgery would be formidable indeed.

Summary

The pre-anaesthetic preparation, anaesthetic technique, and post-anaesthetic management of cases of aortic reconstruction are described.

Blood loss, blood and fluid replacement, and the control of blood pressure are discussed.

The technique of induced hypothermia by the method of surface cooling is described, and some relevant problems are considered.

The importance of maintaining a normal blood chemistry, with particular reference to the blood pH and pCO₂, is stressed.

I thank Professor Charles Rob for giving me access to case notes and statistics of the St. Mary's Hospital Surgical Unit; Dr. Victor Wynn for much valuable assistance and for supplying the graph (Fig. 5) from his personal notes; and Dr. P. N. Cardew, of the St. Mary's Hospital photographic department, for the illustrations and diagrams

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CANICOLA FEVER IN MAN THROUGH CONTACT WITH INFECTED PIGS

BY

JOYCE D. COGHLAN, Ph.D., B.Sc.

Lecturer in Bacteriology, University of Edinburgh

JOHN NORVAL, M.R.C.V.S.

Chief Veterinary Inspector to the City of Edinburgh

AND

H. E. SEILER, M.D., F.R.C.P.Ed., D.P.H.

Medical Officer of Health to the City of Edinburgh

Leptospiral infections in man resulting from contact with infected pigs have been reported from different parts of the world but have not previously been recorded in Great Britain. Thus the benign human leptospiroses known as "swineherd's disease" and "seven-day fever of Queensland" have followed contact with pigs infected with *Leptospira pomona* (syn. *L. suis*) and *L. mitis* (syn. *L. hyos*) respectively, while van Thiel (1948) quotes two published cases of Weil's disease where it seemed likely that the patient had contracted the disease through slaughtering pigs infected with *L. icterohaemorrhagiae*.

Neither *L. pomona* nor *L. mitis* has so far been isolated from man or pig in this country, but occurrences of *L. icterohaemorrhagiae* infection in swine in two rat-infested piggeries in the south-east of Scotland have been reported by Nisbet (1951).

One other type of leptospira—namely, *L. canicola*—has been isolated from swine. Williams *et al.* (1953) found during the investigation of an extensive outbreak of human canicola fever in a rural community in South Georgia that dogs, cows, and swine showed evidence of infection with *L. canicola*. All the human cases had bathed in the water of a creek to which animals had access, and, although the original source of infection remained obscure, it was the first record of *L. canicola* infection in animals other than dogs.

Previously it had been generally accepted that dogs were the only natural carriers of *L. canicola* (van Thiel, 1948) and that infection in man resulted directly or indirectly from contact with these animals. The disease in the dog varies in severity, but, whatever the degree of illness, the infection usually leads to leptospiuria, which

may persist for many months if recovery occurs, thereby providing a source of contamination for other dogs and for man. While the number of known cases of human canicola fever has been steadily increasing, its true incidence is difficult to assess, since it is a disease which must very often escape diagnosis. In some instances of human disease the history would seem to leave little doubt that infection was transmitted from a dog, but others have been recorded where no apparent connexion between patient and dog could be established (Broom, 1951) and where an alternative source of infection seemed probable. It may be that pigs are more frequently infected with *L. canicola* than is commonly supposed, and, if so, the question arises whether they may not act as reservoirs of infection to persons who feed and handle them. Investigations into a number of obscure cases of human canicola fever which occurred between 1950 and 1955 among piggery workers in and around the City of Edinburgh gave rise to a series of interesting results which point to the probable role of pigs in the transmission of canicola infection to man. These form the basis of the present communication.

Laboratory Methods

Agglutination-lysis tests were used for serological diagnoses. The methods of setting up the tests and interpreting the results were those developed by the staff of the Institute of Tropical Hygiene in Amsterdam, and are described in detail by Wolff (1954). Live cultures of leptospires were used, and the sera were diluted from 1 in 10 to 1 in 30,000. Titres of 1 in 100 and over are regarded as indicating past or present leptospiral infections. A few titres as low as 1 in 10 to 1 in 30 have been recorded, but they are to be treated with reserve since their significance is doubtful.

The isolation of leptospires from the organs of infected animals at necropsy was attempted by grinding small pieces of tissue in Korthof's medium in a Waring blender and inoculating tubes of medium with three or four drops of the emulsion so produced. Latterly, however, we were more successful in obtaining positive cultures by inoculating duplicate tubes of Korthof's or Stuart's medium with small pieces of kidney tissue punched out with a Pasteur pipette, after the surface had been seared with a red-hot scalpel blade (cf. Wolff, 1954). The tubes were incubated at 28° C. and examined at weekly intervals up to six weeks before being discarded as negative.

In addition to culture, hamsters approximately 3 weeks old were inoculated intraperitoneally with 0.5 ml. of tissue emulsions or with 0.5 ml. of centrifuged urine deposit suspended in 1 ml. of Korthof's medium.

Microscopical examinations of cultures, urine, and heart blood from experimental animals were made with dark-ground illumination using a 2-mm. objective.

Clinical Observations

The first case to be investigated occurred in November, 1954, when a man employed on a piggery (Farm A) was admitted to hospital with a feverish illness of eight days' duration characterized by severe frontal headache. Blood taken from the patient agglutinated *L. canicola* to a significantly high titre. Although, on inquiry, no history of recent illness was forthcoming, blood from four out of five other persons living and working on the farm gave positive agglutination findings. The results of these tests and other relevant features are shown in Table I.

There were three dogs on the farm, and, to determine whether they might have been responsible for the infection, specimens of blood and urine were obtained. Dark-ground examination of the urine failed to reveal leptospires. Two of the sera agglutinated *L. canicola* to titres of 1 in 30 and 1 in