Table III, where it is compared with that in a normal individual, in a patient with diabetic acidosis, in a patient dying of "chronic uraemia," and with that resulting from the severest degree of acidosis which could be experimentally produced in a normal individual by ingestion of large quantities of ammonium chloride. It is of interest that the severe acidosis of the present case was not accompanied by the slightest hyperpnoea or dyspnoea. It is usually held nowadays that the severe dyspnoea so common in uraemia is the result of acidosis following retention of phosphate by the kidney.

Chloride.—The whole-blood chloride fell definitely, but not excessively, below normal. This fall, which occurred in spite of salines and calcium chloride, accompanied the excessive acidosis described. In most cases of urinary suppression, however, the chloride rises—often to excessive heights if the patient continues to ingest salt. Fishberg¹⁸ reports two cases in which it rose to 1,100 and 930 mg. per cent. respectively. In cases of severe vomiting, on the other hand, the loss of chloride in the vomit is followed by a twofold change in the blood chemistry. There is a marked fall in the blood chloride but a great increase in the alkali reserve, owing to replacement of the Cl ion by CO₂. In the present case the low chloride is undoubtedly the result of vomiting, but the alkalosis that might be expected to accompany it is converted into an acidosis by reason of the complete phosphate retention. Even so, the acidosis is relieved somewhat by the vomiting of free HCl.

Plasma Protein and Cholesterol.—The total whole-blood cholesterol as determined by the colorimetric method of Myers and Wardell was at first low, but rose to 188 mg. per cent. before death. The plasma albumin was somewhat diminished, but the globulin definitely increased with inversion of the albumin:globulin ratio. There does not seem to be any obvious explanation of this curious finding, which is not common in uraemia unaccompanied by albuminuria.

DISCUSSION

In this unfortunate youth, then, we have an example of pure and uncomplicated uraemia which I believe to be comparable in every detail to that observed in experimental animals following total nephrectomy. It will be noted, however, that certain features which are usually regarded as the most typical of chronic uraemia in man are conspicuously absent in this case. Chief among these are the convulsive phenomena. On the other hand, the toxic features of uraemia are very marked. The question, therefore, arises: Do all the features of so-called "chronic uraemia" in man result from renal failure or do they not?

This is a matter which has given rise to endless confusion ever since the days of Bright. There is, however, a marked tendency nowadays to regard the convulsive phenomena which are often associated with renal failure as vasomotor in origin, since they are almost invariably accompanied by vascular hypertension, the actual convulsion being preceded by a sharp rise in blood pressure. This is a fact which can easily be verified by anyone who studies a case of "uraemic fits" with a sphygmomanometer. Such fits, therefore, cannot be regarded as uraemic at all, in the sense that the symptoms of the nephrectomized youth were uraemic. Their separation as a distinct entity is due mainly to the work of Volhard. Their association with the true biochemical syndrome of uraemia, as here defined, is in a sense fortuitous and due to the fact that most renal diseases which are accompanied by renal failure are also accompanied by hypertension and vasomotor phenomena. The term "acute uraemia," which is so often used to describe such fits, is a peculiarly objectionable one. It will be seen that in the case of true acute uraemia here described, both fits and hypertension were absent. It would certainly result in clearer thinking if the term "uraemia" were rigidly confined to the biochemical syndrome following

renal failure, and all other features of different pathogenesis excluded.

There is thus no real difference between the uraemia of one disease and that of another, between the acute process of severe acute nephritis or bilateral obstruction of the ureters and the more insidious process of chronic nephritis or congenital cystic disease. The apparent clinical differences between one case and another are largely the result of the extrarenal factors which usually accompany the true biochemical syndrome, whereas the latter varies in degree only but not in kind from case to case. If this view were more generally adopted we would have less difficulty in understanding the nature of convulsions occurring in acute nephritis and in chronic nephritis, and distinguishing them from the true uraemic phenomena of the same diseases.

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A SURVEY OF 340 CASES OF ACUTE APPENDICITIS

BY

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Between December, 1927, and December, 1931, I operated on 340 cases of acute appendicitis admitted to the Dunfermline and West Fife Hospital. All the cases were operated on within a few hours of admission; delayed treatment of the Ochsner-Sherren type was not attempted. Excluded from this series are cases of subacute and catarrhal appendicitis.

STATISTICAL MATTER

The cases have been divided into three groups according to the type of operation performed. The first table shows the average length of time ill before operation and the mortality rate for each group. The appendix was removed in all cases, with the exception of thirteen, where abscess was present and the appendix inaccessible; in these, simple drainage of the abscess was instituted.

TABLE I.—Results of Treatment

Group	Type	Hours Ill (Average)	Operation	No. of Cases	No. of Deaths	Mortality
1	Acute appendicitis	15	Appendectomy	223	1	Per cent. 0.49
2	Acute appendicitis with local peritonitis or abscess	80	Appendectomy where feasible and local drainage	64	3	4.7
3	Acute appendicitis with generalized peritonitis	56	Appendectomy and suprapubic drainage	53	10	18.8
	Total ...			340	14	4

In Table I the most noteworthy feature is the length of time ill before surgical treatment was sought. In Groups 2 and 3, which comprise 117 cases, it will be seen that more than fifty hours elapsed before these cases arrived for operative treatment. The cause of the delay was investigated, and in all but a small percentage was found to be due to the patient or his friends temporizing with homely remedies before calling in a doctor. It was surprising the large number of cases which had been supplied with castor oil or salts prior to having been seen by a doctor. This free exhibition of purgatives has much to do with the rapid advancement of the symptoms and the pathology of the disease. In several cases the delay was due to difficulty in diagnosis and temporizing until the diagnosis was definite. This was especially the case in patients whose appendix occupied the pelvic position. Of the fourteen fatal cases in this series no fewer than seven suffered from ruptured pelvic appendices, and the average length of time ill before operation worked out at eighty hours for the seven cases (Table II).

TABLE II.—Fatal Cases

No. of Case	Age in Years	Sex	Hours Ill	Position of Appendix	Operative Findings	Subsequent Treatment	Survived
1	5	F.	78	Pelvic	Ruptured; generalized peritonitis	B. welchii; enterostomy	72 hrs.
2	18	M.	82	Retro-caecal	Sphacelated; general peritonitis	B. welchii; enterostomy	7 days
3	51	F.	104	Splenic	Ruptured; general peritonitis	B. welchii	12 hrs.
4	6	M.	144	Pelvic	Ruptured; general peritonitis	Enterostomy	24 hrs.
5	43	M.	106	Lateral caecal	Appendix abscess; drainage only	B. welchii; developed subphrenic abscess	3 weeks
6	47	F.	268	Retro-caecal	Abscess; drainage only	B. welchii	5 days
7	60	M.	72	Retro-caecal	Ruptured; general peritonitis	B. welchii; enterostomy	5 days
8	26	M.	78	Pelvic	Diffuse peritonitis enterostomy	B. welchii	7 days
9	39	F.	80	Pelvic	Ruptured; general peritonitis	B. welchii	13 days
10	25	M.	104	Pelvic	General peritonitis	Enterostomy	6 days
11	16	F.	30	Pelvic	Gangrenous appendix; no drain	Developed portal pyaemia	4 days
12	10	F.	48	Pelvic	Gangrenous appendix; local peritonitis	B. welchii; enterostomy	4 days
13	13	M.	56	Splenic	Ileocaecal abscess drainage	B. welchii	3 days
14	22	F.	48	Lateral caecal	Ruptured; general peritonitis	B. welchii	3 days

Thus out of fourteen fatal cases seven had the appendix in the pelvic position, three in the retrocaecal, two in the splenic, and two more in the lateral caecal. These figures seem to point to the pelvic position of the appendix being the most dangerous. As Cope has pointed out, local signs are few in pelvic appendicitis, and a rectal or vaginal examination is the only method of eliciting local signs in an early case.

TABLE III.—Age and Sex Incidence

Group	No. of Cases	Average Age in Years	Males	Females
1	223	23½	116	102
2	64	31	36	32
3	53	23	38	16
Total ...	340	—	190	150

The youngest in this series was 2 years of age, and the oldest 71. The sex incidence shows that 55 per cent. of the cases were males. This corresponds with what Sherren noted in 1925—namely, in 1903 acute appendicitis was two and a half times as common in the male as in

the female sex. In 1907 there were 65 per cent. males; in 1922, 55 per cent. males.

TABLE IV.—Position of Appendix in all Cases

Position of Appendix	Number of Cases	Number of Deaths
Splenic	69	2
Retrocaecal	86	3
Pelvic	107	7
Lateral caecal	23	2
Iliac fossa and anterior caecal	24	0
Undescended	15	0
Not noted	16	0
Total	340	14

It will be noted that in this series the pelvic position of the appendix appears to be the most common.

PATHOLOGY

As regards the pathology of the disease the outstanding feature was the frequency of the acute obstructive appendicitis. Only ten cases of the 340 operated on showed a total absence of obstructive phenomena. The cause of the obstruction of the lumen, noted and arranged in order of frequency, was:

1. Faecal concretion or foreign body surrounded by faecal material—namely, wheat-ear, particle of enamel, coal, or hair, etc.—31 per cent.
2. Fibrous stricture obstructing the lumen—28 per cent.
3. Kinking the lumen by extra-appendicular adhesions, congenital or old inflammatory—17 per cent.
4. Torsion or kinking of meso-appendix—3 per cent.
5. In several cases the appendix was sphacelated, and it was impossible to tell the primary naked-eye pathology.

Late complications were few in this series, and are shown in Table V.

TABLE V.—Late Complications

Type of Complication	No. of Cases	Outcome
Subphrenic abscess	2	1 died; 1 recovered
Pelvic abscess	2	Both recovered
Faecal fistulae	2	Both closed spontaneously without operation
Post-operative hernia...	3	1 cured by fascial graft; 2 wearing belts

The frequency of general peritonitis is explained by:

1. The acute obstructive type of appendicitis, rapidly going on to gangrene and rupture of the appendix with general infection of the peritoneal cavity. (In one case the appendix was found ruptured four hours after the onset of the first symptom.)
2. The frequent administration of purgatives before surgical aid is sought.
3. The delay in time before surgical intervention.

TREATMENT OF GENERAL PERITONITIS

This, unlike the treatment of acute appendicitis, is not well standardized, and I propose to review the methods adopted in this small series of cases. It is now agreed that in general peritonitis the patient does not die from septicaemia, but from absorption resulting from obstruction of the intestine. Sampson Handley has stated that if peritonitis does not produce obstruction it may become universal without being fatal, and if obstruction has occurred, but is relieved, recovery is the rule. Sir Cuthbert Wallace has made the same statement in these words: "If the bowels can be made to act the patient recovers, if they fail to act he dies" (*Clinical Journal*,

1925). Alvarez (*Mayo Clinics*, 1929) has recently studied ileus, and he believes that ileus in peritonitis is not paralytic, but is due to an excess of nervous inhibition, the intestinal muscle being still capable of active contraction but being held in leash. Hotz (*Mayo Clinics*, 1929) suggest that in peritonitis injury to the muscle is not due to toxins, but to distension with gas. Gas accumulates when absorption is impaired, and distension of the gut closes (a) arterioles bringing the necessary blood to the muscle, and (b) venules of the portal system which normally carry gas away as fast as it is formed. These recent conclusions seem to indicate that the most necessary part of the treatment of peritonitis is to prevent or relieve intestinal obstruction.

The various methods used at different times to prevent or relieve the obstruction were:

1. Operative:
 - (a) Jejunostomy (Victor Bonney); (b) ileo- or jejunocolostomy with caecostomy (Sampson Handley).
2. Antitoxic:
 - Injection of *B. welchii* antitoxin (Williams).
3. Bile enemata (St. Leger Brockman).

OPERATIVE TREATMENT

The cases treated by enterostomy in this series, either at the time of operation or at a later juncture, numbered twenty-one, and among these there were seven deaths. In all the cases in which enterostomy was performed there was present a definite stiffening of the small intestine, with plastic exudate on the serosa and distension of the small intestine.

The enterostomy was performed by Witzel's or Coffey's method, and the catheter in all cases was brought through the omentum. In several cases a double enterostomy was done, one catheter being introduced high up and a second

near the terminal ileum or into the caecum. The toxic content was thus allowed to drain away, distension was relieved, and at the same time 6 per cent. dextrose in normal saline was administered in large quantities directly into the lower bowel. It was often astonishing to see in the successful cases how the pinched, anxious, but perfectly conscious patient was transformed by even one dose of saline into a peaceful condition. The results of the enterostomy operation were quite gratifying when one considers the gravity of the patient's condition. In two cases faecal fistula developed after removal of the catheter, but both closed without operative intervention. In one case especially the life-saving property of this treatment was impressive.

Mrs. C., aged 58, was ill for three days with abdominal pain and vomiting, for which she had taken salts. On admission to the nursing home, seventy-eight hours after the onset of symptoms, she had definite signs of general peritonitis—rigid, distended, tender abdomen, etc. Temperature 102°, pulse 130. Operation revealed generalized peritonitis due to rupture of a gangrenous appendix lying in the pelvis. Appendectomy and suprapubic and local drainage were performed, and an enterostomy carried out. She remained well till the second day after operation, when she became delirious and pulled out the enterostomy tube. On the following day she became distended, vomited copious dark vomit, and looked like dying. Under local anaesthesia a second enterostomy was done, the first distended loop presenting being used. The drainage from this was copious, and in forty-eight hours she became very much improved, and gradually got completely well.

B. WELCHII ANTITOXIN TREATMENT

B. W. Williams, believing that the intestinal paresis might be due to toxin derived from the *B. welchii* which he found to be abnormally increased in the distended bowel, introduced the use of antitoxin in intestinal obstruction and the ileus of peritonitis. It was administered alone, or was given after enterostomy, in twenty-seven cases in this series. There were eight deaths in the twenty-seven cases.

The dose given was 40 c.cm. at the time of operation, repeated twice in twenty-four hours for several days (up to four). The intravenous route was used at the time of operation and the intramuscular afterwards. When given alone before ileus was complete, distension seemed to be relieved and vomiting diminished. The temperature and pulse were lowered, and flatus was passed. In one case it appeared to mask the signs of a spreading general peritonitis until the sixth day after operation.

H. H., aged 18 years, was admitted eighty-two hours after the onset of acute abdominal symptoms. Operation showed a sphacelated retrocaecal appendix, with local peritonitis. Appendectomy and local drainage were carried out, and 40 c.cm. of *B. welchii* antitoxin was administered intravenously and repeated intramuscularly twice daily until the fourth day. The patient had no post-operative vomiting. Temperature and pulse came down to normal, there was no distension, and flatus was passed freely. On the sixth day he complained of pain, commenced vomiting, and the abdomen became rigid. Operation under local anaesthesia revealed a diffuse peritonitis, and, although an enterostomy was performed, he died in forty-eight hours. The difficulty of spotting a spreading post-operative peritonitis is well known, but in this case the signs seemed to be masked by the use of the antitoxin.

BILE ENEMATA

Bile is believed to contain a stimulant to peristalsis, and St. Leger Brockman reported some striking results from its use in intestinal obstruction. In the present series bile was taken from a cholecystostomy case in the ward at the time, or the dressings from a case of biliary fistula were utilized after soaking them in normal saline. One-half to two ounces of bile in six ounces of saline was

TABLE VI.—Enterostomy and its Results

No. of Case	Age in Years	Hours Ill before Operation	Operation*Findings	Treatment	Result
1	15	98	Abscess lateral to caecum leaking into peritoneal cavity; general peritonitis; ileus	Enterostomy; <i>B. welchii</i>	Cured
2	6	144	Advanced general peritonitis with ileus	Double enterostomy	Death
3	62	48	Ruptured appendix; general peritonitis with ileus	Enterostomy	Cured
4	5	72	General peritonitis; ileus	Enterostomy; <i>B. welchii</i>	Death
5	25	80	General peritonitis; ileus	Enterostomy; <i>B. welchii</i>	Cured
6	11	56	Ruptured appendix with general peritonitis; ileus	Enterostomy; <i>B. welchii</i>	Cured
7	11	72	Ruptured ileal appendix; general peritonitis	Double enterostomy; bile enema	Cured
8	59	42	Ruptured pelvic appendix; general peritonitis; ileus	Enterostomy; <i>B. welchii</i>	Cured
9	18	82	Six days post-operation for acute appendicitis; general peritonitis; ileus	Enterostomy; <i>B. welchii</i> ; double enterostomy	Death
10	12	36	General peritonitis; distended ileum	Enterostomy	Cured
11	51	104	Diffuse peritonitis; ileus	Enterostomy	Death
12	60	72	Ruptured appendix; general peritonitis; ileus	Double enterostomy	Death
13	26	78	Diffuse peritonitis; ileus	Enterostomy	Death
14	39	80	General peritonitis; ileus	Enterostomy	Death
15	51	12	Abdomen full of watery pus; not drained. Reopened four days after; plastic peritonitis; ileus	Enterostomy; <i>B. welchii</i>	Cured
16	48	50	Plastic peritonitis; ileus	Enterostomy	Cured
17	55	130	General peritonitis; ileus	Enterostomy	Cured
18	2	120	Undescended caecum; general peritonitis	Enterostomy	Cured
19	42	240	Abscess drained; developed spreading peritonitis; ileus	Enterostomy	Cured
20	58	48	Ruptured appendix; general peritonitis; ileus	Enterostomy; <i>B. welchii</i>	Cured
21	15	72	General peritonitis; ileus	Enterostomy; <i>B. welchii</i> and bile enemata	Cured

administered per rectum every four hours. This treatment seemed to act extremely well in the five cases in which it was tried by the writer. In the *Mayo Clinics, 1929*, Alvarez suggests the use of choline, which is said to be a normal constituent of intestinal muscle and concerned with maintaining peristalsis.

TABLE VII.—Use of Bile Enemata

No. of Case	Age in Years	No. of Hours Ill	Operative Findings and Treatment	Post-operative Treatment	Result
1	11	72	General peritonitis from ruptured appendix; ileus—enterostomy; <i>B. welchii</i> antitoxin	Distension persisted; 1 oz. bile run into bowel through enterostomy tube	Cured
2	14	38	Localized pelvic peritonitis from ruptured appendix—local drainage	Marked post-operative distension and vomiting dark brown fluid; 1 oz. bile in four-hourly enemata	Cured
3	48	48	Ruptured appendix; general peritonitis; 2 free concretions	Post-operative distension; vomiting; 2 oz. bile in saline, four-hourly enemata	Cured
4	14	78	General peritonitis; ruptured appendix—enterostomy	Post-operative distension; vomiting; <i>B. welchii</i> ; 2 oz. bile in four-hourly enemata	Cured
5	15	72	Ruptured appendix; general peritonitis—enterostomy	Post-operative vomiting and marked distension; 2 oz. bile in four-hourly enemata	Cured

SUMMARY

In the treatment of general peritonitis discussed above, successes have been obtained from the use of: (1) operative measures—namely, enterostomy; (2) *B. welchii* antitoxin injections; and (3) bile enemata.

RECTAL PARALDEHYDE BEFORE OPERATION

BY

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The anaesthetic is frequently a cause of much distress to the patient, both in anticipation and during its actual induction. The anaesthetist is not infrequently informed that the thought of the anaesthetic is more distasteful than is the thought of the operation itself. It is not without cause, therefore, that anaesthetists are much concerned with the problem of improving conditions for the patient, in what is almost invariably an ordeal. One obvious method of achieving this consists in appropriate premedication with a view to inducing in the patient before the operation a state of oblivion, or at least of indifference. This can be accomplished, with varying degrees of success, safety, and consistency, by the use of such drugs as hyoscine, nembutal, sodium amytal, luminal, avertin, and paraldehyde. Since the last-mentioned drug has probably been less used for the purpose than have the others, I should like to report my experiences based on a series of some 500 cases in which the paraldehyde has been administered rectally before operation.

The paraldehyde is given in a solution with saline, well shaken together, in the relative proportions of one and a half ounces of saline to each drachm of paraldehyde. The adult dose is 8 drachms of paraldehyde—that is, 12 ounces of solution—except where the patient's weight is less than 8 stone, when the amount is calculated on a basis of one drachm of paraldehyde for each stone of body weight. The average dose of 8 drachms need rarely be exceeded for any patient,

and may often be somewhat reduced without any sacrifice of results. Experience has shown that these relative proportions of paraldehyde and saline must be adhered to; a reduction in the saline content is likely to mean that some of the solution will be returned from the rectum. The properly proportioned solution is nearly always retained without difficulty if its introduction is effected slowly. The paraldehyde used must be fresh. It undergoes chemical change if kept too long, and the use of such paraldehyde may lead to transient diarrhoea after operation; no irritation occurs if the fresh drug is used.

ADMINISTRATION

The procedure is as follows. The evening before operation the patient is given an enema, and a sedative such as veronal, dial, or phanodorm. The following morning, one and a half hours before operation, 1/100 grain of atropine is administered hypodermically (no morphine is given), and immediately afterwards the paraldehyde solution, freshly mixed and thoroughly shaken, is slowly introduced into the rectum by means of a catheter. Retention is rarely difficult; sleep usually intervenes within a few minutes, and this is not disturbed when the time comes for removal of the patient to the operating theatre. The patient remains oblivious to the administration of the anaesthetic, and induction, by any of the usual methods, is singularly easy and rapid, owing to the complete absence of resistance or excitement. The maintenance of anaesthesia is a simple matter and a surprisingly small quantity of anaesthetic agent is required, even where muscular relaxation is called for. After the operation the patient sleeps quietly for a variable number of hours. He can be awakened during this period, but quickly goes off to sleep again. Spontaneous awakening usually occurs from five to eight hours after the operation, the patient remaining drowsy and comfortable for a considerably longer period, and securing, in most cases, a restful first night, often without the aid of morphine.

ADVANTAGES OF PARALDEHYDE PREMEDICATION

The method, observed in a large number of cases, appears to offer certain definite advantages, the chief of which, from the patient's point of view, is the total elimination of the unpleasantness associated with his introduction to the operating theatre, and of the still greater unpleasantness of the anaesthetic. The drug is so consistent in its action that the nervous patient can, if desired, be confidently promised that he will go to sleep quietly in his own room and that he will know nothing of operating theatre or anaesthetic. Post-operative vomiting appears definitely to be reduced, thanks no doubt to the relatively small quantity of anaesthetic required. One-half or one-third of the usual quantity of anaesthetic will, as a rule, suffice to produce the desired anaesthesia—an important advantage in long operations, where the large quantity of a toxic anaesthetic agent normally required is a matter of serious consequence to the patient.

From the anaesthetist's point of view, no greater advantage can be mentioned than the facility with which gas and oxygen can be administered after paraldehyde premedication. Upper abdominal operations are possible without the use of additional ether and with completely adequate muscular relaxation. I believe that, for gas and oxygen, it is the premedication method of choice, and will often serve to afford the patient the advantage of a pure gas and oxygen anaesthesia where this would otherwise be difficult or impossible.

The remarkable degree of post-operative comfort enables the surgeon to limit the morphine administered