I hereby state that after graduating as an M.B. and C.M. from Edinburgh University in 1858, I proceeded to Vienna, Austria, where I was in constant attendance on the hospitals for a period of six months. After five months of travel in Europe I returned to the United States, locating in Washington, D.C. I was engaged in private practice for six months, when I received the appointment of Assistant Surgeon in Charge of Columbia Hospital for Women and Lying-in asylum; the Surgeon in Charge going on a tour to Europe after my appointment. I acted as Surgeon in Charge. After this the Hospital was placed under the charge of a Staff of four Surgeons, and for one year and eleven months I held the position of Resident Physician.

Since resigning from the Hospital I have been in private practice for over five months, and have lately received the appointment as Assistant Surgeon to the Hospital.

I further state that this thesis is entirely of my own work and in my own handwriting.

James Tate Scott.
Hydrothionemia, or the Phenomenon of Sulphuretted Hydrogen Gas in Urine—Four Cases. A review of the literature.

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Thesis for the Degree of
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Washington, D. C., U. S. A.
April 20th, 1893.
Sworn and subscribed to before me this 20th day of April A.D., 1898.

[Signature]

[Notary Public]
Hydrothionuria, or the Phenomenon of Sulphuretted Hydrogen Gas in Urine—Four Cases—A review of the literature.

By

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Within the past three years it has been my good fortune to meet with and demonstrate, chemically, the presence of Sulphuretted Hydrogen Gas in solution in the urine, in no less than four cases. The subject is one which has been almost entirely overlooked, alike by the practitioners and the writers of text-books, though undoubtedly the condition of Hydrothionuria occurs, from time to time, in the practice of many others. Since my first case, over two and a half years ago, I have been constantly on the watch for others, with the result of having met with it three times since.

In three of my cases and in most of those recorded by others, the condition was associated with grave conditions, and I believe that such will usually be the case. When such are met with...
Symptom does occur, it behooves the physician to assume "an attitude of watchful expectancy" at least, even though he may be puzzled in his diagnosis and method of treatment. I will first give my own four cases, with the results of the chemical tests. This will be followed by a complete review of the literature bearing on the subject and, finally, the clinical significance of the condition.

Case I.

M. J., a negress, alt. 14 years and six months; single; primipara. Height, 5 ft. 4 in.; weight, 110 lbs. Admitted to Columbia Hospital Aug. 22nd 1870. The patient was brought to the hospital, at midday, in coma, in a comatose condition, suffering with a well marked case of Purpura Exanthesia. Her friends stated that she had had twelve convulsions since 2 a.m. of that morning. Examination on admission showed labour just beginning, the osdera admitting the tips of two fingers. The position was Recipito - Leva - Interior.
The temperature on admission was 101.6°F; pulse, 112 per minute; respirations, 26 per minute. There was extreme anaemia. The convulsions continuing with awful severity, and not yielding to chloroform anaesthesia or morphia hypodermically, I performed podalic version and delivered her of a full-time dead child weighing 6 lbs. Soon thereafter the eclamptic attacks ceased. The urine, which was drawn by catheter three and a half hours after delivery, was intensely acid and became entirely solidified by both the heat and nitric acid tests.

In the evening of the following day, the nurses came to me complaining of an overpowering odour from the patient which I at once recognized as that of sulphurated hydrogen. This odour was so strong that it nauseated the nurses, and I myself suffered with intense headaches on several occasions while making the analyses on the following days.

It was fortunate that my first case was so exceedingly well marked as to force itself upon my attention at once through
the sense of smell. This led me to perform a series of careful chemical tests, all of which proved satisfactorily the presence of Sulphuretted Hydrogen.

The following were the results of my tests, the reagents conforming to the U.S.P. Test-Solutions.

Urine drawn by Catharina Aug. 23rd '70 (day following delivery):

- Amount = 120 g.
- Sp. gr. = 1015.
- Reaction = acid.
- Albumen = present in enormous amount.
- Odour = penetrating odour of H₂S.

Microscopical Examination: Leary deposit of amnonio-magnesio-phosphate, broken down blood corpuscles, vesical epithelium, and an enormous number of active vibriones in the freshly drawn urine.

Tests for Sulphuretted Hydrogen:

I. 2 ml. of urine in test tube + As₂NO₃ solution (white ppt. of albumen) + HNO₃ = dark grey precipitate at once, becoming rapidly darker and eventually exhibiting a distinct dark zone of silver sulphide, As₂S₃.

II. Urine + excess of lead acetate solution = copious
light grey precipitate. The excess of the reagent precipitates all the albumen in the urine and thus, by admixture of the white albumen with the black sulphide of lead, (PbS), gives a grey colour.

III. Take urine + one drop of lead acetate solution = inky black precipitate of lead sulphide, (PbS), with a dark brown supernatant liquid, no white or greyish precipitate at all being caused.

With this one drop of lead acetate solution, there was only an insignificant trace of albumen thrown down, yet it contained enough lead to unite with all the sulphur of the H2S and convert it into the black sulphide of lead.

This last test, with one drop of lead acetate solution, is one of the most delicate of all.

IV. Fold paper moistened with solution of lead acetate and placed over the mouth of the jar containing the urine gave, in the course of an hour or so, a dull black colour of a leaden lustre.

This is the test I always try primarily as it is exceedingly delicate and easy to carry out.
V. Another characteristic test is made by using a solution of antimony (tartar emetic) to form the orange-red precipitate of sulphide of antimony.  Let urine + Solution of Antimony in excess of HCl = while precipitate of albumen in which, after a few hours, a beautiful orange-red zone of sulphide of antimony unmistakably differentiates itself.  This is a very beautiful test if the H2S is present in large amount, but it is apt to be fallacious as it requires a few hours in which to develop, during which time the urine may be undergoing decomposition in the test jar.

VI. Heller's test: Urine + solution of iron chloride = black precipitate of FeS. If very little H2S be present it only causes a brown discoloration.

In this, my first case, I would draw particular attention to the enormous number of active vibriones in the freshly drawn urine. This point will be considered more fully later on when I take up the clinical significance of the condition. After the appearance of the H2S the bladder
was repeatedly washed out with a solution of Potassium Permanganate. The presence of H₂S ceased soon thereafter.

In this case there is no doubt in my mind that the bacteria liberated the sulphur contained in the albumen which was present in such enormous amount.

Case II.

J. A., female, age 21 years; single.

Admitted to Columbia Hospital Oct. 9th, 1891, with febrile symptoms and a temperature rising every evening to 104° - 105° F.

Laparotomy was done on Oct. 14th, 1891.

A left-sided pyosalpinx was found, containing a foetid, blood-stained pus. The ovary and tube of the right side were normal and not removed.

Before and after the operation the patient was in an exceedingly low condition, with a temperature which ranged for many days after the operation between 104° - 106° F.

She lived however for a year afterwards, eventually dying in another hospital of Phtysis Pulmonalis.

Her urine was tested repeatedly but...
Trace of albumen was ever found either before or after the operation.

On the fifth day after the operation the examination of the urine resulted as follows:

- Sp. gr. = 1017
- Reaction = faintly acid.
- Albumen = none.
- Odour = H₂S.

Microscopically there were no organized deposits nor bacteria.

Now this case is exceedingly interesting—H₂S appears in the urine without a trace of albumen. Probably the hypothesis that H₂S was liberated by the action of bacteria in albuminous urine rests upon a solid foundation in my first case, but that there may be other causes for the presence of this gas in urine is rendered evident by this, my second case in which there was no trace of albumen in the urine at any time for several weeks previous to or after the presence of H₂S, nor were there any bacteria in the freshly voided urine seen. In this case the abdominal wound did not heal for several months, pieces of lifature
silk from the fistula being discharged on several occasions. There was always an offensive, feculent odour to the pus, but no H₂S appeared in the urine after there was an opening established for the discharge of pus.

In this case the appearance of H₂S in the urine may be accounted for in two ways:

A. By a resorption of the H₂S from the pus-containing cavity into the blood, and its subsequent elimination through the kidneys.

B. By an excretion of the gas, developed in the pelvic abscess, directly through the living animal membrane,—the bladder wall.

Case III.

M. J., the same patient who furnished me with my first case, returns in labour twenty months later with another attack of Puerperal Eclampsia! In passing, I may say that this case was one of those rare examples where Puerperal Eclampsia occurs for a second time without the patient carrying twins.
This time labour was artificially induced and forceps applied. She was again delivered of a still-born child.
Examination of her urine showed it to contain albumen in enormous amount, and on the fifth day H$_2$S was present in large volume, all of the tests giving satisfactory reactions.
The urine during the first few days was scanty and very highly albuminuous, but after a free flow was established the H$_2$S disappeared spontaneously. vibrios were present on this occasion also.

Case IV.

E. P., a negro, age 22 years. IV Para.
She was admitted to Columbia Hospital, May 25th, 1892, far advanced in labour, and delivered normally of a 6$\frac{3}{4}$ lb. child in L. O. A. position.
Examination of her urine revealed albumen present in large amount.
On the second day I detected the odour of H$_2$S in the urine, which was also demonstrated by the previous chemical tests.
Examination of urine on day following delivery:

Sp. gr. = 1020

Reaction = strongly acid.

Colour = pale red.

Albunin = present in large amount.

Odour = H₂S.

Microscopical examination = red blood discs, a few leucocytes, detached vesical epithelium; no vibriiones.

Sulphured Hydrogen was chemically demonstrated.

The bladder was washed out with a weak solution of Potassium Permanganate, but I doubt if the rapid convalescence in this case was dependent on the treatment. The patient had an excellent fever, the highest temperature being recorded on the second day and only registering 101.8° F.

This case is of interest, in contrast to the others, on account of the mildness of the symptoms and the complete and rapid recovery.
Review of the Literature.

After looking over the references in the Index Medicus of the Army Medical Library with the greatest care, I have only been able to find these comparatively few cases which I here present.

The presence of Sulphuretted Hydrogen Gas in urine was recognized as far back as 1829 by Chevalier. (Journal de chim. Méd. 1829-2, p. 179) He mentions a case which occurred in a syphilitic woman undergoing mercurial treatment, and regards it as a phenomenon of fermentation.

Höfle. (Med. Anzeiger. Bd. XI, 1845-1, p. 415), found H₂S in the urine in a case which occurred during a small-pox epidemic in 1843-44.

Friedrich Betz was one of the first to devote much attention to the cause of Hydrotartioria. Betz, (Memorabilia, u. 1869-1, 7. f. über den Nachweis und die klinische Bedeutung des Schwefelwasserstoffhaltigen Urins) mentions a case in a man 38 years old, of a strong constitution, who, after a night spent in
Nearly eating and wine drinking, suffered with symptoms of acute gastro-intestinal catarrh. H₂S was present in his expectations and urine. After free evacuation of the bowels, the H₂S disappeared. Betz also records another instance, (über die Quellen und diagnostisch-therapeutische Bedeutung des H₂S im Urin - Memorabilien, 1874-XIX - pp. 66-69). This was the case of a man, aged 79 years, who had H₂S in the urine for a protracted time.

The Post-Mortem Examination showed hypertrophy of the prostate, which interfered with the emptying of the bladder and caused diverticula of the bladder walls in several directions. One of these diverticula, which was exceedingly thin, pressed closely against the rectum which, bulging out like an ampulla, was filled with stagnant faeces. In this case the conditions were peculiarly favourable on account of the exceedingly thin diverticulum which pressed upon the rectum, while the bladder, in its normal anatomical relations, only presents a small area to the abdominal cavity. The fresh urine in this case had a
penetrating, fæcalulent odour of \( \text{H}_2 \text{S} \); weakly acid reaction; Sp. gr. of 1003-5; and contained no albumen.

Microscopical examination showed pus corpuscles, epithelial cells and vibriones. The catheter, which was made of silver, was discoloured black by the \( \text{H}_2 \text{S} \).

Betz found that the faeces in this case contained more than the usual amount of \( \text{H}_2 \text{S} \), and that the quantity of \( \text{H}_2 \text{S} \) in the urine and bowel took a parallel course. It is important to note that the ureters were as thick as a finger and that there was suppurative atrophy of the kidneys. Bett found three theories of causation as follows:

I. The \( \text{H}_2 \text{S} \) develops in the bladder from a decomposition of albuminoid bodies, as pus, blood, etc., — a rare cause.

II. \( \text{H}_2 \text{S} \) appears in consequence of the resorption from the intestine into the blood and its subsequent elimination through the kidneys.

III. The \( \text{H}_2 \text{S} \) appears in the urine on account of the osmosis of the gas from the intestine into the bladder.
A. Emnighaus has fully written up two cases. (Zwei Fälle von mehrfacher Perforation des Verdauungsrohres, und Schwefelwasserstoffgehalt der Urin; Berl. klin. Wochenschr., 1872, H. 4, p. 749.)

His first case occurred in a woman, aged 20 years, who was admitted to the hospital at the latter part of February 1871, suffering with symptoms of gastric ulcer. She died in collapse and with slight convulsions after being in the hospital about a week. At Post Mortem there was found a perforation of the stomach between the pyloric and cardiac orifices near the small curvature, the small intestine was perforated twice, and the sigmoid flexure once.

Four days after the symptoms of perforation H₂S appeared in the urine.

His second case was that of a man, aged 20 years, admitted to the hospital March 19th, 1871. He was so ill that no history could be taken. At Post Mortem examination there were found abscesses in the small intestine, coccic, vermiform appendix and sigmoid flexure. In both these cases of Emnighaus, and in case 11 of Bötz, it would seem that the H₂S appeared in the urine by exomosis through the bladder wall and its absorption.
into the urine.
Johannes Ranke, (Lehrbuch der Physiologie, 3. Aufl. S. 530), made an experiment of very great importance in the study of this phenomenon. He found that if a few drops of the urine, which contained the $H_2S$, were added to other normal urine, $H_2S$ would develop in the latter, and again a third specimen of normal urine could be inoculated from the second.
Ranke says, (translation), "there is no doubt that in the formation of $H_2S$ in urine we are dealing with the phenomenon of fermentation, which I call $H_2S$ fermentation. The development of $H_2S$ in urine can only take place in acid and neutral urine, and not in alkaline."
His experiment would go to prove that the property of developing $H_2S$ was contained in the organic ferments of the urine which contained the $H_2S$ by reason of certain fagous substances peculiar to it.
Müller, (Ueber Schwefelwasserstoff im Harn, Berl. med. Wochenschr. 1887. XXIV. 405-8. 436), verifies the above experiment in every particular, and says that the addition of the very smallest quantity will suffice to cause
the circulated urine to become turbid—Müller says that not every urine can be so inoculated, especially those urines which are highly concentrated, as in infectious diseases, peritonitis, and others, which contain a large quantity of phenol and indigo-forming substances which are toxic to the microorganisms.


Their first case was that of a man, aged 55 years, who had a retro-rectal abscess which communicated through a fistulous opening directly with the bladder. The patient died after a very short course of the disease without affording much opportunity for study. The Post-mortem showed a direct communication of the bladder with an abscess cavity which included the theory of gas diffusion in this case.

Their second case was a most interesting
one in a woman who entered the hospital suffering with pains in the left side under the arch of the ribs, and an irritable bladder, which led them to diagnose the case as vesical catarrh. On the fourth day after admission to the hospital the urine attracted attention by its penetrating odour of H₂S.

From this case they succeeded in isolating little rods by culture which, transplanted in other urine after the method of Rouxke, produced H₂S in the second specimen of urine. The H₂S disappeared spontaneously after eight days, although the bladder catarrh continued. The patient left the hospital, but soon thereafter returned with the same complaint, yet no H₂S could ever be discovered, while under the microscope, there were a number of bacteria identical with those seen before. They do not mention whether the urine was albuminous or not.

Their third case occurred in a woman, aged 17 years, who came into the hospital suffering with parametritis, inflammation of the vaginal mucous membrane and pain on pressure in both inguinal regions. Both tubes and ovaries were thickened.
and surrounded by fusions. There was erosion of the tissue vaginalis uteri. This to me seems most likely to have been a case which was septic and probably gonorrhoeal in origin. In the light of the pathology of peri-uterine inflammations they were probably wrong in calling it a case of parametritis. The vast majority of cases, such as the nature described, are cases of pyosalpinx with a plastic fusion around the ostia abdominale binding together the fimbriae and ovaries. Very probably in the centre of this "mass" or "fusad" there was a degenerating fus-sac which formed the \( \text{H}_2\text{S} \). Soon after the relif of the vaginitis she developed a cystitis and had to be catheterized. The urine was acid, free of albumen, without any leucocytes, turbid from masses of bacteria in it, and smelled of \( \text{H}_2\text{S} \). Soon the urine became clear, the bacteria diminishing but the odour of \( \text{H}_2\text{S} \) continuing. Soon thereafter the \( \text{H}_2\text{S} \) reaction disappeared. After eight days the bacteria again appeared in numerous numbers in the urine and the \( \text{H}_2\text{S} \) reaction again became distinct. Gradually the bacteria disappeared.
and the $H_2S$ reaction as well. 

Here there is a distinct parallel between the presence of $H_2S$ and the quantity of bacteria in the urine. They suggest that the bacilluria might have been caused by masses of bacteria which passed through the bladder walls from the neighbouring inflammatory area.

From this urine they succeeded in isolating a very markedly characteristic species of bacillus which they considered as the cause of the hydrothrombosis.

They convinced themselves by experimentation that this species of bacteria had the power to act reducing upon the sulphur-containing substances in the urine, and, in this above-mentioned case, they consider it to have been the sole cause.

Müller, (loc. cit.), making experiments at the same time and independently, showed that other microorganisms were capable of developing $H_2S$ in the same manner. We will refer to these experiments more fully later on.

Rosenheim and Guttmann further made experiments to show from what substances
present in the urine H₂S was developed. They placed their bacteria in nutrient liquids, to which they added traces of albumen, but were never able to observe the formation of H₂S, while, on the other hand, the same microorganisms placed in other urine free of albumen developed the H₂S reaction. The question then arose to them: "which of the sulphur-containing substances was the source of the H₂S?"

According to Neubauer and Vögel (Anlty. zur Analyse des Harns, II. Auflage. 1856), whom they cite, the source of the H₂S would be in the sulphates which from H₂S in the presence of moist organic substances at a moderate temperature. They quote Pfeffer as saying that, with the presence of sulphates, certain fungi reduce these sulphates to H₂S. They support this by the following experiment: Normal fresh urine was freed from sulphates and then inoculated from urine which contained H₂S. Not a trace of H₂S was developed in it. Miller (loc. cit.), however, did succeed in developing H₂S in urine freed from sulphate by transplantation of bacteria.
Rosenheim and Guttmann believe that the H<sub>2</sub>S-producing sulphur belongs to a class of sulphurs normally existing in urine, and they say that very possibly it may be hyps sulphururous acid which is particularly apt to develop H<sub>2</sub>S with facility.

Friedrich Müller, (Uber Schwefelwasserstoff im Harn. Berliner Klin. Wochenschr., 1887-XXIV, 405-8, and 436), mentions a case of a servant girl, aged 29 years, suffering with Phthisis Pulmonalis. The urine sediment contained numerous leucocytes, bladder epithelium and crystals of triple phosphate. The urine gave a strong H<sub>2</sub>S reaction, and this was more pronounced when it stood in the bladder than when drawn by catheter. In Post Mortem there was found a very small recto-vaginal fistula which had been there more than five years previously. Feces passed through this and probably infected the bladder by passing up the urethra, thus causing a cystitis. Müller found no H<sub>2</sub>S in the urine of patients who had H<sub>2</sub>S in the spula or vomited matters, nor in patients who took
sulphur baths and inhaled large quantities of H_2S, nor did he find it in cases of
gastric ulcer and typhoid fever with perforation. He has never found it where
there has been a pus-sac adjacent to the
bladder, not even if that pus-sac contained
H_2S in large amount. In his experiments
on animals he found that only by
injecting lethal quantities of H_2S, or of
sodium sulphate in solution into the
abdominal cavity, could he cause H_2S to
appear in the urine. He does not believe
that diffusion through the bladder walls
often occurs, but that in every case in which
the urine contains H_2S it has undergone
decomposition, yet not every decomposed
urine contains H_2S.

He further says that hydrothionuria
is a very common phenomenon in all
possible forms of cystitis, not only in the slight degree so often found
in females as a sequela of hemicrurhea,
but also in the more serious cystitic
diseases of the mucus membrane of the
bladder. He says, some specimens of
normal urine left exposed to the air,
especially at a warm temperature, will develop H2S, and from these specimens which to develop H2S other urines can be inoculated.
He found, out of many kinds of microorganisms which develop in urine, two kinds which especially develop H2S.
The first was an oval-shaped coccus, 8 μ in diameter, which often forms diplococci and liquefies gelatin quickly.
The other organism was larger, round, did not liquefy gelatin, and formed H2S very slowly.
He believes that other microorganisms can form H2S, but he only wants to prove that they do form it and not what kinds form it.
Albumin is not the cause, because urine free of albumin and peptones can be inoculated and develop H2S.
Müller concludes that hydrothionuria is in most cases a result of decomposition in urine caused by cutaneous microorganisms.
The appearance of H2S which has been absorbed from other parts of the body, e.g., intestines, kidneys, or from neighbouring pus or gas collections is only very rare, he thinks, and only there if the quantity of H2S
be so great that general topic phenomena resulted. This latter theory, he says, could only be accepted if the urine were examined immediately and no trace of decomposition found.

Sertoli, (Sull' esistenza di uno speciale corpo solfuroide nell'urina. Gazett. med. ital. Lomb. 1869. Ser. VII. p. 179), found that with the addition of any mineral acid and heating to 100° C. any urine would give off H₂S.

Schatz, (Berl. klin. Wochenscr. 1855 - p. 254), mentions a case of hydrothionuria where an error in diet caused a calcareous of the stomach and a general intoxication of the whole system by H₂S poisoning, with the evolution of large quantities of H₂S from the mouth, and it also appeared in the urine in such great quantities that it coloured a visiting card, containing lead black.

J. Vogel, (Neubauer und Vogel, Anleitung zur qualitativ en und quantitativen Analyse des Harns etc. Bearbeitet von Hupfer - 8 Auflage. p. 187 u. 189), says that lead...
had an opportunity for a long time to observe hydrothioruria in a man who had paralysis and who had to be catheterized. The urine was feebly acid, pale yellow, with some sediment, and gave a strong reaction of \( \text{H}_2\text{S} \) with lead acetate.

Löhrich, (Handanalyse, 2. Auflage, 3.354), observed \( \text{H}_2\text{S} \) in the urine in a case convalescing from pyaemic fever. It had no trace of albumen.

C. A. Cameron, (Notes on Pathology of Urine, 1880), had a case of hydrothioruria in a middle-aged man who suffered for two years with \( \text{H}_2\text{S} \) in his urine. I add also another peculiar case, which he mentions, of a young girl who, though in good health, had \( \text{H}_2\text{S} \) eliminated through the perspiration after exercise. The urine examination was not mentioned.

Härtling, (Ueber das Vorkommen vom Schwefelwasserstoff im Harn - 8° Berlin, 1886), presents a case which had gangrene of the right lung and faeculent cystitis.
The sputa contained H₂S. The urine was acid, with a specific gravity of 1.011-1.024. Albumen was present, as well as leucocytes, but no tube casts. Eventually H₂S appeared also in the urine, but after an observation of a month and a half the patient was discharged cured.

Eichwald, (Pathologic u. Therapie, vol. ii. p. 647), says that in certain diseased conditions H₂S appears in the urine and that it can be known by its colouring a silver catheter black. He mentions no cases.

Heller, (Arch. f. Phys. u. path. Chem. u. Mikrosk., 1844—f. 24), found H₂S in the urine of a tuberculous case suffering from pneumonia. He said that it was decomposed urine.

L. Kolipinski, (Med. News, Phil. Feb. 6th, 1892, Vol. IX. no. 6—f. 1574), had a case of hydrothymuria in a man, aged 67 years, who was an inebriate and suffered with chronic gastritis. He had profuse incontinence of urine and an enlarged prostate gland. June 3rd, 1871, the urine examination showed
a red colour; acid reaction; slight turbidity; no sediment; Sp. gr. 1.018; no albumen, bile, or sugar—lead iodate paper gave the H2S reaction.

The urine continued of this same composition for one month, and H2S was always present. "At the end of the month the patient's condition grew worse. \[\ldots\] There now appeared a new light to clear up the mystery of the H2S. The patient began to complain of pains about the arms at the site of a former ischial rectal abscess. There was found on the right side, around a small, circular, cicatrix, a moderate degree of induration, extending forward to the sacral fold, slightly tender and fluctuating. A free incision gave split to about an ounce of pus having a strong odour of H2S. The abscess was thoroughly washed out—and left clean and dry." The night of this day, July 4th, the urine was drawn by catheter twelve hours after the abscess had been opened, and presented the following characteristics: "colour, red; acid reaction; Sp. gr. 1.019; slightly turbid; opaque sediment; a trace of albumen; indigo in excess; granular epithelium, and blood corpuscles. H2S was present."
H2S was not again found in the urine, though on the following day or so he had a severe diarrhoea, with offensive stools and undigested material which showed the existence of gastro-intestinal indigestion. He died July 13th, 1891.

The autopsy, which was confined to the abdominal cavity, showed a dilated stomach, cirrhotic kidneys, the right one containing several small cysts and calcareous infiltrations. The liver was in a condition of fatty degeneration; the spleen was dark and friable but not enlarged; there was no fluid in the abdominal cavity. "The ischio-rectal abscess was thoroughly explored and found empty and granulating. The bladder was contracted, and there was no indurative or inflammatuory in its neighborhood. There was an old ischio-rectal abscess (the residual variety of Paget), for a long time manifesting itself only by the presence of a decomposition-product in a natural secretion."

This case bears some similarity to my second case (page 9), in which there was a pelvic abscess with an offensive faeculent odour to the pus. In that case, as in
the one mentioned by Kołipinskej, no H₂S appeared in the urine after there was an opening established for the discharge of jus.

The bacterial origin of the H₂S in such cases as these, seems doubtful.

Antoine Fluit, (On the elimination of H₂S artificially introduced into the body Med. News - Phil, 1887 - vol. 51 - pp. 610 - 73), made experiments to ascertain the value of the surgeon treatment of pulmonary phthisis by H₂S gaseous enemata, the object being to have H₂S eliminated by the lungs and destroy the tubercle bacilli. He tried gaseous enemata of H₂S to see if it could be eliminated by the lungs. He never succeeded, after the injections, in detecting it in the breath of a human being, but did find it in the breath of a dog on one occasion. A piece of white filter paper moistened with lead acetate solution was held before the mouth for the detection of the gas. In the case of the dog the elimination only lasted for three minutes. He has repeatedly injected H₂S into the veins of dogs, and has always
noted a prompt elimination by the lungs, but this lasted only for a few seconds after the injection was discontinued. He did not find \( \text{H}_2\text{S} \) in the urine of these dogs. Thirt says, “it would appear from these observations, that a certain quantity of \( \text{H}_2\text{S} \) introduced, even in saturated aqueous solution, may be destroyed in some way in the system without being eliminated as \( \text{H}_2\text{S} \).”

Out of all the literature on the subject I have only been able to find three comparatively few cases. It is difficult to actually demonstrate the cause or causes, but we can arrive at pretty certain conclusions from the material presented.

I believe that the condition of hydrothorax is more much more frequently than it is recognized, especially in cases of feculent cystitis. Its diagnosis is of the utmost importance, when it does occur, in leading us to the therapeutic measures to be adopted. The condition, from whatever causes it
originate, demands that the diagnosis be accurate; whether there are neighbouring pus- sores, stagnant and decomposed faces, intestinal calcarcb, perforations, or bacillura.

The possibility of the diffusion of gases through animal membranes is recognized by all physiologists.

Müller (loc. cit.), does not happen to have found H₂S in the urine when there was rupture of the visera with escape of H₂S into the abdominal cavity, nor when there were pus sacs adjacent to the bladder, nor could it cause it to appear in the urine of animals except by injecting lethal quantities of H₂S or of sodium sulphate into the abdominal cavity. But the observations of others antagonize this; for instance, Smaller's case, where there was a general intoxication by H₂S poisoning resulting from an error in diet.

My own case No. II (page 7), where there was a pelvic abscess adjacent to the bladder; the case of Bell, where a very thin diverticulum of the bladder pressed closely against the rectum which was filled with stagnant faeces and contained an unusual amount of H₂S.
the two cases of Cunningham's, (page 15), in
the former of which there were numerous
perforations of the alimentary canal, and
in the latter, abscesses in the small intestines
verniform appendix, cecum and sigmoid
flexure; and Hofjirnski's case, in which
there was an ischium-rectal abscess containing
$H_2S$.

Chas. B. Kelby, in an article on abscesses
round the rectum, (Therapeutic Gazette, Phil.,
vol. XVI., no. 1, Jan. 16th 1893), says that the pres
in abscesses round the rectum often has
a fecal odour from proximity, without
actual perforation. This is known to all
surgeons.

I therefore consider that the diffusion of
the gas from a neighbouring fistula,
or from an intestine containing an
unusual amount of $H_2S$, directly through
the bladder walls is one of the causes
of Hydrothromuria.

I place little credence in Betz' second
theory, that $H_2S$ appears in consequence of
the absorption from the intestine into the
blood and its subsequent elimination
through the kidneys. Müller and
Anstruther Flint, (loc. cit.), have both shown that H₂S can be made to appear in the urine only by injecting lethal quantities either into the abdominal cavity or veins.

Husband, (Forensic Medicine, 4th ed, p.319), says, "when the gas is not slightly diluted, the person becomes suddenly weak and insensible and rapidly dies. The Post-Mortem appearances are, placidity and blackness of the blood, loss of muscular contractility, and a tendency to rapid putrefaction. The bronchial tubes are reddened, and the internal vascular apparatus appear almost black."

In order to be absorbed into the blood and later eliminated by the kidneys, the H₂S would have to be present in such enormous amount as to cause speedy collapse and death,- a condition which was present in none of the cases.

The experiments of Rancke, made also by Müller, and Rosenheim and Guttmann, from conclusively the bacterial origin of a large class of cases of hydrothionuria. Rancke proved that a few drops of urine containing H₂S, on being added to other
Normal urine, caused \( H_2S \) to develop in the latter by a process of fermentation.

Rosenheim and Lützmann (loc. cit.), discovered a bacillus in the urine which developed \( H_2S \)
in other urine, and Müller (loc. cit.), discovered two forms, one an oval-shaped coccus, \( \Phi \mu \)
in diameter, and another larger organism.

In cases I and \( \frac{1}{3} \) of my own series, the urine swarmed with vibrioines and, when these were killed by irrigating the bladder with a solution of Potassium Permanganate, the \( H_2S \) soon disappeared. Müller found that some specimens of normal urine left exposed to the air, especially at a warm temperature, developed \( H_2S \), and from these specimens which did develop \( H_2S \) other urines could be inoculated.

Just what substances the bacteria split-up to form \( H_2S \) it is difficult to say. Extreme albuminuria in eclamptic cases was present in two of my cases, and albumen was present in some of the other cases also.

When we consider the large proportion of sulphur contained in albumen it
is not hard to understand how it may be one of the substances present in urine out of which H₂S may be developed. In the two cases referred to above out of my own series, where there was a dense precipitate of albumen on testing it, and an enormous number of active microscopical organisms, I conceived the idea that the process was analogous to the formation of H₂S during the putrefaction of an egg. But Rosenheim and Gutzmann have positively shown that the presence of albumen is not necessary, and that other substances present in the urine can produce it. These are probably the sulphates, but even this supposition is rendered difficult, since Rosenheim and Gutzmann succeeded in developing H₂S in urine which was previously freed from sulphates; Müller, however, could not cause it to develop in urine freed from sulphates.

I believe that any of the sulphur-containing substances present in the urine may at times be the source of the H₂S.
The clinical significance of hydrothionuria varies according to the circumstances under which its development occurs. In some cases we must reduce the clinical significance of $\text{H}_2\text{S}$ in the urine to a bacilluria; in others to a diffusion of the gas from a neighbouring jux-sac or towel containing a large amount of $\text{H}_2\text{S}$. The decomposition of the urine occurs after its secretion, either in the bladder, ureters, or pelvis of the kidneys, or account of the action of bacteria. It is not essential to find any particular kind of microorganism to explain it, but simply to accept as proved that various kinds have been found to split up the sulphur-containing substances in acid urine.

Küttling (loc. cit.), says that every urine remaining exposed to the air eventually develops $\text{H}_2\text{S}$, so this precludes the idea of assigning the cause to any particular kind of microorganism.

The therapeutic measures to be adopted will depend upon the cause. If the intestinal canal is filled with stagnating gases and faeces, we will
freely purge the patient and administer intestinal antiseptic drugs.
If there are neighbouring abscesses, for instance, Iyo-salping, pelvic abscesses, ischio-rectal abscesses, or tubercular ulcerations of the bowel, bladder, or internal organs of generation, we will evacuate the pus and drain according to surgical methods. If the condition is associated with cystitis and bacilluria, we will in most instances be the case, we will wash out the bladder with antiseptic solutions of Potassium Permanganate, Hydrogen Bicarbonate, or other substances suitable for bladder irrigation.

The End.