Epidemic Arsenical Poisoning in Beer-Drinkers: A Clinical and Public Health Study

Having been Surgeon to the Distillers Company stationed at their Churchill Distillery for three and a half years (the term of my residence in Alva in general practice), I had many opportunities of observing alcoholic suppurative arthritis in varying degrees. I always noted that the most marked and rapidly progressive cases were in distillery workmen who drank very recent spirit; and in those who were sufficiently patient to wait for the hours to come round when they were allowed their "stew," i.e., a place of good and hickey three or four times daily...
Bed shall show in the differential diagnosis section of this account, never once have observed any erythema, erythroderma, herpes, or any pigmentations or other superficial appearances of pathological character.

I left Alva came to Casrith in 1898, and only once since that time, until last October 1900 did see a case of peripheral neuritis; it was unaccompanied by any superficial appearances.

In October 1900, notwithstanding the fact that the temperature was far from high, the weather very dry, a great number of working men began to grumble about having very sweating feet and hands accompanied by great heat, thickening of skin, peculiarly painful red lines of so-called
Inflammation—Very few any
sedentary medical men, chiefly
because several soldiers who
had returned from South Africa
were being frequently treated
to be in hotel parlours while
recounting their adventures, were
among the sufferers. They allowed everybody's necessities
by saying they had had the same
in South Africa, that it was very
"ruined," as nearly every man
in the army in South Africa
had it once or often. It was
then observed as a new mani-
faction of influenza & as it began
to short in some women who
were secret drinkers renewed to
abstain from alcohol, even some
medical men were satisfied
to class the cases as "influenza."
The first case alone was a
"Cooper" in a busy distillery
here & as it happened the Com-
pany was rebuilding a great
part of their premises, they could not brew sufficiently fast to supply the demand, were necessarily forced to hurry their brewing to supply beer at the very shortest possible notice, while over 5 above this they had to buy in a very cheap beer to keep touch with demand.

This copper drank on an average 5 pints a day. Then he came to me complaining of pain tingling running of hands and feet, together with intense thirst. Thirst, running eyes, a peculiar bright, almost scarlet complexion, with a papular rash on forehead. Scalp, I at once jumped to the conclusion that I had before me a case of virulent poisoning from drinking of this recent spirit. So convinced was I that this then the company applied to me for an explanation of his illness.
I shone some of the cases whether or not they should pay for his treatment. I shall not certify that in my opinion he was suffering from his ear allowance containing too crude a spirit. I could not explain it otherwise. This became known to many more cases came under my observation.

I then observed that the cases all got rapidly worse even under treatment. I saw little hole developed a marked eczema variety of extraordinary symptoms. Running eyes, crying with the eye, scarlet, dusky or brown pigmentation. I then traced this part. At this stage I examined to my patients to failing to understand what was behind it all. To October the greater part of November ran on, many cases were now nearly well, but numerous cases appeared.
I noticed particularly that all mistreated cases now end on to severe paralytic symptoms, many resembling head drop, wrist drop, ankle drop.

On November 24th, Mr. Selsman, Reynolds of Manchester startled the country by announcing that he had found arsenic in beer. He once procured samples of the beer in Penworth together with samples of the chlorine used by the brewery, feeling sure that arsenic was also in Penworth, the cause of the epidemic outbreak of instant poisoning symptoms. The application of Kirsch's test quickly proved the presence of arsenic in the beer. I privately informed the officials in the two breweries. I began to tabulate the cases which had severe still coming under my notice.
Extent of the Epidemic.

It appears from published reports that the poisoning had been going on in Manchester and other places for many months, and I will add by Manchester statistics of hangovers, perforated ulcers, alcoholism, and cirrhosis. “Intoxicated adults in one decade together with an account of how excessive intoxication of cheap quality on the market for a long time, that is about a year.

Manchester Statistics
1896 1897 1898 1899 - 1900

Phants

209 246 224 228 340

This represents deaths (total) in each year from the diseases noted. The increase in 1899 - 1900 is at least very suspicious. I conclude
Chol. When describing the Symptoms, especial the Skin condition, it must be accepted as fact that the poisoning has gone on for many months—Liverpool, Manchester, Chester, London, Kendal, Bury, Preston, Lancaster, Ilkley, Leeds, Stowbridge, Highfield, Darlington. Penrith have been all very heavily affected.

In regard to duration of poisoning, I ascertained, from examination of death duties, evidence of the medical practitioners and statements of the assurances, that the first occurrence was on the 19th September 1900. As a result of my suspicions of something wrong with their beer, they ceased to use Ancient's of Liverpool bleach one week before it. Reynolds published his first paper. They had not used this firm's bleach before—
We had no deaths in Scotland due to any of the aforesaid causes, but in England, Tattersall notes that from July to November 1900 there were 41 deaths from incineration, 25 from alcoholism = 66. whereas in the previous seven months of the year there were only 22 deaths.

In 1899 (12 months) 39 only.
1898 .... 31
1897 .... 24

Explanations of how arsenic may be introduced into malt liquor.

Beer is made by fermentation of a solution of crushed malt in which by action of malt diastase, the starch of malt is converted into fermentable sugars and many cases of grain or starchy matter are added. While later on in the process, before fermentation, glucose or invert sugar is added. Invert sugar is often also added to finished beer.
to keep up the "Sparkle" by its continued fermentation.

Glucose may thus be added in the glucose or the invert sugar—
so it is necessary to trace out the manufacture of glucose or invert
sugar, also the conversion of barley
into malt to

(a) Manufacture of glucose—Deoxygenation

This is done by action of Sulphuric acid on starchy matter, chiefly from
maize. The conversion is hastened
by introducing steam pressure. The
formula given for making glucose is
Starch 100, Water 250, 4y 5o. 5

but if a pressure converter be used
the Sulphuric Acid may be reduced
to about 1.5. Therefore in a
wooden vessel with pressure 12
15 to 20 lb. per square inch, provided
steam injector maintains temperature
at boiling point or above? quick
additions of Acid starch—water 15,
destroy maltose composition can
be arrived at in 35 minutes.
maize, rice, etc. Sago root highly sugary, production less than two hours. Finely ground chalk is used to neutralize the acid mannose, the liquid allowed to settle. The clear run off, this is filtered clear of Impurities and is to high colour, is passed thru' animal charcoal towers. Concentrate Sulphurous acid is used to bleach it. The product now concentrated in vacuum pans is complete. Then Sp. Gr. = 1.5. It removes impurities, solids, suspended Tere in hard masses. So far it evident that Sulphuric acid can not sulphurous acid may be the danger, even though the charcoal filter might probably remove some, but not all. The arsenic which might be introduced by milk of chalk or lime used to neutralize acidity may also carry does some arsenic in the deposit. Probably in all, the lowest
(Charcoal) The "hulks" would remove about three quarters of the arsenic introduced. Working on these bases as laid down by Dr. Sharpe, Chemist to Harston Mill Works, Mr. Cooper concluded that the amount of arsenic which might be looked for in khus zero made by sulphuric acid would be about 1.05% 2.1 parts per 10,000. Therefore if 20% might of malt used in a brew in khus zero, that the total malt equals a quarter to 4 Barrels = 2.3 units to a gallon, then we have from 5 to 10 parts per million. Further, it is evident that yeast can remove arsenic from thios. If we allow that 1/8 is so removed, there still remains approx. 28.5-56 pounds per gallon.

If a higher percentage of khus zero, he need use a much greater amount of arsenic will be present. Interest Sugar works out much the same as khus zero as to percentage
A. Brewers—how best to malt—

Definition of malt—

This prepared by steeping raw grain allowing fermentation and removing part of starch changed into saccharine matter—result is "lowered" kiln dried—now the final kiln in the kiln = Anstrains as Cake, may contain arsenic in fact the may contain much or even additives to the grain. Again if a pale beer is going to be brewed, flavour—sulphur is thrown on the fire as if it is a fact that cheap sulphur contains arsenic, this would also deposit it in grani—

how to brew proceed with malt—

- Thoroughly screen the malt free from dust
- Rinse it thoroughly at 160°F for much time, keep two hours, this will complete formation of Dextrin—
- Then it is run off, hopped, cooled, and when beer is ready it added to run into coolers—over on
Thus yeast is added to fermentation vessels, then yeast is liberated from beer in rumint tanks currently for cattle sale.

And Arrenic has been proved by Professor Dixon many times to be present in the “Glucose.” instant syrup, the sheet screened from the malt to the yeast, the yeast, the yeast.”

The explanation is that sulphuric acid made from cheap quality is humanity labeled with Arrenic, so Arrenic in the “Glucose.”

Carbonic acid (cheap quality) are much Arrenic reeefpoint it on flame in the “Kilninig.”

Yeast really contains Arrenic which it has recovered from the Beer. This has been proved by Schru, Rentsberg, Langer, Caccardo and others who have proved that Arrenic is dangerous, hence brewing or other special tendecny to size on any Arrenic while removing some, at some time producing instantly poi-
Converse Ascorinus products.

W. C. J. Witham had shown this Depression in regard to all papers.

Ascorinum (proved) as to the sources of the Ascorin.

(a) In the Chinese Ascorin sugar due to the action of Sulphuric Acid made from cheap Spanish pyrites, which has been found at the Sulphuric Acid works. Analysed found to contain large amounts of Ascorin.

Fortunately only one known instructory means by which this shameful cheap Sulphuric Acid—most evil—was said to enter into the finished product—Chinese—sulphur. Even though having a skilled chemist on premises.

The firm was B. Stolow 17 Liverpool.

(b) On the other or the result of preliminary kiln—drying from the coke—not probably from anthracite.

(c) Possibly also in some years but not proved in Brewing.
(a) Possibly in some hops which are "treated" with cheap sulphuric acid, the producer of the worst of W. H. P. P. manchet who had a popular reach which was traced to his frequently chewing hops. The reason could not account for frequent epidemics.

(b) Might be the Sulphuric Acid (has been proved to in some samples) which is used to bleach hops.

To sum up - hops be grown from malt. Hops in not cheap enough for the heavy beer-drinking working classes bringing Europe = glucose + invert. Sugar are now very cheap. They are made by action of Sulphuric Acid on inferior cane sugar or starch from maize, etc. or from rice, but the king of sugars is directly due to the invert glucose manufacturers using a very cheap commercial Sulphuric Acid to form glucose + invert sugar.

Professor Harold Dixon in conjunction with Professor Delphine + Dr. Sat.
treat of Sulphur (p. 60) has examined many specimens of this Sulphuric acid. His report is as follows:

"I can say that the Sulphuric acid contains more than 1.4 per cent by weight of Arsenious acid.

Also there is very strong evidence that this Sulphuric acid contains fully 0.05 per cent by weight of Arsenious Acid.

The nearest explanation of how the Arsenic comes to be in the commercial acid is that a cheap Spanish Iron pyrites is reacted to sublimine the Sulphur, but at same time this pyrites contains much Arsenic. This also sublimine, & both are condensed. The acid not purified at W. Witham, hitch of Prince College examined 17 Specimens of Beer & 38 Specimens each of Sulphate & Sugar & found the following results. Which show Arsenic in the Beer in amounts ranging from 0.01 to 0.28 grains per gallon."
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**Brewing Sugars**

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Quantitative Analyses

Dr. Reynolds, Manchester, gives the quantities found in Manchester and Salford beers by Professor Delépine to have varied greatly. Various beers were found to contain from 2.5 over 4 parts per 1,000,000. That is from 0.14 to 0.3 grains per gallon.

The burnet sugars he found to contain 0.25 parts per 1,000, the blue one 0.8 parts per 1,000. The Sulphuric acid 1.4 per cent or 4 ounces per gallon.

Quantitative Analyses of Purush Beer

Quantitative

Taking Pure Sesbania Green - 250 c.c.

If beer is evaporated in a porcelain dish over low flame 5100 c.c., then add 25 c.c. of 1% HCl. If the still boiling liquid adds a piece of copper gauge about 1 x 4 inch ball for quarter of an hour - if no darkening of handsome then less than 40 parts of arsenic is present per gallon.

(a)
In testing the glucose 50 grammes are taken and made up to a solution of 100 c.c. with hot distilled water. Steam process gone through, when once 
the copper gauge is finally heated on a flame to reduce the Arsenious Oxi-
dide removed by nitric Acid, well washed and dried.

Both in the case of the Rees and the glucose I got marked darkening or darkening being proved you wash with hot distilled water then with Alcohol dry then roll the copper gauge in a glass tube about 3 inches long heat—next examine under a microscope using 5 inch objective. Then you find octagonal tetrahedral crystals. 

Arsenious Oxi-
dide is 20% ran of arsenic purplish to selenium.

I made out the crystals will join both Rees & glucose gauge.

I did not remove the oxide by nitric Acid —
(b) Quantitative.

Here 250 c.c. or more of the beer is taken evaporated in a porcelain dish to 100 c.c. now add 25 c.c. pure HCl. to a CP5 per gauge 1 in X 3 ½ in. rolled-keep liquid boiling for an hour occasionally stirring. After concentration is marked mark up the bulk with hot distilled water now remove gauge, wash it with hot water (distilled), the first washings being returned to dish, now place each roll in liquid for half hour if this darkens remount rinse it replace first roll after it Arseno has been removed; continue this with rollers until no more darkening is observed.

When washings are completed the rolls are placed in a small beaker ¾ in X 2 ½ in high ¾ in. diam. X 1 ½ high containing 5 c.c. normal 7% HCl. while soas are just to cover the rolls now add 3 in 4 drops 1% a 10% solution of perricid 7 Hydrogen peroxide, moving the
oil up and down in the cold
other mixing solution. Then by letting
it stand the black coating is removed.
Then copper becomes bright again
it is removed washed into another
slanger beaker, all washings being
removed. An each roll is added to
the ha.40 a few more drops of
peroxide are added if necessary
to hurry the removal of coatings.
Now unite the alkaline solution
with the washings shaken out plate for about
an hour, then the copper precipitated
may be filtered away.
Next add 0.01 Frunal Sulphuric
Acid & a little Sulphurous acid solution
until the solution emulls. So 2
may have been concentrated till every
trace of So 2 has gone. Then to the
solution while hot add its own vol-
ume of Saturated 42S water and
5 42S gas. removing to a warm place
allow to stand all night unco-
cered. On following running the
As 2O3 has collected and is filtered off through a very small filter & washed with water till the washings are free from saline matter - The arsenic is now dissolved off the filter with smallest possible amount of hot ammonia-water, evaporated to dryness on the hot plate in a small flat porcelain dish, then cooled. 1 or 2 or 3 drops of hot distilled water are added & drop by drop 1/10 of dilute (1 in 10 or 15) till solution is faintly acid to litmus (this decomposes any arsenious sulphide). Evaporate most of the water to dryness, add 2 or 3 c.c. of 4 N & separate again to dryness, rinse the residue with few c.c. of distilled water & pour this H2O carefully now boil up with CS2 to remove any sulphur-rinses with strong alcohol & afterwards with water, heat on plate till dry, put under a desiccator till quite cold, remove to balance victorious thorough residue recrystallized with hot.
Ammomica water, this dissolving the Arsenie Sulphide by heating attached to dich any Copper Sulphide, put the solution in some gas into a heater reserve dry the capsule on hot plate weigh with some precautions, the difference in weight equals the Arsenie Sulphide.

This process applied to the Bee or Laccare (I had no invert sugar) gave following results:

**Arsenious Oxide in Beer** = 0.616 per gallon.

**Arsenious Oxide in Glucose** = 0.216 per 100 parts

From inquiries at the Brewery I find that 734 gallons of Beer were brewed in the Bee from which I got sample that 224.47 grains of glucose were added for this amount. :: 2316 grains of glucose were used per gallon of beer, this works out to show that 4.86 grains of Arsenic were put into each gallon of mash, in other words the glucose contained virtually 1.63 grains per pound.
had as analysis of beer show:

0.616 g. of arsenic per gallon

Arsenic analysis = 4.85 g. per 100 gallon.

From this it is evident that very much arsenic was removed in process of brewing. Thus:

In 34 gallon brew - Arsenic = 3292.8 g.

But in finished beer - Arsenic = 452.14 g.

Therefore over three-quarters of arsenic must have been removed by the yeast especially the hops.

Unfortunately I could not get any of the "returned yeast" or "draught hops" as these were immediately destroyed, but Professor Telegraph has analyzed yeast after fermentation of beer, also hops, and found them especially yeast to contain about much as 0.05 g. in each gallon of beer equivalent. He has found it much less 2.0 in 100, but also appreciable in the wooden and copper parts of the "inman test". Thus it is conclusively proven that yeast in particular has a great...
affinity for Arsenic - Arsenical or Arsenious Salt.

Possibility of Selenium in Beer.

Selenium being also found near much as 0.3 per cent (= much same as Arsenic) in Spanish Cypriots.

Dr. John Latham of Registrar-General's Dept. says he had Dr. J. L. van't Hoff's Sulfuric Acid examined by Professor Innichitte of King's College who found Selenium = 0.3 per cent. He gave the opinion that Selenium accounted for non tolerence of the Manchester Beers. also for many peculiar symptoms.

This may be so, but as regards the Persian Beers, no Selenium could be found, even by Prof. Macadam in Edinburgh, whom I requested to specially check the Analyses.

Therefore the following Clinical account, which follows is due to describing a history of the effects of Arsenated Beer, especially...
as much as 0.66 per gallon
= .08 percent to .04 per gallon -
and according to Foods Drug
Act, all adulteration over 0.25 per
gallon is liable to prosecution.
It is evident that some very severe
restrictions are necessary in re-
gard to pure beer produce, as it
has been proved that if health
be well screened, all arsenical
dust will be quite removed from
it. This would guarantee pure Beer.

Whereas Beer made sometimes with
as much as 40 parts of arsenic or dust
in beer made from cheap Spanish
or copper arsenide, Selenium laden pyrites,
can never be pure unless very
severe penalties for impurities be
threatened —

In concluding this Public Health part
of the report, I should mention that
many prosecutions have taken place
but the majority have been a
force that has led to nothing.
Part II
Clinical Account.

Subjective Symptoms
Patient invariably complained of.
Sensory phenomena with burning.
Tingling sensations together with a
loss of confidence in their grip.
in their balancing powers. The
latter due to reduction in
such sensation complicated with hypalgesia.
They usually also complained of.
Fast burning sense when in bed
that they were glad frequently to
put their out to cool or dry, owing
to excessive hypodermics.

They also complained of pain
proportional to affected areas, this
strict ankle pain. They said
that cold days intensified the pain
increased the analgesia of sensation,
but aggravated the hypalgesia.
They also complained of shooting
shock pains or general weakness.
They spoke of "walking on hot coals",
"dropping things which they tried to hold"
Three again complained additionally of frontal headache & great burning of
burning of eyes & nose, with a hot metallic dry thirsty feeling in throat which
made them drink often to allay it.
Some complained of hiccoughs severe bronchitis with great shortness of
breath something of rubblos.
Many complained of "Shingles" or "Pimpla", but although nearly every
case showed pigmentation &
some degree, none seemed to notice it
except in the case of one woman
who being sick, or pigmented in the
face thought she was pregnant
(Obraena).

Why two cases complained of sick-nes & diarrhoea, three both shewed typical abdominal spasms after
labor contractions
late seeking advice complained of great thickening of skin of the
Palms & Soles & after worms were thrown out
her because of hair falling out.
Appearances: Aspect

The cases were easily recognized. A heavy puffiness of face, the eyes in many cases running heavily. The eyelids much retracted; the skin of face varying in color from an almost vermilion color, to Scarlett or copper; in some cases much resembling a half-caste. Acne like, papular eruptions were very common, patients usually looked much troubled. Fingers were constantly rubbing hands, fingers or carried hands buried in pockets. The gait was most characteristic in company with facial appearance; it varied from that of a person with cans to that of a paralytic—most walked as if barefoot on coals. Others have lost ankle spring or walk as though flat footed. While more advanced cases with flexus paralysis, resume the slow high stepping gait (heels to toe step). Most advanced cases were most likely to attempt standing would
Despite the chair iron, much at first opportunity, can a rule such cases over, above being ancient in their demeanour, were distinctly sluggish mentally.

Many cases were such emaciated digestive system.

Very few complained of loss of appetite in fact most cases had an increase all complained of the metallic dry thirsty feeling in throat & peculiar sensitiveness of tongue.

In some cases but not all, there was distinct reddening & spurring of the gums & teeth, while the tongue in early cases or corpusca cases showed a fine white moist moist yet not at all flabby or too the indented tongue.

In the later stages this condition was not present, but dry varnish of tongue showed, many similar to a lower congestion patient, often swollen, brown dry necent, but moist at margins.

Vanishing - I only saw this in a few these cases not very marked, but in some
At two places, it has been noted as an early stage symptom with recurrence often every "drink" after meal or meal accompanied by epigastric pain.

Nausea had been a more prominent symptom in early cases. It has usually been characterized by the pain following the material as severe colic which has often produced nausea, retching, or vomiting. Intestinal disturbance has probably been the least marked class of symptoms during the whole epidemic at least those in recurring typhoid.

I should note that W. Reynolds mentions vomiting as a frequent symptom, Manchester. Some of his cases complained of bloody diarrhea, which might have been due to intestinal ulceration, rectal ulceration or to cirrhosis or congestion of the liver, but no one has given any statistics for same cases shown here.
tendencies in the Astites, it is safe to conclude that many cases were Cushing's (hepatic), while owing to the action of Cushing on ductal and mucous membrane, it is surprising how few cases have had severe diarrhoea bloody diarrhea. -- Bramarol mentions Cushing of the liver in pure Cushing poisoning of the cases under Reynolds's account point to interstitial hepatitis such has been proved post mortem.

Respiratory System.

This system shows irritation from end to end, there was the Carpy's, Catarhal laryngitis and Catarhal entere. Larynx with much thickening of vocal cords, almost in one case adenoma, with no distinct Venetian punch in many cases showed marked Simolitus. Some adenoma, such an extent as to produce almost general dullness. This has been proved post mortem. I had no cases of Racemophyria but Reynolds mentions several in non-
phthisical case but he also notes a very striking fact that many cases of tuberculous Peritonitis quickly developed rapid wasting with early formation of many Phrenic cases. He finds a history of latent Peritonitis rekindled by the disease into a severely acute form. This would seem to contradict the present-day treatment of Peritonitis by Aureic -

The case from own showed every cause pulmonarv edema & that secured both Diaphagmore & intracordial pericardium certainty Diaphagmone, as he was in a state of forced respiratory in a very limited way for many hours, with very few inhalation, without pillow -

I have had no cases of Peritoneal-

Circulatory System

In answer, nearly every case developed a low tension pulse, but only two cases developed anything like Jus to in this system. The most Redundant things showing every marked dilatation of heart to the left (to past nipple line) resulting (It is impossible to read the last line clearly.)
Breath to right of sternal line - the heart have once done all right.

Many cases show this particularly in chaster have developed dilatation much past the nipple line for left hand with apex best also extends nipple line such cases produce great post sternal pain dyspnoea on least movements with slight murmurs at apex & base - a "fine piece rhythm" of heart sounds - in chaster this was so frequent & so much accompanied by several body edema, periheal or pleuritic effusion that many cases with thin pigmentation were diagnose as Beri Beri.

Many cases had acids but it is a question whether all were not due to hepatic cirrhosis - all cases developed a low tension pulse & quickening.

In postmortem examination there was very marked dilatation flabby degenerating muscle observed but that was all stomach & plen-
The great majority of cases have thus had, slow tempon pulse, with rapid pulse on slightest exertion. Dyspncea very frequent secondary produced, palpitation & post ophthalmal pain marked dilatation of heart auricular failure has occurred in many cases dilating heart & post murple have giving very slight base impulse, with accentuated second sounds & slight murmurs. Chorstole shortened.

Thus the cardiac failure have been the chief cause of death — Reynolds found edema of feet & hands, or trunk & entire body in 25% of cases. Oedema a few cases & Chloroform Pericardial effusions in a good number especially in Chester.

Bromderl in the Nuremberg Epidemic (Homicidal) found Premortal Poisoning in 1888 found Cardiac Failure to be the chief cause of the numerous deaths.

The sudden deaths showed no distinct symptoms Circulatory beyond loss tension pulse & slight dyspnecia.
Haemopoetic System.

In the severe cases in which occurred jaundice, I examined the blood but could not find any marked change - if anything there was a reduction of red corpuscles & hemoglobin. I made out no increase of the leucocytes - anaemia was not marked.

Urinary System.

In this the anaemia has been very marked in many cases. There have in some instances shown marked increase of leucocytes.

Inexemiable to find any albumen in the urine of any patent only in one did I get a trace of albumen with without any casts. In blood the case cleared up entirely & ait was one severe cardiac failure, I should much doubt the likelihood of any renal inflammation. The patient did merely complained of burning heat at times when menstruating.
Severe cases in man seem to be rare, as shown by frequent detection with lactic acid urine in many cases, and accompanying renal symptoms, as shown by Dr. A. H. Mann, Dr. Abram, and Dr. C. H. Rand. The urine was not found in urine for more than 48 hours after heavy drinking. No cases were on record of albuminuric persisting after cure of alcoholic symptoms, therefore one is justified in believing that no real renal disease has been shown in many cases.

Reproductive System.

In the only married severely paralyzed case in England which came into my hands, I found a total extinction of the sexual reflexes. This man informed me that he was absolutely negative in reaction to any stimulation for two months from time he ceased drinking, his wife had never any remissions.
In examining cases and from
shall refer to circumstances
with acute symptoms in her
child, I found complaint of
exactly similar symptoms. I just
made reference to this by any writers
in the subject of this Epidemic
Integumentary System

This with the hereditary System
were by far the most severely
affected Systems.

As the cutaneous affections have
been the same every where, Pennist
cases showing every variety of
decision, I am largely indebted
to Dr. H. S. Heilie Robert for the
summary of symptoms which I
am now describing.

The patients all complained of swelling
which was flat, hot, painful, red
palm were also frequently tender
there were added tingling, formi-
cation, numbness. This is what
this totally up to the Old Acrodynia
or the Rysthramelaia of
Warhitcull
but there was in addition a very
marked Symmetry taken a great
thickening of the Stratum Corneum
in some cases hyperkeratotic, with-
out however any factor — in
many cases the Dyschromatoplasia
spread to ankles, wrists & between
fingers back of hands — Some again
showed transluscent brown patches
on body, which later on gave a most
unsuspecting designation of a
recurrent nature unilitle yellow
brown pigmentation remaining.
Some showed Phaenomena
taken for Erythema but seen in
brightest red, livid or bluish blotch.
As process sizes, usually quite
separate miliary or even in plaques
of large size, then these began to
designate, like a pernicious patch-

usually the feet, hands showed
these early then limbs & body, but
there was no definite order, in fact
many cases were deeply pigmented
from 7. all.
Nearly every case showed pigmentation, fair people least, dark people most. Very many cases showed vasomotor, pemphigoid, simple papular eruptions.

It has been much noted that the cases which showed most marked erythromelalgia or fever were least affected by paralyses, vice versa.

I should also state here that whereas in Manchester, many cases had severe vomiting, diarrhoea, no such thing occurred in Liverpool, again Liverpool had very little vomiting, Manchester very much - Pethitth little.

Peculiarly the vomiting was also the diarrhoea in Manchester were much due to Scleromycosis.

Brooks has attempted to classify the symptoms under headings of mode of action of arsenic, but has not been able to prevent overlapping.

(a) He described Bryant's toxic condition as due to
(i) Action of Arsenic on Vaccinoma Cutis - thus producing scarlatiniform rashes, erythema multiforme - with circumscribed erythema puris, multifurunculike patches, also papules, vesicles, urticaria, all these in this class terminating in furuncles and desquamation, rarely carbuncles.

(ii) Action of Arsenic on Horse Granks - this makes the great Parasthesia.

(iii) Parasthesia group - accounts for itching, burning, formation of pain, pressure, throbbing, touch sensitivity. The Parasthesia group - this vaccinoma peripheral analgesia gives Flushing, Stutter, edema, analgesia. In horse, motion showing most at pinching, long nerves, arms, hands, feet, suprapubic, also horse-back showing in irritated terminations.

(iii) Action on Intestines of Skin.

This giving pigmentations, dyspideres, circumscribed or (b) hyperkeratization of palms or soles, maybe with surrounding nodules by parakeratosis.
(c) Shaking / Shivering or / shivering / thirstiness / slow growth of nails / (d) Falling out of hair / thinning / hair increased from rich hair / (e) Degenerations / Atrophy / 
(iv) Action in other tissues / 
Liver / Nervous / Tissues / 
Forty degenerations of voluntary / involuntary muscles / nerves, with / degeneration of blood vessels / causing / 
Echymoses / Purpura / 
(v) Combinations of hypertenstion / showing / hypertenstion / Penny / description / whence, / often / 
Peyronie's / with Pigmentation / 
(vi) Effects of Blood on Blood organs / 
Increace of Hæmorrhóblin (whio has / not been the case, except in the reverse) / 
But, also says a tendency / of yellow turning into red, but this / receives no consideration in this / epidemic.
The erythema - much resemble those of this disease, most on trunk & spread on a coppery crust-like stage before disappearing, while they were much accompanied by itching - at first the lesions rhuthiforme like patches were much found about elbows, wrists, knees, sometimes extending from knee to ankle or elbow to wrist. They were red at first, then darkened a blood color red to purplish red. They went into hyperkeratosis & desquamation or only the latter. Commonly around the margin these patches showed popular or papular-septular developments, this eczema-like rash is formed by fell off with an accompaniment of flaky desquamation - The heathen phenomena.

The cause of all this, Vasumater Parasites resulting in itching or edema. \( \text{[1]} \) Face shrinks mostly about face local transient & never inflammatory. The itching also showed stasis producing cyanosis.
of nervousness -

many dermatologic phenomena - Eruptions.

Chief of these being Herpes or pemphigoid patches. Both due to invasion

of nerve trunks. The Herpes was only found in connection with the

cervical spinal roots, sometimes

the eruptions started giving only

pain and no marked eruptions - but

Herpes becomes in the nervous

cases has invariably been bilateral

source was seen accompanying

with any of the Eruptions. The

Pemphigoid Eruptions were seen

most frequently on hands, feet.

Hyperesthesia - was an unusual con-

structed symptom usually accompanied

by any factor. Though usually ac-

panied by Hyperesthesia, yet

in many cases the Hyperesthesia

had all gone before Hyperesthesia

defined. This suggests whether it

was due to Sweat gland cell stimu-

lation or to central irritation.

It was not usually shown on Trunk.
Pigmentation:

Uncommon in nearly all cases, but in some cases, it was more pronounced. In most cases, it appeared on the face and hands, with a characteristic irregularity described by me. In the great majority, there were one or more patches. These were darker than the surrounding skin and were often accompanied by dark circles or spots. The pigmentation was evident in many cases, even in the lower layers of the epidermis. In some cases, it was rubbed off, but in others, it was more persistent. In many cases, however, the pigment was also...
in the true skin, such cases in many instances give promise of permanent clearing.

This pigmentation was shown in any mucous membrane — hyperkeratosis. This, the greatest symptom of chemical poisoning was only general erection of scales on palms though spreading to sides of fingers, knuckles. It showed as an irregular thickening of the stratum corneum, the central triangular portion being less affected — carmo or callositis were much increased, while many cases showed the peculiar flat nodules of hairy tissues, like small hilllocks, all over the palms. Also, in the latter these hilllocks in many cases were like embedded peas — when bore and fell out a deep pit each.

Desquamation varied from rough flakes, thick plaques at first, but all cases finished in a fine early desquamation.
The nails in majority showed marked increases in growth rate, whereas many showed many ridges in transverse lines, some showed longitudinal markings. Others showed subungual keratoma, a few showed keratinisation of the nail bed, and in a few, keratoma of the nail bed. In many cases, keratinisation increased in growth. Others decreased and marked shedding.

Keratinisation: This is a great clinical symptom in its more chronic form. It was in the epidemic outbreak over body, but most on chest and abdomen coming away in large masses as corrugated, flaked and flaked. On the skin, it formed it more fur furaceous on palmar aspects. If sweating very bad, it went on very little, but if very little sweating showed these
the horny layer caused by
hard lacunar teeth.
Degeneration in due to the Arrangeal
action of the Epithelium may possibly
an eliminative process
fatty degeneration.
This has been observed in both
nerve innervated tissue particularly
the heart.

Thus it will be seen that Arsenic
exerts a most powerful action on the
Epithelium in particular, causing
increased granular fluid as to
produce an equally continuous cut-
ting of the superficial layers from
some enrichment = Arsenic, hence
the degeneration.

Yet, whether these effects are the
result of Arsenic or Arsenito, can
not be definitely said, as much
continuity will persist as to the
oxidation action of Arsenic in
the system.
Heroin System

The greater number of the cases had been those of Peripheral neuritis, as many were classed Alcoholic. In some cases, as Bari Bari, I shall first describe the heroin phenomena observed, other cases taken up differential diagnosis from the Alcoholic Bari Bari forms.

The majority of cases have been Peripheral only, but a large number have been Central, as will be shown.

(a) Sensory

These sensory symptoms were most prominent. In every case, this had appeared before any motor trouble.

(b) Tactile thrill - has been a distressing early symptom, particularly in the Psychromegalgin cases. The touch of bedclothes even causing great dread.

(c) Loss of sensation, particularly to much Alchohol has been taken, indicating to Arsenic. This has -
calf symptom has been so very
most characteristic in the muscle
masses of the calves. These firm
gripping produced excruciating
pain; some cases even on slight
superficial pressure were marginal.

Parasthesia was also a very common
symptom. These pin pricks were
as painful as a knife thrust & persisted
very long.

Anacusethria: I cannot find any definite
case recorded. Total Anacusethria
but localized patches have been very
usual = paraesthesias this has been
very common, varying from utmost
tingling, burning, "prick of
needle" or to very marked numbness
in hands, feet, sometimes even to
in the trunk, not far as
recorded, but in a few cases of
Face, Head, such as the 5th nerve
(in Sensory fiber only) as described
by Reynolds.
They have been many cases of
parotid heat disease. There seem a
very hot bottle was described as slightly
heralding of severe degrees have been
succeedingly common that in most
cases have preceded break (see before)

Motor = (summarize in had Alcoholic cases)

In the symptoms were very common
especially from or progressed from
a blunting of power, to loss of tone,
weakening of muscular tone in many
cases a head equal part from the
worse case: a total paralysis with
very marked atrophy. The usual
sequence of affection was, first the
motor, then muscles of the forearm
particularly the extensors, then all
muscles of limb and limbs, but just
as in head Poisoning if the upper
arm muscles were not affected then
the extensor longus escaped,
no "wrist drop" occurred. Many cases
however showed very marked
"wrist drop"

In the lower limbs, the worst symp...
Some were most frequent, the early stage there was irritation of the extensor of the toes, chiefly the big toe, but soon the muscles became paralyzed, wasted, foot wasting, then the intrinsic groups, causing flexion of the toes and foot wrinkled. Patient, the calf muscles became affected, then the thigh group, when rapid muscle wasting set in, quick paralysis often followed. At this stage the muscles on front of trunk failed, patients could not raise themselves. Then diaphragmatic paralysis sometimes came on. Some misfortune? Intercostals. The sphincters were more paralyzed. There was never any Central nerve muscles paralyzed. In many patients got quite incoordinate in their foot movements, but no definite Ataxia has been proved in any case on record. Certainly some sway in balance (sensory) but never as an Ataxic does.
Only one case is recorded of polyneuritis.-

Reflexes - The superficial reflexes have

been impaired as regards

swallowing, bilateral defaecation.

The deep reflexes - These have been

much affected. At times when the

spinal cord showed irritation

in the posterior horn, the knee jerk

was usually exaggerated, but those

were unvaryingly ankle Clonus. When

the calf muscles began to fail over

the thighs, then knee jerks disappeared.

The superficial reflexes were first

exaggerated then went altogether in bad cases.

Graphic Phenomena

There have been noted in the

segmentary system in part,

but omitted to mention the ten-

dency toсудорога, also the cocaine

case of producing sweating from

bottles also the " debtor skin" phenomenon.

Waiting of muscles, knees, back have

been noted in all marked cases
Muscle wasting, the reaction of degeneration has been very definitely dilated.

The early toxic influence in most cases has been noted in mononucleosis. These patients had increased appetite or general feeling of wellbeing. This would support the theory that arsenic is an oxidizer or co-factor in the system, up to a degree.

Central Phenomena

In many cases, particularly heavy alcoholics, there was very marked loss of memory, locality, time, faces. Also marked loss of intuition in sequence of ideas, many were acclimated to fright, horror or day, horror and if only shutting their eyes - this has been coaxed as an hereditary symptom because they always see faces or men, but animals are in the little - but according to Reynolds other patients or many cases, he concludes that arsenic has very little effect.
The Cerebral Cortex —

Secretary System —

The sweating glands have been shown to have their function not much stimulated. Bulk secretion must not be mentioned as regards function but in some cases I watched I found very little if any increase. In fully 80% the other hand it has been conclusively proven that crenic has been excited in the bulk remained severe mental Cerebral

Demisea melancholy.

Temperature —

The majority of cases have shown no definite rule but many cases both Rhythmicans & Paralytic have shown Feverish: Temp. = 101°5 102°

Feverish: Temp. = 102°5 103°F.

This often ceased at end of fortights or three weeks. In Thos. it continued until death & in others again it began early, ceased for a time returned this being a very ominous sign.

(See chart) —
Differential Diagnosis.

(1) Ordinary Simple Arsenic Poisoning:
When small doses are given, affecting appetite, general well-being. Stone nematode enters system the stomach has been the almost invariable rule in this epidemic -

(2) Intestinal Fever or typhoid. Burning pains in abdomen. Phlegm in stomach in 15 minutes, with constipation. Metallic, burning dryness of throat, then intense dyspepsia. Extension from mouth occurring the entire alimentary tract length to rectum over violent purging, vomiting, acute, choleraic discharge gradually commence, urine suppressed, arterial tension falls, respiration labored, by weakening of muscles. Face cyanosed, cramps may be convulsions within death comes in about 30 hours. There is great resemblance between hence arsenic (hyster) poisoning above described & cholera, same in postmortem appearances.
(3) Subacute pneumonia — a more common symptom complex. It is often resistant, especially the vomiting and diarrhea (rinsing brick case). But other characteristics always had no persistent thirst, painful swallowing, scanty urine, red-tinged albuminuria, heartbreak irregular, face cyanosed skin cold blanched overlooking. If the smell of acetone is hydrogen — camphor-scented, but a clear mind. The symptoms frequently remit, but gradually death comes — one large dose if it does not prove fatal quickly usually produces her - to time symptoms = Sweaty such as Eubacterium or typhoid fever. Fever of themic塞西 Paralysis which usually begins in legs, then muscles quickly waste atrophy to lose the function of degeneration early.

These same symptoms follow in subacute cases. Subacute has been taken for a long time — later with symptoms a special case.
When a young man Maddhall
three symptoms became acute:
The first, Patellar Cuneiforme Reflexes disappeared. Then, since these became marked below knee, the fourth advance symptoms as those of multiple heredity -
there was doubt, different organs exhibit selective influence to Arsenic so that acute case shock most damage in hastis, Intestinal, mucous membrane, while chronic forms shock most in skin, liver, kidneys, heart, various System -
regarding skin effect, Hutchinson in 1843 observed a young woman who had developed forward Palace of 3/4th's from Arsenic, when young. Another had developed miliary pustules in a thickened Epidemic - 1846. Lane Alcott have also seen the same, but have found the first and pustules. Eruptions and Brosches mentioned a case of Eczema from using Arsenic tainted playing Coals many writers describe the pigmentation -
I. Hutchinson also mentions herpes as causing mucous membranes from histogenetic stress & quite asymmetrical. He mentions Basanias - even not the syphilitic cancers after Arsenical Keratosis. He mentions a case beginning with pigmentation of skin, progressing to Basanias at elbows, knuckles, palms, then keratoses. The base began benignant, epithelial cancer. He also found it lower cancer epilepsy.

Case is recorded in London in 1884, where a child suffering from slight chronic hemiplegia developed degeneration after fourteen days. Treatment with temperature rising to 101°, histologic examination, settling in every entire body, some segmentation of paralysis following both legs, knee jerks disappeared - numerous from source of infection. Segmentation was easily obtained. She walked with the "dropped ankle gait." They were convinced of the Arsenical cause by ceasing administration. They symptoms immediately left.
...rium and in Acromone Poisoning, hemorrhagic symptoms appearing early, sensory motor weakness, ulcerative, ulcers, mucous membranes also affected. Many seek to abate many who be mine to...The time to take London Refuge go superficial remain...

As regards the early manifestations in Acromone Poisoning, Paralysis. After usually found diseased bowels anterior and posterior paries, plexus cerebra, segmental cord, sensory degeneration, vacuolations, hemorrhage infrom extraneous growth of connective tissue lends the cord, motor in throracic columns. Spinal ganglion cells also frequently degenerate. Blood vessels also degenerate causing hemorrhages...

Glycogenic Polyneuritis

This is common in women of very early years of ovarian function. Contrasting to Acromone form, this relapsing, remitting, irregular bowel. The cause of symptoms of disturbance. Symptoms arise as soon...
cold extremities, muscular twitchings, weakness, thin mental impairments such as loss of memory. The hernia
ocket is becoming a very frequent symptom. Often shows as early as the breast symptoms. It generally comes
on insidiously, often short paresthesiac symptoms, such as exaggerated reflexes, numbness, tingling of fingers
thin corneareceptors, disorders of cold in the extremities, bluish lines, thin muscular spasms, especially at night.
Sciatic fibrous nerve, during pain, such as Lhermitte's, lumbosacral. Thus causes the most characteristic sign
of Ataxia, as hyperesthesia of the muscle masses, especially the calf. Cutaneous localized hyperesthesia are common.
through all general - mostly from knees down. In more advanced cases muscular paralysis extenui may
give Ankle Drift, then Atrophy. Ruiners begin to produceennis
position, movement restrictions of limbs, toe wrist drift, ankle drift,
High stepping gait are established
The trunk muscles diaphragmated
suffer severe paroxysms the facial
muscles, giving loose lips, wide eyes
the phrenic particular become
weakened, while Ptosis, hyposthenia
and ptosis may arise i.e. by Epes et
is to be believed) reaps upon
Symptoms establishes although
as a rule perspires remain widely dilated
True atrocity is much doubted
The Sphincters rarely affected although
when mental influence arrives, dysuria
and retention are common.

These effects are exaggerated in early
stages but soon disappear, while
cutaneous reflexes usually, enfeebled
or abolished.

Reaction of degeneration does not give
distinct sign until late stage
In regard to skin, congestion & their
Tertiary local gangrene have been seen,
while pyogenic pyodermics although
sometimes seen is far from usual
of present mental facts—
Instal advanced cases have the skin becoming very dry & scaly.
Sensitivities to all sorts of substances occur from now to. These changes localized edema is very characteristic of alcoholic neuritis, chiefly on the trunk & upper limbs.

The symptoms usually take two months to develop fully & improvement usually begins in knees at first. In rare cases, some rare cases develop rapidly & kill rapidly much like typhoid fever.

Mental changes are three stages of symptoms: Excitation, lachanolin, convulsions, dementia.

Post mortem - The Spinal cord shows changes are much the same as in Peri-Peri.

Beri-Beri -

This is an endemic epidemic multiple peripheral neuritis, characterized by a special liability to implication of the Encephalitic & Peripheral nerves giving much direct gastric failure. The cranial nerves are otherwise exempt. States causes marked edema of
Connective tissue free of much effusion into the serous thoracic or abdominal cavities.

The organism requires moisture that delights in overrunning bad sanitation poor feeding.

The lesions in this disease are all those usually associated with disease of the peripheral nervous system. There is a questionable symptom except in many acute cases.

Redema is one of the marked phenomena being an invariably prominent symptom commencing over the shins about ankles may even spread up to become general. Then in moderate form it is localized such as in about flanks, neck, sternum—may vary daily in less of a soft that renal redema, while it also differs from edema, renal. This edema most often affecting the extremities sometimes skin localized to hands turns it chorea quickly developing changing appearance area as honey or the edema usually precedes
Parece to anaesthesia.
The urine usually contains a specific gravity remaining very slow. It is sometimes Albuminuric may occur but not necessarily.
Anaesthesia, Paraesthesia; the per- 
creations are much examinable.
Alcoholic & functional neuritis, but 
Paree is a much more prominent 
symptom in Beri Beri. It usually af-
fected most groups, though with- 
face often escape, also Eys, 
achination, & neglect, but the heart muscle 
usually affects very early degenerates 
fast, causing many deaths. 
The test is an in Thae neuritis 
hyoanaesthesia. Nocieus is frequent 
& Cramp common. Atrophy is 
the rule of the sedentary laborer. It is 
also very frequent & a charac-
teristic symptom is that reaction 
Degeneration is usually marked 
before full symptoms of Beri Beri 
develop.
Patellar reflex less normal. Superficially normal, but gives increased sensation of Dorsal sympathetic gland nodes.

The blood pressure less normal, pulmonary early on excitation of brain, innumerable reduplications are common of early palpitation being an early symptom of the accompanying dyskinesis - Cardiac depression is an early symptom.

Cardiac dilation, pulmonary Pericardial effusions are very common.

Dyskinesis an early symptom but constipation is a usual late one.

To Sum up -

Acute alcoholism differs from alcoholic: 1st In the absence of severe incontinence 2nd The skin lesions are absolutely foreign to alcoholic incontinence - (Pigmentation, Kerato角膜, Erythema, Lupus, Erythematosus etc.)

3rd Herpes Zoster, Drug dermatitis, 4th Acne vulgaris, 5th Hypertension, 6th Liver, 7th Menstrual disorders 9th Absence of sweating.
Slum tongue - Conspicuous was the finding of arsenic in urine -

To differentiate Arsenical Poisoning from Beri Beri the signs and symptoms are quite ample While the early edema localized to feet and ankles redness of skin, redness of conjunctivae, and urination of red blood, symptoms of perineal effusion are the remitting recurrent symptom must be excluded at once diagnosis Beri Beri from Arsenical Poisoning.

Haring thus observed the manifester in the two chief medical which may resemble Arsenical Poisoning having drawn attention to the distinguishing points it is very clearly seen that this epidemic has been Arsenical in its effects although many cases have been augmented by the alcoholic element chiefly in cases of alcoholic "Soakers"
From the foregoing account it is
easily evident that crescentia in
Crescentia paroxysm affect the
Skin, hence, Systems which have
a change also marked on the De
data Cardiac, urinary or systems
which include here or. Many argu
that its effects are from nourishing
from action, owing to its poring
properties, others again hold that
it is a constant force, simple
can explain its presence in skin
or as due to freeehiniation.

Regarding Sequence of Symptoms
Bernard's table is quite confirmed
by Reynolds' theory:

1) Digestive Symptoms
2) Ischemic
3) Cardiac
4) Acute Skin Symptoms
5) Disturbances of Sensitivity
6) Mental Paralysis
7) Pigmentation
8) Tenderness - Reynolds mentioned
Widal's data in hypero epidemic
as follows.  Feb 8. Sore trouble
with shivering, was 4. Acute autumn
symptoms. Pandemic cough...
Runny eyes & nose — March 31.
Sensory disturbances of limbs, then a few days later, fasces of the upper limbs. Then slowly restored
in one year.

Prognosis.
The full symptoms of the case run
away slowly severe, but with
some mild cases, the objective
respiratory & sensory symptoms
soon pass. Also the skin becomes
normal except leaving only a
pale brown pigmentation, but in
some cases, the hypermelania has
persisted for months. Regarding the
sensory or motor symptoms, it is
safe to say they will persist
for a year or more in balance.
And myself have seen cases
with slight persistent overlying
parasthesia. While one case
Whose chart I enclosed has
every likelihood of going on to
an ascending Paralysis —
The general mode of death is...
by sudden or gradual heart failure
or by acute pneumonia, Cir-
rhosis of Liver or Post-Emigration Pneumonia.

Classification of Cases in Leucitis
In 44 cases which have passed
this year, I can classify them
as follows:

(1) Those with Sensory Motor Symptoms
    almost entirely

(2) Those with Acute Symptoms

(3) Those with Respiratory Cardiac
    Symptoms chiefly

(4) Those with Acute Symptoms
    Enteric Intestinal chiefly or only.

This includes one infant.

(5) Those with Complete Sequence
    of the Subacute or Chronic Symptoms

Descriptions of Typical Cases
In beginning this treatise I had
intended giving many detailed
cases, but finding it better to
describe the Disease fully in
each system by extracts
from my various cases to,

[Further text not clearly legible]
I have been able to give an absolutely complete account of the cases clinically and medically describe the case from each group I have arranged. I had no deaths in birth therefore cannot give my Pathological Anatomy, neither can I find any published up to date.

Sensory Motor Disturbances entirely.

Within Sanders 42 - Married, stout, shapely complained on Dec. 2nd 1901 of loss of confidence in his grasp or touch, also a feeling of wearing too thick stockings or RCS. He only drank about 3 pints per day with the mentioned desire as cause he was incredible. On any minute examination I found his appetite very good, his digestion excellent. Cardiac Respiratory System quite clear, urine normal - his Skin tanned not even pigmentation - his Choppa, no Anemia - J3 enlarged liver - his temper perfect very normal up to first joint in hand swell on 2nd joint in foot but only far as then.
His patellar reflexes were quite absent.

The plantar was very faintly elicited at the superfi cial surface.

The ankle tendon very faint.

The plantar sense was very much affected in that he could not distinguish cold from hot, but being merely indistinct toucher he complained of cold extremities being very anxious and much dependent.

He said the manure had been in for 4000 feets, and he was much on light duty.

He had more hyperventilation muscular manner, especially calves although reaction of degeneration was not altogether shifted.

I am aware that he would have developed it soon unless I had put him to bed with massage and other treatment.

He was perfectly well in 6 months.

He never allowed any temperature.

His joint was "hot"—laboured, but he had no unrest as ankle drop.

He had no restless ankle peroneal pain.

He was not an alcoholic.
In a quick he unusually indicated very virulent encumbrance. I bear premature to my present others cases also drank at same lieu, but did not partake it exclusively as he did.

Case of Cutaneous lesions.

I found cutaneous lesions in over thirty of my cases, but the following was the truly remarkable case.

George Dobson, aged 32, complained on Dec. 14 of "pimple on head and face". I examined and found him suffering from a small papular eruption on scalp, face, and neck, very few on face, but he also showed a dirty dusky pigmentation of almost his entire body, especially on neck, around nipples, axillae, and other prominent prominences. The nipples were absolutely black. He also complained of severe pain neuralgic down front of left forearm taking line of left b. intercostal space. He had very slight constipation, but no digestive
Cardiac, urinary, respiratory, loco-
rectory symptoms - In three days
he developed very marked Shingles
in regions of pain at same time he
complained for first time of thickening
This lumbosacral, which developed
slight Rhizomelalgia, but not at all
severe, followed on by severe hyper-
keratosis, with horny nodules, great
hyperidrosis - His motor symptoms
were virtually nil. His deep reflexes
only showed exaggeration, while
his sensory symptoms were very
mild, only amounting to "dulness"
of touch - a bare sensation of pins
and needles in fingers. He had no muscle
pain. He soon began to throw off
very thick epidemics. This has since
been followed by repeated desquam-
ations of a fine flaky nature. The
sweating has been his longest con-
tinued symptom, but has been
aggravated by his occupation
of a Blacksmith. He complained
of Hypokinetic up to end of February.
Case of Respiratory Cardiac effects prominently but without marked pigmentation of skin affected.

John Harman, aged 64, sculptor, came on Jan 2 after Christmas, complaining of intense thirst, shortness of breath, and bronchitis.

On examination the man was suffering from severe headaches, fever, dry skin, marked congestion and congestion of tachycardia. Frenular congestion is more on each side of the neck. Hoarse voice, intense dry metathoracic thyroid tenderness. The entire head of the patient was almost reduced to the superficial capillary hemorrhages, were red and very much swollen, especially posteriorly, hoarseness. Tongue had a dry brown coating, with white at sides. Bronchitis was very marked on January 5, a few days' pulmonary edema of both sides, severe need to acquire deeper inhalations. The cardiac condition rapidly got worse with occlusion dilatation both to right and left. Pulsation at base, hipple line occurred with general soft systolic murmurs.
Everywhere a timepiece rhythm? Heart beats, the second usually being almost uniform. He - his pulse was 79. Low tension, large volume - 108. When street, very much more if many way excited, even sitting up gave most alarming dyspnea. There was marked cyanosis. J. figures for lips - same color of foot but indurated patches on mental face - left much - no local edema. Thinner, discutendums being converted of general dark "half earth" pigmentation general over body, but most on knees. Presence, pellagraoid areas on both elbows & very irritating eczematous lesions - His hands feet showed the usual erythematosis with marked hyperkeratosis. J. figures, sides, feet, hands - very profuse manifest hypodermic, a marked transverse ridging of the hair, with splitting and very marked filling out of hair, He showed very slight motility or any phenomena - his breath
were not seen gone, but unusual
his part every fair, but for tenderness
I felt very little real paracenthesia
femoral deep, from heat no mus-
tenderness. I found he had
a trace of pneumonia, but as his
temp. was 100 to 102. His cardiac
condition alarming. He has a cast
of blood in urine. I believe he had
no renal mischief. The clearing
up entirely, proves this. He had no
numbness, but his appetite was gone
she had diarrhoea but with only
slight after-cramps.

I consider this an acute case,
to account for it, that this man's
thirsty burning metallic thirst con-
sumption becomes so unquenchable
that, unwashing in his yard, 10 yards
from an "infected" inn, he sud-
to much his daily beer allowance
from 2 pints to upward of 15 or 20
daily. This weight ten days if the
American beer just coming to the
inn to be sold, he had been think
ing heavily for ten days when he came to me, so that I reckon he was severely under auspicie for three weeks when I took him in hand—
the got entirely well—oxygen inhalation was given while I was not sure at first time Digitalis later on I gave small amounts of Marmarey Digitalis and Drakenberg. So I finally gave him Strophanthus or the Hydrocortisone acid. He was ill until beginning of February but the skin is faintly brown pigmented now. April 22, 1501.

Case of Acute Enteric Intestinal Symptoms
I saw this most marked in two cases only—one a young man of 18 years an apprentice Painter, who when he fell the piper one throat sickness thought he had head Colic other symptoms her symptoms were produced in a few days probably May 18. He came 21st 24. characteristic marked emaciation, jaundice, very faint abdominal tongue & slight dyspnoea respiratory.
With vomiting after every drink or meal because diarrhea, with very painful after opium. He had no thin line unguent. He was not sure to them he quickly got all right.

The most interesting case in a child 75 months. He was thin, very fond of start drinking 4 or 5 pint daily. The child showed very marked cyanosis. The tongue, marked redness of diarrhoea with after colic. I suspected that was wrong put mother on to drink an eat malt start very quickly the child got well, then the mother. She had by this time got much pigmented with several patches like Erythema Paries (very painful), marked corrugation of the joint of the paries and the skin. The room was given up alcohol entirely. Both mother and child are perfectly well. I did not try to find arsenic in the mother until.
Breathing became symptoms -

S succeeded Cooper at 82 - came to

me in second week of October 1901,
complaining of pain, tingling sensation
Phenol. feet, severe thirst, burning throat
burning eyes opened his brightness. Scarlet
ness rash on face with papules almost acne-like eruption. Though it

warmed eardrums as well as forehead

Face - I advised him to change his hair
thinning it was too crude in alcohol
but very quickly I found him putting
much voice rest I was puzzled.

He knew I was beginning to drink and
another Benj. Parliamentary Bear.

Neurur waxing but had gained appetite
no thirst. He had marked Coryza,
congested Ears - In Respiratory Symptoms
beyond faint hemorrhage. His Carotic
Symptoms much pronounced. Palsi-
flotation + slight shortness of breath
with low tensive pulse, quickened by exertion.

His blood was quite normal -
his heart enlarged + distinctly tender
but had been so before.
primary system was all right but for dysuria & reduction of amounts. He showed a peculiar loss of ophthalmic oils in their own negative all otologic for two months after being seen by me. Skin rash symptoms were numerous beginning with a patch on the back of the face, then erythemato, reddish, like patches began at the ophthalmic knuckled of the lower back, abnormally time he developed erythromelalgia & hypertelorite with the fever one had also marked painful wasting deep trophic, serosal, & exudary. Then his pigmentation quickly affected from verminous to dirty brown around the neck & black. Then he became very ill by an acute febrile, trunca paralysis, mental symptoms, & insomnals that showed plentiful red over both his front of the face. This was accompanied by very great pain in all muscles, severe headache, no sense of the hands & feet.
The influence of alcohol must in this case be taken into consideration. However, there was general numbness but no anesthesia in any marked degree in tingling, burning, prickings, or Thursts. But such gradually lessened to almost complete numbness below knee level, having trunk head and face his last recuse was just under such that blankets were given him for too much sensation. He frequently put fags into air. Then again his hands would be "too cold" vice versa. In fact one time, but the robe would scarcely he felt another time would feel chilly.

Then motor symptoms progressed very rapidly from back tone to almost complete paroxysm, having less power muscular wasting with reaction of degeneration. This especially so in intersosseous, branches of forearm, especially extensor. Then producing "dropped" upper arm paroxysm, corresponding symmetrical muscle in lower limbs. Further decrease in ankle drop; high stepping.
certain fact was established first. Then he became confined to bed.
This instance of affection of the lungs much
than his respiration both deep and superficial
sharply. But he failed no further.
His diaphragm, intercostals, con-
cap. Sphincters escaped restriction although difficult to keep all
night, esophagus deficient in restriction.
Respiration seemed not at all affected
as regards skin.
He showed no mental changes.
This temperature only ranging about
101-102° for about 10 days.
He got very far towards well under
improvement but at present moment
is still deficient in his intercostal
muscles. Great change of temperature
especially cold) very recently in
hands, feet - he decomposed first
in large plaques then as a fine
flake for some weeks. The pernio-like
patches following exist. Lesions
will be several months yet
before lasting recovery. Orthostatic coma normal.
### Dr. Maw's Clinical Chart

**Name:** J. Brown  
**Sex:** Male  
**Age:** 30  
**Occupation:** Farmer

**Disease:** Arterial Lesions  
**Complications:** Multiple Lesions

#### Dates of Case

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- **Pulse Rate**
- **Respiration Rate**
- **B.O.**
- **Amount of Urine**
- **S.P. Gr.**
- **Albumen**
- **Sugar**
- **Reacts**

**Entered Stationers Hall:**

S. Maw, Son & Thompson, London.
I have put the chart of an alcoholic American case which began on Dec 6 simple with skin lesions and paroxysms of temperature, all was going well till February when temperature rose again and it is going more irregular. The show many symptoms from early recording analysis.

Treatment:

The great point here is not to give Bismuth derivatives. So that other wise it becomes a case of treating symptoms, diet of all alcohol and arsened water, re-cupration then treat the respiratory conditions with ammonium reseratants, morphia to check secretions. Belladonna Balsam found useful. The gastric and intestinal symptoms are easily overcome with simple Bismuth Arsenic Acid mixture. The cardiac symptoms require digitalis and certain like Phenaacetin. The intense pains require morphia, the lesser ones Phenacetin.
The Skin lesions must be treated as per symptoms — caution to those frequent in pregnancy.

In the late menitis I found very small doses of Soda and Potassium in Ammonia given with Anchoa and Staphanthon to give quick relief. Whenever the proximal pains and temperature have gone begin very gently with smallest doses of Staphanthon in addition, gradually remove the Soda. Then when once that all active menitis has ceased you may increase Staphanthon and begin gentle drainage. There is very little in the treatment provided always that one avoids Cardiac depressants, remember not to dream of using Staphanthon or Electricity until every sign of menitis has gone. Regarding electric treatment it is best to look to muscles at first stage of the treatment. 

Thence later —
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