Beryllium metallicum

"A second group of clinical cases has been reported in which, following exposure to certain beryllium compounds in fantastically small amounts after a period of time varying from months to as long as seven years a chronic form of beryllium poisoning results."
(Harriet L. Hardy, 1950)

"In our study of cases of chronic beryllium poisoning, we have been impressed and stimulated by the observation that women who have been exposed to beryllium compounds may show no evidence of disease by sign or symptom until a successful pregnancy has been completed. I want to emphasize that the pregnancy, itself, appears, if anything, to be a helpful process, but when the child is 4 to 6 months old the patient may notice inability to gain weight, shortness of breath, and cough."
(Harriet L. Hardy, 1954)

"When in 1948 Hueper proposed that the sarcoid pulmonary manifestations of berylliosis might be followed by outright malignant lesions in the lungs, this suggestion was received with a great deal of scepticism."
(Wilhelm C. Hueper, 1955)

"Beryllium and beryllium compounds are carcinogenic to humans."
(International Agency for Research in Cancer, 1993)

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Smaragd, Glycinum und Beryllium - F. Zippe

„Unter den Mineralien, welche die Alten **Smaragd** nannten, befindet sich eines, welches noch gegenwärtig diesen Namen führt, nachdem alle übrigen, die mit ihm nichts gemein haben, als eine grüne Farbe, als Abänderungen anderer Mineralspezies erkannt wurden. Dagegen gilt der Beryll, der vom Smaragd als gänzlich verschieden betrachtet wurde, gegenwärtig bloss als eine Farbenvarietät desselben. Smaragd und Beryll wurden im Jahre 1798 von Vauquelin zerlegt; man hatte sie früher für Verbindungen von Kieselerde und Tonerde gehalten, er entdeckte darin eine eigentümliche Erde, welche **Glycinerde** genannt wurde, weil sie mit Säuren süß schmeckende Salze bildet; die deutschen Chemiker nannten sie **Beryllerde**, welche Benennung insofern nicht ganz passend ist, weil die Mineralspezies nicht Beryll, sondern Smaragd genannt wird. Das Metall wurde im Jahre 1828 von Wöhler und Bussy dargestellt; so wie die Erde erhielt auch dieses zweierlei Namen: **Glycium**, wofür man auch **Glycinum** schreibt, und **Beryllium**. Es zeigt sich als dunkelgraues Pulver, das durch den Polierstahl Glanz erhält, ist sehr strengflüssig, in der gewöhnlichen Temperatur an der Luft beständig, ebenso im Wasser; bis zum Glühen erhitzt, verbrennt es mit lebhaftem Glanze.

Im Mineralreiche findet sich die Glycinerde nur im Smaragd, Chrysoberyll, Phenakit, Euklas, Helvin, Leukophan, Melinophan und Gadolinit Alle kommen nur in kristallinischen Gebirgsmassen, teils als Fremdlinge, teils sporadisch, teils auf besonderen Lagerstätten vor und gehören zu den wenig verbreiteten, zum Teile auch zu den sehr seltenen Mineralien.

Von den Glycinerde enthaltenden Mineralien ist der **Smaragd** eine Verbindung von 13,84 Glycinerde (welche 5,1 Glycium enthält) und 18,75 Tonerde mit 67,41 Kieselerde; er findet sich fast stets in sehr netten, mitunter sehr grossen Kristallen als Fremdling im Granit, Gneus, Glimmerschiefer, Talkschiefer, auch in Drusenräumen und Nestern im Granit, seltener auf Zinnerzlagerstätten. Die schönen zu Schmucksteinen tauglichen Smaragde kommen bei Santa Fé de Bogota in Peru mit Kalkspath auf Gängen im Tonschiefer und Hornblendeschiefer vor. Die Alten erhielten den Smaragd aus Oberägypten; in neuester Zeit sind dort am Berge Zabarah die Gruben aus denen er gewonnen wurde, zwar wieder aufgefunden werden, geben aber keine Ausbeute mehr. Plinius rühmt besonders die skythischen und baktrischen Smaragde, von deren Vorkommen gegenwärtig nichts bekannt ist, wenn man nicht etwa die in neuester Zeit bei Katharinenburg aufgefundenen bisher zählen will; die Kristalle finden sich dort im Glimmerschiefer, sind oft einige Zoll gross, jedoch unrein und nicht zu Schmucksteinen tauglich. Kleine Krystalle kommen, ebenfalls im Glimmerschiefer, im Heubachthale in Salzburg vor, sie sind jedoch äusserst selten rein. Viel häufiger finden sich die mit dem Namen **Beryll** bezeichneten Abänderungen, deren Farbe verschiedentlich grün, blau, gelb und weiss, aber nicht lebhaft und rein grün, (nicht smaragdgrün) ist. Die durchsichtigen Berylle, als Schmucksteine **Aquamarin** genannt, kommen an mehreren Orten in Sibirien und in Brasilien vor; nach Plinius erhielt man den Beryll aus Indien. Sehr grosse trübe Kristalle finden sich im Granit bei Fahlun und an mehreren Orten in Nordamerika, kleinere sind in Irland, Frankreich, Bayern, Schlesien von Schlackenwald und von Ullersdorf in Mähren bekannt.

Euklas, ein sehr seltenes Mineral besteht aus 23,92 Glycinerde mit 32,4 Tonerde an 43,68 Kieselerde gebunden, kommt auf Klüften im Chloritschiefer bei Villa ricca in Brasilien vor.

Phenakit (mit 1,66 Glycium) enthält 45,1 Glycinerde und. 54,9 Kieselerde, ist als Seltenheit

blos von Perm im Glimmerschiefer, vom Ilmengebirge auf Nestern im Granit und von Framont in den Vogesen im Brauneisensteine bekannt.

Chrysoberyll, Verbindung von 19,75 Glycinerde und 80,25 Tonerde, wurde zuerst in Ceylon im aufgeschwemmten Lande, in Brasilien im Sande einiger Flüsse, welche auch Diamant führen, gefunden; später fand man Kristalle im Granit bei Haddam in Connecticut und im Glimmerschiefer bei Katharinenburg; bei Marschendorf in Mähren hat er sich als Fremdling in zerstreuten losen Blöcken, sogenannten Feldsteinen, die aus Gneus stammen, gefunden. Der Chrysoberyll der Alten war eine Varietät des Berylls.

Leukophan enthält 11,51 Glycinerde mit Kalk und Natron an Kieselerde gebunden, findet sich sporadisch im Syenit in Norwegen, so auch der **Melinophan**, der nur 2,2 Glycinerde enthält. Im **Gadolinit** sind 2 bis 11 Prozent und im **Helvin**, auf einem Lager von Blende und Kupferkies im Gneuse bei Sehwarzenberg in Sachsen, dann bei Modum in Norwegen vorkommend, sind 9 bis 11 Prozent Glycinerde enthalten.

Dargestellte Verbindungen sind über 40 mit unorganischen und 10 mit organischen Substanzen bekannt worden.“
(F. X. M. Zippe, Geschichte der Metalle, Wien 1857, Glycium, S. 358-360)

A piece of ore set in gold - M. Vasilyev

“The ore, or rather the mineral, is beryl, the exquisite beryl. It occurs as a multitude of gem stones-emeralds and chrysoberyls, aquamarines and euclases. Exquisitely faceted and set in precious metals, they adorned the crowns of emperors and the fingers of the rich. Yet, beryl is just a silicate of beryllium and aluminium, the sole source of beryllium metal.

Endless stories can be told about the pieces of beryl so highly valued by man for centuries. Much blood and sweat has been poured for these attractive but otherwise useless stones. Many lives have been lost and many underhand schemes have been devised because of them. However these are all bygone things about beryllium. Its future is altogether different.

The first man to run on beryllium was the French chemist Vauquelin who had spent years investigating gem stones. In his report to the French Academy about his discovery in 1798 he suggested the name "glucinium "from the Greek for sweet, as beryllium salts tasted sweet to him.

Beryllium metal was for the first time obtained in 1828. The tiny pinchful of dark-grey powder contaminated by impurities was not enough for the properties of the new metal to be established. It was not until 70 years later, at the turn of the 20th century, when beryllium was obtained in a relatively pure state by electrolysis, that investigations into its properties began.

The properties have proved truly remarkable. Probably only titanium can vie with beryllium in the happy combination of properties as useful to man.

Beryllium is a hard, steely-white metal closely akin to magnesium. It has a very low specific gravity (1.82) and melts at 1284° C, or at a temperature much higher than magnesium or aluminium does. This is a great advantage, because in the future planes will fly at hypersonic speeds and, as a result, in the face of intense heat. Yes, heat. The gentle breeze that caresses your cheeks when you're walking turns into a springy mass when you're cycling. Its pressure builds up when you change to a motor car. And it becomes almost solid to "your hand if you thrust it out of a plane flying at 200 to 250 kilometres an hour.

Do not try to stick your hand out of a supersonic plane, though. You will lose it. A flier who had to bailout of his plane unprotected was chopped up as if he had gone through a meat-grinder. The air scalped him and broke his legs, arms and ribs. The man had to stay in hospital

for many months. He was lucky, because other fliers were killed in similar situations.

Striking at a plane at high velocity, air heats the plane's skin. This is why heat-shields have to be provided around the cockpit on high-speed aircraft. The heating is far more severe in the case of missiles and rockets. The V-2 missiles the Germans bombed London with towards the end of the second world war were at a cherry-red heat at the moment of impact because of the resistance of air. It is for this reason that so much emphasis is placed on high heat resistance and high melting point in the metals to be used in aircraft and spacecraft in the future.

Beryllium has all this and high strength at elevated temperature into the bargain. When heated to 400° C duralumin loses four-fifths of its strength, and beryllium only a half.

In terms of the strength-to-density ratio beryllium leads stainless and high-strength steels. For beryllium the ratio is 26, and for the two classes of steel the respective figures are 10 and 20. The framework of a multistory building made from beryllium will be stronger than from the same weight of steel. A plane built of beryllium will have a range 40 per cent greater than an aluminium plane solely because of the reduction in weight. This is why beryllium has become a coveted metal to every aircraft and spacecraft designer.

Not that the interest in beryllium is limited to the aircraft industry. Well before aircraft designers came to think of it, beryllium was used for windows in X-ray tubes. This is because beryllium, hard and unmanageable, is highly transparent to X-rays.

Then metal-makers took interest in beryllium. They found that small additions of beryllium would markedly improve the properties of many metals and alloys. At one time car springs were made of plain carbon steel. After 800 to 850 thousand load cycles they would fail because of fatigue. Then a small amount of beryllium was added, and the steel came by a wide margin of strength. Now car springs can stand up to 14 million load cycles without so much as a sign of failure. This endurance has been imparted by beryllium.

Beryllium does not combine with magnesium. Yet an addition of as little as one part of beryllium in ten thousand makes magnesium alloys highly resistant to oxidation both in air and in water. Even when heated to 700° C, such an alloy will not ignite. And again this fire resistance has come with beryllium.

Beryllium is a very efficient deoxidizer for steel. Neither aluminium nor magnesium can do the job better. Beryllium is the most important, however, as a component of what are called beryllium copper alloys. Its content is only two to two and a half per cent, but its effect is truly miraculous. Highly critical parts, such as springs, spring contacts, gears and bearings operating at high speed, pressure and temperature, are made of beryllium copper alloys. This is because after a suitable heat treatment they become as strong as the best grades of steel. They are very resilient and conduct both electricity and heat well. On top of all, they resist abrasion. Because beryllium copper never gives out sparks when it strikes a stone, it is made into picks, hammers and saws used in mines, gunpowder factories, and other jobs where fire hazard is present.

Beryllium copper ages almost in the same way as some aluminium alloys do. For ageing, beryllium copper is heated to 700-800° C, quenched in water and then held for several hours at 250 to 350° C. After quenching the metal becomes very ductile and as pliable as clay. The subsequent tempering turns it into a very durable and strong metal with a yield point of up to 128 kilograms per square millimetre, as against the 16 kilograms after quenching.

Still another important application for beryllium in metallurgy is the surface hardening of steel. The process is called beryllization and consists in that the steel to be hardened is placed in a container holding beryllium or its compound in powder form and heated to about 1000° C. The surface layer picks up beryllium and becomes very hard and resistant to oxidation

at up to 800° C.

In addition to these important uses in metal-making, beryllium has attracted the attention of nuclear physicists. As it has turned out, beryllium can retard the neutrons passing through it without catching them, a very important property for a material used in nuclear reactors.

To cut the long story short, there is a great number of uses for beryllium in science and technology. Unfortunately beryllium is in fact a semiprecious metal. To quote the *New Scientist,* it costs about £20-£30 a pound for the raw (unworked) metal and from £50 to £500 a pound as components, compared with £200 a pound for gold. Beryl, its sole source, carries as little as about five per cent beryllium. Its abundance in the Earth's crust is as low as a few ten-thousandths of one per cent.

Is this to mean that beryllium, so attractive in its properties, is to remain the cluster of grapes from the fable about the Fox and the Grapes? Hardly so. After all, beryllium is twice as abundant in the Earth's crust as lead or cobalt.

The figures on its output seem to strike an optimistic note. In 1932 the Siemens Werke in Germany produced two tons of this semi-precious metal. In 1937 the United States manufactured about seven tons of beryllium alloys. In 1959 the beryllium output (leaving out the socialist countries) was 180 tons.

Not that beryllium is free from shortcomings. It is very hard and will scratch glass at room temperature (just try and work it by cutting). While rather ductile when made very pure, it is very brittle and will go to pieces at a hammer's blow. This is why beryllium cannot be rolled, forged or drawn. Only if it is raised to a dark-red heat can it be forged, and then to a limited extent. In the manufacture of beryllium sheet, billets have to be rolled inside hermetically sealed steel containers at 300° C. So beryllium is a very difficult creature. But metal-makers know how to "break in" such "wild horses". One way is by using the techniques of powder metallurgy.”

(M. Vasilyev, Metals and Men, Moscow 1967, p. 167-170)

Vergiftung / Poisoning

Bertrandit

Vergiftung / Poisoning

Übersicht / Overview

I. Toxikologie des Beryllium / Toxicity of Beryllium
1946 - The toxicology of a new metal - Lawrence T. Fairhall
1950 - Beryllium and its compounds are toxic even in fantastically small amounts - Harriet L. Hardy
1955 - Beryllium and Cancer - Wilhelm C. Hueper
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II. Vergiftungsberichte / Cases of Poisoning
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I. Toxikologie des Beryllium

1946 - The toxicology of a new metal - Lawrence T. Fairhall

A few articles have been added to the recent literature concerning the industrial hazards associated with the production of beryllium, but during the past three years no work has been reported on the physiological action of beryllium itself. Shilen, Galloway and Mellor (1944) reported the health hazards incident to the extraction of beryllium. Kress and Crispell have reported cases of chemical pneumonitis in men working with fluorescent powder containing beryllium. Van Ordstrand, Hughes, De Nardi and Carmody have recently reported the dermatological effects which they have observed in the beryllium industry during the past four years, and which were reported under the heading of 'beryllium poisoning'. The fatal cases described in this report resulted from chemical pneumonitis. A similar chemical pneumonitis developed in an additional 33 workers at various occupations in these plants. While the hazards due to dust and fume that existed in the plants was undeniable, the term 'beryllium poisoning' is a misnomer, since no toxic action can be directly attributed to the beryllium ion itself, and the beryllium in these cases merely acted as an adjuvant.

In connection with the manufacture of fluorescent lamps, a number of cases designated as sarcoidosis were reported in 1943 in the Massachusetts area. Since beryllium oxide is one of the constituents of the fluorescent powder, and since very little was known concerning the toxicity of beryllium, this substance was suspected as the aetiological factor in the production of sarcoidosis.

The inhalations of beryllium sulphate dust was shown to be injurious to guinea-pigs, 67% mortality in one exposure, in experimental work reported by Hyslop et al. in 1943; but, when the more neutral potassium beryllium sulphate was substituted, the animals were able to tolerate much larger doses; there was no mortality in larger doses daily for seven days. Later experimental work, using rabbits, has confirmed this earlier finding.

Beryllium sulphate is so extensively hydrolysed that hydrogen is evolved when zinc is placed in a solution of the salt. It is understandable that the inhalation of beryllium sulphate dust would prove strongly irritating, owing to the local production of sulphuric acid in contact with the delicate tissue of the lung substance. The production of a pneumonitis in the victim exposed to such irritating material is readily understood. Potassium vomiting sulphate, on the other hand, is soluble in cold water, is nearly neutral in reaction, and yields all the reactions characteristic of the beryllium ion. It is not bound as a complex, which might be expected to have different properties from other beryllium salt solutions. If beryllium were a protoplasmic

poison, both beryllium sulphate and potassium beryllium sulphate should show similar effects. With reference to the ulceration produced by beryllium sulphate entering cracks in the skin - a condition which occasionally occurs with employees handling this material - it would be of interest to try the effects of beryllium salts or more inert acids, such as beryllium citrate or beryllium malate.

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(Lawrence T. Fairhall, The toxicology of the newer metals, British Journal of Industrial Medicine vol. 3 (1946), October, p. 207)

1950 - Beryllium and its compounds are toxic even in fantastically small amounts - Harriet L. Hardy

Beryllium Uses

"Beryllium is the fourth lightest element, with an atomic weight of 9.02. Since the 1920´s in this country beryllium has been used in alloys with nickel, aluminium, copper for many purposes because of its great lightness and resistance to stress and heat. In the late 1930´s scientists developed a powder containing zinc, manganese, and silica, as well as beryllium, which fluoresced when exposed to ultraviolet light. From this grew the huge fluorescent-lamp industry. During the war beryllium was used in many ways, especially as an alloy with steel in engines. Physicists, knowing that beryllium emits neutrons when bombarded with alpha particles, used this element as a tool in atomic-energy research. Perhaps the greatest potential use is as a moderating material in nuclear reactors.

History of Beryllium Toxicity

From 1933 to 1942 (see Refs. 3, 18, 20, 34, 51, 55), European scientists described illness in workers handling beryllium compounds. The reported toxic effects resulted chiefly in a disease of the lungs, sometimes resulting in death. Considerable animal study was done in these years. Bone [22], liver [7], and pulmonary lesions [17,34] were produced. Reports of illness in the United States among workers in the beryllium industry first began to appear in 1943 and have appeared annually since (see Refs. 2, 24, 28, 29, 47, 48). The American reports describe pulmonary disease and indicate involvement of the eye, nose, throat, skin, liver, kidney, spleen, and lymph nodes as well. In view of these reports the Manhattan District and later the Atomic Energy Commission [15,45] developed means of making industrial operations as safe for the workers as possible. This job was made different in part because of the necessary secrecy and in part of the failure of American industry and its technical advisers to accept the toxic possibilities of beryllium and its compounds until recently (1947).

Clinical Character of Beryllium Poisoning

In order to appreciate the scope of the problem, some details are given here as to the character of beryllium poisoning. The greatest number of cases of this disease have come from the plants extracting beryllium from the ore, beryl, which reaches the United States from South America and India, and which is also found in a few western and northeastern states. Exposure to beryl ore has never been known to produce illness. Beryllium extraction involves the use of many chemicals and the development of vapours and dust containing beryllium compounds. A serious skin irritation may be the only effect; or, if certain beryllium compounds,

even in small amounts, get under the skin [48], occasionally a wound occurs which is difficult to heal. The skin effects arising from a wound contaminated with beryllium received much newspaper publicity in 1949 because of nonhealing wounds received by children playing with discarded fluorescent lamps containing zinc manganese beryllium silicate [21,49]. Methods of fluorescent-lamp disposal under water or on large remote dumps by men well protected by heavy protective clothing and proper respirators have been generally adopted to correct this situation.

An eye effect, frequently observed in beryllium extraction plants, is conjunctivitis [48]. Irritation of the upper respiratory tract occurs and may be accompanied by bronchitis or a still more serious form of acute beryllium effect, a chemical pneumonia, if the worker continues to work in the beryllium atmosphere or the exposure is intense [48]. Before the seriousness of acute beryllium poisoning was recognized several deaths were reported from beryllium pneumonitis. Immediate removal of the worker from the contaminated atmosphere, hospital care, and oxygen therapy have now made fatalities rare [48].

Chronic Beryllium Disease

A second group of clinical cases has been reported in which, following exposure to certain beryllium compounds in fantastically small amounts after a period of time varying from months to as long as seven years a chronic form of beryllium poisoning results [23,24]. About 120 cases have been reported up to date. Patients ill for an average of three years; complete recovery is unknown, but a few patients have returned to work. The mortality rate is at present about 25 per cent [31]. Such cases have occurred in fluorescent-lamp plant workers, in brass workers using copper-beryllium alloys where master alloys of 4 per cent beryllium are used, in workers manufacturing beryllium alloys, in government laboratory workers, neon sign manufacturing workers [52], and in a small number of individuals who have lived in the vicinity of beryllium plants, so called 'neighborhood cases' [15]. In 1946 the first cases of chronic beryllium poisoning was reported on an atomic-energy project. Since that time several other patients have been reported from Commission-sponsored activities, including research and metallurgical workers.

The most striking feature of chronic beryllium poisoning is the delay between the time of the exposure and the onset of symptoms of the disease. In some cases this may extend up to eight years or longer. Confusion in diagnosis between miliary tuberculosis and chronic beryllium disease may be made by the physician unless he is aware of the possible effect of beryllium. Differential diagnosis between sarcoidosis and chronic beryllium poisoning is made by the greater pulmonary disability, frequency of gastro-intestinal symptoms, lack of X-ray changes in the bones, and poor prognosis of the latter. The patient has great weight loss, cough, shortness of breath, and a chest X-ray showing changes involving both lung fields throughout. Chronic beryllium poisoning may also involve lymph nodes, kidney, liver, spleen, lung, skin, and probably bone. [24,31,32]

Before the cause and character of beryllium poisoning were understood, some workers in beryllium extraction plants and in plants manufacturing alloys suffered more than one attack of acute beryllium pneumonitis. Removal from the beryllium atmosphere and proper bed care resulted in cure, but a second attack might occur after return to work with beryllium compounds. A few such cases, removed from exposure to beryllium compounds, later developed the symptoms of chronic beryllium poisoning without further contact with beryllium compounds. There are instances of workers exposed to beryllium compounds of known toxic effect who show no ill health but whose chest X-rays indicate marked changes. There are also workers similarly exposed to the same conditions as those suffering from chronic beryllium disease who have symptoms of slight dyspnoea on exertion, mild loss of strength and weight, and characteristic chest X-ray changes. The term 'beryllium effect' has been adopted for this group. These individuals may develop disabling symptoms, at which time experience shows they follow the pattern of chronic beryllium poisoning.

No specific treatment exists for chronic beryllium poisoning. Oxygen, good nursing care, high protein diet, and indicated antibiotics are helpful. The possibility that cortisone and ACTH may be an adjunct in the treatment of chronic beryllium disease is currently being explored (1950).

Control of Toxic Effects of Beryllium

For control of the disease, engineering methods have been developed to reduce the amount of beryllium in the workers´ breathing zones to the vanishing point. Laboratory workers using beryllium even on a small scale now handle the material in special equipment that is enclosed to prevent contamination of air with even small amounts. Experience shows that extremely small quantities of beryllium in the air are sufficient to cause disease in some individuals. The figure of less than 2 µg if beryllium per cubic meter of air an 8-hour working day is the present level suggested by the committee to advise the Commission on beryllium toxicity problems [57]. In order to measure these quantities, very delicate chemical and spectrographic methods have been developed at the University of Rochester, the Kettering Laboratory of the University of Cincinnati [8], and the Trudeau Foundation, Saranac Lake, New York. The Rochester Atomic Energy Project has made several on- and off-site air surveys in the vicinity of beryllium-refining plants in order to relate actual condition with the onset of the chronic disease [14,15]. This study was done because of the development of a few cases of chronic beryllium poisoning in individuals who lived near plants or in whose home beryllium workers lived. Impressive amounts of beryllium compounds of known toxic potential have been found by measurement after shaking out workers´ clothes. A number of detailed clinical studies of workers have been made. Much of this work is done in collaboration with private doctors and individual physicians in communities and in industries where cases of chronic beryllium poisoning exist. Some of these data have been made available to the medical profession [6]. Radioactive beryllium (beryllium [7]) made by the cyclotron, has been used to study the effects of small amounts of beryllium in animal experimentation. These studies indicate that once beryllium is in the body, undoubtedly it has general as well as local effects. Scientists in several laboratories are also using animals to try to learn which beryllium compounds are poisonous and how much of each compound is necessary to produce toxic effects. The late Dr. Leroy Gardner, working at the Saranac Laboratory, produced pulmonary lesions in small animals very like those seen in autopsy material from cases of chronic beryllium poisoning. The group at the University of Rochester has satisfactorily reproduced in animals the lesions of acute beryllium poisoning [43]. This work, much of which is reported in the medical literature, continues. Conclusions are difficult to draw in view of the great differences in species. Moreover, the mode of action of the toxic compounds in the body is obscure and requires further study. The problem of the exact beryllium compounds - their physical and chemical state - capable of producing beryllium poisoning still has to be solved. Preliminary steps have been taken by means of electron microscope studies at several Commission laboratories.

A committee of Commission and contractor personnel formulates recommendations for medical and engineering control of the toxic effects of beryllium. This group meets to review new clinical, experimental, and engineering data and reports its findings to the Commission. Such assistance is also made available to industrial physicians and others responsible for the health of workers in industry [57].

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(Harriet L. Hardy, M.D., Beryllium Poisoning, United States Atomic Energy Commission AECU-569(Rev.), Massachusetts Institute of Technology 1950)

1955 - Beryllium and Cancer - Wilhelm C. Hueper

"Beryllium is a metal which has found significant industrial use only since about 1920. It was not until about 1940 that beryllium and its compounds were extensively employed for numerous purposes and products (beryllium-copper, beryllium-aluminium, and beryllium-nickel alloys, glass, phosphors in fluorescent lamps and neon tubes, atomic energy products, ceramics, refractories, X-ray tube windows, vitreous enamel, radio tubes, textile fibers, gas mantles). It is evidently for this reason that untoward effects in persons exposed to the inhalation of dusts and fumes of beryllium and its various compounds have been recognized only during the last decade. These manifestations were of both acute and chronic nature as far as the respiratory organs were concerned (acute beryllium pneumonitis, chronic pneumoconiotic granulomatosis, berylliosis). Some investigators used the term "sarcoid" in describing the histologically peculiar, pulmonary manifestations. It is remarkable moreover, that chronic berylliosis has appeared not only among exposed workers, but also among persons living in the neighborhood of fluorescent lamp factories and inhaling their beryllium-containing effluents (Eisenbud, Berghout and Steadman [1]; Eisenbud, Wanta, Dustan, Steadman, Harris, and Wolf [2]).

Similar observations on occupational berylliosis were reported from Germany, Italy, England, Russia, and Canada. Not infrequently, similar granulomatous lesions have been observed in other parts of the body after the usually traumatic introduction of beryllium dust, especially of beryllium phosphors from broken fluorescent tubes. The skin of the fingers and hands was the most frequent extrapulmonary location of these reactions. Beryllium granulomas have also been found in the nose and in the anterior ocular structure.

It is noteworthy that beryllium apparently once inhaled is retained over a long period of time in the human body, since beryllium has been detected in the urine up to 10 years after cessation of exposure (Klemperer, Martin, and Van Riper [3]) and has been demonstrated in the lungs of rats 1 year after the inhalation of beryllium oxide (Dutra, Bargent, Cholak, Hubbard, and Roth [4]) as well as in their bones (Stokinger, Steadman and Root [5]; Barnes [6]), where it may replace calcium. The skeleton retains the bulk of the beryllium in the body (50-80 percent) if the inhaled aerosols are soluble compounds, such as beryllium sulfate and beryllium fluoride; the lungs retain the bulk of beryllium if the compounds are insoluble, such as beryllium oxide. Experiments of Aldridge, Barnes, and Denz [7], moreover, have shown that beryllium ions react rapidly with certain tissue proteins and form complexes with plasma proteins when introduced into the blood. These complexes protect the beryllium from being precipitated by phosphate ions.

The metabolic peculiarities of beryllium compounds obtain special importance in view of the fact that Gardner [8] in 1946 reported the production of osteogenic sarcomas in rabbits injected intravenously with insoluble beryllium-containing powders (beryllium phosphate, zinc beryllium silicate). Other investigators subsequently confirmed these results with the same and other beryllium compounds (beryllium oxide, beryllium silicate, metallic beryllium) introduced into rabbits by the intravenous or respiratory routes (Sissons [9]; Barnes, Denz and Sissons [10]; Hoagland, Grier and Hood [11]; Nash [12]; Dutra, Largent and Roth [13] [14]; Barnes [6]). The preparatory period for the sarcomas was 11-24 months.

Commenting on the successful production of osteogenic sarcoma in rabbits after inhalation of beryllium oxide, Dutra, Largent, and Roth noted the fact also that the bones of persons dying with berylliosis contained not inconsiderable amounts of beryllium. They came to the following conclusions: 'During the last 20 years, considerable numbers of persons have been exposed to dusts of poorly soluble compounds of beryllium in various industries throughout the United States. Despite the fact that cases of cancer of this type have not been reported, it is possible that the inhalation of poorly soluble compounds of beryllium may eventuate in osteogenic sarcoma in man. Presumably, the incubation period of such tumors would be

considerably longer in man than in rabbits, and observations may be required over a period of years before it will be known whether persons who have been exposed to beryllium are prone to have such tumors.'

Barnard [15] also suggested that osteogenic sarcoma from compounds of beryllium 'might possibly be another industrial hazard.' So far, only rabbits have responded with the development of osteogenic sarcoma following the administration of beryllium compounds. The direct introduction of powdered beryllium metal into the femoral cavity of rats, into the pleural cavity, and into the paranasal sinuses failed to elicit a single neoplastic response at the site of injection in any 1 of the 85 animals used within an observation period of 2 years (Hueper).

When in 1948 Hueper proposed that the sarcoid pulmonary manifestations of berylliosis might be followed by outright malignant lesions in the lungs, this suggestion was received with a great deal of scepticism. The recently reported successful production of bronchogenic carcinomas in the lung of rats which, over periods of more than 1 year, inhaled dust of soluble and insoluble beryllium compounds (Vorwald [16]), however, makes the appearance of such delayed malignant sequelae in man a distinct possibility, especially as several cases with coexisting berylliosis and cancer of the lung have recently been observed (Kahlau [17]). In view of the established occupational as well as general environmental occurrence of human berylliosis, it may be pointed out that the discovery and identification of this pneumoconiosis was definitely facilitated by the distinctive and definitive histological features of the disease. If these manifestations should be followed by the development of cancers of the bones and lungs, the establishment of causal relations between a previous exposure to beryllium and the subsequently appearing cancerous reaction would appear to be rather easy.

The studies on the toxicity and carcinogenicity of beryllium compounds indicate that the toxic and cancerous manifestations are to be considered as responses to the action of beryllium itself and not as the result of the associated anions of its acidic salts (Stokinger, Sprague, and Hall [18]). In considering possible future carcinomatous developments in persons with previous exposure to beryllium, some consideration also may be given to the toxic effect exerted by beryllium on the liver leading to the development of cirrhosis and to an impairment of the metabolic and detoxicating function of this organ (Aldridge, Barnes, and Denz [7]; Hoagland, Grier, and Hood [11])."

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(Wilhelm C. Hueper, A Quest into the Environmental Causes of Cancer of the Lung, Public Health Service Publication No. 452, Washington 1955, p. 30-31)

1958 - Toxische Wirkungen des Berylliums - William Gutman

"Bronchitis, Bronchiolitis. Atemnot bei Anstrengung. Husten, feuchtes Rasseln. Leichte Temperaturerhöhung. Cyanotisches Aussehen. Schmerzen unterhalb des Brustbeines, Appetitlosigkeit, Ermüdung. Gewichtsverlust. - Ähnliches Bild wie bei Viruspneumonie. Pathologisch tuberkelähnliche Granulome in der Lunge. In der Haut Geschwüre, Granulome, Fisteln. - Röntgendurchleuchtung der Lunge zeigt ein Bild ähnlich miliarer Tuberkulose ('Schneesturmbild'), oder diffuse Trübung, auch Bilder ähnlich denen bei Influenzapneumonie oder Pneumonie nach Keuchhusten und Masern. - Im toxikologischen Tierexperiment: disseminierte pneumonische Herde, Blutungen in der Lunge. Im Experiment zeigte sich eine Vermehrung der tuberkulösen Infektion bei inokulierten Meerschweinchen nach subcutaner Injektion kleiner Dosen von Berylliumsalzen. Keine Tuberkulose wurde gefunden bei Sektion von Arbeitern mit Beryllium, noch zeigten Beryllium-Arbeiter bei Untersuchung Zeichen von Infektion, wenn sonstige Lungenerkrankung vorlag. (zusammengefasst nach Hamilton, Industrial Toxikology)

Atemnot, größer als nach den physikalischen Zeichen zu erwarten gewesen wäre. Lungenkapazität in Vergiftungsfällen sehr bald reduziert, sehr rasche, oberflächliche Atmung. Ursache ist ungenügende Sauerstoffsättigung infolge Verhinderung der Sauerstoffpassage durch die Alveolarmebran (Wright, 6th Saravan Symposion). Charakteristische Verzögerung des Beginnes der ersten Symptome nach Aufgabe der Arbeit mit Beryllium (von 6-9 Jahren). Chronischer Verlauf der industriellen Vergiftung, 1-6 Jahre, mit Remissionen und Exacerbationen. Bemerkenswert seltenes Vorkommen von positiven Tuberculin-Test. (zusammengefasst nach Hardy, Oxfordmedicine, Vol. 4)

Nach allem Vorangegangenen scheint es, daß Atemnot außerhalb jeden Verhältnisses zu den physikalischen Zeichen ein führendes Charakteristikum bei Erkrankungen des Atmungstraktes ist, in denen Beryllium besonders angezeigt scheint.

Die charakteristische Verzögerung im Beginn der Symptome (vergleichbar einer Inkubationszeit), die sehr langsame Entwicklung und der chronische Verlauf, die charakteristischen Röntgenbilder, die pathologisch-anatomischen Befunde, weiters die Verstärkung der künstlichen Tuberkuloseinfektion im massiven Tierexperiment, auf der anderen Seite die auffallende relative Seltenheit positiver Tuberculum-Reaktionen bei Arbeitern mit Beryllium, sowie die Seltenheit von tuberkulösen Obduktionsbefunden bei Beryllium-Arbeitern - all dies legt Beryllium nahe als ein Mittel für Tuberkulosefälle, wenn die führenden Symptome vorhanden sind. Die toxischen Symptom weisen auch auf atypische und Viruspneumonien hin, neben unspezifischen aber charakteristischen Bronchial- und Lungenerkrankungen.

Prüfung, Toxikologie und der zitierte Fall weisen auf Beryllium als ein wichtiges, neues Arzneimittel hin."
(W. Gutman, Beryllium, Allgemeine Homöopathische Zeitung Bd. 203 (1958), S. 436-437)

2003 - Harriet Louise Hardy - Fighting Man-Made Disease

"A physician and industrial toxicologist, Harriet Hardy was a blazing force in industrial medicine. In a landmark study in 1946, she identified beryllium as the cause of chronic respiratory disease. In 1952, she established the National Beryllium Registry, one of the first registries to collect long term data on a chronic disorder.
A tough talkmaster, she engaged industry and government in fierce argument, yet her essential impulse was practising medicine. During her 88 years, Hardy was a staunch advocate for workers in clinical care, writings, and court testimony. Just knowing Hardy was an expert witness, at times, persuaded plaintiffs to settle. She insisted on the difficult path of joint union-management decisions.

"…unless there is definite commitment of executive authority, government agency, industry, or academic institution, occupational medicine and hazard control cannot thrive."

Hardy´s diverse studies included: anthrax, arsenic, asbestos, benzene, beryllium, cadmium, carbon tetrachloride, coal worker´s lung disease, cyanide, lead, mercury poisoning, mesothelioma, pesticides and radiation. She was among the first to recognise the connection between asbestos and cancer. Despite ill health, she lectured widely on the importance of fighting man-made diseases."
(D. F. Salerno and I. L Feitshans, Harriet L. Hardy, fighting man-made disease, J Epidemiol Community Health 2003 57:924)

II. Vergiftungsberichte

1954 - An unusual case of joint pains and fever - Howard B. Sprague and Harriet L. Hardy

"Dr. Sprague: Dr. Hardy, we are going to discuss this morning the case of our patient, Mrs. George C., whom I first saw in September, 1953, and later referred to you for observation and treatment.

She was a 32 year old married woman who had never been ill prior to February of 1953. Her first baby had been born in September of 1952. In February, for the first time, she began to have redness and swelling of the ankles with considerable pain. This arthralgia migrated to the elbows and to the hands, and she also had pain in the back and shoulders. Her temperature rose to 100.4 F., and she was laid up for about seven weeks. During this time she had some small, red, desquamating nodules over her body, but particularly the arms and legs. She suffered also from shortness of breath, cough, and a tachycardia at a rate of 120 beats per minute. X-ray films of the spine were said to show arthritis. She was treated with bed-rest, oil of wintergreen locally, and sodium salicylate by mouth.

Her cough and dyspnoea continued after she got out of bed, and she found herself unable to care for her house and the new baby. There was also fatigue and some sore throat, and she lost 15 to 20 pounds. For a time she was given penicillin by mouth and by inhalation.

When I examined her, she was a thin, ill-appearing, young woman, obviously dyspneic on slight exertion and even at rest, with slight cyanosis. She coughed vigorously on lying down, and it was necessary to examine her in the sitting position. There was no increase in the venous or arterial pulsations in the neck. The heart was not enlarged to percussion. The sounds were of good quality. The pulmonic second sound was louder than the aortic. There were no murmurs. The rhythm was regular at a rate of 100, and her blood pressure was 105/65. I could not make out any dullness of her lungs to percussion, and there were no rales. The liver was not enlarged, and there was no edema of the ankles. There were numerous red, slightly scaly spots over the arms and legs, three or four millimetres in diameter.

Electrocardiogram showed prominent P waves in leads II and III and inverted T wave in lead III and lead aV_F. There was an RR^1 complex in the precordial lead V_1 with low T waves across the precordium, and inverted in V_1 and V_2.

Fluoroscopic examination showed a striking abnormality in the lung fields with an extremely dense mottling throughout both sides. There was no abnormality in the size or the shape of the heart.

With this clue, I investigated her history of industrial exposure more thoroughly. She first said that she had worked as a stenographer, but it appeared on further questioning that in 1943 and 1944, she had worked for a company making fluorescent lamp bulbs and had been exposed to beryllium dust. Radiologic study of the chest showed the following: 'Fluoroscopic examination and films of the chest show diffuse linear nodular densities scattered throughout both lungs from apex to base. The heart is normal in size and shape, and the oesophagus is normal in position and appearance. No gross enlargement of the mediastinal nodes is present. Conclusion: The diffuse reticulation in both lungs suggests berylliosis. Without the history, Boeck´s sarcoid would also be a good possibility, despite the lack of gross hilar adenopathy.' Dr. Hardy, would you be good enough to let us know what investigations you undertook when this patient was hospitalized?

Dr. Hardy: Because of this particular patient´s apprehension about her prognosis, she did not have preliminary pulmonary function studies, as we intend all our cases should. We studied the liver function with the conventional tests and did a variety of biochemical determinations which have been of interest to us, such as blood calcium and phosphorus, and alkaline phosphatase, We are were careful to do tuberculin testing, sedimentation time, and sputum studies, because it is likely we shall one day mistake miliary tuberculosis for beryllium disease.

Dr. Sprague: I was particularly interested in bringing out the point here that this woman was considered to have rheumatic fever, and her dyspnoea was attributed to rheumatic heart disease. Would you please tell us, Dr. Hardy, about the relationship between the pregnancy and the activation of her beryllium reaction, and also about your experience with joint and skin manifestations of this disease?

Dr. Hardy: In our study of cases of chronic beryllium poisoning, we have been impressed and stimulated by the observation that women who have been exposed to beryllium compounds may show no evidence of disease by sign or symptom until a successful pregnancy has been completed. I want to emphasize that the pregnancy, itself, appears, if anything, to be a helpful process, but when the child is 4 to 6 months old the patient may notice inability to gain weight, shortness of breath, and cough, as did Mrs. C.

In answer to your question about experience with joint and skin manifestations of this disease, our observations include another case with a similar history to Mrs. C. The original diagnosis was rheumatoid arthritis and later proved to be beryllium disease with the joint picture a part of the chemical intoxication. In addition, we have a definite group of patients with mild to moderately severe beryllium poisoning who have intermittent joint pain without x-ray evidence of joint changes or any increase in sedimentation time. These patients´ symptoms are relieved with aspirin. Skin manifestations of beryllium effect are divided roughly into three categories: first, the reaction of irritation following direct contact with acid salts of beryllium; second, a subcutaneous granuloma associated in some cases with introduction of beryllium compounds accidentally or, in animals, experimentally; and finally, as in the case of this patient, a small but definite group of cases in which spontaneous skin lesions of great variety simulating Boeck´s sarcoid appear as one manifestation of chronic beryllium poisoning.

Dr. Sprague: During the time that this patient was in the hospital, the question was again raised as to the significance of the evidence of some degree of right-sided heart strain. How

much cor pulmonale have you seen in your patients with beryllium poisoning?

Dr. Hardy: Cor pulmonale is a complication in most cases of beryllium disease of any severity. Failure of the right heart is the usual cause of death.

Dr. Sprague: You have also explained, Dr. Hardy, that your concept of berylliosis is that it is a completely generalized process. In what other organs and tissues have you found beryllium granuloma or abnormal concentration of beryllium?

Dr. Hardy: Beryllium was found at autopsy and biopsy in the lung and also the liver, spleen, kidney; cervical, hilar, and abdominal lymph nodes; bone and skin: The so-called beryllium granuloma, which incidentally we believe is only one part of the pathologic reaction to certain beryllium compounds, has been described in lung, lymph nodes, liver, spleen, kidney and skin.

Dr. Sprague: Another point which I should like you to discuss is the relationship between the disease and Boeck´s sarcoid. It is my impression that there has been an opinion that sarcoid is commoner in places in the world where the natural concentration of beryllium in the environment is relatively high? Is there anything to support this contention?

Dr. Hardy: Dr. Sprague, you are referring to a very interesting but as yet undeveloped observation that the incidence of Boeck´s sarcoid can be correlated with the patient´s residence in an area where certain soil is found. The original discovery brought out the fact that this particular soil is described as containing small amounts of beryl, the ore from which beryllium comes. However, studies of water, air, and vegetation in these so-called 'sarcoid belts' has not borne out the idea that beryllium is present in a detectable concentration. It is too soon, however, to know whether, with increased knowledge of detection of trace materials in soil, this may or may not be an important lead in the understanding of granulomatous diseases.

Dr. Sprague: I have, of course, been immensely pleased at the favorable reaction to cortisone in this case. What is your general experience about the effectiveness of this new therapy? I see that you have cautioned her local physician to have her urine tested once a week for sugar because of the possibility that cortisone might produce glycosuria.

Dr. Hardy: We are very much impressed with the effectiveness of cortisone in the management of chronic beryllium poisoning. We do not believe that we are curing the disease although it has been shown in one or two cases by biopsy that the drug has made some change in the granulomata. Klemperer has shown that there is no change in the excretion of beryllium after cortisone therapy. However, if we persist in this form of treatment, our patients are amazingly improved in their general health, an observation which has been checked objectively by pulmonary function studies, showing that there is actual improvement in the oxygen saturation of the blood. We have seen several transient glycosurias in cortisone-treated cases of beryllium poisoning. We have, however, never had to stop treatment for this reason. We have learned that the cortisone must be continued in most cases and medical supervision is required, especially in the presence of infection and when there is any evidence of irritation in the upper intestinal tract.

Dr. Sprague: Thank you very much, Dr. Hardy, for discussing these very interesting points. Here, then, is a patient in whose history appear fever, joint pains, and swelling, skin nodules, dyspnoea, cough, orthopnea, tachycardia and fatigue. The electrocardiogram suggests some right ventricular strain, and the whole process was originally mistaken for rheumatic carditis. The differential diagnosis actually, however, was not difficult because of the negative findings in the heart on auscultation and the characteristic pulmonary pathology on x-ray examination. The delay of almost 10 years in the appearance of beryllium reaction to the level of clinical symptoms is a discouraging fact in the natural history of the disease. It certainly means that all persons thus exposed must continue under medical observation for many years,

and the industrial compensation liability is one of indefinite duration. However, there is real hope of successful therapy by cortisone."

(Clinical conferences: Howard B. Sprague and Harriet L. Hardy, An unusual case of joint pains and fever. Beryl-liosis and pulmonary hypertension mistaken for rheumatic fever. Circulation 1954:10:129-132)

Prüfungen / Provings

Aquamarin

Prüfungen / Provings

1951 - Provings of Beryllium metallicum - W. Lees Templeton

The number of provers - 7
The number of controls - 4
Potencies used - 3x, 6x, 7x. 12x, 30th.
The proving substance was specially prepared by Nelson´s.

Head
Frontal aching, on waking.

Throbbing, worse heat, worse coughing, occiput to mastoid, → left to right, worse jarring, movement, cough, holds it: worse rising, worse light, worse movement.

Throbbing, splitting, better lying that side, better fresh air, hot drink, worse mental emotion.

Occipital, worse jarring, worse movement, throbs.

Light headedness, feeling of unreality, as if would faint, as if things would disappear, with weakness and worn-out feeling.

Eyes
Eyes feel dusty.

Nose
Fullness, worse open air, worse evening.

Fluent discharge, worse warm room, better open air.

Thin discharge excoriating nostrils.

Stuffy, better cool air.

Fullness, better open air.

Blocked on rising, worse warm room.

Ears
Pain, worse swallowing.

Mouth
Ulcers tip of tongue and inside lower lip.

Lips dry, swollen, burning red, throbbing, worse heat.

Ulcer inside lip.

Dryness, lips, mouth and throat, lips dry and cracked, dry, burning, worse waking, rough, dry, loss of taste.

Ulcer inside lip: upper lips sore to touch as if chapped.

Throat
Sore, worse saliva, worse hot fluids, worse evening.

Burning, sore, worse saliva, solids, hot drinks. Better cold drinks, bright red injection pharynx and palate, worse coughing.

Worse swallowing anything: worse coughing, like knives, looks glazed (palate and pharynx). *Must* swallow.

Dry, as if coated, keeps on clearing it. Sore waking, better eating or drinking.

Sore, better eating.

Stomach
Loss of appetite, worse sight or smell of food, fullness before meals, drowsy after meals.

Nausea (riding in bus), better lying down, with loss of appetite, tight epigastrium, worse inspiration. Nausea riding in bus, better eating.

No desire food. No appetite, averse sweets.

Sick feeling, averse food. Full up yet still hungry. Full up with no appetite.

Pain worse standing, better doubling up.

Diarrhoea after rising.

Respiratory
Cough from sternum. *Dry*, worse open air, worse cold air, better warm room.

Pain right base with constriction of chest, worse breathing; persistent cough, *painful and useless*; *sweet* tasty expectoration, cannot get deep enough cough, worse a. m. rising.

Breathless with palpitation on running upstairs, had to sit down to recover.

Pharynx looks dry; *sweetish* sputum, creamy phlegm retched, dry evening, loose a. m., p. m.

Upper sternum like *knives* on coughing, worse smoke, worse bending head backwards.

Expiratory wheezing.

Heart
Palpitation with faintness and weakness legs.

Back and Extremities
Stitching pain lumbar and mid dorsal, worse sitting and bending head forward, better walking.

Darting pains, worse first movement, worse lying down, worse night.

Soreness right elbow as if bruised, as if strained, worse grasping, tender muscles forearm with lack of power.

Cold sensation right buttock and right lumbar reg.

Stabs of pain first metacarpal right, synchronous with heart beat.

Jabs of pain left forearm, powerless feeling, worse touch and pressure, better rest.

Pains thighs, worse pressure, walking, crossing legs.

Aching, right arm tired.

Cramp toes, muscles, front of legs when walking.

Temperature
Shivering, sweating, worse slightest movement, worse night in bed, worse warmth.

Burning like a blush, base of neck and between shoulders, worse warmth. No strength legs.

Skin
Small red itching papules left foot, worse scratching, worse warmth, worse bed.

Rough red patch right wrist, no itching.

Symptomatology of Beryllium from Provings

Head
Throbbing splitting sensations.
Worse heat, coughing, jarring, movement, light (*Bry*.).
Better fresh air, lying that side.
Direction: → left to right. → Occiput to mastoid.

Nose
Coryza thin, acrid discharge: fullness, better open air, worse warm room.

Mouth
Dryness lips and mouth, cracked lips: burning, rough, sore, as if chapped.
Ulcers tip painful tongue and inside lower lips.

Throat
Sore, burning, like knives, worse saliva, hot fluids, coughing, evening; better cold drinks, eating.
Must swallow (*Merc*.): must keep on clearing the throat.
Red **glazed** appearance of palate and pharynx.

Stomach
Loss of appetite. Averse all food, averse sweets.
Fullness before meals: full up yet hungry (*Lyc*.); fullness with aversion food. Drowsy after meals: tightness epigastrium worse inspiration.
Nausea, worse sight and smell of food, riding in bus, better lying down and eating.

Respiratory
Cough from sternum, cannot get enough. Dry, painful, upper sternum (*Bry*.), like knives.
Non-productive: worse cold air, smoke, bending backwards, better warm room.
Sputum tastes sweet.
Dyspnoea (with palpitation) on exertion.
Constriction chest on inspiration.

Heart
Palpitation with faintness and weakness of legs.

Back and Extremities
Stitching pain mid dorsal and lumbar region.
Worse sitting, bending head forwards, first movement, lying down. *Better* walking.
Elbow, forearm, metacarpals and phalanges, as if bruised, as if strained, *with loss of power*.
Cold sensation lumbar and gluteal regions.
Weakness legs.

Fever
Shivering in bed worse slightest movement.

Skin
Itchy papules, worse scratching, worse warmth.

Symptoms of Beryllium Poisoning

Dermatitis (contact), skin ulcer, oedematous, weeping, itching, burning, small, indurated papule surrounded by area of erythema. Pathology subacute, necrotizing ulceration. *Skin symptoms a measure of susceptibility to bronchial or pulmonary irritation*.

Nasopharynx: soreness of nose and throat manifested by mild epistaxis, metallic taste, diffuse swelling of mucous membrane with vascular engorgement. Tracheobronchitis; cough, râles both lungs, non-productive except for occasional blood-stained mucus.

Mild dyspnoea. Râles fine, then coarse, *vital capacity reduced*, low grade fever.

Chemical pneumonitis.

Cough with occasional blood stained sputum, *substernal burning pain, dyspnoea on exertion, cyanosis, anorexia, rapid pulse*, no temperature, reduced vital capacity, râles fine to coarse all over both lungs.

Sign of infection absent. E.S.R. normal. Temperature not much elevated.

Bronchoscopy: hyperaemic mucous membrane, patchy exudate.

X-Rays, diffuse, haziness, irregular areas of infiltration with prominent peribronchial markings. Discrete, following upon absorption of soft infiltration, large and small conglomerate nodules. X-rays cleared before symptoms cleared!!!

Necropsies, large number of plasma cells, no polymorph infiltration, diffuse pulmonary oedema and haemorrhagic extravasation.

Oxygen and rest of most benefit.
(Van Ostrand, *Journ. Amer. Med. Assoc*., Vol. 129, No. 16, Nov. 15th, 1945)

Beryllium poisoning shows signs as follows: acute and chronic lung disease, and skin disease.

Cough, dyspnoea, undue fatigue and loss of weight.

Lung X-ray show three stages: granularity, reticulation, nodulation.

Early appearances suggested miliary TB, later suggesting sarcoidosis, but later pathology showed focal *granulomatous lesions* in lungs and *liver*.

Acute pneumonitis can be caused by sulphate, fluoride, oxide, and metallic dust. Exposure to fluoride for 20 minutes is known to have produced three cases of acute pneumonitis.

Subacute granulomata have been produced in persons who have cut themselves on fluorescent lamps.
(M. A., 1949, p. 230)

Summing up

This metal is said to resemble aluminium in its effects, though more poisonous, being absorbed by the alimentary canal and excreted by the kidneys. A disease entity has been described in workers, the effects being ascribed to inhalation of fumes and dust and affecting chiefly the skin and respiratory tract. This proving has been somewhat disappointing in that few or no symptoms apart from those described in the poisonings were obtained., though it is interesting that such should have been produced by mouth, by dilutions from 3x to 30^{th} c, suggesting that the effects are not entirely due to local irritation. The fact that in this proving no

mental nor vital effects were obtained only emphasizes the value of such symptoms when produced by *other* drugs.

Dunham suggested that drug action is of three kinds: 1. Chemical; 2. Mechanical; 3. Dynamic or vital; and though the symptoms of the latter are of the greatest value, still many of our provings have been examples of 1 and 2 only, and many are essentially prescribed on local symptoms and signs. Ulceration of mucous membrane, the common aphthae of the mouth, for example, are very difficult to cure, as many of you will know, and this might be just a remedy for such local conditions when so-called deeper drugs seem to have no effect.

The throat symptoms resemble *Lachesis* in its modalities, the digestive symptoms those of *Lycopodium*, and so Beryllium might be considered when these drugs seem to bear a superficial resemblance to the case. The **glazed** streptococcal appearance of the pharynx and palate was especially distinctive and should be a guide when prescribing.

Combining the poisonous symptoms with those of provings it is possible to envisage the use of this drug in chest conditions such as one finds in influenza especially in the cases, which in some epidemics are not uncommon, where **dyspnoea, more marked than one should expect from the physical signs**, is encountered. If viewed from this angle the drug which it resembles in the premonitory stages of influenza is *Rhus tox*., with its coryza, tracheobronchitis and muscular aching which is better for movement and indeed, even the digestive symptoms are quite similar with its lack of appetite, nausea and fullness after meals and drowsiness after eating. At any rate, in a case which resembles *Rhus* in general and where *Rhus* fails this remedy might be considered, and just as *Baptisia* is often suggested for a more severe attack of influenza than what *Gelsemium* covers, so, where the symptoms are those of *Rhus* but the symptoms seem (and *especially those of the lungs*) more severe, then *Beryllium* might be useful.

It might be valuable if members were to insert into their repertories the symptoms obtained in this proving, thus adding to their repertories a new drug for such symptoms as nausea riding in a vehicle and at the sight and smell of food. Though *Colchicum* is frequently suggested for the latter symptom I myself have never found it of the slightest value.

So though it would seem that *Beryllium* is not a distinctive drug in its own right, it might take its place with other 'local' and 'acute' remedies as an alternative to better known remedies.

Most of the head symptoms resemble *Bryonia* as do also the cough symptoms, so once more, as is so useful, there is a discrepancy between the symptoms in one region and those in another, for example, here the symptoms are like *Bry*. and those of the back and limbs resemble *Rhus tox*.

But one thing must be said only if other provings of Beryllium confirm those I have described to-day should they be accepted. This, I feel, is absolutely vital. There must be confirmation and no one proving accepted without such independent confirmation.

If for no other reason, the fact that symptoms produced by the inhalation of fumes and dust have been reproduced by the ingestion of potencies would justify the work involved."

(W. Lees Templeton, Report on Beryllium Provings, The British Homoeopathic Journal vol. 43 (1953), p. 78-84)

Heilungen und Klinische Hinweise / Cures and Clinical Hints

Smaragd

Heilungen und Klinische Hinweise / Cures and Clinical Hints

Übersicht / Overview

1958 - Akute Tracheobronchitis mit ungewöhnlich großer Atemnot bei einem 6jährigen Mädchen - W. B. Griggs

"6 Jahre altes Mädchen mit akuter Tracheobronchitis. 'Einer der ärgsten Fälle, die ich je gesehen habe.' Erstickender, hochklingender Husten mit mehr Atemnot, als nach den physikalischen Zeichen zu erwarten gewesen wäre. Nach überaus heftigen Hustenanfällen kommt nur wenig Auswurf, der dick, leimartig, zähe ist. *Kalium bichromicum* hatte keine Wirkung. Der Husten bekam einen kruppartigen Klang. *Hepar sulf*. hatte ebenfalls keine Wirkung. Hierauf *Beryllium* C6, stündlich eine Dosis für 8 Stunden. Hierauf Auswurf etwas leichter, aber Atemnot noch immer unverhältnismäßig groß in Anbetracht der physikalischen Zeichen. *Beryllium* 30. Potenz alle halben Stunden eine Gabe. Nach der 6. Dosis Auswurf viel dünner und Atemnot bedeutend vermindert. Heilung in 3 Tagen. 'Ich habe noch nie in meinen 60 Jahren solche Atemnot und so zähen Auswurf bei einem jungen Kind gesehen, aber die Heilung war vollständig und der Patient erfreut sich heute guter Gesundheit.' "
(W. B. Griggs, The Hahnemannian Monthly Vol. 90, No. 4; zitiert in: W. Gutman, Beryllium, Allgemeine Homöopathische Zeitung Bd. 203 (1958), S. 436-437)

1962 - Sarkoid der Lungen - W. B. Griggs

"Sarkoid der Lungen bei einem jungen Mann, klinisch bestätigt. Geheilt durch Beryllium."
(W. B. Griggs, Philadelphia, zitiert von Dr. W. Gutman, Allgemeine Homöopathische Zeitung Bd. 207 (1962), S. 157)

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