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TRENDS IN INDUSTRIAL LUNG DISEASE: YESTERDAY, TODAY AND TOMORROW

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The inhalation of vapors, smokes and dust with disease-producing potentialities has been of deep concern to man over many generations. "The potter turneth the wheel" and "cleaneth his furnace" are Biblical references to the early beginnings of industries with potential siliceous hazards. Mark Ruffer, studying exhumed Egyptian mummies of the XIII dynasty referred to the injurious marks left by silica in lymph nodes draining the lungs. One of the finest historical medical reports is by Georgius Agricola in his *De Re Metallica* which was written in the sixteenth century. The English translation was made in 1912 by Herbert Hoover mining engineer at that time. This translation was made some 45 years before the advent of "modern Republicanism!"

Terminology

Before launching into this review of occupational lung diseases due to dusts or chemicals, a word on terminology. The term pneumoconiosis is employed much as Leroy Gardner used it, meaning, in essence, all chronic lung changes directly attributable to the inhalation of dusts and chemicals. The label pneumoconiosis is a non-specific term, not a disease entity! From the standpoint of clarity, in the interests of both legal and medical professions, the specific type of an occupational lung disease should be spelled out. If, for instance, it has been clearly demonstrated that a certain coal dust is responsible for a characteristic lung condition, it should be so recorded; *e.g.* "Pneumoconiosis in Anthracite Coal Loaders, Northern Missouri Coal Fields, Mine #4. . . ."

The creation of hybrid names or exotic terms for occupational dust diseases is to be deplored as the terms introduced are often without significance; or as Merewether says about ptilosis, a lung disorder allegedly due to the inhalation of ostrich feathers, "What this definition really means only the bird knows."

Early Days of Silicosis

A. J. Cronin, writing in *The Citadel,* with the scene cast in the 1920's had this to say on the paucity of knowledge of dusty lung and its ultimate sequelae.

> There was evidence, mostly conflicting, from South Africa upon that red rag of Rand Labour troubles, gold miners' phthisis, which was undoubtedly due to dust inhalation . . . but beyond that, nothing!

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The fundamental lesion in silicosis is familiar to all who have studied the pathology of the lung. It has a consistent pattern which holds for a number of different mining areas in which there has been exposure to high concentrations of free silica. The dust hazard responsible for this pattern between the years 1890-1935 was very great. Nesbitt in his book *Gold Fever* writing of the years 1912-1915 said,

> Men worked out their eight years or a little more and duly died, as though dutifully conforming to a strange custom of an underground country. No insurance company would do buiness with a miner.

With the disintegration of tin ore mining in England at the beginning of the 20th century, these disbanded workers sought employment in the South African gold fields and in the diverse mining areas of the United States and Mexico. They brought with them poor mining habits, dormant tuberculosis, and nomadism, the miner's prerogative. These three features no doubt played a prominent role between 1900 and 1935 in spreading the "scourge of the mine" in this country and in other countries in which rich ore areas were being ripped apart. A highlight on the destructive nature of silicosis is recalled on considering the rapid onset and dissemination of silicosis and tuberculosis as observed in construction workers on the Gauley Bridge project in West Virginia.

The Changing Tale of Silicosis

From my own observations, and from leading investigators contributing to the medical literature, it seems probable that severe degrees of silicosis such as were described in the first 30 years of the 20th century are now no longer produced. Not only has the severity decreased but the length of exposure needed before silicosis develops has greatly increased. Smith stated that in 1916 the average underground service of the new silicotic was 8½ years. During the 6 years immediately prior to 1947 the average was 20 to 21 years. The most striking feature is that the nodules are fewer in number, localized and practically confined to the upper and posterior parts of the lungs. The disease, as it were, does not now invade beyond these strongholds.

My earlier experience with silicosis was derived from investigating silicosis in gold miners working in northern Ontario gold fields. In some 50 cases analyzed over 10 years ago, tuberculosis was a concomitant complication in over half of them, and death in another 20 per cent was due to cardiovascular difficulties. Most of the cases observed recently have been in foundry workers. In these workers the complication of tuberculosis appears to be reduced and confluence of the silicotic nodules is uncommon. The workers die from other conditions, such as Bright's disease, malignant hypertension or malignant growths unrelated to the lung. The problem today is to assess the degree of disability and determine, if possible, that the limited degree of micronodular silicosis produced pulmonary disability either through fibrosis or adaptive bronchovascular changes or that death was hastened. The large lung section is of inestimable value in correlating disability with structural alterations and anatomic extent.

This lessened severity of silica pneumoconiosis and sharply curtailed incidence are attributable to excellent housekeeping in the majority of manu-

facturing plants, careful tracing of tuberculous carriers, and thirdly, with the knowledge that widows now control 70 per cent of the wealth in the country, there is no need on their part to marry again, infecting their second and third husbands with tuberculosis! This was the common story 50 to 100 years ago!

There are two other facets of silica pneumoconiosis which might be touched on; one is the differing susceptibility of individuals in developing silicosis, and the other is the possibility of eliminating silicosis completely as an occupational hazard in siliceous industries. Some years ago Riddell worked out the following time table on the incidence of silicosis in certain occupations.

Gold mining	24 men working 10 years	1 develops silicosis
Foundry	228 men working 10 years	1 develops silicosis
Porcelain	933 men working 10 years	1 develops silicosis

The differing incidence of silicosis in the different industrial areas can be explained by the varying concentrations of dust. But the interesting aspect which is worthy of future investigation is that many men escape the crippling disease, yet apparently working under similar conditions as the individual afflicted with silicosis.

As to the second aspect, in modern plants probably dust suppressive measures are as effective as it is possible to make them. A preventive or therapeutic feature directed against the development of silicosis but which needs further research is the possibility of introducing an antidotal component into the lung, to prevent the pernicious micro-particles of silica from going into solution and causing aggressive nodular scars within the lung. This, in essence, is the most satisfactory explanation for silicosis; antidotal neutralization of this action may lead to the eradication of the disease and its disappearance from the roles of workmen's compensation acts. On this point, it has been commented on in medical-legal circles that a paradoxical relationship exists between incidence of disease and litigious interest! The current position of silicosis is an illustration of this expression.

The question of slowly developing complications following upon remote exposures to noxious agents or pernicious dusts represents one of the plagues which afflict the medical and legal professions. It is one that requires careful analysis, with the expenditure of every effort to reconstruct the chain of events, before any precipitate legal decision is appended. An alleged relationship between lung cancer and silica pneumoconiosis has been put forward. On this it should be remembered that the incidence of lung cancer is quite high in the general population and unrelated to the siliceous industries. A recent survey of 1438 cases of silicosis showed an incidence of 0.70 per cent lung cancer, and in 1393 non-underground population cases, there was an incidence of 0.93 per cent. Only in hematite miners, in which there is a heavy admixture of iron with silica, has there been any evidence adduced for an increment in lung cancer.

Years	*Lung Cancer %*
1932-1947	4.5%
1948-1955	15.4%

The frequency of lung cancer in male non-miners (hematite area) was reported as 2.0 per cent. This recent study by J. S. Faulds indicates that several factors may be operating in the development of lung cancer under these particular circumstances.

Coal Workers' Pneumoconiosis—"Black Twin of Silicosis"

A. J. Cronin, 1937, in *The Citadel* says:

> Without jumping to any immediate conclusion . . . the incidence of pulmonary trouble amongst the anthracite workers was positively in excess of that existing in the other underground workers in the coal mines.

The years 1800 to 1875 were those of indecision as far as the role of coal was concerned in the production of a genuine occupational lung disorder. As early as 1834, the "scourge of the mine" was accompanied by "black spit" and autopsies showed a soft black lung. Despite these early attempts to grapple with the problem, it was not clear whether there was a single pathological entity or a group of entites, *e.g.*, "bronchitis, interstitial pneumonia, asthma, etc."

The years 1875-1940 were those of "innocence." Mavrogordato, long a leader in the investigation of the dusty trades wrote in 1922 that "Colliers do not now develop miners' lung diseases"; and a great investigator, Haldane, stated that "The dust the miners breathe protects them from serious danger."

The years 1940-1957 have been crucial years of re-examination. Cummins and Sladden had considered the shortness of breath in miners to be due to a form of silicosis modified by the accumulation of coal dust; yet these findings were in miners who had done no "hard heading" (drilling through siliceous rock). At the same time the anthracite coal fields of Pennsylvania were surveyed and the lung shadows of the miners on the basis of radiographic studies were graded as I, II, or III degrees of *anthraco*-silicosis. Although it had been noted that in these miners' lungs the percentage content of free silica was low, the radiological changes were attributed to the long exposure of dustier systems of mining, producing a high total storage of silica. The conclusion of this report from the anthracite Pennsylvania fields was hedged by the remark that "Silica is usually the fibrosing agent, although adherence to the general term pneumoconiosis still seems desirable." Then Gough uncovered in 1940 that in laborers loading steamships with bituminous coal developed a distinctive pneumoconiosis that was directly attributable to the action of coal, not silica. This lead to a complete survey of the South Wales coal field by a number of investigators, *e.g.*, Fletcher, Gilson, Heppleston and others, firmly establishing that coal dust itself can produce pulmonary fibrosis. In England now coal miners' pneumoconiosis is an accepted entity recognized by both the medical and legal professions.

In the United States, the extent of this disease is not known, but there are a few scattered reports that indicate its existence cloaked under the terms "anthracosis" or "miner asthma." Although the controversial aspects and the factors which hinder the investigation of this condition are recognized, it was felt that an effort to label "coal workers' pneumoconiosis" as a genuine disease entity was warranted.

It is customary in textbooks and other writings to quote silica as a hazardous dust and to regard coal as a harmless dust. But Gough says;

> In our experience the dust of commercial coal is harmful when breathed in the quantities in which miners and other coal workers in Wales have been exposed during the last 30 years.

In coal workers there appears to be three distinct forms of pneumoconiosis involving three different pathological processes. First, there is the relatively uncommon classical silicosis seen in men engaged wholly or mainly in drilling or hewing rock and cutting through strata which separate the coal seams. Hard heading is the name given to this occupation. On the other hand, there are two distinctive phases of "coal workers' pneumoconiosis." First, there is the so-called "benign pneumoconiosis," as referred to by most writers, in which foci of coal dust occur throughout the lung and in relation to these, focal emphysema develops. The pathological implication is that the emphysema is mechanically produced by the deposition of coal dust. The other phase is a late developmental one of massive fibrosis superimposed on "simple" pneumoconiosis. These massive lesions appear to be the result of infection developing in a dust-laden lung and the infection appears to be tuberculosis. This third form is called "infective massive pneumoconiosis."

In this third type of infective massive pneumoconiosis, because of the great amount of fibrous tissue, the progress of the disease is slow and death usually results from congestive heart failure. This is directly attributable to the encroachment of the fibrous tissue upon the pulmonary vessels which increases greatly the work-effort required of the right heart, eventually ending in failure. The rank of coal appears to influence the disease process. Extrapolating from the evidence put forward by the South Wales investigation, one would anticipate that the anthracite coal fields of Pennsylvania would produce a higher incidence of coal miners' pneumoconiosis than the bituminous fields of Utah.

To determine the true cause of death of coal miners dying in their fifties and sixties requires a post mortem examination with *detailed quantitative studies* of the heart-lung alterations. It is only by such studies that the true nature of the shadows in the chest can be determined. Shadows in the chest in the coal worker do not necessarily mean an occupational disease. Far too frequently such an indiscriminate diagnosis leads to unnecessary legal entanglements, raises false hopes of compensability and burdens the administration of management.

On dusts, akin to coal in that they too were previously regarded as innocuous, it is conceivable that inhalation of iron in insulting quantities over many years, or barium, or graphite, or carbon black, could produce a disabling lung disorder. But such cases have to be carefully evaluated, as certain elements, because of their high atomic number, as iron and barium, are opaque to X-rays and produce shadowings of the chest fields. This, of itself, does not mean disability!

Silicate Pneumoconioses

Most silicates in the natural state are not responsible for fibrotic contractures of the lung, although in the packing and shipping sections of industrial

plants concerned with the commercial preparations of such substances, the atmospheres are frequently dusty. Sherlock Holmes in the *Case of the Engineer's Thumb* says that "Fuller's earth is a valuable product" as a filter and color removal agent; sillimanite in porcelain manufacturing, and China clay for the pottery and ceramic industries. All can be given as far as producing irreversible lung changes a "clean bill" of health.

(a) *Non-Fibrous Pneumoconioses*

With diatomaceous earth widely used in the United States and extensively mined, sacked, graded and treated in southern California, the problem may be somewhat different. Diatomaceous earth has many uses as a filter for dynamite, abrasives, polishes, extenders and building materials. The crude native diatomaceous earth (salt water type) from California, exists as amorphous hydrated silica (opal). Daily inhalation of this dust over some years may produce diffuse linear lung shadows. Fresh water diatomaceous earth does not appear to have this stimulatory property.

With calcination (heating to 1800°) or flux calcination (heating with soda to 1800°) of the diatomaceous earth, large percentages of cristobalite are produced. This latter heat-produced constituent has greater fibrogenic powers than its natural occurring kin, quartz. In the cases which I have personally observed, one can recognize two basic reactions: a marked cellular increase along the bronchovascular pathways due to the native earth and, secondly, an equally distinctive nodular response due to man-induced alterations in the diatomaceous earth.

Between the years 1930-1940 the complications observed in the workers of diatomaceous earth might well, at least as far as the California field was concerned, have been attributable to the adventitial factor of a high rate of indigenous tuberculosis within its workers. Since that time, with the advent of modern methods of dust control, and after a "second look" at the nature of the dust, the working environment has improved greatly in this industry. Because of this marked improvement in plant conditions, the intensity of exposure to such dusts has been sharply reduced. With the falling away of tuberculosis as a major disease, the complications that might be anticipated, because of the anatomical placement of the dust along bronchovascular channels, are bronchial obstruction with emphysema and compromisation of the pulmonary vessels with the late sequelae of pulmonary heart failure.

(b) *Fibrous Pneumoconioses*

Asbestos, a mineral of unparalelled properties was used long ago as a crucible for burning tapers before Vestal Virgins and was an important ingredient in the funeral robes of kings. Both these professional trappings have long since disappeared, but the same unconsumable properties, recognized by the ancients, are now utilized in insulation and fire resistant products such as roofing materials, theater curtains, heating elements and refractory products.

The mineral is mined out of limestone, crystalline or quartz schists in association with iron and alkaline earths. The chief geographical locations of the veins are southern Quebec, southern Rhodesia, New York, California,

Arizona, Georgia, Brazil and Russia. Russian asbestos is usually of the "harsh" variety; this is a mineralogical term!

From the physical properties listed below, it is readily apparent why certain forms of asbestos are more frequently chosen for commercial products, *e.g.*, white Canadian chrysolite, because of its long flexible fiber, is highly desirable for fabrication purposes. These same physical factors are also responsible for the development and severity of the lung disorder.

Asbestos Types	Tensile Strength	Spinnability	Surface Area	Heat Resistance	Fibre Diameter
Amosite	90,000	++		++++	
Anthophylite	4,000	+		++++	
Chrysolite	100,000	++++	220,000	++++	.0000008
Crocidolite	300,000	+++		+	
Tremolite	8,000	0		++	
Actinolite	1,000	0		+	
Comparison Purposes					
Carbon Steel	150,000				
Cotton Fibre	75,000				.0004
Glass Fibre	150,000				.00026
Human Hair					.0016
Nylon				3,100	.0003

At the mine head and rock crushing houses, the lung hazard is less frequently encountered than in the carding and spinning factories, the site of fabrication for finished products, although even here the incidence is low. From 1900 till 1924, working conditions in some fabrication plants were, to say the least, unsatisfactory. The dust was so thick that "the hand could not be seen in front of the face." Since that time, conditions have been vastly improved through rigidly enforced plant hygiene methods.

The best of evidence indicates that with inhalation the silicate fiber of asbestos is trapped in the terminal bronchial ducts in the bases of the lungs and acting as a foreign body which elicits an inflammatory and subsequently an incarcerating scar reaction. This reactive fibrosis and the asbestos bodies present a highly characteristic appearance under the microscope. In the X-ray of the chest, all that is seen in granular shadowing in the bases of the lungs. The two complications of asbestosis frequently seen years ago were bacterial pneumonias and tuberculosis engrafted on the prepared soil. In manufacturing areas in England it has been held that 30 per cent of individuals in the early years of the asbestos hazard developed tuberculosis either due to implantation of the tubercle bacillus upon the asbestotic pneumoconiosis or a lighting up of a dormant childhood infection.

Since 1950, of the workers who were exposed in the "dusty, glorious '20's," one late alteration within the lung which has been commented on by investigators such as Perry, Middleton and O'Donnell has been the development of primary lung cancer. This sequential development pattern would appear, judging from individual case reports, to be an interesting one. Several investigators claim that 15 per cent of individuals suffering from asbestos pneumoconiosis develop primary lung cancer.

The second possible complication in these individuals who have had exposures to asbestos of thirty years or more, even in controlled, reasonably hygienic plants appears to be the development of thickening of the pulmonary arteries leading to pulmonary hypertension and terminal right heart failure.

Vegetable Pneumoconiosis

In the handling of agricultural products, directly or indirectly, thousands of workers are involved. Respiratory tract symptoms and signs, loosely labeled "asthma, bronchitis and unresolved pneumonias," are often alleged to be due to the inhalation of such materials as hay, grain, tobacco, pepper, sugar cane chaff, cotton and corn products. Louis Bromfield in his story of Malabar Farm gives an excellent clinical description of "farmers' lung."

Dock workers and shredders of dried Louisiana bagasse (stalkings remaining after the sugar juice has been extracted) sometimes developed sudden shortness of breath, blood tinged sputum, and shadows in the X-ray plate. Owing to the self-limited nature of the disease, pathological studies are rare. No definite incitant has been uncovered; but the prevailing concept is that fungal contaminants in the drying bagasse are responsible for this enigmatic pneumonia. "Silo disease" and "Monday morning" or "factory fever" encountered in the cotton spinning factories, may be an allied disease state. This pulmonary condition encountered in cotton factories may have an allergic background with the termination being emphysema and pulmonary heart disease.

Clinical Manifestations

A recent case which was studied illustrates the difficulty of incriminating a vegetable fiber as being responsible for minute radio-opacities in the lung fields of a worker in a rug-weaving factory. Fibers derived from the fibrous portion of the vegetable were inhaled over a period of 23 years, with the pathological findings being suggestive of a long standing struggle between the defenses of the body and allergenic vegetable products. To validate the establishment of a genuine occupational disease required an analysis of both plants, vegetable and industrial; the effects of the dust upon biological tissues and the establishment of a probable relationship between symptomatology and pathological findings. This is the approach, rather than the hasty allegation and uninformed hypothetical question. Awards should not be made on surmise or speculation.

Metallic Pneumonitides

Beryllium

In 1931 only 2 tons of beryllium ore were used in the U.S.A.; in 1946 the consumption had increased to 2,000 tons. Much of this increase came about through heavy demands during World War II for an alloy with an extremely high melting point, lightness and great elasticity. Beryllium became the glamor child of metallurgy and has found wide usage in radio-television tubes, precisions tools, incandescent and fluorescent lamps, and atomic energy products.

Workers exposed to the beryllium or its bonded acid salts, either in the extraction or manufacturing processes, may develop diseases of the eyes, skin and lungs. Two forms of lung disease exist; an acute "chemical pneumonitis" and the chronic granulomatous form.

The chronic pulmonary disease first showed up as a baffling lung condition in the closing years of the war, being first described in Massachusetts as "Salem sarcoid." This type of granulomatous tissue response is now considered by some investigators as a sensitized tissue response, with beryllium being the sensitizing agent. The minute amounts of beryllium recovered from lungs chemically analyzed would support the belief that sensitization through a previous exposure may play a prominent role in the development of the lung alterations. I need only repeat that a worker overhearing the buzzing of a fluorescent lamp does not constitute an exposure!

The sharp curtailment of the amount of beryllium incorporated into alloys, and its abolition as a coating for fluorescent tubings are two public health measures introduced which have effectively suppressed the development of new cases, a triumph accomplished in a few short years, through the cooperation of manufacturers, insurance carriers, medical professions, chemical engineering and public health authorities!

Aluminum

Shortly after the finish of World War II, a distinctive form of a crippling lung disorder appeared in furnace operators and crane men working above bauxite fumes. Bauxite is a highly essential ingredient needed in the manufacturing of abrasives. From my observations on some 10 cases of this unique scarring of the lung, the high concentrations of amorphous aluminum oxide apparently selectively damaged the air sac walls. This diffuse scarring profoundly interfered with the vital exchange of gases in the lungs, with the workers dying of pulmonary insufficiency. No increased incidence of tuberculosis or lung cancer has been observed in this lung disease. The diffuse scarring within the lungs comprised the small bronchi and the small vessels of the lung with the workers developing emphysematous air sacs which often ruptured, precipitating acute massive collapse of the lungs, or death resulted from congestive heart failure. To the best of my knowledge, this condition has virtually disappeared.

An understanding of these two conditions offers a framework for investigating other patterns of lung disease, which have appeared already or may appear on the medico-legal horizon. Cadmium, nickel, chromate, manganese and many other valuable metals being used, either in their elemental state, bonded or incorporated into carriers, produce or may produce a "metal fume fever," or a progressive scarring of the lung. It is unfortunate that scarring, a healing process for infections, produces a crippling broncho-vascular disease in the earth worker. A late pathologic possibility following exposure of the worker to these enzyme inhibiting substances is induction or promotion of a new growth in the lung. With the rapid expansion of industrialization, it is from this latter group of chemicals that development of future occupational lung disease might be expected.

The fast moving chemical and technologic developments reflected in

modern production processes means associated problems for industrial medicine. Unfortunately too often industrial medicine has to await the development of a destructive event before the pattern of disease is understood. Frequently, the legal profession expects informed answers even before the event has occurred! A solution to this general dilemma has not yet been offered. The understanding and settlement of these potential problems will only come through careful investigation, not hasty litigation.

A word of caution on the creation of new and modern pneumoconioses. Far too often, exuberance and enthusiasm on the part of the medical profession, unrestrained, have hinted at or directly led to the establishment of occupational lung diseases—in harsher words, an attempt to establish an industrial lung disorder without evidence. Russell has recorded that ascertainable truth under the best of circumstances in "piecemeal, fragmentary, uncertain and difficult to glean." Progress and understanding of all forms of disease closely parallel this philosophic conclusion. This is particularly exemplified by the halting advances manifested by the industrial lung disorders covered this afternoon. The valid establishment of pneumoconioses in the future will be no different.

This word of caution is directed to all groups concerned in the health of the individual worker. Clinical coloring woven into a chest X-ray interpretation; failure of industrial commissions or workmen's compensation boards to test the probity of the expert's evidence; a facile correlation between alleged exposure and simulated disability; these are all false salients which can only lead to the unjustified creation of a spectral lung disorder . . . which may return to haunt its creator! Coal miners' emphysema may be genuine, but it is equally true that genuine emphysema exists in the coal miner!

Conclusion

In the future, not only will the conventional patterns of pneumoconioses be present, but there will be modifications through admixture of other dusts. New lung hazards will alter the industrial lung panorama, perhaps due to the inhalation of newly discovered volatile materials, rare earths, "innocent" silicates, exotic plant material or newly created alloys or plastics. All this will substantiate the quotation, "In this harsh world, draw thy breath in pain." This utterance is from Shakespeare, not from the tongue of a union-shop steward.

As a pathologist I would make a plea, to both claimant and defendant, that every channel be explored in the investigation of a potential case of an occupational disease; by correlative pulmonary function studies and needle biopsy of the lung, or the autopsy, *"mortui vivos docent,"* or even breaking the strange isolation of death with an exhumative study. The changes in the lung may be the "last clear chance" to obtain justice, redress a wrong or write new law.

To paraphrase an earlier reference of Thomas Belt writing in 1942: The lung is a dossier filled with incriminating evidence. In a very real sense an occupational log book; it holds an indelible record of mineral particles breathed during life; and after-death constitutes a sort of palimpsest of the industrial history. The lung of the worker in dusty trades is a parchment,

a fabric often inscribed over from top to bottom. As for the inks, they may be the black dust of coal: the glittering tracery of silica or the red of tubercle.

I trust that this recapitulation of the fumblings in the past on dusty lung disorders, present hesitancies, and possible future trends will not remind too many of this audience of Hernik Ibsen's writing in *Peer Gynt:*

> This is a tale turned topsy-turvy,
> prinked and tinselled out,
> decked in plumage new and fine,
> till none knows its lean old carcass.
> That is just what you've been doing,
> vamping up things, wild and grand . . .
> and with all the other horrors,
> filling me with speechless dread,
> till at last I recognized not
> what of old I'd heard and known!