FILE NAME: Brakes (BRK)

DATE: 1937 Jan

DOC#: BRK005

DOCUMENT DESCRIPTION: Article - New England Journal of Medicine - The Value of Sympathectomy in the Treatment of Vascular Disease

The New England
Journal of Medicine

VOLUME 216 JANUARY 28, 1937 NUMBER 4

VERMONT STATE MEDICAL SOCIETY

THE VALUE OF SYMPATHECTOMY IN THE TREATMENT OF VASCULAR DISEASE*

BY R. H. SMITHWICK, M.D.†

CONSIDERABLE progress has been made in the treatment of vascular disease as a result of a better understanding of the relation between the vascular and the sympathetic nervous systems. Our knowledge of this subject is by no means complete, but is increasing rapidly from year to year. In the brief time at my disposal I shall discuss the results of sympathectomy in Raynaud's disease, thromboangiitis obliterans, angina pectoris and essential hypertension.

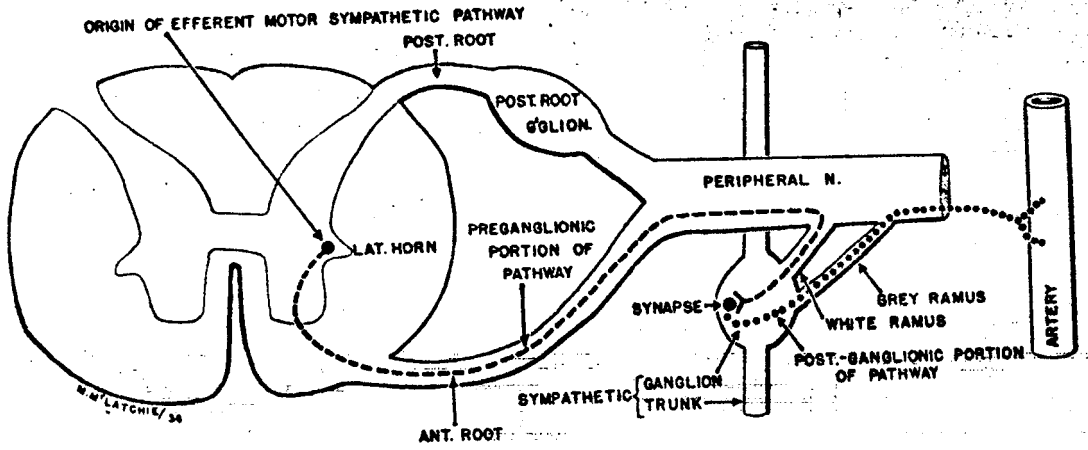

FIG. 1. Diagrammatic representation of an efferent vasoconstrictor pathway from its origin in the lateral horn to its termination in the arterial wall. The devious pathway makes its interruption possible in many different places from an anatomic point of view. Note the pre- and postganglionic portions of the pathway. This is of great physiologic importance, as described in the text.

Anatomy. Before turning to the clinical aspects of the subject, I wish to mention certain anatomic and physiologic facts, which must be appreciated in order to understand the effect of sympathectomy on blood vessels. We are concerned primarily with the motor impulses to arteries, their origin, pathways and the most accessible points at which they can be interrupted. The most important of the motor impulses result in vasoconstriction, although vasodilators are also present. Langley first clarified the efferent pathways of the visceral nerves from the central nervous system.[1] The neurons of the vasoconstrictors arise in the lateral column of the gray matter in the entire dorsal and upper two or three lumbar segments of the spinal cord (fig. 1). Their axons leave the cord by way of the anterior roots, reaching the outlying sympathetic trunk by way of the white rami communicantes, which run from each peripheral nerve to the corresponding ganglion of the sympathetic trunk. These white rami arise close to the intervertebral foramen. This portion of the pathway will be referred to as preganglionic. The axons terminate in some ganglion of the sympathetic chain where they form synapses and then continue on to the blood vessel wall as postganglionic fibers. The postganglionic axons leave the ganglion of the sympathetic trunk by way of gray rami and then join the peripheral nerves, leaving them at various levels along their course to supply the arteries in question. It therefore becomes obvious that the efferent vasoconstrictor pathway

*From the Surgical Services and Circulatory Clinic of the Massachusetts General Hospital.
Presented before the One Hundred and Twenty-Third Annual Meeting of the Vermont State Medical Society, October 16, 1936.

†Smithwick, R. H.—Assistant Surgeon, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 180.

DANGEROUS DUSTS*

BY JOHN B. HAWES, 2ND, M.D.†

THE subject on which I am to speak is important for two reasons. In the first place, this question of dust, dangerous or otherwise, constitutes the most important problem in the whole field of industrial medicine and, secondly, dust is the source of one of the most vicious "rackets" inflicted on this country. This latter I will describe briefly.

The basis of this racket is founded on (1) ignorance on the part of the workingman, (2) ignorance on the part of doctors as to what dusts are dangerous and (3) unscrupulous and mercenary lawyers. The average workman is well aware that the granite cutter, who has been exposed to granite dust and has been disabled thereby, quite properly receives compensation, as does the sandblaster and, in many instances, the foundry worker. Naturally, the laborer is in no position to make a distinction between the various varieties of dust, innocuous or dangerous. All dusts look alike to him. Therefore, if in the course of his work in a lime kiln, a textile factory or flour mill or elsewhere, he comes down with bronchitis, bronchiectasis, asthma, tuberculosis or something similar, he very naturally argues that it was the dust that caused it and claims damages. This brings us to the second reason for this racket, which is likewise based on ignorance—ignorance on the part of the medical profession as to what constitutes a dangerous dust and as to the difference between the general term, pneumoconiosis, and the specific one, silicosis. Of the many physicians whose testimony I have listened to in such cases I know of only one that I feel is actuated by mercenary motives for testifying as he does. In the vast majority of instances of this kind, doctors are testifying for their clients with honest and sincere motives. A few examples of this might well be given here.

A physician, whom I have known for 30 years as an intimate friend and a high-grade general practitioner, testified in my presence that a certain man who had been working in a woolen mill had acquired a chronic asthmatic bronchitis because of the dust to which he was exposed in the course of his work. He had never inspected the mill itself. Aside from the fact that wool dust is perfectly innocuous and never has caused any serious disease, I personally had inspected this mill which was situated in one of the suburbs of Boston and had found the conditions there to be the most hygienic that one could expect. There was practically no dust.

Another case was that of a man who had developed tuberculosis. He had worked as a cutter of cotton cloth with scissors and with a machine in one of the most high-grade private charitable enterprises for the handicapped that I know of in Boston. He was under the care of a well-known and competent physician. In going over the files of this case I read a letter from this physician to the effect that his patient's exposure to this cotton dust, inhaled in the course of his work, might well have led to pneumoconiosis and should therefore be considered a possible cause of his tuberculosis. Naturally, I had inspected the premises where this man worked and knew that the dust was conspicuous by its absence and that the quarters were light, airy, clean and well ventilated; by no conceivable chance could the dust have been in any way a cause of his illness.

These are examples of what I believe to be ignorance on the part of physicians desiring to help out their patients. The following case is rather different. A man in a certain very large industry in this country may be exposed in the course of his work to minimal amounts of rust or iron dust, which is beneficial rather than harmful, equally minimal amounts of lime, which contains a microscopic amount of a harmless silicate (lime in fact is often advocated in treatment) and some powdered soap, one of the ingredients of which is a silicate, perfectly harmless in such minimal quantities. The powdered soap is used for lubricating iron or steel wire as it is drawn through a die. It should be borne in mind that this soap was a *lubricant* and in no way an *abrasive*, as an abrasive soap would have ruined the wire. The physician I have in mind testified in five of these cases, in which I likewise appeared. In one case the autopsy showed that the man had died of cardiorenal disease with no trace of silicosis in his lungs, but it was claimed that the man had had *silicosis* and that this was responsible for his death. His testimony in the other cases was along similar lines. Of this nothing further need be said.

The third factor, and the most important one in the development of this racket, is the crooked and mercenary attorney. One of the very large industries in this country, that of cement-making, where workers may be exposed to one of the harmless silicate dusts, has been subjected to attacks on this basis with the result that verdicts of $15,000, $20,000 and $25,000 have been awarded to the plaintiffs, with cases said to amount to over $12,000,000 now pending! Clever lawyers with able assistants, a hired group of witnesses and unscrupulous physicians were able to carry on this remunerative occupation for a very long time before the manufacturers com-

*Read before the meeting of the American Clinical and Climatological Association at Richmond, Virginia, October 27, 1936.

†Hawes, John B., 2nd—President, Boston Tuberculosis Association. For record and address of author see "This Week's Issue," page 180.

bined resources to defeat them. So much for one aspect of this question.

Now I return to my subject, "dangerous dusts." The title is a bit of a misnomer as there is only *one* dust that may be dangerous and that is the dust which contains silicon in some form. All the organic dusts and those inorganic dusts which do *not* contain silicon are harmless. Lest there be a storm of dissent from those who wish to contradict me when I make this statement, may I first define the word, "dangerous", as used here. By "dangerous" I mean a dust that, either by itself or in combination with something else, injures or irritates the lungs or leads to a fibrous tissue reaction which reduces the individual's resistance to tuberculosis and other bacterial diseases of the respiratory tract. Naturally, I do not include chemical poisons.

Let us first consider the organic dusts. Street dust, although it may contain small amounts of silica, in itself is harmless. The only danger incurred in breathing street dust comes from the germs that may be present, especially in winter. There has been a great deal of loose writing about the danger of organic dusts. For many years the high mortality among the workers in tobacco factories was notorious and was blamed on the dust. The truth of the matter is that tobacco dust is not dangerous but that the work involved in this industry called for little or no physical exertion and, therefore, invited weaklings, while, in addition, the hygiene of the workers was notoriously low. Consider the textiles, cotton and wool. Anyone going into the sorting room of either a cotton or a woolen mill will usually find the air full of dust. Here the bales of raw material are opened and the contents "fluffed up", as it were. Armfuls of the cotton or wool are then put down an immense chute by the workmen, where it is carried on to the next department. In the majority of instances, anyone entering this room for the first time, unless he is absolutely nonallergic, will soon notice a tickling of his nose and a strong tendency to rub or scratch that organ and, sooner or later, will experience some sneezing. Strongly allergic persons will have this to a marked degree. Certain individuals of this type often come down with a severe acute rhinitis or a bronchitis during the first 10 days or so of their work in the sorting room, which soon passes off, however, leaving no ill effects. All this does not mean that wool or cotton dust is dangerous in the slightest. And yet one of the most marked cases of silicosis that I ever saw was in a workman whose job for many years had been in the sorting room of a large factory where woolen carpets were made. A large part of the raw material in this room came from Egypt or some other foreign sandy district. Amazed at finding silicosis in a wool worker when I knew that wool could not be the cause, I investigated the plant and in this room found an eighth to a quarter of an inch of brown desert sand on the floor, some of which, in the course of the work, was of necessity breathed in—hence, this man's silicosis.

Another marked case of silicosis occurred in a man who had worked for many years in the Walter Baker Chocolate Factory in Milton, and yet chocolate and cocoa dust are certainly innocuous enough. This man's sole job, I found on investigation, was to resurface the granite mill wheels used to grind the cocoa bean. Another very marked and eventually fatal case of bronchiectasis occurred in a worker in a large lumber mill where every workman was exposed to varying amounts of wood dust. In this case, however, the man fell down a chute into which sawdust was poured and was taken out, unconscious, an hour later from the mass of sawdust which had buried him. Here again there is nothing to prove that wood dust is harmful. And so I might go on through all the organic dusts.

Now a word about the non-silicon-containing inorganic dusts. The worker in the soft coal mine develops anthracosis, which shows up strikingly on an x-ray, but it is *not* a disabling disease, nor does it predispose him to tuberculosis. If he gets silicosis, as some of the workers in the hard coal mines occasionally do, it is because of the granite or silicon-containing rock in which the coal is imbedded. I have no doubt but that the slate and marble workers will present changes in their lungs after years of exposure to this dust, but such exposure certainly does no harm. Exposure to lime even seems to do good.

Granted, then,—I feel sure that many of you will not grant it—that the only dangerous dust is the one that contains silicon in some form, what are the factors that determine the degree of danger in the exposure to such silicon-containing dusts? These may be given as follows:

(1) The *amount* or *proportion* of silica present in the dust. Silicon is such a common element that it might be said to be ubiquitous. It is present in small amounts in practically all street dust. The stiffness of the fibers in hay, straw and cereals and that of the feathers of birds is due to silicon. It is a normal constituent of lung ash up to 1 per cent. It is only when it is present in excessive amounts, such as in granite dust and sand, which are nearly pure silicon, that it constitutes a menace.

(2) The *form* of silicon present in the dust. Free silicon, SiO_2, as found in granite and sand, is infinitely more dangerous than combined silicon, as found in the silicates with one exception—asbestos.

Except for asbestos the silicates may be described as comparatively harmless. Consider tale, for instance. Tale, a magnesium silicate, is the basis of almost all toilet powders and is used extensively in the rubber industry. It rarely, if ever, has been known to cause a disabling fibrosis in the lungs.

(3) The *size* of the dust particles. Particles of less than 5 microns are most dangerous. Particles over that size do not enter into the finer tissues of the lungs and are, therefore, practically innocuous. The rate of the fibrous reaction in the lungs is proportional to the fineness of the dust.

(4) The *density* of the dust. If the number of particles of silicon-containing dust per cubic foot of air exceeds twenty million such a dust may be said to be dangerous.

(5) *Intensity* of the exposure. The granite cutter working with a surfacing machine is exposed to infinitely more dust than the foundry worker, who is only occasionally in certain types of work exposed to much dust. A survey in Massachusetts showed a silicosis incidence of 15 per cent in granite cutters and only 8 per cent in foundry workers.

(6) *Length* of the exposure. It naturally follows that the longer the exposure the greater the inhalation of dust. An exposure of 12 to 15 years is the average time required to give a granite cutter a disabling silicosis, while one of 2 to 3 years can cause it among asbestos workers.

(7) Finally, and I might call this the most important factor of all, we come to that indeterminable question, the *individual resistance* to dust. Just as certain persons are extraordinarily resistant to tuberculosis, so are others, for no known reason, able to breathe in large amounts of a dangerous dust over a long period of time without damage to their lungs.

Each one of the above points must be taken into account. Bearing them in mind, let us now consider the diagnosis of silicosis. This has been left far too long entirely in the hands of the roentgenologist. If the x-ray man in court or at the clinic says that the plate shows a diffuse, nodular fibrosis and makes a diagnosis of silicosis, no other factor pro or con, up to the present time, is apt to be given serious consideration. I object strongly to this.

The diagnosis of silicosis, just as the diagnosis of tuberculosis, depends upon various factors, the first of these being the *history of exposure*. Whenever you are called to give an opinion upon such a case, I would beg that you do not take the statement of the patient or that of his friends or, indeed, of his family physician entirely for granted, but that, wherever possible, you go through the plant and see for yourself the exact work that the man or woman did and how much actual dust he or she was exposed to in the course of said work. This, of course, does not mean that you must take a dust count or make a chemical analysis, which should be obtained from experts, but it does mean using one's common sense in sizing up the situation. The second point to be considered in making a diagnosis concerns the *symptoms*. The most striking of these is shortness of breath; cough may be, but often is not, present, while loss of weight and strength, blood-spitting and general weakness are often conspicuous by their absence. Shortness of breath *by itself*, without any other condition to explain it, is an important point.

Third to be considered are the *physical signs* in the lungs. These, again, may be conspicuously absent; but, in well-advanced cases, generalized dulness, most marked at the bases and extending upwards, front and back, harsh, prolonged and often diminished expiration and occasional fine, high-pitched rales, as if two pieces of fine sandpaper were being rubbed together, are important to bear in mind. In addition to these signs there is one on which I am coming to place much importance, namely, a marked restriction of chest expansion on taking a deep breath. This is especially true in asbestos workers. I have seen nearly 75 men and women that have been exposed to asbestos dust and have found that the one striking sign present in those in whom I have made a positive diagnosis has been a greatly diminished chest expansion, rarely over *one inch*. One man of nearly 70 years came in to see me who had worked for many years in this particular factory as a night watchman and who by the very nature of his work had been exposed to little or no dust. He had marked arteriosclerosis and considerable emphysema and complained of shortness of breath, but nevertheless he was able to expand his lungs nearly *three inches*. X-ray showed normal lungs except for a moderate emphysema.

Finally, we come to the *x-ray*. This, in advanced cases of granite cutter's silicosis or that of the sandblaster or indeed in well-marked asbestosis, speaks for itself. The borderline cases, however, are very difficult to decide upon, so much so, that I know of no two x-ray men who would agree. Of course, the presence of a complicating tuberculosis, with a history of exposure to a dangerous dust and even slight signs in the lungs physically or by x-ray, would tend to justify a positive diagnosis, which without these would be impossible.

Asbestosis is a relatively new disease, con-

cerning which we know remarkably little except that it does not seem to correspond to the laws governing silicosis in general. There is not the slightest doubt, however, in my mind at least, that asbestos dust is the most dangerous of all dusts. Asbestosis is able to produce total and permanent disability in a remarkably short length of time, even after only 2 or 3 years' exposure and occasionally less, as compared with the much slower and more gradual action of pure silicon and the great majority of silicates. Asbestos is an aluminum or magnesium silicate or a combination of these or others and, in its chemical analysis, agrees closely with that of talc, cement and certain other silicates. The fact remains, however, that asbestos is extremely dangerous and fatal, while talc, cement and other silicates are comparatively innocuous except under prolonged, intense exposure along with a very greatly lowered individual resistance.

The points emphasized in this brief talk on a very big subject are the following:

(1) That there is only one dangerous dust—that containing silicon in some form;

(2) That the diagnosis should be based, not purely on x-ray, but on a history of exposure, on the clinical symptoms and on the signs in the lungs, along with the x-ray; and,

(3) That, whenever possible, a personal inspection of the claimant's work should be made in order to see for one's self just how much dust exposure there is, thus insuring a correct and fair decision.

PRECISE EVALUATION OF ULTRAVIOLET THERAPY IN EXPERIMENTAL RICKETS*

BY JOHN W. M. BUNKER, PH.D.,† AND ROBERT S. HARRIS, PH.D.†

IN a previous communication, Bunker and Harris[1] recorded the opinion that light radiations of wave lengths other than those in the region, 2800-3100 Angstroms, which at that time were generally regarded as constituting a "vital band" of light, may assist in the biochemical processes of rickets therapy. This opinion, based on preliminary qualitative studies, was strengthened by the findings of Sonne and Rekling,[2] who suggested 2530 to 3025 as an antirachitic region.

Since 1930 but few reports bearing upon this subject have appeared. Knudson[5, 6] reported cure and prevention of rickets in rats by radiations from either a quartz mercury vapor arc or from a GE S-1 bulb and defined the gram calories of these heterogeneous radiations necessary to cure a rat whose skin had been depilated. No attempt was made to evaluate different parts of the spectrum. Gorter[3] reported that only 2950 to 3100 Angstroms are active. Knudson and Benford[7] reported in abstract form that the antirachitic effectiveness of monochromatic ultraviolet light does not correspond to that producing erythema, but details are lacking.

Our own study, continuing with optical filters of special glasses, quartz and solutions of nickel sulfate and copper sulfate, enabled us to investigate spectral zones of decreasing width. One handicap of the filter method is the difficulty of eliminating longer wave lengths while retaining shorter, since increasing absorption is generally first obtained at shorter wave lengths. Another difficulty lies in the fact that such filters do not cut off sharply, but generally allow definite increments of longer or shorter wave lengths to be present in a selected band, which increments must always be of unknown relative importance, if in fact there be a difference in the relative effectiveness of various wave lengths.

By the filter method it was determined that a band made up principally of light from two lines of the mercury arc spectrum, namely, 3025 and 3130 angstroms, showed some therapeutic value in experimental rickets in rats. Subsequently, by the use of a water-cell monochromator, described by Harrison,[4] we were able to eliminate from further consideration the line, 3130, which was completely ineffective at an applied energy of approximately 1.75 gram calories per rat.

All the subsequent work was done with a Bausch & Lomb quartz monochromator (type No. 33-86-05) with prisms 8 cm. by 8 cm., which transmits sufficient energy from a 6 inch, 220 volt Uviare to permit treatment of rachitic rats with single lines of light.

Experimental

The arrangement of the equipment used in the phase of the investigation dealing with monochromatic radiations is shown in figure 1.

In a white transite housing, B, open at top and bottom and raised from the bench on legs, a vertical 6 inch Uviare, A, was placed in the

*From the Biological Research Laboratories, Massachusetts Institute of Technology.

Contribution No. 75 from the Department of Biology and Public Health, Massachusetts Institute of Technology, Cambridge, Mass.

†Bunker, John W. W.—Director of Biological Research Laboratories and Professor of Physiology and Biochemistry, Massachusetts Institute of Technology. Harris, Robert S.—Research Associate in Biology, Massachusetts Institute of Technology. For records and addresses of authors see "This Week's Issue," page 150.