patient learns this the addiction becomes confirmed and of the most serious character.

The other cocaine-like substances which might lead to habit are stovaine, tropacocaine, and perhaps holocaine, though cases of addiction to these are unknown to me.

To some extent fashion or opportunity determines the type of drug used. In New York heroin is used almost entirely, in Chicago morphine is the common narcotic, and in cities with a large coloured population cocaine is in vogue.

TREATMENT.

The first and principal fact to remember in treatment is that the addict is not a mentally normal man, and the problem is not so much whether we can temporarily stop the addiction as whether we can educate his mind so that temptation can be withstood—in other words, to alter his character. Simply to control conduct so that choice cannot be exerted is to court disaster.

The following examples will show the effect of custodial care alone.

The Department of Health of the City of New York publishes statistics and details by medical experts of nearly 8,000 cases of drug addicts during ten months; 1,580 received at the clinic were willing to accept treatment and withdrawal of the drug. Those treated were kept six weeks and discharged in good physical condition "cured." In three months a large proportion had already relapsed, in spite of all efforts made to assist them.

Dr. Doane, speaking in November, 1920, stated that there had been treated in the Philadelphia Hospital 3,500 cases of drug addiction, four-fifths of which were heroin and cocaine. These patients were mostly from the lower strata of society. Dr. Doane thought that 3,400 or 3,500 had relapsed. In Los Angeles the Board of Health, with the assistance of the municipal authorities, started a narcotic clinic where addicts got their supply at a nominal price without criminal risk. This system had several advantages: the victims were able to work again; illicit peddling was reduced to a minimum; the addicts became known; but the system was much abused and cures were not effected.

It is generally recognized that of all addicts those that contract the habit as a result of disease are easiest to cure. All addicts desire to be cured but fear the withdrawal symptoms. An addict may beg to give up the habit of his own free will and submit to restraint, but as soon as his wishes are acceded to he will endeavour to render restraint unavailing, and he is so well versed in the literature of the subject that he finds a ready reason for anything he does. He is quite unable to resist an opportunity to cheat if the occasion offers. To ensure even a temporary cure restraint and custodial care are therefore essential.

The chief reason against restraint is that our object is not so much the temporary suppression of the morphine as the cure of the desire for it, and the best way of effecting this is by the re-education of the dormant will.

Under restraint the drug can be gradually reduced, and generally ten days will suffice to bring it down to *nil*. Some authorities substitute rectal administration for the injections, and some codeine for morphine. Many of these neurotics would do anything rather than diminish their injections; they take a morbid pleasure in injecting something under the skin. During the cure intensive purgation should be carried out. Morphine is the only alkaloid excreted by the alimentary canal, and it is well to get rid of it and prevent reabsorption.

The drug which is perhaps most useful in the treatment of addictions of all kinds is atropine, or, better, hyoscine. Hunger and to a smaller degree thirst are caused by conditions of the stomach, and atropine and hyoscine control the motor nerves to the stomach and intestine. It is more than likely that morphine craving has some association with the stomach. Many other drugs are, however, employed: nitrites and sparteine to lower blood pressure during the withdrawal symptoms, alkalis to combat the dyspepsia, and such drugs as valerian or asafoetida to produce a profound psychical impression. De Quincey found much benefit from valerian. To effect a cure the patient must be made to live a methodical life, to partake of temperate meals, and to observe orderliness in everything, all of which are loathed by the neurotic and morphinist.

In conclusion I desire most strongly to protest against the modern tendency to regard these people as criminals; they are and should be regarded and treated as mental cases,

addiction being the prevalent symptom at the time. The Harrison Narcotic Law has proved a failure in America, yet our own authorities have legislated on parallel lines, and time only will show whether the cure is not worse than the disease.

Since this lecture was delivered an amending bill to the Dangerous Drugs Act of 1920 has been read a second time in the House of Commons and referred to a Standing Committee. It proposes that these narcotic drugs shall be purchased only by registered medical practitioners in actual practice, whatever that may mean. I suppose all registered medical men are in actual practice, though many of us are not in practice for profit. Very large quantities of morphine and cocaine are used by the various chemical and pharmacological laboratories throughout the kingdom. Medical students may be required to detect morphine in solution and to investigate its properties on isolated mammalian tissues. Alkaloidal chemistry without morphine and cocaine is indeed unthinkable.

The second objectionable provision is that the practitioner may not prescribe for himself; horrible penalties are attached to infringement of both these provisions. Almost one is tempted to ask, Can legislators be so foolish as to attempt to pass laws directly concerning medical men which are against the whole feeling of the profession and which are calculated to cause intense resentment? If the drug traffic is to be exterminated then all our hope should be centred on the registered practitioners in this country, and to make bad law by legislating for a hard case or two will do infinitely more harm by alienating the majority of the profession than the mite of good.

Hunterian Lectures

ON

MAN'S POSTURE: ITS EVOLUTION AND DISORDERS.

GIVEN AT THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

BY

PROFESSOR SIR ARTHUR KEITH, F.R.C.S., F.R.S.,
CONSERVATOR OF THE MUSEUM.

[Abstracts.]

LECTURE III.—THE POSTURAL ADAPTATIONS OF THE SHOULDERS AND THORAX.

At the end of a long day's tramp most of us have become aware of a vague aching pain in the region of the shoulders. Nor need we feel surprise when we remember the heavy burden we have had suspended from our neck throughout the day—the load represented by the upper extremities. In a man of average weight the shoulders and arms represent a burden of about 20 lb. They are suspended from the cervical and upper dorsal regions of the spine—chiefly by the trapezius muscles and particularly by the fibres inserted in the neighbourhood of the tip of the shoulder. Then there is another composite sheet of muscle, made up of rhomboid and serrate parts, which is also concerned in maintaining the shoulders in position. By the rhomboid muscles the vertebral base of the scapula is supported from the lower cervical and upper dorsal spines; by the serratus magnus the base of the scapula is steadied on the side of the thorax because that muscle springs from the eight upper ribs. We may look on the rhomboids and serratus magnus as forming a continuous muscular sheet passing from spines to ribs; within it the base of the scapula is embedded and steadied. The levator of the angle of the scapula, originally part of the serratus sheet, is also concerned in balancing and supporting the shoulder from the neck. Now and again, once in a hundred subjects, we see another muscle helping in the support—the levator claviculae. It is strange that man is the only one of the higher primates in which this muscle has become vestigial; it is also noteworthy that in the chimpanzee this muscle is small and tends to disappear. The sterno-mastoid and pectoral muscles also play a subsidiary part in supporting and balancing the shoulder.

SUSPENSORY MECHANISM OF THE SHOULDERS.

When we stand or sit a postural reflex mechanism is brought into action which keeps the shoulder muscles constantly at work until we lie down again. How complex this

mechanism must be inferred from the observation of Dr. Gordon Holmes; he noted that when the cerebellum was injured on one side, the shoulder of the same side had dropped downwards and forwards, because the postural tone of the supporting muscles was partly lost. Professor Wingate Todd¹ has stated that, in passing from infancy to adult life, a progressive dropping of the shoulders takes place; both outer and inner ends of the clavicle descend, but the descent is greatest at the outer ends.

DROPPING OF THE STERNUM AND RIBS.

The falling of the shoulders is not an isolated occurrence, but part of a process in which the whole thoracic region participates. This dropping or ptosis, which may be recognizable by the end of childhood, becomes clearly evident by the end of the third decade. The sternal ends of the ribs and the sternum itself come to take up a respiratory position at a level of one or more vertebrae lower down than in childhood. The level of the domes of the diaphragm descends, the oesophagus becomes elongated, the lateral segments of the ribs more oblique in position, the angles formed by the ribs with their cartilages more oblique. With the descent of the ribs the back-to-front diameter of the thorax is necessarily diminished; Wintrich found that in people who had passed middle life the front-to-back diameter of the thorax had become reduced by 33 mm. in men and 26 mm. in women. The diminution in side-to-side diameter is much less.² An exaggerated degree of this normal dropping of the shoulders, with a diminished or inspiratory poise of the thoracic walls, must be regarded as a result of an exhaustion, or partial exhaustion, of the postural muscles which support the shoulders and ribs when the human body occupies the upright position.

PRESSURE ON THE BRACHIAL PLEXUS.

If the tonus action of the supporting muscles of the shoulder should fail, then a strain must fall on the brachial plexus, particularly on the lower cord as it crosses the first rib. This is especially likely to be the case if the lower cord has to cross a rib attached to the seventh cervical vertebra. Bramwell and Dykes state that not more than 5 or 10 per cent. of cervical rib cases develop symptoms of nerve pressure. Carriers of cervical ribs are certainly more liable to pressure symptoms than those of normal development; the essential circumstance which gives rise to symptoms of plexus pressure is not the presence of a cervical rib, but a failure or partial failure of the mechanism evolved for the support of the shoulders in the erect posture.

A LATE PRODUCT OF EVOLUTION.

The postural mechanism for the maintenance of man's shoulders is a human character of late evolution; it must have come with the adaptation of his lower limbs as the sole organs of support and progression. Man is a flat-backed and broad-shouldered animal; his shoulder blades are placed on the back rather than on the sides of his chest. In these features, however, man does not differ from other members of the orthograde group of primates—the gorilla, chimpanzee, orang, and gibbon. He differs from them in this circumstance—that in the climbing, moving arboreal anthropoid the arms and shoulders have to sustain or help to sustain the weight of the trunk, whereas in man the trunk has to sustain the weight of the shoulders and arms. In the anthropoid shoulder all the essential structural features of the human shoulder are already present; we have only to suppose the perfecting of a supporting reflex postural mechanism for the shoulders and a reduction in the length of the arms to realize how the anthropoid condition might become human. Suspension of the upper extremities is a late product of evolution and must be regarded as a human characteristic.

SUPPORTS OF THE PRONOGRAD THORAX.

In tracing the evolutionary stages which led on to the human spinal column we found that the great break in the series came not between the human phase and the great anthropoid phase, but between the lowest of the orthograde apes—the gibbons—and the highest of the pronograde monkeys. When we seek for the stages which lead on to the human position of shoulders and shape of thorax, we find the real break in the series again occurs at the passage from the pronograde to the orthograde form. If we would understand the changes involved in the evolution of the human shape of thorax and the human mechanism of respiration, we

have to go back a long way in our geological history and formulate a theory which will account for the origin of the orthograde gibbon. One has to note particularly the manner in which the thoracic part of the body is supported on the arms or forelimbs of pronograde monkeys. The back is narrow and the shoulders are set by the side of the thorax, coming close together on the back almost as in a dog. The ventral or sternal aspect of the thorax is also narrow; the body of the sternum itself is a jointed rod. The lateral walls of the anterior part of the thorax are flattened and the scapulae are placed against them. By a series of muscular slings or hammocks the weight of the body is transferred to the upper limbs and shoulder girdles. Chief of these muscular slings or supports are the right and left great serrate muscles, which, rising from the lateral aspect of eight anterior ribs, ascend to be inserted to the vertebral borders of the shoulder blades. The two levator muscles to the angles of the scapulae continue the serrate hammock into the neck; they arise from all the transverse processes of the neck. The rhomboid sheet binds the vertebral border of the scapula to the spinous processes of the neck and upper dorsal region; the trapezius muscles also assist. A secondary muscular hammock is formed by the right and left pectoral sheets (pectoralis major, pectoralis minor, pectoralis externus). They pass from the sides of the sternum and adjacent ends of the costal cartilages to find insertions to the upper ends of the humeri. The flattening of the chest from side to side and the approximation of the shoulders are adaptations to the pronograde manner of progression and of thoracic support.

SHAPING OF THE ORTHOGRAD THORAX.

Many occasions in the arboreal life of a monkey arise when branches have to be clutched by the hands and the whole weight of the body supported from the arms. The muscular slings which attach the shoulder girdle to the body in these circumstances take on a new action; the thorax of the animal now becomes compressed between the ventral and dorsal muscular slings and so tends to be flattened in a back-to-front direction. Now this method of arm suspension, which is only occasional in pronograde primates, has become habitual in gibbons—the arms are the main means of support and progression. The thorax has become flattened before and behind, its sides are rounded, the ribs are drawn up as if they were in a position of extreme inspiration. The pectoralis major—a main part of the muscular sling—has no longer a mere sternal origin as in pronograde apes, but has extended its base of attachment upwards to the clavicle and outwards on the fifth and sixth costal cartilages, almost to the bony ribs. The origin of the pectoralis minor has become shifted from the side of the sternum outwards almost to the costo-cartilaginous junctions. The trapezius, rhomboids, and latissimus dorsi form the dorsal part of the muscular sling for supporting the weight of the body from the arms. In pronograde monkeys the latissimus arises merely from the spinous processes of the back, but in gibbons its origin has been extended forwards, so that it has a fascial origin from the iliac crest and direct muscular origins from the three or four lower ribs. The serratus magnus, now strong and highly specialized, plays an important rôle as a muscular sling. It exerts a traction on the side of the thorax tending to increase the lateral convexity of the ribs and the side-to-side diameter of the thorax. The shoulder, instead of being placed laterally as in pronograde forms, becomes drawn outwards and backwards and thus comes into being the broad-shouldered figure which characterizes all orthograde forms, including man.

ARE THE EFFECTS OF USE INHERITED?

Some thirty years ago I was of opinion that there was no other way of explaining the peculiar features of the gibbon's thorax except by supposing that some pronograde primate had adopted a brachiating habit of progression and that the chest, by inheritance of the effects of use, under the suspensory action of the shoulder muscle slings, had become transformed from a pronograde into an orthograde type. This idea I have abandoned for the following reasons: No alteration in use can cause a migration in the origin or insertion of a muscle—such as we see in the attachments of the muscles directly concerned in slinging the thorax from the shoulder in orthograde apes. During the period of development we see the process of muscular migration actually taking place; it is a developmental process carried out by a force the nature of which we do not yet comprehend, but we cannot conceive a way by which it could be influenced by inheritance of effect. Further, we note, particularly in certain kinds of pronograde monkeys,

tendencies for certain muscles to migrate in a certain direction—tendencies which have become exaggerated and take place regularly in orthograde apes. We see a process, which is tentative and incipient in more primitive apes, become fully effective in the later and more specialized orthograde forms. In such manifestation we have clearly to do with a developmental or growth mechanism; it is one which affects not only the attachments of the upper limbs to the body and the shape of the thoracic walls, but involves a simultaneous alteration of all the structures concerned in respiration—lungs, bronchi, trachea, pleura, diaphragm, the great blood vessels in the heart itself. It is these latter structures which now demand our attention.

EVOLUTION OF THE FLAT BROAD STERNUM.

Amongst the most remarkable changes which took place during the evolution of the orthograde posture was the transformation of the sternum. In the higher pronograde monkeys the breast bone, all save its first part or manubrium, is made up of rod-like segments, usually five in number. In all orthograde primates—the gibbon, orang, chimpanzee, gorilla, and man—the sternal segments ultimately become fused and form a stout, plate-like structure—the short sword sternum. The widening and stiffening of the sternum has greatly strengthened the anterior wall of the orthograde thorax. It is easy to see why the sternum should become strong and plate-like. When the upper limbs become used for the purposes of suspension and prehension greater stresses must fall on the thorax—particularly the parts to which the pectoral muscles are attached. Although the primary reason for the transformation of the sternum is connected with posture and locomotion, such a complete change in the structure of the sternum must have reacted on the mechanism of respiration. It led to the development of upper thoracic or chest breathing.

THE IMPORTANCE OF THE INTERCHONDRAL FACETS.

Often the consideration of minor points of structure will guide the anatomist towards important conclusions. Medical men must often have noted the articulations between the fifth and sixth and sixth and seventh costal cartilages; the upper cartilage throws down an elbow, which comes into contact with the cartilage below; at the point of contact there is a synovial facet. In only three animals do we find these elbows of cartilaginous contact—man, the gorilla, and chimpanzee. They are not present in either gibbon or orang; in them the suspensory use of the upper limbs has become predominant in locomotion, and in consequence they have become "pigeon-chested"; nor are they present in pronograde apes or monkeys. We obtain a clue to the functional meaning of the intercartilaginous facets if we consider the mode in which the diaphragm acts in pure thoracic breathing. The diaphragm then exerts its power, not in depressing the abdominal viscera but in raising the anterior or ventral ends of the ribs to which it is attached—namely, the lower six. Through the cartilaginous heels on the fifth and sixth cartilages its power is extended to the solid plate-like sternum, which it also raises. As the sternum is elevated it will be found that a bending or flexion takes place at the joint between the body of the sternum and the manubrium or presternum. Further, we note in the highest of the orthograde primates, particularly in the human thorax, that the first pair of ribs and manubrium become more and more welded together to form a single functional structure, for which I have proposed the name of "thoracic operculum." The thoracic operculum is set obliquely; it is hinged behind to the first dorsal vertebra; it rests in front on the body of the sternum; it is almost stationary if the respiratory movements are purely of the abdominal type, as in pronograde apes; it becomes a freely moving structure when respiration is of the thoracic type.

RISE IN IMPORTANCE OF THE APICAL REGION OF THORAX.

That the evolution of the orthograde posture gave an enhanced respiratory importance to the upper thorax is seen from another line of evidence. The straight muscles of the abdomen are the flexor muscles of the spine. In the thoracic region they are attached to the ventral ends of ribs; behind, to the ventral or pubic end of the pelvic girdle. These structures serve as powerful spinal levers; through these levers the straight muscles are more than a match for the extensors of the spinal column. Now, in the human body, as in that of all orthograde primates, the straight muscles take hold of the fifth, sixth, and seventh costal cartilages. In the older pronograde

forms the straight muscles extend their origin forwards to the whole length of the sternum and to the first pair of ribs—as is the case in all the more primitive vertebrate mammals. With the evolution of the orthograde posture the origin of the straight muscles was withdrawn from the four upper ribs, thus setting them free for a new kind of movement. This retreat of the straight muscles was accompanied by that of the external obliques and scalenus medius. Thus all the structural transformations which occurred during the evolution of the orthograde posture led to the freedom and increased respiratory importance of the apical region of the thorax. We shall see that these changes in the walls of the thorax were accompanied by corresponding modification of the apical region of the lung.

POSTURAL CHANGES WITHIN THE THORAX.

Up to this point our attention has been concentrated on the modifications of the thoracic wall involved by the change from a pronograde to an orthograde posture. We are now to enumerate, as briefly as possible, the structural alterations which took place within the thorax. The dissection which best suits our purpose is one often practised by human anatomists; extensive windows are cut in the lateral walls of the chest and through these the root of each lung is exposed, then cut through and the lungs removed. The mediastinal wall is thus exposed, and we can note the exact form of each pulmonary cavity. When such a dissection is made of one of the commoner pronograde monkeys, we are at once struck by the oblique position in which the diaphragm is set; its domes slope from the lower end of the sternum towards the eleventh pair of ribs. If we make a similar dissection of a gibbon we find the diaphragm arranged much as in man; the central tendon has become cordiform in shape and the domes of the diaphragm nearly horizontal in position. The flattening of the dorsal and ventral wall of the orthograde thorax has altered the contour of the diaphragm, giving it a wide cordiform shape. The chief structural changes concern the muscular fibres of the crura of the diaphragm. In pronograde monkeys the crural or spinal fibres are widely separated from the costal fibres by an interval filled by a fibrous membrane. In the gibbon, as in all orthograde forms, the fibrous interval is obliterated by the outward migration of crural fibres; the origin of the outer fibres has become shifted from the bodies of the upper lumbar vertebrae, over the sheaths of the psoas and quadratus lumborum muscles. We have here another example of muscular migration playing a part in an evolutionary movement.

Fixation of the Heart.

When we compare the thoracic dissections of the monkey and gibbon we see that the orthograde posture has entailed an alteration in the position and also in the fixation of the heart within the thorax. The point which particularly catches our attention in the monkey's thorax is that the inferior vena cava, instead of at once entering the pericardium on appearing above the diaphragm, as it does in man and in all orthograde apes, has a course of some two inches before it enters the base of the heart. Only the apex of the pronograde heart comes in contact with the diaphragm. Between the heart and diaphragm passes a sinus or diverticulum of the right pleura; the mouth of this subcardiac sinus lies internal, or medial, to the inferior vena cava; it is occupied by the azygos lobe of the right lung. When the diaphragm contracts the azygos lobe is distended, this inspiratory descent being accompanied by a stretching of the highly elastic inferior vena cava. In the gibbon the heart is bound more closely to the diaphragm, but an azygos lobe and a reduced subcardiac sinus are clearly recognizable. In the great anthropoids and man the base of the heart has become firmly bound to the diaphragm; in all of them a vestige of the azygos lobe of the right lung and of the subcardiac sinus are unmistakably present. There is no better proof of man's pronograde ancestry than the evidence he carries in his right lung.

Development of the Apical Region of the Lung.

As the orthograde heart settled on the upper surface of the diaphragm it lost the tapering form seen in pronograde animals, and assumed the blunt conical form we are familiar with in man's body. But why does the base of the heart become so closely attached to the diaphragm in orthograde forms? It is because of the increased development of the apical region of their lungs; the suprardicular parts of the lungs can be thoroughly aerated only if the roots of the lungs

participate in the respiratory movements of the diaphragm. The heart and pericardium are fixed to the roots of the lungs; in orthograde forms the pericardium becomes bound to the diaphragm.¹ Thus with the evolution of the great anthropoids there came about a new relationship between the diaphragm and roots of the lungs; the central tendon of that muscle became yoked to the roots of the lungs through the pericardium. In the chest of man this relationship is even more intimate than in anthropoids. For the proper aeration of the apical part of man's lungs diaphragmatic movements are essential.

Other Respiratory Changes.

It would take us too far afield were we to analyse all the intrathoracic changes which mark the evolution of the orthograde posture. The arch of the aorta and the manner in which it gives off its great branches became altered; the human arrangement is linked on to the pronograde type by a series of intermediate stages seen in the great anthropoids. The lung assumed a conical shape, with the wide and hollow diaphragmatic base with which we are familiar in human anatomy. The fissures of the lung become less deep; they have almost disappeared from the lungs of the orang. The apical lobes become larger, the inferior lobes, particularly the parts which fill the hinder and lower recesses of the pleural cavities, smaller. The distribution of the bronchial tree was changed, the apical supply becoming increased.

SUMMARY.

It will thus be seen that the evolution of the orthograde posture was attended by a structural revolution in all those parts which are concerned in setting up a tidal flow of air to the lungs. Ribs, vertebrae, sternum, body wall and spinal muscles, diaphragm, pleurae, lungs, and heart underwent a simultaneous and harmonious adaptational transformation. To account for such a structural revolution we must postulate a much more elaborate mechanism controlling developmental processes than any we have knowledge of as yet. Further, it is evident that the square shoulders, the flat back, the wide chest, the cordiform diaphragm, we are familiar with in man's body, are of ancient origin, having been evolved with the appearance of the earliest of orthograde primates.

REFERENCES.

¹T. Wingate Todd: *Anat. Anz.*, 1912, vol. 41, p. 337. ²For data relating to these age changes in shoulders and thorax see Wingate Todd, loc. cit.; C. B. Rodes, *Zett. f. Morph. u. Anthropol.*, 1905, vol. 9, p. 103 (Wintrich's observations are here included); E. Mehnert, *Ueber topographische Alterveränderungen des Atmungsapparates*, Jena, 1901. ³For fuller details see Keith, *Lancet*, 1903, i, p. 622; 1904, i, p. 559.

The Goulstonian Lectures

ON

THE NATURE OF ARTERIO-SCLEROSIS.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS
OF LONDON

BY

GEOFFREY EVANS, M.D.CANTAB., F.R.C.P.LOND.,
ASSISTANT DIRECTOR, MEDICAL UNIT, AND ASSISTANT PHYSICIAN,
ST. BARTHOLOMEW'S HOSPITAL.

LECTURE III.—CAUSATION OF DIFFUSE HYPERPLASTIC SCLEROSIS.*

THE description of diffuse hyperplastic sclerosis formed the subject of the first lecture; the process concerned in its production and the opinion that it was one of inflammation was presented in the second; the matter of its causation remains for consideration.

Since diffuse hyperplastic sclerosis has not been produced experimentally only clinical evidence on causation is available, and before this is discussed the clinical signs of diffuse hyperplastic sclerosis need to be established. The clinical diagnosis is nearly always a matter of inference, since there is rarely the opportunity for microscopical examination of a piece of tissue; though the study of retinal vascular disease holds out the prospect of the accurate diagnosis of diffuse hyperplastic sclerosis during life the correlation of ophthalmoscopic findings with histological lesions is not as yet complete. The clinical picture can be established, however, on the basis of the naked-eye *post mortem* indications of the presence of the lesion.

NAKED-EYE POST-MORTEM EVIDENCE OF DIFFUSE HYPERPLASTIC SCLEROSIS.

The identity of diffuse hyperplastic sclerosis as an affection of the larger vessels has not been established, hence it can only be recognized by microscopical examination of tissues cut fresh or suitably embedded to retain their fat. Its presence is inferred from the appearance of thickening of the smaller arteries, particularly of the kidneys and spleen, and the vessels are seen to protrude like little quills (in the words of Hilton Fagge) on the cut surface of these organs. When there has been a raised blood pressure during life the walls of the larger arteries such as the splenic and iliac arteries are obviously thickened, and the hypertrophied media shows as small transverse ridges on the inner surface of the vessel wall.

Cardiac hypertrophy, especially affecting the left ventricle, when not due to disease of the heart and lungs, is termed "idiopathic hypertrophy" for the sake of present convenience, and it is the most important evidence, though not proof, of diffuse hyperplastic sclerosis. Its significance in this respect has been tested by two lines by inquiry. In the first place, an analysis of twenty consecutive years of medical *post-mortem* records of St. Bartholomew's Hospital¹ showed that idiopathic cardiac hypertrophy of more than 15 oz. weight was significant of cardio-vasculo-renal disease. Thus the records of the great majority of cases with idiopathic hypertrophy showed death due to cerebral haemorrhage, heart failure, or uraemia; or when death was due to other cause the records made reference to arterio-sclerosis or some form of renal sclerosis. The few exceptions included a small group of cases of pernicious anaemia (Cabot has also called attention to cardiac hypertrophy in this disease), a few cases of alcoholism,² and obese heart. In the second place, I have found diffuse hyperplastic sclerosis in all cases of idiopathic hypertrophy that I have examined microscopically. The hypertrophy of the heart and media of the arteries is to be regarded as a compensatory phenomenon, physiological expressions of increased activity during life. The only essentially pathological sign of diffuse hyperplastic sclerosis is renal sclerosis in one of its several forms—a matter that will be referred to later. In short, the essential *post-mortem* picture of diffuse hyperplastic sclerosis is limited to the cardio-vasculo-renal system; it consists of left ventricle hypertrophy, the appearance of medial thickening, and renal sclerosis. This picture is often dominated by one of the complications or sequelae of vascular disease, such as cerebral haemorrhage, thrombosis, heart failure, or septicaemia; it may also be one aspect of a coincident condition, such as nephritis when death is due to uraemia, and it is often overlooked when death is due to an intercurrent disease such as pneumonia or cancer.

CLINICAL PICTURE OF DIFFUSE HYPERPLASTIC SCLEROSIS.

Post-mortem evidence establishes the clinical diagnosis of diffuse hyperplastic sclerosis on idiopathic cardiac hypertrophy and renal sclerosis. The clinical diagnosis of the former is not without difficulty, since heart disease, particularly myocardial disease, is often difficult to exclude. From a comparison of blood pressures taken during life and heart weight determined after death a close relation has been established between raised systolic pressure and idiopathic hypertrophy. The direct relation shown in my first paper on arterio-sclerosis¹ has not been borne out by a series of 32 cases collected since. The two series are not, however, directly comparable, since the second series was merely compiled from clinical notes and *post-mortem* records. Other factors than mere increase in blood pressure are concerned in determining the degree of cardiac hypertrophy. Of the 32 cases of idiopathic hypertrophy that form my second series (heart weight more than 15 oz.) the systolic blood pressure during life was 180 mm. Hg or over in 84 per cent., and was 170 mm. Hg or over in 94 per cent. These cases furnish further evidence of the association of raised blood pressure and idiopathic hypertrophy, which is already generally accepted. In choosing an arbitrary figure of systolic pressure as having a pathological significance it is very necessary to take a conservative line, since the blood pressure is liable to considerable variation in health,³ and in conditions of fatigue, excitement, pain, dyspnoea, and the like, but my experience has been that the systolic pressure does not reach 180 mm. Hg under such conditions; and having regard to the relation of diffuse hyperplastic sclerosis on the one hand, and of a systolic blood pressure of 180 mm. Hg to idiopathic

* Abstract of lecture delivered on March 13th.