venes, ice to the head, revulsives, sinapisms or turpentine stupes to the legs and trunk, and stimulating enemata, in which thirty grains of quinine may be combined, or a few leeches to the mastoid processes, may be useful. Blisters are sometimes applied. Under the influence of quinine the fit may terminate like ordinary ague. Stertor, congestion, coma with high temperature, suggest active measures, but not of a depleting character. When there are gastralgia and vomiting, I have found opium afford great relief. Time does not admit of dwelling on numerous other antiperiodics; none are comparable to cinchona and its alkaloids. The mixed alkaloids now prepared in India from cinchona grown in the plantations at Darjeeling and other hill stations, have been submitted to trial and found very efficacious, though certain objections were made that they caused nausea; it seems as though this and other objections are not so serious as at first imagined, and that the drug will come into general use. Though economically it may have advantages, it will not supersede the sulphate. Dr. De Vry brought this preparation to notice. Dr. Verckhuysen says quinetum is of great value as a febrifuge, but takes longer to act, and will not replace quinine in pernicious fever. It has the same apyretic effect as quinine, but is less powerful; larger doses are, therefore, required at longer intervals before the paroxysm than quinine. It produces no unpleasant effects, no noises in the ear, and can be taken by those who cannot take quinine. It is more efficacious in chronic cases as a tonic, whilst in masked malaria it is incomparably superior to quinine. Arsenic is a valuable febrifuge and antiperiodic. When quinine does not succeed, arsenic sometimes will do so, given in doses of five drops of the liquor arsenicalis. Care must be taken not to continue its use until symptoms of gastric irritation are set up. In the treatment of the early conditions of malarial fever I have never found it equal to quinine; but in cases of chronic malarial poisoning, with frequent returns of fever, neuralgia, or other indications of the chronic action of malaria, I have seen benefit arise from the continued use of arsenic in small doses—three or four drops of the liquor potassæ arsenitis twice a day, after food. The antiperiodic powers of opium are probably the chief reason why opium-eating and smoking has become so widely-spread a habit in China and India. There is little doubt that it does possess such a power, and that in the earlier stages it gives great relief; it relieves pain, soothes and breaks or stops the periodic return of fever; and it seems to assist those exposed to malarial influences in resisting them; it has been used for this purpose since the time of Galen. Trotter, Lind, and others, in the last century, prescribed it, and there may be cases where it might be expedient to use it now; it would probably be hurtful during the hot stage, yet in the cold and sweating stages it might be beneficial. Waring says he has seen it cut short the cold stage like a charm, and mitigate the severity of the following hot stage. I have had no experience of it as a febrifuge, and as there are so many others that would better fulfil the purposes required, except in intercurrent conditions, which might complicate malarial fever, I should not resort to it. Many other drugs are spoken of both in the officinal and native pharmacopœia, but they are inferior to the cinchona alkaloids; arsenic, gelsiminum, biberine, salycine, strychnine, atees (aconitum heterophyllum), piperine, ilicin, bonduc nut (fruit of *Cesalpinia Bonduchi*), salts of iron, zinc, picric acid, the mineral acids, and a variety of native drugs—the hyposulphites and alcohol. These, or some of them, especially iron, may be of service in certain stages of the fever, or in the cachexia following it. Atees is much used in native practice, and is a valuable drug, as an antiperiodic, tonic, or as combined with gentian, chiretta, or other vegetable bitters, but it can in no way take the place of quinine, quinetum, or arsenic. I may not omit to mention the tincture of Warburg. I have never found it better than quinine, though it certainly possesses febrifuge and diaphoretic properties. Dr. Maclean and others speak highly of it, and as its composition has been declared, the objections to its use have been removed; but I must leave it with this brief notice. In the treatment of malarial cachexia, with enlarged liver and spleen, the most important step is change of climate, the judicious use of preparations of iron and quinine (sulphate is best), and attention to the state of the portal system. I do not mean excessive purgation, but gentle action by salines combined with quinine and vegetable bitters. Carefully regulated and nourishing diet, and protection from all vicissitudes of climate must be enjoined. In such cases benefit may be derived from the saline and ferruginous waters of Germany, and from measures that tend to improve the general health. I may add that a prolonged absence from the country in which the mischief originated is necessary. In conclusion, I would remark that though mercury is especially to be deprecated in the treatment of disease connected with malariously enlarged spleen, local application of the ointment of red iodide of mercury, applied as it is done in India for goitre, is often successful in reducing the spleen; and that it does not appear to incur much if any

risk of mercurialism being induced. Professor Maclean speaks favourably of it, and I can endorse the opinion he has expressed. One word in regard to a matter I have omitted. In advanced splenic cachexia the patient should be very careful not to make any exertion; the result may be rapid dissolution with all the symptoms of pulmonary obstruction. The last instance in which this was impressed on my attention was in the case of a young Englishman of eighteen or twenty who had returned from India in a profound state of cachexia—the spleen descending nearly to the ilium, and with all the symptoms of anæmia in the most advanced condition. Under the influence of quinine, iron, and nourishment, he was improving, and there was hope of further progress. One day, in spite of warnings, he rose, walked to the window, and tried to raise it. He went back to bed exhausted and breathless, and died in a few hours.

In my next lecture, I hope to describe some forms of continued fever in India.

THE GULSTONIAN LECTURES ON PULMONARY CAVITIES: THEIR ORIGIN, GROWTH, AND REPAIR.

By WILLIAM EWART, M.D. Cantab., F.R.C.P.,
Assistant-Physician and Pathologist to the Brompton Hospital for Consumption;
Physician to the Belgrave Hospital for Children; Demonstrator of
Physiological Chemistry at St. George's Hospital.

LECTURE III. PART III.—*Concluded.*

OF the alterations in shape suffered by cavities, I have already spoken at sufficient length. The relations of cavities to surrounding parts are also subject to variations. When superficial, and when at the same time closely adherent to the chest-wall, cavities are only susceptible of very limited alterations in their position; but the intervention between them and the pleura of a moderate thickness of spongy tissue enables them to undergo a gradual but decided displacement. This shifting of cavities, as the result of age, takes place in a determinate direction. In all cavities the natural tendency is to a recession towards the root of the lung; and the compensatory development of the healthy portions of the lung usually assists by substitution the normal retraction. Thus, I have sometimes found in the mid-dorsal region, connected with a thickened and shortened bronchus, the remains of a cavity which had obviously retreated from the surface, under the influence of the super-inflation of the lung-tissue which had grown up, as it were, around it. The retraction upwards of cavities at the base is often very striking, but greater practical interest belongs to the history of cavities at the apex.

The direction of the *shifting* of the apex-cavities is remarkably constant. Unless rigidly fixed by adhesions, they invariably tend to retreat from the front of the chest towards the back, and from the sub-clavicular into the axillary region. Consequently, the chronic cavities, frequently detected after death at the outer apex, should not be taken to have necessarily originated there. The remarkable shifting to which I have alluded was pointed out by Dr. C. T. Williams, in his lectures on the Various Modes of Contraction of Cavities in Phthisis Pulmonalis (*Lancet*, 1873, vol. i, pp. 298 and 369). I fully agree with the view which he expresses concerning the special mechanism at work. It cannot be doubted that, on the right side, the retraction is mainly favoured by the great expansion of which the middle lobe is capable. I would not, however, restrict this action to the right middle lobe. Every portion of spongy tissue in the sternal region is possessed of similar capabilities for expansion; and, on the left side, the ultimate results are not far behind those observed on the right.

The recognition of the changes to which I have alluded has a direct bearing, not only upon our diagnosis, but upon the prognosis which we may form in individual cases. Unless we have carefully examined the upper axillary region, we cannot pronounce a chest to be free from excavation. The signs of disease have sometimes entirely disappeared from the anterior aspect of the chest, when cavernous sounds are still plainly perceptible in the axilla. The discovery of a vomica in this situation frequently throws upon the history of the case a clearer light than is derivable from the patient's own statements. On the other hand, in the early stages of subclavicular disease, the rapidity with which the signs are observed to recede may be accepted as a measure of the favourable chances. From this rapid shifting, we derive an

assurance that the vomica has ceased to extend, that its contraction is active, and that the lung is sufficiently free from adhesions to acquire the compensatory development which is the first essential towards recovery.

The changes in the position of cavities which I have indicated are usually bound up with very definite alterations in the configuration of the lungs. Upon this third result of the contraction of cavities, I would dwell at some length, finding that hitherto the changes in question have received but little attention, although hinted at by Laënnec, and pointed out by my colleague, Dr. Douglas Powell. The retraction which has been shown to take place in the line of the bronchus continued, leads, unless rigid fixation have resulted from adhesion, to a dragging inwards of the surface of the lung at a corresponding point. Thus a deep puckering found at the surface of the lung after death, is often an indication of a subjacent cavity. Similar puckerings may, however, result from a variety of circumstances. The fibrin of an infarct, syphilitic fibrosis, or the common fibro-tubercular masses, will all induce a retraction of the surface; and it may be here noted that those cavities which are formed at the expense of hæmorrhagic deposits occur, as pointed out by Dr. R. E. Thompson, subsequently to the original retraction. This does not, in my estimation, preclude a subsequent contraction of the cavity and a secondary retraction; and, in accordance with this view, it is customary to find the highest degree of retraction over those cavities which can be traced to hæmorrhage.

Minor differences can be pointed out between the retraction due to cavities and the puckering from other causes. The puckering due to tubercle or to hæmorrhagic nodules (exemplified in the specimens under your inspection) consists in a shallow depression of the surface over which the pleural membrane is roughened by irregular and rigid wrinkles. These puckers are determined by the peripheral position of the consolidation, and by the concentric mechanism of its contraction. Cavities, on the other hand, being drawn in towards the root of the lung, give rise to a slit-like infolding of the surface, invariably deeper than the hæmorrhagic pucker. As an extreme instance of this condition, I submit to you a photograph, and the specimen from which the photograph was taken. The lungs were removed from the body of a young man who died of chronic phthisis, under Dr. C. T. Williams, at the Brompton Hospital. When first admitted, in November 1874, he was puny and delicate for his age (then fourteen years). He had suffered from cough for twelve months, but inherited no predisposition to phthisis; neither was there any history of hæmorrhage, nor of any important disease. Diffused crepitations were the only signs audible in the chest. In January 1875, however, a marked cavernous gurgle was heard in the right subclavicular and subscapular regions. When discharged, in April of the same year, he had much improved in weight and in general appearance. The phthisical sounds, however, still indicated the presence of excavation at the right apex, coupled with creaking sounds in the right scapular region; and, at the left apex, bronchophony was noted in addition to crepitation. He appears to have enjoyed comparatively good health until six months prior to his second admission, in February 1881. On this occasion, dulness and humid crepitations were present at both apices externally; and, at the back, cavernous sounds were found between the spine and the right scapula. Anteriorly, dulness and humid crepitations occupied the upper half of the left lung and the upper third of the right. The patient died with symptoms of dyspnoea, soon after his admission. The specimen presents more points of interest than I can venture to enumerate. I would call your attention incidentally to the remnants of the old apex excavations remarkably fibrosed and contracted, and to the presence at the left outer apex of a cavity of emphysematous origin, such as I have described to you in my first lecture. The most remarkable feature, however, is the puckering of the lung-surface over the contracting cavity, especially the deep antero-posterior involution of the base of the left lung (Fig. 9). The visceral pleura can be clearly followed along both sides of this V-shaped incisure (measuring two inches and three-quarters in length), the summit of which is continuous with a thick fibrous scar two inches and a quarter in length, and one-third of an inch in thickness, which it is difficult not to consider as resulting, partly at least, from the cicatrization of a cavity. This scar terminates above in a thick-walled fibrous cavity. I believe this cavity to have originally extended to within a short distance of the middle of the base, and by its gradual healing to have caused the extreme retraction you witness, whilst the healthy tissue on either side became expanded in a proportion nearly equal to the loss incurred by involution. The elongation of the left lung in consequence of the flattening from side to side should also be noticed.

Involutions of such large extent are extremely uncommon; they are most frequently due to the retraction of cavities. This, however, is far from being the only source of a recession of the pulmonary surface.

The scars very commonly found at the apex in old age, are often independent of any deposit, and due to a simple infolding of the lung-surface, which may be considered as a senile change. I look upon this variety of involution simply as an indication of distributed proportion between chest space and bulk of lung.

Fig. 9.—Involution directly induced by Retracting Cavity. The Involution at Base roughly corresponds to description given in text.

Whenever the thorax is too small for a lung, or the lung abnormally large for the size of the thorax, the superfluous surface must be reduced by involution; and a "reef taken", as it were, in the visceral pleura. In emphysema, involution is never absent. We find in this disease some of the most extreme instances of the latter. The large-lunged emphysema, sometimes observed in old age, leads to essentially similar conditions. The thorax shrinks relatively earlier than the lung; and the unsubstantial pulmonary expansion, kept up under the influence of chronic bronchitis, necessitates an adjustment by means of involution. Similar changes are apt to occur in youth, and even in childhood; they are here almost invariably due to alveolar collapse, with subsequent collateral emphysema. But I have more specially referred to the senile conditions, because in them we possess the simplest illustration of the changes which so commonly result from excavation.

The flattening of the upper part of the chest, induced by excavation and fibrosis of the upper lobe, can seldom take place without some degree of flattening of the lower thorax. The base of the lung, which is usually the seat of compensatory distension, becomes, relatively to the thorax, hypertrophied; and involution follows after the simple mechanism which I have described.

Fig. 10.—Flat Chest. Contraction of Cavity, with Fibrosis at Left Upper Lobe. Transverse Involution at Left Base from Compensatory Hypertrophy of Lower Lobe.

The shape of this involution is governed by the shape of the thorax. If the latter be flattened from front to back, the lung will become affected with *transverse involution*; if from side to side, the sulcus of involution will follow an *antero-posterior* direction. In both these instances, the horizontal section of the base is lessened in diameter; and, the divergence of the opposite sides of the lung being diminished, the lung tends to elongate.

Occasionally, the folding at the base occurs in several diameters; and the lower surface of the lung, which is usually, under these circumstances, adherent to the diaphragm, is hollowed out into a *dome-like involution*. (This condition is imperfectly depicted in Fig. 12.)

The chief importance of the secondary changes which I have described lies in the loss of breathing surface which they occasion. Involution of the pulmonary surface, and the loss of respiratory tissue incidental to it, are capable of being promoted by artificial interference, especially where pleurisy co-exists. Respiration is influenced by pleurisy in a very definite manner. The natural excursions of the ribs are reduced; the lessened amplitude and power of the diaphragmatic contractions shorten the range of vertical displacement of the lung; and the thorax, as a whole, for the avoidance of pain, assumes a position almost of expiration. Adhesions being favoured by comparative rest, and completed at this stage of imperfect expansion, the base of the lung, in a slightly collapsed and infolded state, becomes connected with a restricted surface of the convexity of the diaphragm. I consider it probable that this tendency may be materially encouraged by the mechanical appliances so often used in the treatment of pleurisy; and that the respiratory excursions may become shortened even beyond natural and instinctive limits. The ultimate results on the outline of the lung are readily perceived. The contraction and the descent of the diaphragm cannot take place without inducing a folding or involution of the basic surfaces. On the other hand, the adherent fringe of the lung is not at liberty to glide into its accustomed groove; but, whilst it becomes curved inwards, its place is supplied by a lateral expansion of the side of the lung. Pleurisy at the base is an extremely common event in phthisis; and it often intensifies in the manner which I have described the involution arising from the contraction of cavities, and from compensatory hypertrophy.

Fig. 11.—Narrow Chest. Fibrous Shrinking of Upper Lobe around a Contracting Cavity. Antero-Posterior Involution at Base from Compensatory Hypertrophy of Lower Lobe.

The rough outline which I have given of this subject, would not be complete without a brief allusion to a form of involution indirectly due to excavation. Great shortening cannot take place in one lung, as a result of the contraction of a cavity, without occasioning some degree of shortening in the other lung. Especially striking is this rise in the

Fig. 12.—Short Chest. Extensive Contraction of Right Lung: Basic Involution, with Invagination of Diaphragm. Secondary Shortening and Involution affecting the Hypertrophied Left Lung.

main level of the diaphragm, whenever the latter, as depicted in the diagram, has become invaginated into the lung—an occurrence of which I have seen a few instances, and which would naturally result where

basic involution had been preceded by intimate adhesion between the lung and the diaphragm. In cases of this kind, the sound lung, whilst it expands in breadth under the influence of compensatory hypertrophy, is restricted as to vertical space. This leads to the formation of a transverse involution, which often occupies the middle third of the axillary surface, and which, as it were, accommodates the sound organ to the curtailed dimensions of its fellow. The interest of the change which I have just described lies in the fact, that the results of excavation are here visited upon the healthy lung. *Ceteris paribus*, the shortening of the hypertrophied lung will favour its encroachment into the diseased side of the chest.

From what has been stated in this lecture, concerning the ascent of the diaphragm in cases of vertical contraction of cavities, and concerning its depression, where the contraction, being horizontal, tends to narrow and elongate the lung, it is easy to divine the direction and extent of the secondary displacements of the abdominal organ. Upon this part of my subject, I need not further dwell; but I would direct your attention to the more important alterations which occur in the position and in the shape of the thoracic organs, and especially of the heart and large vessels.

The aorta, doubtless, owes to the strength of its walls, and to its close connection with the vertebral column, its immunity from the effects of pressure to which other vessels are subjected. The superficial position of the pulmonary artery, and its comparative thinness, render it especially liable to suffer. The shrinking of cavities at the left apex is almost inevitably followed by exposure of the pulmonary artery, and not infrequently by some degree of pressure upon its walls: the clinical signs of these conditions, when present, supply valuable testimony in favour of the diagnosis of contracting vomica. The large veins accommodate themselves with wonderful ease to the great shifting which they occasionally undergo under the influence of excavation, and we seldom notice any serious complications arising in this direction.

Of much greater interest to the practical physician are the alterations in the position, and in the relations of the heart. An attention to these changes is often rewarded with valuable diagnostic results; and I confidently assert that, in some cases of phthisis, the examination of the heart supplies us with evidence more conclusive than is obtainable from an auscultation of the lung.

As a guide to the existing level of the diaphragm, a determination of the position of the heart-beat is of the greatest assistance. The heart's apex, I need scarcely state, does not always coincide with the strongest impulse, perceptible to the hand; nor does it necessarily occupy, as in health, the lowest position. But, should we succeed in ascertaining, by a combination of the methods of palpation and of auscultation, the exact position of this lowest point, we derive absolute information concerning the height of the diaphragm—a subject of the greatest practical importance.

Again, the extent of the exposed cardiac area sometimes throws much light upon the degree of expansion or of retraction of the lungs. Excavation occurring at the left upper lobe, especially in its anterior segment, is apt to be followed by retraction of the spongy substance which normally covers the greater part of the heart's surface. Excavation, in a similar position, on the right side, although it may lead to exposure of the right side of the heart, is usually accompanied by the encroachment of the left sternal fringe over the normal cardiac space, and the presenting cardiac surface is not as a whole increased.

Where the excavation, instead of being anterior and superficial, extends into the depth of the lung, and involves some of the bronchi which are distributed towards the shoulder, and where, moreover, the pericardial surface is adherent to the lung, considerable displacement of the heart outwards takes place on the side affected.

It is obvious that the same contraction will lead, on the left side, to a lateral position, much more extreme than is possible on the right; and that it will also raise the heart to a higher level than follows from right-sided disease. This was long ago pointed out by Dr. C. T. Williams, in his lectures on the contraction of cavities. The specimens and the photographs before you carry their own explanation; you will find in them illustrations of extreme dislocation of the heart towards the left.

The displacement of the heart towards the right is less frequently described, not owing to a less frequency of the apex excavation, but rather owing to the fact that compensatory hypertrophy of the right middle lobe usually supplies in the right half of the chest the place of the tissue destroyed; and also, from the circumstance that adhesions of the right lung to the pericardium are less common than happens on the left. I have frequently met with this displacement; it is well shown in the photographs submitted to you, where I have roughly mapped out the area of the displaced heart and the position of the cavities which have induced dislocation.

Time forbids that I should attempt a detailed description of the rotation which accompanies lateral displacement of the heart, and of the mechanism to which the rotation is due. For a full treatment of the subject I would refer you to Dr. Sibson's article on the Position and Form of the Heart and Great Vessels in Dr. Russell Reynolds's *System of Medicine*. From the special standpoint of the healing of cavities I would, however, introduce slight modifications in the account there given of the mode of production of this change. A purely secondary influence is to be ascribed, in my opinion, to the aorta, to the pulmonary artery, and to the superior vena cava, in determining a rotation to the left in left lateral displacement of the heart, and to the right in right lateral displacement. The relative length of these vessels enables them rather to suffer than to exercise the influence in question. Within the pericardium, which limits its movement in a very definite manner, the position of the heart is controlled by three attachments closer than all others. I refer to the pulmonary veins on either side, and to the intrapericardial portion of the inferior vena cava. The shortness of these trunks occasions at the base of the heart the same movements which the vessels themselves undergo. The influence exercised upon the heart by a retraction of one or of the other lung is comparable, in direction, to the movement imparted to a heavy upturned sack in emptying its contents by a tilting of its corners. At the corners are the right and the left pulmonary veins, which, as they recede with the lung, tilt the heart towards the one or the other side; but the resulting movement is modified by the other cardiac attachments in a manner which cannot be understood without referring to a special case. Let us take the contraction of a cavity in the left upper lobe; owing to the retraction of the right lung upwards, outwards, and backwards, the left pulmonary vein raises and draws backward the left auricle, whilst the relatively forward position of the root of the right lung, and the high level of the diaphragm and of the orifice of the inferior vena cava, loosen the reins for the right auricle, which readily, with its ventricle, rotates towards the left. Similarly, if the right auricle be raised, the rotation towards the right will be favoured by the looseness of the attachment of the left auricle; for it should not be forgotten that retraction of the root of one lung is almost invariably accompanied by an advance of the sound lung towards the middle line. In this movement of lateral displacement the "hinge" of the heart appears to me to coincide, not with the line drawn between the upper and the lower venæ cavae, as suggested by Dr. Sibson, but rather with the lines passing through the inferior vena cava and the right and left pulmonary veins respectively.

These remarks must conclude the subject of the anatomical results of the contraction of cavities, which, in common with the other parts of these lectures, I can only pretend to have roughly sketched. The small measure of what has been achieved would have been yet smaller but for the helpful friendship of many. For the loan of specimens and of drawings my thanks are due to Dr. Andrew Clark, to Professor Marshall, and to the authorities of St. George's Hospital; for the elaboration of diagrams, to my friend, Mr. A. H. Hallam Murray, to Mr. Francis Compton, to Mr. Arthur Grayling, and to my predecessor as Pathologist at the Brompton Hospital, Dr. Reginald E. Thompson; for much encouragement to all my colleagues, and specially to our senior physician, Dr. Symes Thompson, and to Dr. Pollock, by whose recent elevation to the consulting staff we lose more than it befits me to say; and, lastly, for untiring aid, to my brother, Mr. Charles Ewart.

In bringing these lectures to a conclusion, I feel that they have contained little information which can be recapitulated in the shape of propositions. The difficult study of cavities has repaid me rather in suggestions than in facts. These suggestions I have attempted to lay before you. The origin of cavities and their progress, for better and for worse, and the influences which they exercise and which they suffer, have supplied the humble material for my demonstrations. Of "treatment and of cure," which are the aim of all our labours, I have refrained from speaking, remembering that, before we attempt to guide the forces of nature, we should clearly understand the ways in which they are put forth. But I venture to express a confident hope that the treatment of pulmonary cavities may look forward to a better future if it seeks its foundation in a sound pathology.

The method which I have adopted in my endeavour to gain a clearer insight into phthisis, has been based upon a study of functional relations. I am conscious of the meagre results which I have obtained, and of the great amount of work which remains unachieved. But if I have in any way contributed to the adoption of a plan of investigation which may become productive in other hands, these lectures, and the indulgent attention with which you have received them, will not have been in vain.

ABSTRACT OF LECTURES ON THE MORPHOLOGY OF THE MAMMALIAN SKULL.

Delivered at the Royal College of Surgeons of England.

By W. K. PARKER, F.R.S.,
Hunterian Professor of Anatomy in the College.

LECTURE VIII.—THE VISCERAL ARCHES IN THE AMPHIBIA AND ABRANCHIATA: THEIR MODIFICATIONS IN RELATION TO THE SENSE-CAPSULES.

IN Fishes, there is no secondary opening of the infero-lateral face of the auditory capsule to form a fenestra ovalis; consequently, there is no part that can be called a *stapes* or auditory operculum. This specialisation begins in the Urodeles, in the majority of which there is only a small cartilaginous nucleus, which fits into the fenestra. Proteus retains the most archaic condition; the stapes becomes an oblong bone like the bowl of a spoon, and it is connected with the hyoid arch by a thick mass of ligamentous fibres. Below, this ligament is attached to a thick epihyal, which remains soft; but the cerato-hyal ossifies as a phalangiform bone, and has no separate hypohyal. The stapes itself is a rudiment of the pharyngo-hyal.

In Menobranchus, Siredon, and most of the Caducibranchs also, there is no epihyal, and only one nucleus answering to the pharyngo-hyal; but in Cryptobranchus, Menopoma, Spelerpes, and Desmognathus, there is a second anterior nucleus related to the auditory capsule; and in the large types there is a separate epihyal, which coalesces with the suspensorium. The epihyal also exists in Siren. The second pharyngo-hyal nucleus is generally ossified at its proximal narrow end by the ectosteal growth of the first or stapedial plate; but in *Desmognathus fuscus* the two are separately ossified. The stapes is formed some weeks before the other piece, which is the *incus*; together these form a columella, which is an anticipation of that seen in the Sauropsida.

In most kinds, the distal end of the basi-branchial remains after metamorphosis as a median thyro-hyal, as in birds, supporting the larynx.

The Urodeles early close their first cleft. Thus they have no tympanic cavity; only the hyoid arch assists in forming the ear-chain. They have no *annulus*. In such forms as Cryptobranchus and Menopoma, the segmentation of the hyoid arch is almost exactly like that of the two first branchials of Acipenser; viz., two pharyngo-branchials, one epibranchial, and one cerato-branchial; but in these Urodeles the epihyal is broken up into three pieces, and there is no basi-hyal.

In some Anura—viz., Bombinator, Pseudophryne, and some kinds of Phrynisus—there is only one upper segment of the hyoid arch—a pharyngo-hyal or stapes; and the cleft is nearly suppressed, even inside. The annulus tympanicus is generally absent or quite rudimentary in these forms. In the most typical and perfect species of Rana, there are a large tympanic cavity, and an annulus, the horns of which are confluent above to form a perfect ring. The stapedial chain is composed of four segments—stapes, interstapedial, medio-stapedial, and extrastapedial; the latter having an ascending process—the suprastapedial—which is generally confluent with the skull inside the emerging seventh nerve, as is also the epi-cerato-hyal band. In some cases, however—viz., in Phyllomedusa and Acris—the latter coalesces with the short pedicle of the suspensorium. A little bony deposit appears on the stapes, but none on the interstapedial. The medio-stapedial becomes quite bony, and sometimes the extrastapedial (*e.g.*, in *Rana pipiens*) ossifies also.

The short primary hyoid elongates into an epi-cerato-hyal band; the basi-hyal and basi-branchial are confluent in the adult, and the narrow hypobranchials become the thyro-hyals. The other arches, outer and inner, are absorbed. The stapes of the common frog appears when the tadpole is a little more than half grown; the rest of the auditory chain arises as a single rod at about midsummer; by autumn, the other segments are formed. Here, again, the posterior pharyngo-hyal nucleus is the first to appear, being the most important. In Pipa, everything is finished before they emerge from the maternal pouches; and in Pseudis the shaft of the columella appears before transformation is finished. In no Amphibian is there a fenestra rotunda.