Percussor stethoscope / by B. Wills Richardson.

With B. W. Richardson's Comp.
2 North Frederick St
Dublin

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ART. X.—*Percussor Stethoscope.* By B. WILLS RICHARDSON, F.R.C.S.I.

IN the twenty-sixth volume of the Dublin Medical Press, 1851, page 382, I described a form of stethoscope which admitted of being used as a substitute for Dr. Winterich's copper hammer. I considered that an instrument combining both the stethoscope and percussor in one, might be received with favour[a].

The description was not accompanied by an illustration; but as this form of instrument was subsequently approved of and adopted by some of our most practised stethoscopists, I have been induced to publish the annexed drawing of it.

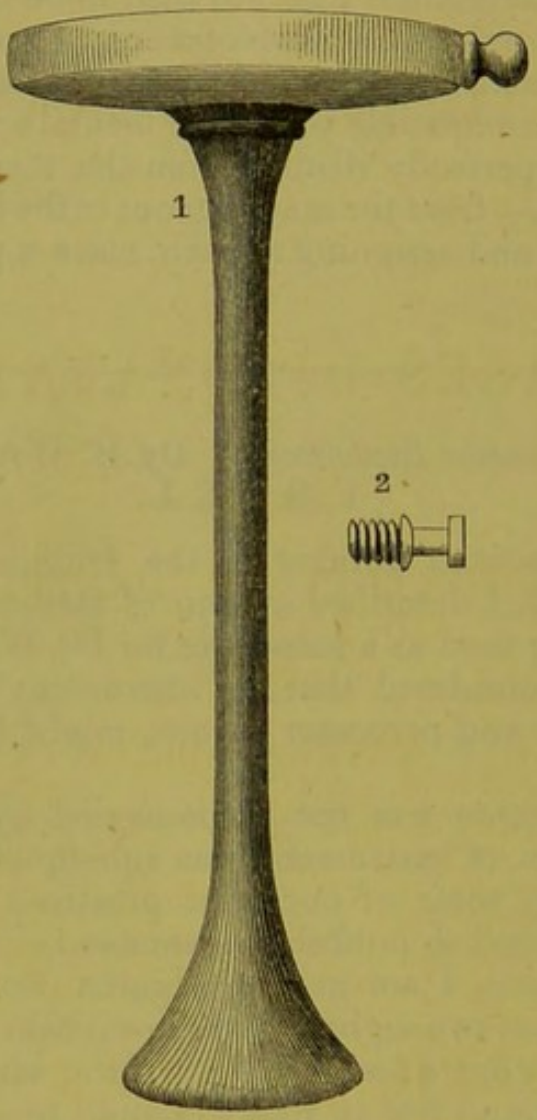
The ear-piece, I am now of opinion, should be made of ivory, from about two-eighths to three-eighths of an inch thick. Into its outer edge a small nipple of the same material, and similar in form and size to Fig. 2, should be screwed and properly cemented. A piece of vulcanized Indian-rubber, of about an eighth of an inch in thickness, and of the same diameter as that of the percussing end of the nipple, should next be glued to this end, and then another piece be drawn tightly over the nipple, and tied securely round its neck. Fig. 1 shows the instrument complete.

Some makers, instead of the nipple, have substituted that part of Dr. Winterich's hammer which contains the Indian-rubber; but I prefer the former, as it may be used with either the pleximeter or naked finger.

It is almost superfluous to say, that the stethoscope should

[a] Sir Henry Marsh was the first to introduce a stethoscope with this combination. His instrument consists of an ear-piece grooved on its outer edge, into which there is inserted a solid vulcanized India-rubber ring; the shank is made of wood.

be well made; for if the percussing parts are not firmly put together there will be a rattle when it is used.



The shank may be formed of wood.

Mr. Read, of Parliament-street, made the instrument from which the engraving is taken.

ON THE USE

OF AN

ARTIFICIAL MEMBRANA TYMPANI,

IN

CASES OF DEAFNESS,

DEPENDANT UPON

PERFORATION OR DESTRUCTION

OF THE

NATURAL ORGAN.

BY

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A COMPLETE TREATISE

ON THE

DIAGNOSIS AND TREATMENT

OF THE

DISEASES OF THE EAR.

F. Shoberl, Printer to H.R.H. Prince Albert, Rupert Street.

PREFACE

TO THE

SECOND EDITION.

Since the publication of the first edition of the present memoir, I have had frequent opportunities of testing the value of the artificial membrana tympani. The result of my own experience, added to that of other medical men (as shown in the cases appended), is confirmatory of its entire success. Further researches have given additional support to the views laid before the Royal Society, on the action of the muscles of the Eustachian tube, and on the functions of the tympanic cavity—views which first induced me to suggest the practicability of the use of an artificial membrana tympani.

18, Savile Row.
March, 1854.

ON

AN ARTIFICIAL MEMBRANA TYMPANI.

It is well known that the partial or complete destruction of the membrana tympani affects, more or less, the power of hearing. In those cases where the orifice in the membrana tympani is small, and the mucous membrane of the tympanum continues healthy, but little inconvenience is experienced: when, however, the orifice is large, and the mucous membrane of the tympanum has become thickened, the capability of hearing is so much impaired that the patient is entirely excluded from the advantages of general conversation.

In the present paper I purpose to describe the results of my attempts to provide a remedy in cases of perforated membrana tympani, by the introduction of an artificial substitute for that important membrane. I shall divide the subject into the following sections:—

I. On the structure of the healthy membrana tympani.

II. On the functions of the membrana tympani, tympanum, and Eustachian tube.

III. On the formation and use of an artificial membrana tympani.

I. ON THE STRUCTURE OF THE HEALTHY MEMBRANA TYMPANI.

In a paper published two years since[1] I demonstrated that the healthy membrana tympani consists of five laminæ, which, commencing externally, are as follows:—

A. The epidermis.
B. The dermoid layer.
C. The radiate fibrous layer.
D. The circular fibrous layer.
E. The mucous layer, with its epithelium.

A. The *epidermis*, as is well known, forms a *cul-de-sac* at the inner extremity of the external meatus, from whose surface it is easily removed by maceration.

1

(Figure 1.) In the course of dissecting, I have more than once observed this delicate epidermis to be the only layer remaining over portions of the membrana tympani, varying from a line to a line and a-half in circumference, and yet appearing sufficient to close the tympanic cavity, so as to render the power of hearing nearly perfect. The knowledge of the fact that this delicate layer is occasionally all that is left of the membrana tympani, ought to induce caution in the use of the syringe, as its application may cause a rupture of the epidermis.

[1] On the Structure of the Membrana Tympani in the Human Ear. (Phil. Trans., Part i., 1851.)

B. The *dermoid layer*, as its name implies, is continuous with the dermis lining the meatus, and it is situated between the epidermis and the radiate fibrous layer. It is extremely thin, and is the seat of the exquisite sensibility possessed by the membrana tympani; it also secretes the epidermis. (Figure 2.) The latter, previous to the publication of the paper just alluded to, was supposed to be secreted by the outer surface of the radiated fibrous layer; but there is now no doubt of the existence of the dermoid layer, which is more easily visible in cases of hypertrophy.[1]

2

C. The *radiate fibrous layer*—hitherto usually described, in conjunction with the circular layer, as "the fibrous lamina of the membrana tympani"—was wrongly considered by Sir Everard Home to be muscular. The fibres of this lamina are attached, externally, to a circular cartilaginous ring, which is received into a groove of the osseous meatus and centrally to the malleus. (Figure 3.) The most attenuated portion of this layer lies between the posterior part of the long process of the malleus, and the circumference of the organ. This lamina is continuous with the periosteum of the meatus.

3

D. The *circular fibrous layer* is attached to the radiate lamina by fine cellular tissue, and the two

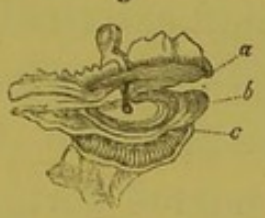
[1] In the specimen from which figure 2 was taken, the dermoid layer was slightly hypertrophied. It is seen passing from the surface of the meatus, and in some degree concealing the malleus.

structures may be readily separated. It consists, as its name implies, of circular fibres, which are firm and strong at the circumference of the organ, but towards its centre become so attenuated as to require care in order to detect them. (Figure 4.) The strong fibres at the circumference form a complete circle, and are attached to each side of the body of the malleus, as well as to the sides of the upper third of the processus longus. The circular fibrous layer is continuous with the periosteum of the tympanic cavity.

4

E. The *mucous layer* lines the inner surface of the circular fibrous layer. Like the rest of the mucous membrane of the tympanum, it is very thin and delicate, and the epithelium covering it is composed of cells with ciliæ. It will be observed that of all the laminæ composing the membrana tympani, none are proper to it, but they are continuations of other structures. (Figure 5.[1])

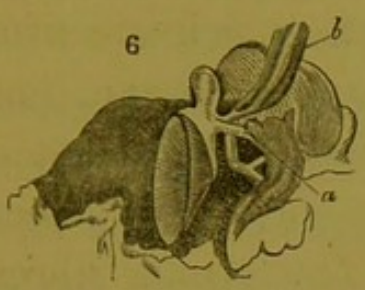
5



In order to give a complete idea of the membrana tympani, it is desirable to add, in this place, a few words relative to the *tensor tympani ligament*. This structure is about three-fourths of a line in length, and is attached, internally, to the cochleariform process, and externally to the inner surface of the malleus at the junction of the long process with the neck.

[1] *a*. The layer of the mucous membrane.
b. The circular fibrous layer.
c. The radiate fibrous layer.

The interior, which is tubular, forms a receptacle for



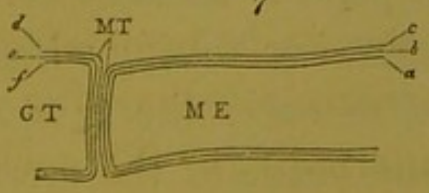
the tendon of the tensor tympani muscle. (Figure 6.[1]) This ligament performs the important function of drawing the membrana tympani inwards, and thus, by antagonism with the circular and radiate fibres of the organ, which tend to draw the membrana tympani outwards, the organ is kept in a state of moderate tension, and adapted to be acted upon by the tensor tympani and stapedius muscles. In cases of apparently total destruction of the tympanic membrane, the ligament in question still performs the function of keeping the chain of ossicles in a due state of tension, drawing the malleus inwards at the same time that that bone is pulled outwards by the few fibres of the membrana tympani that happen to remain attached to the body of the malleus.[2]

[1] *a.* The tubular tensor tympani ligament.

b. The tensor tympani muscle, the tendon of which has been drawn upwards from within the tubular ligament.

[2] The accompanying diagram gives an idea of the relations of the several laminæ of the membrana tympani.

7



ME. Meatus externus. *MT.* Membrana tympani.
CT. Cavitas tympani. *a.* The epidermis of the external meatus.
b. The dermis of the external meatus.
c. The periosteum of the external meatus.
d. The periosteum of the tympanic cavity.
e. Mucous membrane. *f.* Epithelium.

II. ON THE FUNCTIONS OF THE MEMBRANA TYMPANI, TYMPANUM, AND EUSTACHIAN TUBE.

During the present year, I have been engaged in some investigations into the functions of the membrana tympani and tympanic cavity, the results of which have been laid before the Royal Society in a paper.[1] In that communication I have shown that, contrary to the usually received opinion, the Eustachian tube does not remain always open, and that so far from allowing an uninterrupted communication between the cavity of the tympanum and that of the fauces, the guttural orifice is always closed except during the momentary action of swallowing.[2] In this latter case, the muscles of the Eustachian tube, the tensor and levator palati muscles, open the guttural orifice of the tube, afford free egress to the mucus secreted by the lining membrane of the tympanum, and allow air to enter or leave the tympanic cavity. The closure of the tube, excepting during the act of deglutition, can be experimentally proved. To those accustomed to descend in a diving-bell, it is well known that the unpleasant sensation in the ears, amounting sometimes to positive pain, is capable of instant removal by the act of swallowing, during which the condensed air being allowed to enter the tympanum and come in contact with the inside of the membrana tympani, the pressure on its outer surface is relieved by being counterbalanced. Again, if an attempt is made to

[1] On the muscles which open the Eustachian tube.

[2] Hyrtl and Wharton Jones had previously stated that the walls of the guttural portion of the tube are in contact.

swallow while the nostrils are closed by the finger and thumb, a sensation of fullness and pressure is experienced in the tympanic cavity, in consequence of air having been forced, during the act of deglutition, through the open tube into the tympanum; and this sensation continues until, by another act of swallowing, the tube is re-opened and the confined air escapes into the fauces. In the above paper an account was given of the muscles of the Eustachian tube in mammalia, birds, and reptiles; and in every animal examined it was quite apparent that the guttural orifice of the tube was closed excepting during muscular action. In some mammalia the tube is opened by the muscles of the palate; in others, by the superior constrictor of the pharynx. In birds there is a common membranous Eustachian tube, into which the osseous tubes open at the base of the skull, and this common tube descends between the two internal pterygoid muscles, to the internal surface of each of which the circumference of the tube is firmly attached by dense cellular tissue; and it is only during certain actions of these muscles that the tube is opened.

Having shown that the guttural orifice of the Eustachian tube in man and animals was closed, except during certain muscular actions, I next showed by a modification of Mr. Wheatstone's experiment, suggested to me by Mr. C. Brooke, that the sonorous vibrations communicated to the bones of the head appear much louder when the meatus is closed, than when its orifice is open. If, for instance, a tuning-fork be made to vibrate, and it be then placed in contact with

the head, the sound proceeding from it will, in a few seconds, cease to be heard; but if, directly on this cessation of sound, the experimenter close the entrance of the meatus in one ear, so as to convert it into a shut cavity, he will immediately hear a renewal of the sound of the tuning-fork; from which it appears most probable that the sonorous vibrations communicated to the external meatus impressed the membrana tympani much more powerfully when confined to the cavity of the meatus, that when allowed free communication with the external air. Considering the result of this experiment in connexion with the preceding fact of the ordinarily closed state of the tympanic cavity, it appeared to me highly probable that the sonorous vibrations imparted to the cavity of the tympanum, could only make their due impression on the membranes of the labyrinth, when strictly confined to the tympanic cavity and were not allowed to expend themselves in the cavity of the fauces. This conclusion was strengthened by the recollection that all the walls of the tympanic cavity appear constructed for producing resonance, having an investing mucous membrane of such tenuity as scarcely to be detected, save by the touch, or by the use of a magnifying glass, and also by observing that this peculiar condition of the mucous membrane was restricted to the tympanic cavity itself, and to that portion of the Eustachian tube which forms a portion of the resonant walls of the tympanic cavity.[1]

[1] In a paper published in the British and Foreign Medico-Chirurgical Review, No. 21, January, 1853, I have endeavoured to show that a

If the view here advocated be correct, and if, for the perfect performance of the function of hearing, it be necessary that the sonorous vibrations should be confined to the tympanic cavity, it is clear that the analogy usually cited as existing between the musical instrument, the kettle-drum, and the tympanum of the human ear, to the effect that in both, the air within should be allowed to communicate with that without, is incorrect; and it is also evident that an opening in the membrana tympani must, in a degree, diminish the power of hearing.[1] Upon the examination of patients affected with a simple perforation of the membrana tympani, this diminution in the ability to hear can, in fact, always be detected; although, as has been stated, if the orifice be small and the organ otherwise healthy, the difference is inconsiderable.

leading function of the membrana tympani, and the muscles and ossicles of the tympanum, is to act as the analogue of the iris in the eye. The tensor tympani muscle not only, as its name implies, renders tense the membrana tympani but also compresses the fluids of the labyrinth, while the stapedius muscle has a directly opposite action in relaxing the membrana tympani, and in placing the contents of the labyrinth in a state to be affected by the most delicate sonorous undulations. The base of the stapes moves to and fro in the fenestra ovalis, as a piston in a cylinder. There is no doubt in my mind that the fenestra rotunda is the chief medium for the passage of these undulations to the labyrinth, for the chain of bones may be incomplete without the hearing power being affected to scarcely an appreciable extent. Another very important function of the membrana tympani is to form part of the resonant walls of the tympanic cavity.

[1] Müller has shown that for the production of sonorous undulations it is not requisite a small drum should have an orifice for the communication of the air within and that without; and Mr. C. Brooke states that such orifice is only required where the air is considerably displaced, which is the case only in the more simple vibrations of the membranes.

In the greater number of cases, however, where perforation of the membrana tympani has existed, other lesions of a serious character have accompanied it—as thickening of the mucous membrane of the tympanum; pressure on the membrane of the fenestra rotunda; derangement of the articulation of the stapes with the fenestra ovalis; or injury to the nervous expansion in the labyrinth. Under any of these circumstances, it occurred to me that as an orifice in the membrana tympani, by preventing the sonorous undulations from being concentrated upon the membranous labyrinth, owing to their diffusion in the meatus, might be the direct cause of the diminished power of hearing, so it was probable that increased power would be the result of an artificial stoppage of the orifice.

III. ON THE FORMATION AND USE OF AN ARTIFICIAL MEMBRANA TYMPANI.

As a consequence of the preceding train of reflection, I was led to attempt the construction of an artificial membrana tympani, which it was hoped might serve as a substitute for the natural membrane, so far, at least, as its function of closing the tympanum and of rendering its walls resonant was concerned.[1]

[1] My hopes of success were strengthened by the result of some observations I had made upon cases of perforate membrana tympani. When these cases are not complicated with any other serious lesion of the organ, it must have been remarked, by others as well as by myself, that the patient, from some inexplicable cause, at times suddenly hears perfectly well, or nearly so. This improved hearing sometimes remains a few minutes only, at others for one or more hours. Having found this improvement to follow the use of a syringe and tepid water, or even of the pocket-hand-

After some experiments I tried vulcanized india rubber and gutta percha, making use of the thinnest layers of them that were procurable. With both these substances I succeeded in making a rude kind of artificial membrana tympani, by cutting a portion about the size of the natural membrane, and passing through it a piece of thread, by means of which and a fine tube it could be passed down to its proper situation. The tube was then withdrawn, and the thread alone left in the external meatus, by which the artificial membrane could be withdrawn at the pleasure of the patient or the operator. The disadvantages attaching to this apparatus were, difficulty of applying it on the part of the patient; liability of the material to be torn by the thread; and unsightliness of the latter hanging down from the meatus. The experiment, however, was sufficiently satisfactory to induce me to request Messrs. Weiss to construct one, the centre of which should consist of two very fine plates of silver, having a diameter of about three quarters of a line, between which the layer of vulcanized india rubber or gutta percha might

kerchief, I examined the ear in certain patients, after these operations had been effected, and I found in the former case that a bubble of water, and in the latter of discharge, filled up the orifice in the membrana tympani. Upon destroying the bubble, the improvement in the hearing at once disappeared. In one patient I was able to keep up the improved hearing by the use, from time to time, of a solution of gum acacia in water. Upon reconsidering these facts, since I completed the observations upon the closed state of the tympanic cavity, I have arrived at the conclusion that the bubble of water, discharge, or mucilage acted beneficially by again confining the sonorous undulations to the tympanum, and this conclusion has been strengthened by subsequent observations. (See Appendix.)

be placed, and to the outer surface of one of these plates a silver wire was to be attached. The artificial membrana tympani made by Messrs. Weiss, from these directions, has hitherto been perfectly successful. As supplied by them, the portion of vulcanized india rubber or gutta percha is about three quarters of an inch in diameter, which leaves sufficient margin for the surgeon to cut out a membrane of any shape that may seem to him desirable, and to leave the silver plate, either in the centre or towards the circumference, at his discretion.[1] (Figure 8.)

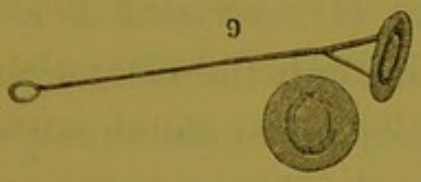
8

The silver wire is of sufficient length to admit of the membrane being introduced or withdrawn by the patient, but is not perceived externally except upon especial observation. A second kind of artificial membrane is made by fixing the layer of gutta percha or vulcanized india rubber between two very delicate silver rings from the eighth to the sixth of an inch in diameter; these rings are rivetted together, leaving a portion of the membrane drawn moderately tense in their centre; a margin of the membrane is also left beyond the circumference of the rings, so as to prevent the latter being in contact with and irritating the tube of the ear. To the surface of one of these rings the silver wire is fixed by two branches, and they should be joined so that the outer surface

[1] I now invariably use vulcanized india rubber, not much thicker than ordinary brown paper.

of the rings should look obliquely outwards and forwards instead of directly outwards, thus imitating the direction of the natural membrana tympani. This kind of membrane is often preferable to that previously described, if the meatus is sufficiently large to admit of its passage. (Figure 9.[1]) A pair of forceps is made whereby the artificial membrane can be more easily introduced and withdrawn. (Figure 10.)

9



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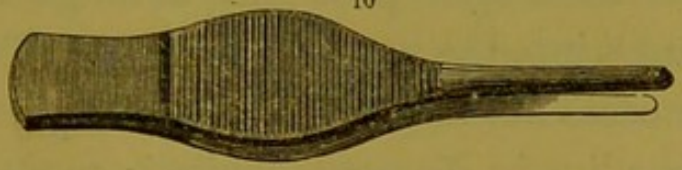

Before proceeding to speak of the mode in which the artificial membrana tympani should be applied, it is necessary to premise a few words on the diseases which usually cause perforation or destruction of the natural membrane, and upon the condition of the structures which remain. The most frequent of these diseases is catarrhal inflammation of the mucous membrane lining the tympanic cavity. It is one of those usually styled otorrhæa, of which a more particular account will be found in the paper cited in the margin.[2] This disease generally follows an attack

[1] In some cases, however, it produces a loud noise as if it were too tense; it would, perhaps, be desirable to have it made with only one branch, so that the surgeon may be able to alter the angle of the membrane with the stem, according to the case.

[2] On the Nature and Treatment of those Diseases of the Ear which have hitherto been designated Otorrhæa and Otitis. Transactions of the Provincial Medical and Surgical Association, vol. xviii. 1851.

of scarlet fever, scarlatina, measles, or any ordinary cold, and it usually occurs in children having a tendency to enlargement of the glands. The tympanic mucous membrane becomes thickened, and secretes so large a quantity of mucus of so viscid a character, that it cannot escape through the Eustachian tube; consequently, it gradually distends the tympanic cavity and presses upon the inner surface of the membrana tympani, a portion of which, generally posterior to the malleus, begins to ulcerate, and an aperture is at length produced, through which the mucus exudes into the external meatus. This orifice is in some cases not larger than a small pin's head, in others it is a line in diameter, while in many cases the entire membrane is destroyed, with the exception of a margin at the circumference about half a line in diameter, which, being composed of the combined fibres of the thickest portion of the circular and radiate laminæ, generally remains. This margin is deepest at the upper part. In some rare cases, the long process of the malleus continues entire after the complete destruction of the membrane to which it was attached; but, as a general rule, the whole of this process is gradually absorbed, leaving merely the head of the bone which articulates with the incus, the neck, and the body which receives the attachment of the tensor tympani ligament internally; anteriorly and posteriorly the fibres of the remnant of the membrane are attached, and externally the processus brevis remains. It will therefore be understood that, in cases of so-called destruction of the membrana

tympani, a margin is generally left, to which the body of the malleus remains fixed, and to the inner part of which the tensor tympani ligament and muscle are attached, affording the means by which the small bones and muscles of the tympanum are still enabled to perform their functions. In cases of general *ulceration* of the mucous membrane of the tympanum, which fortunately seldom occurs, the incus is generally discharged, and sometimes the malleus also; but even in these cases, if the attachments of the stapes to the circumference of the fenestra ovalis remain uninjured, the power of hearing may be much improved: should the stapes however be removed, total and irremediable deafness ensues.

The other disease through which an orifice in the membrana tympani is usually effected, is *ulceration of the fibrous laminæ*. The disease itself is commonly the result of inflammation of the dermoid layer, which spreads first to the radiate fibrous and thence to the circular lamina. The laminæ, being weakened by the ulcerative process, fall inwards as far as the promontory, to which they often ultimately adhere, and, when an orifice has been thus produced, its margins are not unfrequently drawn into the shape of a funnel, whose inner part adheres to the tympanic walls. In ulceration of the membrana tympani, proceeding from the dermoid layer, the entire organ is very rarely destroyed, but an orifice merely is produced.

The cases in which the artificial membrana tympani is of the greatest benefit are those where there

is a well defined aperture in the natural membrane, or, if it be entirely absent, where there is simple hypertrophy of the mucous membrane of the tympanum, with or without discharge from its surface. In these cases, it will be found that the organ has by no means entirely lost its power of discerning sounds; as a general rule, the human voice is heard when the mouth of the speaker is situated within about a foot of the patient's ear, and when the words are spoken slowly and distinctly. The diminished power of hearing just noticed, while it entirely excludes the sufferer from the advantages of general conversation, is, however, greatly aggravated when, to the affection of the membrana tympani and mucous membrane of the tympanum, the stapes has become ankylosed to the fenestra ovalis, or the nervous expansions have been injured. In such cases where the patients require to be shouted to close to the ear, the artificial membrane will not prove of any service.

The Mode of Applying the Artificial Membrana Tympani.

As in cases of perforation or destruction of the membrana tympani there is so frequently catarrhal inflammation of the mucous membrane of the tympanum, it is obviously important that no foreign substance should be placed in contact with that membrane; and, as there is always a margin of the membrana tympani remaining, the object of the Surgeon should be to keep the artificial membrane external to the latter. After carefully noting the

size of the inner extremity of the meatus to which the natural membrana tympani was attached, the operator should then cut the artificial membrane as nearly of the size and shape of the natural one as possible, taking care at the same time to keep the margin quite smooth and regular.[1] The patient must then be placed with the head inclined to the opposite shoulder, while a strong light is thrown into the meatus, which if liable to discharge should have been previously syringed. The operator will now take the artificial membrane, and, having moistened it with water, pass it, by means of the silver wire, gently inwards, until it has reached what he considers the natural position. This he will ascertain by the occurrence of a faint bubbling sound, caused by the escape of the slightly compressed air beyond it; he will also feel a slight obstruction offered to its further passage by the remnant of the natural membrane. Should he attempt to pass the artificial membrane beyond this point, the patient will complain of pain, which until then had not been felt. The most certain test, however, of the artificial membrane having been properly placed is the sensation of the patient, who discovers, by the sound of his own voice, or that of the surgeon, or by the movement of his tongue and lips, that his hearing has been suddenly much improved.

It will be imagined that great care must be taken to cut the membrane so that it shall fit the inner ex-

[1] In cases where only a small border of the natural membrane remains, it is often desirable to cut the artificial membrane of a size larger than the inner extremity of the tube, so that its edge may turn outwards.

tremity of the meatus with exactness, since if too large it would cause discomfort, and if too small it would not fulfil its purpose of rendering the tympanum an air-tight cavity. It is not easy, in all cases, to fit the artificial membrane exactly to the inner extremity of the meatus, so as not to allow of any communication between the air in the tympanum and that in the external meatus; this is, however, the object which should always be sought to be attained. At first, the patient should be instructed not to use the artificial membrane for more than two hours daily; and, if he complains of an uncomfortable feeling, one hour, or even half an hour, will be sufficient.

It would, perhaps, be expected that the contact of a foreign body, like the artificial membrana tympani, with the wall of the external meatus would soon become intolerable; such, however, is not the case, and several patients have left my room without being able to say, from the sensation in the ear, whether any foreign body were there; many have now worn this apparatus daily, during several months, without having suffered the slightest pain. The explanation of this circumstance may be found in the fact that the most sensitive part of the meatus externus is about its centre, the membrane in the immediate vicinity of the membrana tympani not being so abundantly supplied with nerves: another explanation is that the circumference of the artificial membrane presses with extreme gentleness against the wall of the meatus.

The results of the application of the artificial organ

have been much more satisfactory than I had reason to anticipate. I have already used it beneficially in nearly fifty cases. The substitution of a thin layer of vulcanized india rubber or gutta percha, for so exquisitely delicate a structure as the healthy membrana tympani, would be expected to afford but trifling aid; such, however, is not the case, for among the patients relieved by it most have heard the human voice perfectly across an ordinary sized room, and in one case the voices of boys in the open air were heard at a distance of between one and two fields. Surgeons, who have paid careful attention to diseases of the ear, will not be surprised at the efficient substitute the artificial membrane offers, as they will bring to mind many cases in which the natural organ has been greatly hypertrophied, especially in chronic inflammation of its dermoid layer, with but a very slight diminution of the power of hearing.

The surgeon having ascertained that the artificial membrane is beneficial to the patient, if no pain is experienced, it may be allowed to remain in the ear for a few hours, and gradually increased to the whole day: it is often desirable that the use of the membrana tympani should be preceded, or accompanied, by vesication over the mastoid process, whereby the thick mucous membrane of the tympanum may be rendered more healthy. In all cases, the artificial membrane should be removed at night, and, when there is any discharge, the ear ought to be syringed each night and morning with tepid water.

CASES.

Deafness for sixteen years, discharge from each ear for six years, aperture in each membrane tympani; power of hearing restored.

Peter Turnbull, æt. forty-three, formerly in the army, from which he was discharged on account of his deafness, was admitted, under my care, at St. Mary's Hospital, on the 12th of January, 1852. He stated that sixteen years ago, without any other assignable cause than a cold, he became slowly dull of hearing, and five or six years since he perceived a discharge from both ears, which has continued up to the present time. The power of hearing has been gradually diminishing, so that, at present, he requires speaking to loud, close to his head. Upon examination, an aperture between one and two lines in diameter was observed in each membrana tympani, and the mucous membrane of the tympanum, which was the source of the discharge, was more thick and red than natural.

The treatment consisted in keeping up counter-irritation over each mastoid process, and in the use of an injection composed of three grains of acetate of zinc, to an ounce of water. Under this treatment, he somewhat improved, but the hearing still remained so defective that he was precluded from following any avocation. In the commencement of June, I experimented on this patient with the first artificial membrana tympani, composed of vulcanized india rubber, and the good effect was at once decided. When it was placed over the surface of the original membrane, so as wholly to close the orifice, the patient made a movement of his lips, and said, "I hear as differently as possible from what I have done for many years; everything sounds clear!" This patient went away with the artificial membrane in his ear, hearing conversation perfectly. The following morning, he came to my house, saying that he had accidently moved what I had left in his ear, and that he was "as dull as ever." I replaced the artificial membrane—he again heard well; and being supplied with one which he could introduce or

remove at pleasure, he has worn it during the day, ever since —a space of between three and four months—and he has never complained of pain or discomfort from it. Latterly, he has found the hearing so much improved that he has been able to dispense with the use of the artificial membrane for a few hours daily; but he hears much better with than without it. As a proof of the great amelioration that has taken place, this patient told me that while in the country lately, and using the membrane, he heard voices at a distance, and upon going towards the place from which they appeared to proceed, he found some boys under a hedge, more than a field distant from the spot where he heard them. He is going back into the army.

This patient was shown at a meeting of the Pathological Society of London, in February, 1853; the following is the published report:—"The artificial membranes having been removed, the members of the Society had the opportunity of observing the perforate condition of each membrana tympani. After the removal of the membranes, he could not hear, unless loudly spoken to; but, when he had replaced them, which he did with apparent readiness, his hearing was excellent."—*Medical Times and Gazette, February* 12, 1853.

Each membrana tympani destroyed by measles at four years of age. Hearing restored by the artificial membrane. Very sensitive to sounds.

Miss B., æt. twenty-one, consulted me on November 9, 1853, on the recommendation of Dr. Grindrod of Seaforth, near Liverpool. Her health was good.—*History of case.* At four years of age she suffered from an attack of measles, which was followed by discharge from each ear; this has lasted to the present time, so that the ears require syringing every day. Since the measles, the power of hearing has been so much deteriorated, that it is requisite for her to be spoken to distinctly within the distance of a yard. Upon inspection, it was found that the membrana tympani of each ear had been destroyed, and that the only vestige of it was a very narrow margin. The mucous membrane lining the tympanic cavities was very red, and much thicker than natural; it was covered by a mucous discharge.—*Treatment.* An artificial membrana tympani was introduced into each ear, the effect of which was to improve the power of hearing at once and so greatly, that the patient heard my voice perfectly

well across my room, with my back turned towards her. Ordered to wear the artificial membranes during the day, to take them out at night, and to syringe the ears with warm water twice daily.—13th. Has been wearing the membranes every alternate day, and has heard perfectly while they were worn. Indeed, the only drawback to her comfort has been the circumstance that her friends still speak loud to her, which causes considerable uneasiness in the ears from the very great sound.—16th. Continues to hear well, but has been obliged to remove to a quiet street, as the sound of carriages passing through the street has been annoying. She complains of the "intolerable rustling" of her silk dress, of which she was never before conscious. The patient left London after some further watching, hearing quite comfortably. I received a letter from her in December, from which I subjoin an extract:—"I am thankful to say the improvement in my hearing has increased almost daily, and I now hear general conversation easily, and feel quite a different person from what I did a short time since. I am still sensitive to sounds, but not merely so much distressed with them as I was at first. I found the noise of the organ at church too great the first time I went, and came out almost as soon as the service commenced. I find no pain in my ears, and am in all respects in the enjoyment of good health."

Deafness of twenty years' duration perfectly relieved by the artificial membrane.

The following particulars of a case about which we corresponded, were sent to me by Dr. Shearman of Sheffield:—"I tried the false drum in one ear, the whole of the membrana tympani had been destroyed, and the cavity of the tympanum so bared to the view, that it was difficult at first to ascertain whether the drum membrane had gone, or was obscured by polypoid, or other growths; however, the probe came down upon the bone. The false drum gave such relief, that the hearing distance was increased from actual contact to twelve, and subsequently to eighteen inches; the patient is now able to manage the contrivance herself.

The other membrana tympani of the same patient is yet so covered with polypous growths, that I cannot make out the precise condition of the drum; however, inflation of the tympanum shows that the membrana tympani is perforated. The deafness in this case is of nearly twenty years duration,

is perfectly removed on the left side, and although the whole of the left membrana tympani is destroyed, the false one acts perfectly."

Destruction of each membrana tympani: stricture of the meatus.

Miss S., æt. twenty-four, not in very good health, states that at four years of age she suffered from an attack of scarlet fever, subsequent to which she was so hard of hearing, as to require to be spoken to distinctly within the distance of a yard. This hardness of hearing is increased during cold and damp weather; has had discharge from both ears, but at present it is only very slight from the left, which is the better ear; complains of no pain, but of a noise. Lately, from not being in good health, has been more than usually dull.

Examination: Right ear. The hearing distance of the watch is half an inch.[1] The central part of the meatus is so contracted that it is not more than half its natural size. By means of a very strong light thrown beyond the contracted portion, a portion of the mucous membrane of the tympanum could be detected; there was no appearance of the membrana tympani.—*Left ear.* Hearing distance one inch; meatus contracted like that of the right ear; the membrana tympani was not seen, but in place of it was observed the shining tympanic mucous membrane.

At first sight it appeared that the presence of the stricture would offer an obstacle to the introduction of the artificial membrane, I nevertheless tried a small one to each ear, passing it through the stricture, and then moving it gently to and fro, so as to allow it to recover its plane surface. Having done this, I moved it slightly inwards to the situation of the natural membrane, and immediately the patient found that she heard perfectly all that was said at any part of the room. There was not much difference between the hearing power of the two ears. Care was required in the management of the case from slight tendency to irritation of the meatus, but the patient left me hearing well. In the middle of February, 1854, the mother of this lady being in London, called to thank me for the benefit produced in her daughter's case. She said that her daughter "continued to hear perfectly, and that she was quite an altered person."

[1] I have used the same watch for many years; the natural "hearing distance" is three feet.

Deafness from scarlet fever during five years. Hearing entirely restored by the use of the artificial membrane.

Miss G., æt. fourteen, was brought to me in August, 1853, by Dr. Grindrod. Health good.—*History of case.* When between nine and ten years of age, suffered from scarlet fever, since which time has had a discharge from both ears, attended by a diminution of the hearing so as to require to be spoken to loud, near to her. She has lately been to a school at Brussels, where her defective hearing had greatly arrested her progress. Upon *inspection*, it was found that the membrana tympani in each ear was absent, the mucous membrane of the tympanum was thick and red, and poured out a mucous secretion. An artificial membrana tympani was applied to each ear, and the result was so complete a restoration of the hearing power, that the patient could hear all that was said in different parts of a large room. This patient returned to school at Brussels, and in about six weeks afterwards I received a letter from the father, a medical man, from which the following is an extract:—"We have had the most pleasing intelligence from my little daughter at Brussels respecting her hearing. I think I cannot do better than give it in her own words—'I have had three German doctors and one French one to see me, or rather the *artificial membranes.* I am quite a new creature, my hearing is so greatly improved.' The father adds—'This is very satisfactory evidence as to the successful operation of your beautiful invention, after nearly five years' deafness, to an extent that she was unable to hear a word in church the whole of that time.'"

Deafness for twenty years from measles and scarlet fever. Greatly improved by the artificial membrane.

Mr. M., æt. twenty-three, consulted me, December 20, 1853. Health good; no relatives deaf.—*History of case.* At three years of age, had scarlet fever and measles at the same time, accompanied by much discharge from each ear; was totally deaf for some months after the attack, but slowly improved, so as to hear a loud voice spoken near to the left ear; the right ear nearly useless; lately has heard better at times with left ear for two or three hours. To-day, requires to be spoken to loud within two feet of the left ear, and is about the same as ordinarily.

Upon examination of the *right ear*, the meatus was found

to contain a collection of mucus and epidermus, which being removed, the membrana tympani was seen to be white, like paper, flat and thick; posterior to the inferior third of the malleus is a small orifice, about three quarters of a line in diameter, through which mucus oozed from the tympanic cavity. The watch was not heard, even when pressed against the ear; the crack produced by two finger-nails was distinguished.—*Left ear.* The meatus contained a shready discharge; the membrana tympani was absent; the mucous membrane of the tympanum red and much tumefied. Watch heard when in contact with the ear. Although the hearing power of the left ear was somewhat less than in the majority of cases, where there is an absence of the membrana tympani uncomplicated with any other disease, I nevertheless determined to try the artificial membrana tympani, the effect of which was to improve the hearing considerably, although not to the same extent as in the majority of cases.

December 21. Ordered to wear the membrane for four hours.

December 22. Upon the use of the membrane to-day, heard my voice distinctly half across my room.

December 27. Says that he never remembers to have heard so well as yesterday; heard everything that was said at dinner, and his own voice was quite distinct; the sound of the rustling of a lady's silk dress quite astonished him; towards the latter part of the day did not hear quite so well; last night, after removing the drum, was exposed to cold, which caused some pain. Without the drum, to-day could not hear my voice, unless I spoke into his left ear; with it, heard much better, but not so well as yesterday: this arose from the mucous membrane of the tympanum being much tumefied. This tumefaction gradually subsided, and this patient left me a few days after, hearing quite well. He inserts the artificial membrane himself, which requires a little careful adjustment. On one occasion, when he started for a walk in the street, after having inserted the membrane, his hearing was far from good, but as he walked on the pavement, a sudden movement took place in the ear, and he heard perfectly.

In February, 1854, in a letter, this gentleman says, "I have much pleasure in informing you that the artificial membrane continues to be effectual; my friends are much gratified at the improvement you have been able to effect."

Miss H., æt. seventeen, was brought to me in November, 1853, by Mr. Jeffree, of Lambeth. At seven years of age, had a severe attack of scarlet fever, since which time she has had a discharge from each ear, and is so hard of hearing as to require to be spoken to distinctly in a raised voice within the distance of two yards; she has not been able to hear general conversation. About two years since, a portion of carious bone was removed from the back of each ear.

Upon examination, no membrana tympani was observed in either ear, and the mucous membrane of each tympanic cavity was thick and red. The application of the artificial membrane gave immediate relief, and the patient was soon able to wear it during the whole of the day.

The following is an extract from a letter written by the patient, in February, 1854:—"By the use of the artificial drum, I am happy to say that I am now, and have been since I saw you, perfectly capable of hearing at church, and taking part in conversation, with as much facility as if I had never been deprived of the power."

S. H., Esq., æt. twenty-one, was sent to me by Mr. Fergusson, on December 4th, 1853. Between six and seven years of age, he had an attack of scarlet fever, since which he has had, at intervals, discharge from each ear, attended by so considerable a degree of hardness of hearing, that he is obliged to lean forward whenever he is spoken to, so as to be within a yard of the speaker. Upon examination, the membrana tympani was found to have disappeared from each ear; the watch was not heard by the right ear, and at a distance of five inches from the left. By the aid of the artificial membranes, he was able at once to hear me talk across my room, and he soon heard general conversation perfectly. He went into the country, and in the end of January, I had the following confirmation of the persistence of the benefit: "My hearing is quite optional to me; that is, I can hear or not, just as I choose. The artificial drums I use quite well—can put them in or take them out without assistance. My debt of gratitude to you I can never repay."

Lieut. L., æt. twenty-seven, consulted me on January 1st, 1854. He stated that he had been dull of hearing for fifteen years, especially in the right ear; complains of a singing

noise in both ears, but especially in the right. During the last year, he has been getting worse, so that he is now inconvenienced in society. On examination, the membrana tympani was found to have disappeared from the right ear, and the mucous membrane of the tympanum was red; the watch was not heard, unless in contact with the ear. In the left ear, the membrana tympani was white, like paper, and at its upper part was a small polypus. Upon the application of the artificial membrane to the right ear, the patient heard well at once, although he never remembers to have heard with it before. He was soon able to put it in for himself, and to hear admirably well. At the end of February, he thus wrote:—"I yesterday, by accident, lost overboard the artificial membrane, which I have been wearing since I saw you. I find it now rather uncomfortable to leave off wearing it, and I am sure the hearing has been greatly improved, as, although I have had a bad cold lately, I doubt if any one in my new ship has discovered that I am deaf."

N. M., Esq., æt. seventeen and a half, consulted me on August 15th, 1853.—*History.* He had measles when young, since which time he has had a discharge from each ear, accompanied by so great a difficulty in hearing, that he requires to be spoken to distinctly within the distance of a yard. Hears somewhat better in frosty weather. Upon examination, a considerable orifice was discerned in each membrana tympani, The mucous membrane of each tympanic cavity is thicker than natural. On applying the artificial membrane to each ear, the hearing power was at once restored; he said that he "heard painfully well, and that his own voice sounded like a trumpet." He soon learned to put the membrane in himself, and he continues to hear quite well. At the end of February, 1854, he wrote from college:—"I have as yet been perfectly able to hear almost every lecture, except those of one professor, who speaks very indistinctly."

APPENDIX.

Several writers have alluded to the beneficial results following the treatment of a perforate membrana tympani by means of the introduction of foreign bodies, especially of lint and cotton wool. Thus Itard cites a case in which the deafness was completely relieved by the introduction of a portion of cotton wool to the bottom of the meatus. Deleau speaks of a patient who greatly improved his hearing by the introduction of a piece of wood or the central part of an onion. Mr. Tod describes "the relief derived from the mere introduction of a little lint into the external meatus, in those cases where the membrana tympani has been ruptured or destroyed by disease. So great, indeed, is the improvement which takes place from the application of this simple remedy that patients will frequently appear astonished on being so easily relieved."[1] In the year 1848, Mr. Yearsley published a pamphlet entitled "On a new mode of treating Deafness when attended by partial or entire loss of the membrana tympani; associated or not with discharge from the ear." In this pamphlet, he advocates the application of cotton wool in a moistened state, in cases of partial or entire loss of the membrana tympani, the object of this substance being, as he has subsequently stated, "to support the remaining portion of the membrana tympani, or the ossicula."—*Provincial Medical and Surgical Journal,* August 18, 1852.—With respect to the mode of applying the wool, the following are his instructions:—"A small piece of wool differing in size according to the case, and fully moistened in water, is introduced through the speculum to the bottom of the meatus, and adjusted superiorly, inferiorly, anteriorly, or posteriorly, according to the situation of the perforation, and other circumstances connected with the case; but care must be taken that the entire opening be not covered, otherwise the experiment will not succeed. It is also indispensable to success that the moisture of the wool should be preserved." He also says, "It is far from my wish to discourage the attempts of others to placea right 'these magical bits of wool,' but truth compels me to add that, simple as it may appear, it is an operation requiring the most delicate tact to manipulate with success, which great experience only can confer."

In all cases of perforate membrana tympani, where the introduction of a foreign substance is productive of benefit, I believe it will eventually be proved that such benefit is the result of a partial or complete closure of the orifice.

[1] Anatomy and Physiology of the Organ of Hearing, pages 105-6. 1852.

ON THE

WEIGHT AND DIMENSIONS OF THE HEART

IN

HEALTH AND DISEASE.

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ON THE

WEIGHT AND DIMENSIONS OF THE HEART

IN

HEALTH AND DISEASE.

The following paper contains a series of observations with calculations based upon them, intended to illustrate the weight and dimensions of the heart in health, and the changes which it undergoes in different forms of disease.

The observations have all been collected by myself within the last few years; some have already been published in connection with a series of tables of the weight of the brain,[1] but most of them have not previously appeared in print, and all the weights of the diseased heart and the measurements, are now first collected and analyzed. For the purpose of presenting the facts in a more complete form, and in order to furnish the means of ready comparison between the results here arrived at, and the data from which they are deduced, I have preferred to give the whole of the observations together, rather than to refer to the former papers for those previously published.

The mode in which the observations were made is as follows:—The heart having been removed from the body, and the aorta and pulmonary artery cut across about an inch above their origin, the left ventricle was laid open by an incision commencing near the aortic orifice, and continued across the apex to the posterior wall, so as fully to expose the cavity, without interfering with the attachments of the valves. The right ventricle was exposed by a similar incision extending from near the origin of the pulmonary artery to the apex, and the auricles were laid open transversely. The organ was then deprived of coagulum, washed, wiped, and weighed.

The measurement of the girth of the heart externally was taken with a piece of tape or string, from the line of the septum in front, to the corresponding situation behind. The capacity of the orifices

[1] Monthly Journal of Medical Science, vol. vii. (New Series, vol. i.) 1846.

was ascertained by the passage of graduated balls numbered from 1 to 15, and measuring from 21 to 63 French lines—each

WEIGHT AND DIMENSIONS

No.	Age.	Sex.	Weight of Body.	Weight of Heart.	Girth of Right Ventricle.	Girth of Left Ventricle.	Length of cavity of Right V.	Length of cavity of Left V.	Thickness of Walls of Right Ventricle.		
			lb	oz. dr.	lines.	lines.	lines.	lines.	Base.	Mid-point.	Apex.
1	7 days	F.	7	0 13	25	19			2 lines		
2	10 months	M.		1 10	29	25			1 line		
3	1 year	F.	15	2 0	34	32	22	19	2	2	$1\frac{1}{2}$
4	3 years	F.	22	1 10	35	35	21	21	1 line		
5	3	M.		2 15	35	35			1 line		
6	$4\frac{1}{2}$	M.		2 9	36	30	25	22	$1\frac{1}{2}$	$1\frac{1}{2}$	1
7	8	F.	56	5 1	42	36	36	30	1	2	1
8	8	F.	24	3 0	36	30	27	24	1	$1\frac{1}{2}$	1
9	11	M.	43	4 6	38	37	34	28	2	2	1
10	13	F.	$46\frac{3}{4}$	6 2					$1\frac{1}{2}$	2	$1\frac{1}{2}$
11	18	F.		7 15	52	48	34	31	$1\frac{3}{4}$	2	1
12	20	M.	97	11 0	49	48	48	39	2	2	$1\frac{1}{2}$
13	24	M.		8 8	50	46	40	34	$1\frac{1}{2}$	$1\frac{1}{2}$	$1\frac{1}{2}$
14	24	M.		9 $1\frac{1}{2}$	60	60	34	32	2	2	$1\frac{1}{2}$
15	24	M.		9 0	60	54	42	40	2	$1\frac{1}{2}$	1
16	25	M.		8 8	60	38	35	32	2	2	1
17	26	M.	101	8 11	54	48	45	36	$1\frac{1}{2}$ line		
18	27	M.	120	11 12	52	52	45	42	2	$2\frac{1}{2}$	$1\frac{1}{2}$
19	30	M.		8 $15\frac{1}{2}$			40	36	$1\frac{1}{4}$	2	1
20	31	M.		9 4	48	53	46	37	2	2	2
21	37	M.		6 $4\frac{1}{2}$	50	42	36	36	2	2	$1\frac{1}{4}$
22	66	M.		10 8	60	46	45	37	2	2	1
23	20	F.		7 4	49	38	40	32	2	2	1
24	24	F.		10 11	60	48	48	42	$1\frac{1}{2}$	3	$1\frac{1}{2}$
25	47	F.		10 3			42	36	$3\frac{1}{2}$	$2\frac{1}{2}$	2
26	60	F.	87	11 0	52	40	42	36	2	2	1
27	66	F.	70	7 0	48	42	42	27	$1\frac{1}{2}$ line		

number being 3 French lines more in circumference than the former. The capacity of the orifices having been ascertained, and the state

OF HEALTHY HEARTS.

Thickness of Walls of Left Ventricle.			Thickness of Septum.	Circumference of Apertures.				Cause of Death.
				Tricuspid	Pulmonic.	Mitral.	Aortic.	
Base.	Mid-point.	Apex.	lines.	lines.	lines.	lines.	lines.	
2 lines				13	13	13	13	
3	3	$1\frac{1}{2}$		22	19	22	19	Acute hydrocephalus and pneumonia.
3	3	2	4	28	22	25	$17\frac{1}{2}$	Bronchial phthisis.
$3\frac{1}{2}$	4	$1\frac{1}{2}$	3	26	21	25	18	Burn, 4 hours.
3	4	2		31	24	28	22	Diseased kidneys and dropsy ; empyema.
3	4	$1\frac{1}{2}$		36	25	30	20	Phthisis, 4 valves to pulmonary orifice.
3	4	2		33	30	33	27	Burn of abdomen.
$3\frac{1}{2}$	$3\frac{1}{2}$	2		36	27	30	24	Abscess of liver; diseased mesenteric glands.
5	5	2		39	33	36	24	Fractured skull.
$4\frac{1}{2}$	5	$1\frac{1}{2}$		42	36	42	30	Fever; enlarged spleen.
7	7	3	6	48	42	45	30	Phthisis and morbus renum.
4	7	2	6	43	29	37	26	Phthisis.
5	6	$2\frac{1}{2}$	6	45	39	42	36	Meningitis.
5	6	3	5	60	42	48	39	Ascites and diseased liver.
6	5	2	6	60	34	42	31	Fever; foramen ovale not entirely closed.
4	6	2	6	54	45	51	42	Disd. kidneys and bladder.
5	6	3		60	42	48	36	Ulceration of intestines.
5	7	2	7	50	32	38	28	Fever, cirrhosis hepatis.
$5\frac{1}{2}$	$3\frac{1}{2}$	2	$3\frac{3}{4}$	51	45	48	36	Abscess of brain.
6	7	$2\frac{1}{2}$	$6\frac{1}{2}$	54	48	51	42	Phthisis.
6	5	$2\frac{1}{2}$	5	51	39	48	36	Phthisis.
5	7	$2\frac{1}{2}$	6	51	39	39	36	Fever.
6	8	2	4	42	33	37	29	Variola.
$3\frac{1}{2}$	5	2		60	45	48	36	Ac. capillary bronchitis.
7	5	$2\frac{1}{2}$		54	39	45	30	Ac. asthenic bronchitis supervening on chronic.
4	7	2		40	34	42	39	Bronchitis.
6	7	2		51	42	48	33	Phthisis.

of the valves observed, the orifices were laid open, and the length of the ventricular cavities measured from the attachments of the aortic and pulmonic valves to the apex, and the thickness of the parieties ascertained at three points; near the base, where the walls begin to narrow; at the mid-point between the base and apex; and at the

WEIGHT OF HEALTHY HEARTS—MALES.

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death.
		lb.	oz. dr.	
28	6 mths.		0 13½	
29	2 „	24	2 10	Burn, 21 days.
30	2 yrs.	31½	2 12	Burn, 6 hours.
31	4		4 4	Burn, 12 hours.
32	5	21	3 0	Coma, succeeding cholera.
33	8	48	6 4	Scarlatina.
34	10	45	4 4	Cholera.
35	11	42	8 8	Typhoid fever.
36	11		5 2	Typhoid fever.
37	11	38	4 0	Chorea.
38	11		6 0	Phthisis.
39	14		9 0	Amputation of toe; morbus renum.
40	14		6 8	Typhoid fever.
41	15	88	8 0	Laceration of internal organs from injury.
42	15		6 0	Phthisis; copious hæmoptysis.
43	16	47	5 8	Phthisis.
44	16		7 0	Bronchial phthisis.
45	16		6 8	Typhoid fever.
46	17		8 8	Typhoid fever.
47	19		11 0	Typhoid fever.
48	20	100	10 0	Diphtheritis.
49	21		8 8	Strumous pyelitis.
50	21	106	9 8	Phthisis.
51	22	127	8 0	Phthisis.
52	24		8 0	Phthisis.
53	24		10 0	Fever.
54	24	90	7 8	Phthisis.
55	24		10 4	Phthisis.
56	25	103	10 0	Phthisis.
57	25	125	10 8	Fever; white spot on pericardium.
58	25		9 0	Fever.
59	26		8 12	Lunbar abscess.
60	27	145	9 0	Phthisis.
61	27		11 4	Variola.
62	28	108	8 8	Fever.
63	28	97	9 8	Phthisis; softening of brain.
64	28	108	10 0	Fever.
65	28	97	7 8	Morbus renum.
66	29		11 0	Morbus renum.
67	29		11 0	Phthisis; white spot on pericardium.
68	32		11 8	Double pneumonia.
69	32		11 8	Morbus renum.
70	33		10 8	Ulceration of cartilages of knee.

pex. The septum was cut across about half way between the base nd apex, and measured across its centre.

No organs were weighed in which there was an undue proportion f fat on the surface, and none were measured which, whether from lisease or post-mortem change, were very flaccid. The weight em-

WEIGHTS OF HEALTHY HEARTS—MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.		Cause of Death.
	Years.	lbs.	oz.	dr.	
71	33		9	0	Diseased liver.
72	33	99	8	6	Phthisis; aorta atheromatous.
73	33		11	0	Apoplexy; aorta atheromatous.
74	35		8	0	Morbus renum.
75	35	100	7	8	Phthisis.
76	35	87	7	4	Morbus renum.
77	35		6	12	Phthisis.
78	35		11	8	Laceration of aorta, from violent muscular exertion.
79	37	112	8	0	Fever, white spots on pericardium.
80	37	90	9	10	Abscess on buttock and secondary deposits.
81	37		9	0	Pneumonia.
82	37	84	9	4	Phthisis.
83	37		9	0	Fever.
84	38		8	8	Fever.
85	38		10	0	Meningitis; pneumonia.
86	38	100	10	12	Fever, chr. bronchitis.
87	38		8	8	Erysipelas of head.
88	38		11	0	Delirium tremens; softening of brain.
89	39		6	0	Carcinoma pylori.
90	39		11	0	Phthisis.
91	39		9	12	Phthisis.
92	40		11	0	Phthisis, two valves to aortic orifice.
93	40		11	12	Fever.
94	40		11	8	Cirrhosis hepatis.
95	40	130	11	8	Delirium tremens.
96	40		10	4	Phthisis.
97	41		9	8	Phthisis.
98	42	115	11	12	Pneumonia.
99	42	114	8	8	Phthisis.
100	45	79	9	8	Phthisis.
101	45		10	0	Phthisis.
102	46	90	10	0	Injury; purulent deposits; white spot on pericardium.
103	47	129	10	0	Phthisis.
104	50		9	0	Fever.
105	50		9	0	Fever.
106	51		9	12	Pleuropneumonia.
107	52		10	8	Bronchitis; white spots on pericardium.
108	53	115	10	8	Complicated dislocation of astragalus; white spot on pericardium.
109	53	108	5	0	Cirrhosis hepatis and morbus renum.
110	54	146	11	8	Meningitis and apoplexy.

ployed is avoirdupois, and the measure French lines and inches—the French line being.0/888 of an English inch, and the French inch equal to 1.065 English inch, or rather more than $\frac{1}{15}$ in. longer. The French line is equal to 3.25 millimetres. The French measures were made use of as having been those employed by M. Bizot and some other observers.

WEIGHTS OF HEALTHY HEARTS—MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.		Cause of Death
	Years.	lb.	oz.	dr.	
111	54		9	0	Pleuritis; peritonitis.
112	55	80	8	8	Tertiary symptoms.
113	55		8	0	Phthisis.
114	56		11	8	Cut throat.
115	56	84	7	0	Phthisis.
116	60	102	11	8	Disease of stomach.
117	60	101	10	0	Fractured ribs; pneumonia.
118	60	129	11	0	Phthisis; white spot on pericardium.
119	60		11	8	Phthisis.
120	60		11	0	Chronic dysentery.
121	62	99	10	8	Phthisis.
122	70		11	8	Fever; white spot on pericardium.

WEIGHT OF HEALTHY HEARTS—FEMALES.

123	10 wks.		1	3	
124	1 year and $\frac{3}{4}$th	$13\frac{1}{2}$	1	8	Subacute hydrocephalus.
125	5 years	28	2	14	Cholera.
126	6		2	12	Burn, 14 hours.
127	7	32	2	3	Fever.
128	7		3	0	Fever.
129	11	36	3	14	Cholera.
130	17	59	4	8	Phthisis, necrosis.
131	17		7	0	Phthisis.
132	18	113	9	8	Fever.
133	18		10	8	Bronchitis; phthisis.
134	19		7	12	Typhoid fever.
135	19		8	0	Cholera.
136	20	90	9	0	Phthisis.
137	21	86	5	12	Phthisis.
138	21		10	8	Morbus renum.
139	21	97	10	0	Phthisis.
140	22		10	4	Fever.
141	23	85	6	12	Phthisis.
142	23		8	8	Disease of ear and brain.
143	24		11	0	Acute capillary bronchitis.
144	25	86	9	12	Pneumonia.
145	25		5	8	Phthisis.
146	25	66	8	0	Tertiary syphilis.
147	25	117	9	0	Variola.

The tables include 183 observations of the weights of organs regarded as healthy, and 145 observations of the weights of hearts presenting either primary or secondary disease. The measurements are of 27 healthy and 41 diseased organs. The whole series is a careful selection from a larger number of observations, of which those only are given which are regarded as accurate and complete.

WEIGHT OF HEALTHY HEARTS—FEMALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.		Cause of Death.
	Years.	lb.	oz.	dr.	
148	26		9	0	Acute phthisis.
149	28	97	9	8	Fever.
150	28		7	4	Morbus renum.
151	28		7	12	Phthisis.
152	28		7	4	Morbus renum.
153	29		9	8	Disease of liver.
154	30		8	8	Pneumonia; morbus renum.
155	30		10	0	Cholera.
156	31	104½	9	0	Delirium tremens.
157	31	89	9	12	Fever.
158	32	107	9	12	
159	33	84	8	8	Fever.
160	34	88	7	8	Phthisis.
161	34	103	11	0	Phthisis.
162	34	71	6	8	Morbus renum.
163	35	58	5	8	Phthisis.
164	36		8	8	Ac. supervening or chronic bronchitis.
165	36		10	12	Fever.
166	38	79	11	0	Phthisis.
167	39		8	8	Scrofulous abscess of liver.
168	39		10	0	Pleuritis; morbus renum.
169	39	73	7	7	Phthisis.
170	39	95	9	0	Phthisis; puerperal mania.
171	39		9	8	Morbus renum.
172	40		9	0	Sudden death during convalesence from fever.
173	40		9	4	Pneumonia; gangrene of os uteri after fever.
174	40	75	8	0	Relapsing fever.
175	45	109¾	11	0	Phthisis.
176	45	91	8	0	Tubercular peritonitis.
177	47	109	9	12	Morbus renum.
178	50		7	0	Phthisis.
179	51		8	8	Phthisis; white spot on pericardium.
180	52	74	8	0	Fever.
181	53	101	10	12	Phthisis.
182	56		10	8	Fever.
183	58	66	8	0	Gangrene of leg from obliteration of aorta.

WEIGHTS AND MEASUREMENTS

No.	Age.	Sex.	Weight of Body.	Weight of Heart.	Girth of Right Ventricle.	Girth of Left Ventricle.	Length of Cavity of Right Ventricle.	Length of Cavity of Left Ventricle.	Thickness of Walls of Right Ventricle.			Thickness of Walls of Left Ventricle.		
									Base.	Mid Pt.	Apex.	Base.	Mid Pt.	Apex.
	Yrs.		lb.	oz. dr.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
1	25	F.		9 0	54	40	44	32	4	4	2	4	4	2
2	5	F.		3 12						2		4	4	2
3	15	M.		10 0	54	42			2	to	$5\frac{1}{2}$	$3\frac{1}{2}$	to	$4\frac{1}{2}$
4	20	M.		12 0	66	42			4	7	3	6	5	3
5	$8\frac{1}{2}$	F.		6 8	54	42			4	3	1	5	6	2
6	45	M.	149	17 8	56	58	47	52	2	2	2	6	9	3
7	33	M.		12 8	60	54	42	40	$1\frac{1}{2}$	$1\frac{1}{2}$	$1\frac{1}{2}$	6	6	$1\frac{1}{2}$
8	35	M.	78	10 12	54	48	42	36	3	3	2	6	8	3
9	44	M.	127	12 12	54	50	48	40	2	$1\frac{1}{2}$	1	5	7	2
10	40	M.		12 8	60	50	48	41	$2\frac{1}{2}$	2	1	$5\frac{1}{2}$	6	3
11	50	M.		14 0	60	56	48	40	2	2	2	4	7	3
12	65	M.		12 8			36	36	3	$2\frac{1}{2}$	1	7	7	$2\frac{1}{2}$
13	65	M.	137	12 0	48	39	51	40	2	2	1	6	8	2
14	65	M.		40 12	96	86	62	51	3	$2\frac{1}{2}$	2	8	11	$3\frac{1}{2}$
15	71	M.		21 0	72	66	48	42	2	3	$1\frac{1}{2}$	10	11	$2\frac{1}{2}$
16	72	M.		14 0	76	54	51	36	3	$4\frac{1}{2}$	$1\frac{1}{2}$	6	6	$2\frac{1}{2}$
17	78	M.		18 0	54	66	42	36	3	5	2	6	9	4
18	60	F.		14 0	60	60	48	42	2	2	2	5	7	$2\frac{1}{2}$
19	62	M.		14 8				36	2	3	$1\frac{1}{2}$	8	9	4
20	62	M.		16 0			42	36		$2\frac{1}{2}$		$6\frac{1}{2}$	7	$3\frac{1}{2}$
21	24	M.		15 8	62	62	50	49	2	$1\frac{1}{2}$	$\frac{1}{2}$	$7\frac{1}{2}$	$7\frac{1}{2}$	$1\frac{1}{4}$
22	75	F.		10 12	48	45	40	38	2	2	2	8	8	3
23	33	M.		17 12	54	62	60	48	2	3	2	6	10	3
24	32	M.		16 0			56	42	2	$2\frac{1}{2}$	$\frac{3}{4}$	4	7	$1\frac{1}{2}$
25	42	M.		16 0	53	63	50	44	$1\frac{3}{4}$	$2\frac{1}{4}$	1	$6\frac{1}{2}$	$5\frac{1}{2}$	2

OF DISEASED HEARTS.

Thickness of Septum.	Circumference of Apertures.				Cause of Death, and Form of Disease.
	Tricuspid.	Pulmonic.	Mitral.	Aortic.	
lines.	lines.	lines.	lines.	lines.	
	51	39	45	30	Chronic bronchitis, with deformed spine.
	39		33	25	Abnormal septum in right ventricle.
	39	13	36	33	Abnormal septum in right ventricle ; aorta arising from both ventricles ; pulmonary or. contracted.
	45	12	36	30	Great contraction of pulmonary orifice, with open foramen ovale.
	42	36	39	19	Open foramen ovale ; thickening of valves.
11	58	43	56	39	Double pneumonia ; no valvular disease.
	60	48	51	39	Plenopneumonia ; some tendency to excavation at apex of left ventricle.
	60	42	48	39	Diseased hip ; malformation by defect of aortic valves, with thickening of valves.
7	42	35	39	34	Fever.
$6\frac{1}{4}$	54	48	54	42	Fever and bronchitis ; slight thickening of valves.
5	60	48	51	42	Erysipelas of head ; slight thickening of aortic and mitral valves.
6	54	48	48	39	Intussusception ; valves somewhat thickened, and aorta dilated.
6	57	42	45	39	Fever, with icterus and hæmorrhage from bowels ; no material valvular disease.
10	63	54	60	42	Sudden death ; slight atheromatous degeneration of aortic and mitral valves, and of aorta.
8	60	42	51	36	Bronchitis ; thickening of aortic and mitral valves ; dilatation of aorta.
4	60	45	54	36	Chronic bronchitis ; atheromatous disease of aorta and pulmonary artery ; thickening of valves.
	60	45	54	39	Scalp wound ; thickening of aortic and mitral valves; great dilatation of aorta.
	60	39	45	33	Some thickening of aortic valves ; disease and dilatation of aorta ; fatty degeneration.
	57	42	48	33	Contraction of aortic orifice, and thickening of the valves ; rupture of septum ventriculorum.
	54	42	48	36	Mass of bone projecting into the aortic orifice ; valves ossified ; recent pericarditis.
	57	45	51	42	Apoplexy ; aortic valves much thickened and decurtated ; atheromatous dis. of mitral valves and of aorta.
					Extreme contraction of aortic orifice from malformation and ossification of valves ; death from strangulated hernia.
	60	42	54	30	Injury of aortic valves from violent exertion.
	60	48	60	42	Incompetency of aortic valves, with recent endocarditis and fibro-cartilaginous degeneration of muscular structure.
	60	45	51	36	Extensive disease of aortic valves ; obstruction and incompetency ; aortic disease.

WEIGHTS AND MEASUREMENTS

No.	Age.	Sex.	Weight of Body.	Weight of Heart.		Girth of Right Ventricle.	Girth of Left Ventricle.	Length of Cavity of Right Ventricle	Length of Cavity of Left Ventricle.	Thickness of Walls of Right Ventricle.			Thickness of Walls of Left Ventricle.		
										Base.	Mid Pt.	Apex.	Base.	Mid Pt.	Apex.
	Yrs.		lb.	oz.	dr.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
26	50	M.		23	0	66	60	66	50	$2\frac{1}{2}$	$3\frac{1}{2}$	$2\frac{1}{2}$	7	7	3
27	55	M.		34	0	90	81	72	60	$2\frac{1}{2}$	5	2	10	$5\frac{1}{2}$	1
28	56	M.		15	4	58	55	48	36		$3\frac{1}{2}$		6	5	$3\frac{1}{2}$
29	57	M.		11	8	44	60	38	38	2	3	1	7	7	$2\frac{1}{2}$
30	46	F.	91	16	0			52	52	2	2	$1\frac{1}{2}$	6	7	3
31	27	F.		8	8	60	48	42	36	2	2	1	4	5	2
32	23	F.		10	8	59	47	44	39	3	2	1	5	5	2
33	39	F.		15	12	75	52	51	42	2	3	$1\frac{1}{2}$	5	5	2
34	40	F.		12	8	66	50	48	42	2	2	1	6	7	3
35	68	F.		12	0	54	48	45	36	2	2	2	5	5	2
36	8	F.		10	8	60	48	36	36	2	2	$1\frac{1}{2}$	5	5	3
37	11	F.		7	8	41	45	33	33	$1\frac{1}{2}$	2	$1\frac{1}{2}$	5	$6\frac{1}{2}$	$2\frac{1}{2}$
38	12	F.		12	0	54	60	36	42	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	5	5	$2\frac{1}{2}$
39	18	F.		13	8	72	54			2	2	1	6	6	3
40	39	F.		22	0						4		9	11	3
41	63	F.		22	0	55	55	43	42	2	2	$1\frac{1}{2}$	7	10	2
42	37	F		9	0					$1\frac{1}{2}$	$1\frac{1}{2}$	$1\frac{1}{2}$	4	4	4

OF DISEASED HEARTS—*continued.*

Thickness of Septum.	Circumference of Apertures.				Cause of Death, and Form of Disease.
	Tricuspid.	Pulmonic.	Mitral.	Aortic.	
lines.	lines.	lines.	lines.	lines.	
	60	48	54	45	Retroversion and perforation of aortic valves; some disease of mitral and of aorta.
	60	51	60	45	Incompetency from adhesion of valves and dilatation of orifice; aortic disease.
	54	45	54	39	Incompetency; recent endocarditis.
9	48	39	42	36	Softening of brain and apoplexy; old pericarditis; thickening and incompetency of aortic valves; dilatation of aorta.
		37		29	Thickening and decurtation, and incompetency of aortic valves; aorta dilated and diseased.
4	57	39	39	30	Peritonitis, from passage of gall-stones. Pericardium entirely adherent from old attachments; mitral valve thickened and rigid.
6		29	22	25	Great contraction of aperture, and induration and thickening of mitral valve.
5	54	39		33	Sudden death. Very great contraction of mitral aperture, and induration and thickening of valves.
5	40	30	24	27	Fever. Great contraction of mitral aperture, and some thickening of aortic valves.
	54	36		30	Great contraction of mitral aperture, and induration and thickening of valves.
	36	33	45	30	Free regurgitation through mitral aperture, from dilatation of the orifice; valves slightly opaque and thick. L. aur.; 1½ to 2 l. thick.
5	42	30	36	30	Combined aortic and mitral disease, chiefly mitral; sequence of rheumatism of two years' duration.
	48	33	36	30	Great aortic and mitral disease; sequence of rheumatic fever 9 years before.
5	42	36	42	27	Thickening and contraction of mitral, with some of aortic aperture and valves; old pericarditis; sequence of rheumatism 15 months before.
	51	39		30	Very great contraction of mitral aperture, with thickening of the aortic valves; aorta atheromatous.
	51	32	43	35	Contraction of mitral aperture; crescentic thickening of aortic valves; tricuspid valve thickened; chronic bronchitis.
	21	34	18	30	Very great contraction of tricuspid and mitral valves; slight thickening of aortic valves.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
43	14		16 12	Dilatation and hypertrophy of walls of left ventricle, without valvular disease; recent lymph on pericardium.
44	18		28 0	Destruction of aortic valves occasioning incompetency; malformation.
45	18		12 8	Pericarditis; purulent deposits after amputation.
46	22		12 0	Compound fracture.
47	25		12 0	Laceration of walls of heart from injury.
48	22		12 0	Phthisis; some thickening of tricuspid valve; hypertrophy and dilatation of right ventricle.
49	24		14 8	Combined mitral and aortic valvular disease.
50	23		13 0	Pneumonia; hypertrophy of left ventricle, without valvular disease.
51	21		24 0	Incompetency of aortic valves, with thickening of mitral valves.
52	25		12 0	Phthisis.
53	29		14 4	Left ventricle hypertrophied and dilated; aorta dilated.
54	26		15 0	Incompetency of aortic valves, with thickening and opacity of mitral.
55	26		13 0	Delirium tremens; some thickening and opacity of left valves.
56	27		15 0	Fever; chronic bronchitis of left valves and emphysema.
57	29		12 8	Phthisis; no valvular disease.
58	29		14 0	Cholera; great contraction, thickening, and induration of mitral valve.
59	38		21 0	Aneurism of septum of left ventricle, with hypertrophy and dilatation of both ventricles.
60	38		12 0	Phthisis and pneumonia; some opacity of pulmonary valves.
61	39		21 0	Apoplexy; great hypertrophy of left ventricle, but no valvular disease; aorta much dilated and diseased.
62	32		12 8	Phthisis; no valvular disease.
63	33		21 0	Incompetency of aortic valves, from malformation by defect; thickening; morbus renum.
64	36		13 0	Pneumonia; hypertrophy of left ventricle; white spots on pericardium; no valvular disease.
65	38		15 0	Chronic bronchitis and emphysema; no valvular disease.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.		Cause of Death, and kind of Disease.
	Years.	lbs.	oz.	dr.	
66	30		24	0	Phthisis, pneumonia ; aneurism of transverse arch ; no valvular disease.
67	37		14	8	Delirium tremens ; slight opacity and thickening of left valves.
68	34		16	0	Fever ; no valvular disease.
69	32		14	8	Incompetency of aortic valves ; diseased kidneys and spleen.
70	38		19	0	No material valvular disease ; dilatation of aorta.
71	38		11	8	Phthisis ; aorta dilated and diseased ; some thickening of walls of right ventricle.
72	34		20	0	Hypertrophy and dilatation of right ventricle, without valvular disease.
73	30		14	8	Pneumonia ; hypertrophy and dilatation of left ventricle, without valvular disease.
74	30		13	8	Chronic bronchitis ; no valvular disease.
75	36		23	8	Aortic valves incompetent ; some thickening of mitral and diseased aorta.
76	36		17	8	Mitral valvular disease ; some thickening of aortic valves ; hypertrophy, and dilatation both of right and left ventricles ; hemiplegia.
77	33		14	0	Morbus renum ; recent pericarditis ; no material valvular disease.
78	34		22	4	Incompetency of aortic valves ; slight thickening of mitral.
79	36		12	0	Fever ; no valvular disease.
80	36		24	0	Incompetency of aortic valves.
81	32		18	0	Old pericarditis and disease of aorta, without material valvular disease ; dysentery after typhoid fever.
82	40		12	0	Aortic valvular obstruction ; malformation.
83	40		16	0	Recent pericarditis ; aortic and mitral valves somewhat thickened ; aorta dilated and diseased.
84	40		15	0	Fever, with jaundice ; some thickening of mitral valves, and hypertrophy of left ventricle.
85	44		15	8	Phthisis after fever ; no valvular disease.
86	45		11	8	Fever ; white spots on pericardium ; some thickening and opacity of aortic and mitral valves.
87	42		14	12	Chronic bronchitis ; some thickening of aortic and mitral valves.
88	45		13	8	Fever ; hypertrophy, with dilatation of left ventricle ; no valvular disease.
89	43		13	8	Chronic bronchitis and emphysema ; no valvular disease.
90	40		16	0	Pericardium universally adherent ; aortic valves healthy ; mitral somewhat thick.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
91	42		13 6	Fever ; no valvular disease.
92	43		14 8	Recent pericarditis ; chronic bronchitis; hypertrophy of right and left ventricles.
93	44		17 4	Old pericarditis ; no valvular disease ; great hypertrophy of left ventricle.
94	45		16 4	Chronic bronchitis ; thickening of mitral and aortic valves ; dilatation of aorta.
95	46		10 0	Phthisis ; white spots on pericardium ; no valvular disease ; aneurism of aorta.
96	44		20 0	Obstructive and regurgitant disease of aortic valves; rupture of aorta ; dissecting aneurism.
97	40		16 8	Obstruction of regurgitant disease of aortic valves.
98	50		11 12	Pleuropneumonia ; old pericarditis ; thickening of mitral valve.
99	51		13 0	Pericarditis ; bronchitis ; aneurism of coronary artery ; dilatation of cavities, with atheromatous disease of aortic and mitral valves, and of aorta.
100	51		13 12	Fever; no valvular disease.
101	51		21 8	Extensive thickening, and contraction of mitral, aortic, tricuspid and pulmonic valves ; thickness, 4, 5, and 2 lines ; great hypertrophy of right ventricle, old pericarditis ; morbus renum.
102	53		11 8	Chronic bronchitis ; white spots on pericardium and adhesions.
103	55		23 0	Aortic valves incompetent and some disease of aorta.
104	52		12 8	Chronic phthisis ; no valvular disease.
105	58		12 8	Hemiplegia ; no valvular disease.
106	50		23 8	Aorta much dilated and diseased ; no material valvular disease.
107	59		14 8	Aortic valves thickened and ossified, and aorta diseased.
108	53		10 8	Compound dislocation of astragalus ; recent pericarditis.
109	57		13 0	Cut throat—no valvular disease ; dilatation and hypertrophy of left ventricle ; aorta dilated and diseased.
110	52		14 8	Morbus renum ; mitral thickened; left ventricle hypertrophied.
111	51		21 8	Mitral valvular disease, with some thickening of aortic valves and diseased aorta.
112	58		23 0	Much obstruction at aortic orifice and some regurgitation ; mitral valve slightly thickened ; aorta dilated.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
113	67		13 0	Chronic bronchitis and emphysema ; thickening of mitral valve and hypertrophy of left ventricle.
114	60		11 8	Ulcer of stomach ; pneumonia ; opacity of aortic and mitral valves; aorta dilated and diseased.
115	57		16 0	Phthisis ; valves healthy, but mass of bone projecting into upper part of orifice from walls of aorta.
116	65		18 4	Apoplexy; thickening of mitral and aortic valves ; hypertrophy and dilatation of left ventricle; diseased aorta.
117	62		14 0	No valvular disease ; hypertrophy with dilatation.
118	71		23 0	Diseased prostate ; no valvular disease ; hypertrophy, with dilatation of right and left ventricle; aorta dilated.
119	74		19 8	Convulsions ; thickening and ossification of aortic valves.
120	78		21 0	Thickening and ossification of aortic valves; some thickening of mitral; dilatation of aorta.
121	80		14 8	Fractured arm and delirium tremens ; white spots on pericardium ; no material valvular disease.

WEIGHT OF DISEASED HEARTS, FEMALES.

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
		lbs.	oz. dr.	
122	5 mos.	11	2 4	Malformation ; aperture in septum of ventricles, with contraction of tricuspid aperture.
123	14 yrs	95	11 12	Morbus renum.
124	17		11 0	Fever ; some thickening of mitral valve, with hypertrophy and dilatation of left ventricle.

WEIGHT OF DISEASED HEARTS, FEMALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
125	16		11 8	Lobular pneumonia; thickening and opacity of mitral valve.
126	24		15 8	Morbus renum; no material valvular disease.
127	29		11 8	Fatty degeneration of heart and kidneys.
128	21		18 0	Great contraction of mitral orifice and thickening of valves, as well as of pulmonic and tricuspid; aortic orifice small.
129	28		12 0	Morbus renum; coma; hypertrophy, with dilatation of left ventricle.
120	29		8 8	Phthisis; aneurisma aortæ.
131	34		15 0	Phthisis; pneumonia; general hypertrophy and dilatation without valvular disease.
132	34		12 8	Chronic bronchitis and emphysema; hypertrophy of right ventricle; no material valvular disease.
133	36		12 0	Fever; no valvular disease.
134	36		13 12	Morbus renum; no valvular disease.
135	30		12 8	Phthisis; no valvular disease; aorta greatly dilated.
136	40		17 0	Combined mitral, aortic, and tricuspid valvular disease, chiefly aortic; regurgitation.
137	44		23 0	Great incompetency of aortic valves; orifice dilated and valves thickened and indurated.
138	50		11 8	Chronic bronchitis; no valvular disease; hypertrophy and dilatation of right ventricle.
139	53		23 0	Combined mitral and aortic valvular disease; pleurisy.
140	57		12 8	Chronic phthisis.
141	60		14 0	Dissecting aneurism of descending aorta; no valvular disease.
142	60		17 8	Dilatation of aorta; and great hypertrophy and dilatation of left ventricle, without valvular disease.
143	66		18 0	Injury of head; no valvular disease; dilatation of aorta; hypertrophy and dilatation of left ventricle.
144	66		8 12	Recent endocarditis destroying aortic valves; pneumonia; meningitis.
145	67		18 8	Contraction of aortic orifice, and induration and thickening of valves.
146	70		13 0	Apoplexy; aortic valves thick and rigid; aorta dilated and atheromatous; hypertrophy of left ventricle.

PART I.—*Weights of the Healthy Heart.*

TABLE I.

Showing the weight of the healthy heart in males and females, at different periods of life, as deduced from 155 observations; males 94; females 61.

Ages.	Males.		Females.	
	Nos. Weighed.	Mean Weight.	Nos. Weighed.	Mean Weight.
Years.		oz. dr.		oz. dr.
15 to 20	9	8 $2\frac{6}{9}$	9	8 $1\frac{6}{9}$
20 to 30	27	9 $0\frac{4}{27}$	21	8 $10\frac{9}{21}$
30 to 40	31	9 $7\frac{20\cdot5}{31}$	19	8 $13\frac{16}{19}$
40 to 50	9	9 $11\frac{1}{9}$	5	9 3
50 to 60	15	9 12	6	9 $7\frac{3}{6}$
60 to 70	3	10 $13\frac{1}{3}$	1	7 0
	94		61	

TABLE II.

Showing the different weights of the healthy heart in males and females, between 20 and 55 years of age, deduced from 125 observations.

Weights.	Males.		Females.	
	Nos. Weighed.	Per Centage.	Nos. Weighed.	Per Centage.
oz. oz. drs.				
5 to 6	2	5·2	3	12·2
6 to 7	2		3	
7 to 8	10	39·4	10	44·8
8 to 9	20		12	
9 to 10	18	39·4	13	42·8
10 to 11	12		8	
11 to 11 12	12	15·7		
	76		49	

	oz. dr.
Average weight of the healthy heart in 76 males from 20 to 55 years of age, ...	9 $8\frac{56\cdot5}{76}$
Ditto ditto in 49 females ditto	8 $13\frac{8}{49}$

From the above table all organs weighing more than 11 oz. 12 dr. are excluded; if the cases, in which, without other disease, it weighed 12 oz. be calculated, the observations are extended to 83 males and 51 females, and the average weight of the heart in males is 9 oz. $11\frac{81\cdot5}{83}$ dr.; in females 8 oz. 15 dr.

Table III.

Weight of the healthy heart, in persons from 20 to 55 years of age, in cases of phthisis, and of all others, exclusive of bronchitis and morbus renum, specifying the average weight in acute and chronic diseases, separately.

	Nos. Weighed	Mean Weight	Extremes	Mean Age
Males.		oz. dr.	oz. dr. oz. dr.	Years.
Phthisis, . .	27	9 $3\frac{11\cdot5}{27}$	11 0 and 6 $4\frac{1}{2}$	34·03
All others, . .	44	9 $9\frac{30}{44}$		36
Acute cases only,	30	9 $13\frac{3\cdot5}{30}$	11 12 „ 8 8	36
Chronic cases only,	14	8 $14\frac{10\cdot5}{14}$	11 8 „ 5 0	35·9
Females.				
Phthisis, . .	17	8 $6\frac{11}{17}$	11 0 „ 5 8	34·4
All others, . .	23	9 $2\frac{9}{23}$		31·4
Acute cases only,	16	9 $5\frac{3}{16}$	11 0 „ 7 4	34·3
Chronic cases only,	6 [1]	8 $9\frac{2}{6}$	9 8 „ 8 0	30·2

The above table contains only the cases in which the weight of the heart did not exceed 11 oz. 12 dr.; if those in which it weighed 12 oz. be included, the result will stand as follows:—

Males,	Phthisis,	30	.....	9 oz. $7\frac{16\cdot5}{30}$ dr.
„	Others,	47	.....	9 „ $12\frac{6}{47}$ „
Females,	Phthisis,	17	.....	8 „ $6\frac{1}{7}$ „
„	Others,	24	.....	9 „ $4\frac{7}{24}$ „

Table IV.

Weights of the heart in all cases of phthisis, bronchitis, and morbus renum, compared with the weights of the healthy heart in all other cases, hypertrophy, pericarditis, and morbus aortæ excluded.

Weights.	Phthisis.		Bronchitis.		Morbus Renum.		All others.	
	Males.	Fem.	Males.	Fem.	Males.	Fem.	Males.	Fem.
5 oz. to 6 oz		3					2	
6 „ to 7 „	2	2				1		
7 „ to 8 „	6	3			3	2	1	5
8 „ to 9 „	2	4		2	1		17	7
9 „ to 10 „	10	1				3	8	9
10 „ to 11 „	6	4	1	1	1	1	4	2
11 „ to 12 „	4		1	1	1	2	10	
12 „ to 13 „	3	2	2	1				
13 „ to 14 „			3			1		
14 „ to 15 „		1	3		1			
15 „ to 16 „	1					1		
Mean age, . .	34·1	35·3	46·4	38·4	30·1	33·4	36·5	31·4

[1] One case, in which the disease causing death is not stated, is excluded from this calculation.

Table V.

Weights of the heart in males and females under 20 years of age.

Age.	Males. No.	Mean Weight.	Heaviest.	Lightest.	Age.	Females. No.	Mean Weight.	Heaviest.	Lightest.
		oz. dr.	oz. dr.	oz. dr.			oz. dr.	oz. dr.	oz. dr.
6 months	1	0 13½			7 days	1	0 13		
10 months	1	1 10			2½ months	1	1 3		
2 and 2½ yrs.	2		2 12	2 10	3 months	1	1 0		
3 years	1	2 15			1 year	1	2 0		
4 and 4½ yrs.	2		4 4	2 9	1¾ years	1	1 8		
3 years	1	3 0			3 years	1	1 10		
8 „	1	6 4			5 „	1	2 14		
10 „	1	4 4			6 „	1	2 12		
11 „	5	5 10⅘	8 8	4 0	7 „	2		3 0	2 3
14 „	2		9 0	6 8	8 „	2		5 1	3 0
15 „	2		8 0	6 0	11 „	1	3 14		
16 „	3	6 5⅓	7 0	5 8	13 „	1	6 2		
17 „	1	8 8			17 „	2		7 0	4 8
19 „	1	11 0			18 „	3	9 5	10 8	7 15
					19 „	2		8 0	7 12
	24					21			

Of the observations included in this table, all but 11 were cases of acute disease, and the results would not have been materially different had the cases of chronic disease been excluded from the calculation. The difference in the weights of the heart at different ages, in young persons, is chiefly due to the relative vigour of the children and the greater or less rapidity with which their growth proceeds; and in this respect the rate of growth of the heart corresponds with that of the brain, and indeed of the body generally.

INFERENCES.

1. The weight of the healthy heart in persons from 20 to 55 years of age, averages in males 9 oz. 8 dr., and in females 8 oz. 13 dr. The mean difference between the weights of the organ in the two sexes being thus 11 drachms. This calculation is based only upon the observations in which the heart weighed less than 12 oz., if those in which it attained the weight of 12 oz. be added, the average becomes somewhat higher, or 9 oz. 11 dr. in males, and 8 oz. 15 dr. in females, and the difference between the weights in the two sexes is 12 drachms.

Calculations of this description must always be to a certain extent arbitrary, for as the heart is found in some cases to be considerably above its ordinary weight, without the proportion of its walls and cavities being materially altered, or the organ being otherwise diseased, it is not easy to say at what point it ceases to be healthy. The estimates of the weight of the healthy heart here given differ in some degree from those of other observers. Bouillaud[1] infers that the average weight of the heart is from 8 to 9 oz. (systeme usuel, or from 8 oz. 10 dr., to 9 oz. 11 dr. avoird.), and may amount to 10 or 11 oz. (10 oz. 12 dr., and 11 oz. 14 dr. avoird.), but his observations refer to only 14 cases, and include individuals of both sexes and of various ages from 16 to 38. The mean weight of 8 adult hearts given in his table is 9 oz. 2 dr. (9 oz. 15 dr. avoird.), the extremes being 7 oz. 6 dr. (8 oz. 5 dr. avoird.), and 11 oz. 3 dr. (12 oz. 4 dr. avoird.)

Dr Clendinning[2] estimates the average weight of the male heart in persons from 20 to 60 years of age, at $8\frac{1}{2}$ oz. avoird.; the female heart at $7\frac{2}{5}$ oz. avoird., and his calculations are based upon 118 observations (58 males and 60 females). Dr Reid's[3] researches give the average weight of the heart in 89 males, from 25 to 55 years of age, as 11 oz. 1 dr., and in 53 females, as 9 oz.; the difference being $2\frac{1}{2}$ ounces.

2. The weight of the healthy heart differs in different forms of disease, being greater in persons who die after short periods of illness, and less in those who have suffered from protracted and emaciating diseases.

This result is illustrated in table 3, from which it will be seen that the weight of the heart in adult males who sank under acute diseases, averaged 9 oz. 13 dr., while in those who had died of chronic diseases, phthisis, bronchitis, and morbus renum being excepted, it averaged 8 oz. 14 dr. In females the heart weighed in those who had died of acute diseases, 9 oz. 5 dr., and in those who sank from chronic diseases, 8 oz. 9 drachms.

Dr Reid found the mean weight of the heart in 9 adult males, who had died from accidents, to be 12 oz. 6 dr., while, as before stated, in the adult males who sank from disease, it weighed on an average 11 oz. 1 dr.; but in both these calculations the weights of hearts are included, which exceeded what I have regarded as the limit of health, either in cases of accident or disease.

3. The general inference to be deduced from these observations, would appear to be that in adult males who have died from acute diseases, or from the effects of accidents, the ordinary weight of the healthy heart is from 9 to 11 oz. avoird., and in those who have

[1] Maladies du Cœur, 2me Ed., 1841, t. i., p. 25.
[2] Med. Chir. Trans., vol. xxi., 1838, p. 55.
[3] Lond. and Ed. Monthly Journal of Medical Science, 1843. Also, Physiological, Pathological, and Anatomical Researches, Ed. 1848, p. 376.

died from chronic diseases from 8 to 10 oz. In females the ordinary weight of the heart in acute cases may be regarded as from 8 to 10 oz., and in chronic diseases from 7 to 9 oz. Occasionally, however, in persons of small and delicate frame, who have died from exhausting diseases, such as cancer of the stomach or chronic affections of the liver, the heart will be found to weigh only 5 or 6 oz.; and in large and powerful persons, of the male sex, who have been suddenly killed or have died after a very short illness, the organ may weigh 12 oz. or perhaps even more, without exceeding the limit of health.

4. The heart increases in weight with the advance of life.

This inference is borne out by the facts collected in table 2, for the exception formed by the single case in advanced life, in females, is unimportant. It is also confirmed by the observations of Drs Clendinning and Reid, and by a table, compiled from their data, in Dr Sharpey's edition of Quain's Anatomy.[1] It is not, however, clear, to what period of life this increase extends, and whether in very advanced age, there is not, as is shown to be the case with the brain, a more or less marked decline in weight.[2] Hospitals do not afford the means of ascertaining satisfactorily the weight of the heart in aged persons, for comparatively few old people die in them, and of these a large proportion labour under some form of cardiac disease; it is therefore very desirable that medical men having charge of public charitable institutions for the aged, should direct their attention to this point. From some observations which I have made, I have been led to suppose, that when entirely free from disease, the heart, so far from continuing to increase in weight to the term of life, undergoes a decrease in advanced age; and I have more especially noticed this to be the case in females, in whom the decrease of weight of the brain in advanced life, is noticed at the earliest period and to the greatest extent. The opposite result arrived at by other observers, I cannot but suspect to have been due to organs, not strictly healthy, having been included in their calculations.

5. *Weight of the Heart in Phthisis, Bronchitis, and Disease of the Kidneys.*—The weight of the heart in persons who have died of phthisis, is less than in those who have sank from other diseases; but the decrease of weight after death from that disease is usually not so marked as in persons who have died from other chronic affections, unconnected with disease of the lungs.

This inference is deduced from the facts which appear in table 3, there being, however, an exception as to the relative weight of the heart in phthisis and in other chronic diseases, in females; but the latter cases are too few in number to form a satisfactory basis for generalization.

[1] Vol. ii., p. 1124.

[2] See observations by Dr Reid and myself, in Lond. and Ed. Journal, 1843, and 1846, and by myself in the Lond. Journal of Medicine.

The conclusion here drawn as to the relative weight of the heart in phthisis and in other diseases, differs considerably from that arrived at by Dr Clendinning, that the weight of the heart in phthisis is greater than its weight in other diseases, in which the organ is healthy, I have therefore carefully compared the two series of observations with the view of ascertaining the cause of the discrepancy. After re-calculating the data collected by Dr Clendinning, separating from them all the weights which exceeded the limit of health, and classifying the remainder according to the acute or chronic character of the disease occasioning death, the results are as follows :—

		oz.	dr.
Males.	Weight of the heart in cases of phthisis	9	9
,,	,, ,, of acute disease only	9	1
,,	,, ,, of chronic disease only	8	0
Females.	Weight of the heart in cases of phthisis	7	13
,,	,, ,, of acute diseases only	8	8
,,	,, ,, of chronic diseases only	7	8

It will thus be seen that it is only in reference to males that the observations collected by Dr Clendinning, bear out the inference which he has drawn from them, that in phthisis the heart is heavier than in other diseases; while in females, the results are similar to those deduced from the larger series of data contained in the paper, and show that the weight of the heart in phthisis, is ordinarily less than in acute diseases, but greater than in other chronic diseases, unconnected with obstruction in the lungs. The correct explanation most probably is, that in phthisis there is a tendency to enlargement of the heart, which, though counteracted to a greater or less extent by the emaciation, prevents the organ declining so much in weight as would have been the case were no source of obstruction present. This inference, though different both from the conclusions of MM. Louis and Bizot and Dr Clendinning, is quite compatible with the correctness of their observations, and the different results arrived at by them and by myself, is doubtless due to their having compared the condition of the heart in phthisis with its state in *all* other diseases, instead of with its weight and size in *chronic* diseases *only*. There is certainly nothing in phthisis which prevents the heart becoming considerably hypertrophied, as is shown by two cases included in Dr Clendinning's paper in which it weighed 13 oz. 4 dr. and 13 oz. 8 dr. in males; and by others in my own in which it weighed 15 oz. 8 dr. in a male, and 15 oz. in a female.

6. The facts collected in table 4 show, that in chronic bronchitis the heart ordinarily acquires a considerable increase of weight. In two cases only of persons who had died of this disease did the organ possess its average weight in chronic diseases, while in 9, 8 males and one female, it exceeded the weight regarded as the extreme limit

of health; and, in three of them, all of whom were males, it weighed from 15 to 16 oz. Though the number of cases contained in the table is so few, yet the results are too marked to be regarded as accidental.

7. The condition of the heart in cases of disease of the kidneys is also shown in table 4; and from this it will be seen that in 7 cases, or 4 males and 3 females, the weight of the organ was below the average in other chronic diseases; while in 11, 3 males and 8 females, it exceeded the average, attaining in one male the weight of 14 oz. 8 dr., and in 2 females of 13 oz. 12 dr., and 15 oz. 8 dr. The facts, therefore, confirm, to a certain extent, the observations of Dr Bright,[1] that the disease of the kidneys has a tendency to produce enlargement of the heart, unconnected with valvular disease, or disease of the aorta.

Part II.—*Weight of Diseased Hearts.*

Table VI.

Weight of the heart in cases in which it exceeded the limit of health, but in which there was no obvious source of obstruction; and in cases of old adhesions of pericardium, and disease of the aorta (including atheromatous deposit, dilatation, and aneurism), unconnected with valvular defect.

Weights.	Hypertrophy without Obstruction.		Adhesions of the Pericardium.		Aortic Disease.			
	Males.	Age.	Males.	Age.	Males.	Age.	Females.	Age.
8 oz. 8 dr.							1	29*
10 „ 0 „					1	46*		
11 „ to 12 oz.			1	50	2	38* and 60		
		Mean.						
12 „ to 13 „	5	37·2			2	57 and 65	1	30
13 „ to 14 „	5	50					1	60
14 „ to 15 „	4	46·7			1	29		
15 „ to 16 „	1	34	1	40	1	45		
16 „ to 20 „	2	45 and 34	2	44 and 32	4	38 to 78	2	60 and 66
						Mean 56·4.		
20 „ to 24 „					5	30* to 71		
						Mean 52·2.		
40 „ 12 dr.	1	65						

[1] Guy's Hospital Reports, vol. i., 1836, p. 380.

In 11 out of the 18 cases of hypertrophy included in the table it is stated in the reports that there was no valvular disease, and in the others any slight thickening, opacity or atheroma which existed was inadequate to explain the increase of weight. In all the cases, also, there was an absence of material chronic disease of the lungs and aorta.

In two females the hearts weighed 11 oz. and 12 oz. in persons of 17 and 36 years of age: in the former, though there was slight thickening of the mitral valves, it was inadequate to explain the unusual weight of the heart for a person of the age, and in the second there was no valvular disease.

From the table all the cases in which there was recent pericarditis are excluded, and one, in which the pericardium was universally adherent by old attachments, but in which there was chronic bronchitis, is also omitted. In the case of the male, 32 years of age, in whom the heart weighed 18 oz., there was not only adhesion but extensive ossification of the pericardium. The cases marked with an asterisk were aneurisms. In two of these in which the heart weighed 8 oz. 8 dr. and 24 oz., the patients were phthisical, and this was also the case in the female in whom the heart weighed 12 oz. 8 dr., and the aorta was dilated.

Table VII.

Weight of the heart in cases of aortic valvular disease, whether obstructive, regurgitant, or both.

Weights.	Males.			Females.		
	Obstruct.	Obst. and Regurgit.	Ages.	Obstruct.	Obst. and Regurgit.	Ages.
			Years.			Years.
8 oz. and 12 dr.					1	66
10 „ and 12 „				1		75
11 oz. to 12 oz.	1	1	40 and 57			
12 „ to 13 „				1		70
14 „ to 15 „	2	2	44.7			
15 „ to 16 „	3	3	45.5		1	46
16 „ to 20 „	1	3	47.7	1		67
20 „ to 25 „	1	8	44.5		1	44
28 „		1	18			
34 „		1	55			

In several of these cases there was also dilatation or atheromatous disease of the aorta, and, generally, some thickening of the mitral valve.

Table VIII.

Weight of the heart in cases of mitral valvular disease.

Weights.	Males.	Age.	Females.	Age.
		Years.		Years.
8 oz. and 8 dr.			1	27
10 „ and 8 „			1	23
11 „			1	17
12 „ and 12 oz. 8 dr.			2	62 and 40
14 „ and 14 „ 8 „	2	29 and 52		
15 „ and 12 dr.			1	39
17 „ and 8 „	1	36		
18 „			1	21

Of the cases included in this table the male in whom the heart weighed 14 oz., and the two females in whom it weighed 8 oz. 8 dr. and 11 oz. died respectively of attacks of cholera, peritonitis and fever.

In one case not entered in the table, there was free regurgitation through the left auriculo-ventricular aperture from dilatation, without disease of the valves; and the organ weighed 10 oz. 8 dr.; the subject being a girl 8 years of age. In several cases the tricuspid valves were also slightly thickened.

Table IX.

Weight of the heart in cases of combined aortic and mitral valvular disease.

Weights.		Males.	Age.	Fem.	Age.
oz.	dr.				
7	8			1	11
12	0			1	12
13	8			1	18
14	8	1	24		
17	0			1	40
18	4	1	65		
21	8	1	51		
22	0			2	39 and 63
23	0			1	53

In the three cases in which the organ attained the greatest weight, the predominant valvular disease was mitral. In that in which the heart weighed 21 oz. 8 dr., the pericardium was also universally

adherent, and there was considerable thickening of the tricuspid valves, and disease of the aorta; and in some of the other cases the tricuspid valves were also slightly thickened.

INFERENCES.

1. The weight of the heart may very greatly exceed the limit of health without the existence of any material valvular, aortic, or pulmonic disease, adhesions of the pericardium or other obvious source of obstruction.

Thus it will be seen in Table VI. that in 18 males, the weight of the heart exceeded 12 oz., and in 5 cases it amounted to 15 oz., 16 oz., 17 oz. 8 dr., 20 oz. and 40 oz. 12 dr., the last being the heaviest heart weighed. In all these cases there existed no source of obstruction, either in the heart itself or in the aorta or lungs, which appeared sufficient to explain the great increase of weight, and in 11 of them it is expressly stated in the reports, that there was no valvular disease. In the case in which the heart weighed 40 oz. and 12 dr., though there was slight atheromatous change in the aortic and mitral valves and in the coats of the aorta, the lungs were healthy, and nothing sufficient to explain the hypertrophy was detected.

The process by which the heart attains this great increase of weight in cases where there is no marked obstruction to the circulation, is most probably a slow one, but the data given show that there is no relation between the age of the individual and the amount of hypertrophy. The heart is found to weigh very considerably above the average in comparatively young persons, and the more extreme degrees of hypertrophy are not more common in advanced age, than in persons at earlier periods of life.

Only two cases which could be regarded as instances of simple hypertrophy in females are included in the tables, and, in neither of these did the organ exceed 12 oz. in weight;—facts which show the infrequency of this form of disease in females, and the very slight increase of weight which the female heart attains, when there is no serious source of obstruction.

2. The tables contain only four cases in which there were old adhesions of the pericardium, without valvular, aortic, or pulmonic disease, or recent pericarditis;—and this number is too small to warrant any decided inferences. It will, however, be seen that in one instance the weight of the heart did not exceed the limit of health, being only 11 oz. 12 dr., while, in the other three cases, it was very considerably greater, or 16 oz., 17 oz. 4 dr., and 18 oz. These facts do not confirm the opinion that adhesions of the pericardium, so far from leading to enlargement of the heart, rather tend to produce atrophy; but I have examined other hearts in which the pericardium was entirely adherent by old cellular attachments, without the organ being larger than natural. The person in whom the heart weighed 18 oz. was a male, 32 years of age, and, in this in-

stance not only was the pericardium much thickened and adherent, but there was also a considerable formation of bone.

3. The heart most generally acquires very great increase of weight in cases of disease of the aorta, whether consisting in atheromatous deposit and ossification in the coats, dilatation, or sacculated aneurism; but in none of the cases of this description did the heart attain so great a weight as in one of those in which there existed no obvious source of obstruction to explain the enlargement.

The last column in Table VI. shows that in one female the weight of the heart did not attain the average, being only 8 oz. 8 dr. In one male also it only slightly exceeded the average, being 10 oz.; and in two other males it did not exceed the limit of health, weighing in each 11 oz. 8 dr. In the remaining cases (seventeen in number—thirteen males and four females), it very considerably passed that point, weighing from 12 oz. to 24 oz. in males, and from 12 oz. 8 dr. to 18 oz. in females; the greatest weights being, in males, 16 oz. 4 dr., 18 oz. 4 dr., 19 oz., 21 oz., 23 oz., 23 oz. 8 dr., and 24 oz.; and in females, 17 oz. 8 dr., and 18 oz. It must, however, be observed, that in the male in which the heart weighed only 10 oz., in a second in which it weighed 24 oz., and in the female in whom it weighed 8 oz. 8 dr. in addition to the aneurisms, the lungs were tuberculous; and in the female in whom the heart weighed 12 oz. 8 dr., the aorta was dilated and the lungs tuberculous, so that, in these instances, the hypertrophy of the heart resulting from the aortic obstruction, may have been to some extent counteracted by the emaciation from phthisis.

As in the cases of hypertrophy only, so also in this form of disease, there is no just relation between the ages of the individuals and the increase in the weight of the heart.

4. The heart usually becomes very greatly enlarged in cases of aortic valvular disease, whether obstructive or regurgitant, or both; and the increase of weight is apparently greater in these cases than in those of disease of the aorta; but in no instance did the organ acquire from this cause so great a weight as in one of the cases of hypertrophy unconnected with obvious obstruction.

These inferences are deduced from Table VII., from which it appears that in males the weight of the heart exceeded the average in all the cases; but, in two instances, in one of which there was obstruction only, in the other regurgitation, it did not pass the extreme limit of health, weighing only 11 oz. 8 dr., and 12 oz. In seven other cases of obstruction its weight ranged from 14 to 21 oz., the greatest weights being 16 oz., 16 oz., 19 oz., and 21 oz.; and in eighteen cases of regurgitation, from 14 oz. to 34 oz.; the greatest weights being 20 oz., 21 oz., 22 oz. 4 dr., in three cases 23 oz., 23 oz. 8 dr., in two cases 24 oz., 28 oz., and 34 oz.

In females, the increase of weight in cases of aortic valvular disease also obtains, though to a less degree. If a case in which one of the segments was destroyed by acute endocarditis, and in which death

rapidly ensued, and the organ weighed only 8 oz. 12 dr., be excluded, the lightest heart in the table weighed 10 oz. 12 dr., or within the limit of health; yet, in this instance, there was most extensive thickening and ossification of the valves, probably originating in malformation, and causing extreme obstruction: and as the subject of the case was 75 years of age, the progress of the disease must have been very slow. In two other cases of obstructive disease the heart weighed 13 oz. and 18 oz. 8 dr., and in two cases of regurgitation 16 oz. and 23 oz.

It will thus be seen that, though the weight of the heart in cases of aortic valvular disease was greater than in the cases of disease of the aorta, the heaviest heart in the former weighed nearly 7 oz. less than in the remarkable case of hypertrophy without valvular or aortic disease before mentioned.

The table also shows that the weight of the heart in cases of aortic valvular disease is by no means proportionate to the age of the subjects; the organ having attained a weight of 28 oz. in a boy of 18, and 23 oz. in a female of 44.

It is not possible to ascertain the respective effects of obstructive and regurgitant disease in increasing the weight of the heart, for the two generally coexist, and the latter is very frequently only the final stage of the former; the influence which is due to each cause cannot therefore generally be assigned. In the case, however, in which the heart weighed $17\frac{3}{4}$ oz. in a male 33 years of age, the disease was the result of rupture of the angle of attachment of two of the valves, from violent muscular exertion, 27 months before death, and though there must have been some obstruction to the flow of blood from the ventricle into the aorta, the great increase of weight must have been chiefly due to the incompetency of the valves. A still more remarkable instance of enlargement from regurgitation, is afforded by a case of rupture of the valves, related by Dr Quain, in which the heart acquired the weight of $22\frac{1}{2}$ oz. in a period of two years which elapsed between the occurrence of the accident and the death of the individual. In both these cases the heart was most probably healthy till the accidents occurred.

In all cases of regurgitant disease, on whatever cause dependent, the increase of weight probably takes place rapidly; thus, in the boy of 18, in whom the organ weighed 28 oz., the duration of illness had only been two years and ten months; on the contrary, in cases of obstructive disease, the enlargement of the heart is probably a slower process. A striking example of the length of time during which obstruction may exist without the heart acquiring great increase of weight, is afforded by the case of the elderly female, before referred to, in whom the organ weighed 10 oz. 12 dr., but in this instance, it seems probable, that a decrease of weight may have occurred with the advance of life. The case, however, with many others which might be quoted, shows the absence of any just relation between the degree of obstruction and the amount of hypertrophy.

5. In cases of mitral valvular disease the heart ordinarily exceeds the limit of health, but does not attain so great an increase of weight as in cases of aortic or aortic valvular disease.

Of the cases included in Table VIII., three, that of the male in whom the heart weighed 14 oz., and two females, in whom it weighed 8 oz. 8 dr., and 11 oz., death did not result from the direct effect of the disease, but from other causes. If, therefore, these cases be excepted, it will be seen that in one female the heart weighed only 10 oz. 10 dr., or within the limit of health, while in the six other cases it greatly exceeded that point, weighing, in males, from 14 oz. 8 dr. to 17 oz. 8 dr., and in females from 12 oz. to 18 oz. These weights are, however, much less than those in the cases of aortic and aortic valvular disease, and still less than that of the heart in which the hypertrophy was not dependent on obvious obstruction. It will also be observed, that in these cases there is not the great difference in weight between the male and female heart which is observed in other forms of disease, but the cases are so few, that the inferences cannot be fully depended upon.

The weight of the heart in cases of mitral valvular disease bears no certain proportion to the age of the individual, as is shown by the subject of the case in which the heart weighed 18 oz. being a female only 21 years of age.

6. In disease of the mitral and aortic valves combined, the weight of the heart is generally intermediate between that in cases of aortic and mitral disease alone; the organ being lighter than in aortic valvular disease, and heavier than in mitral valvular disease.

It will be seen from Table IX., that if the case in which the heart weighed 7 oz. 8 dr., in a girl 11 years of age, be omitted, in all the observations, including two females of 12 and 18 years of age, the weights of the heart exceeded the extreme limit of health; and that in three males it weighed from 14 oz. 8 dr. to 21 oz. 8 dr., and in three females from 17 oz. to 23 oz.

7. The tables contain only one observation of the weight of the heart in extensive disease of the mitral and tricuspid valves. In this instance the disease was probably congenital. The organ weighed 9 oz., and the subject was 37 years of age, but she was peculiarly ill-formed and stunted, and did not appear more than 16 or 18 years old.

8. In the tables the weights of four hearts are given in which there were congenital malformations. Of these cases that which presented the least important deviation from the natural structure, was one in which the foramen ovale was unclosed, in a girl 8½ years of age, and the heart weighed 6 oz. 8 dr. A second case was that of a girl aged 5 years, who died of hemorrhage from the throat or stomach, during an attack of scarlet fever, and the only malformation was the existence of a septum dividing the infundibular portion of the right ventricle from the sinus, and the heart weighed 3 oz. 12 dr. In a third case, that of a boy 15 years of age, in addition to a similar source of obstruction in the right ventricle, the

pulmonary artery was small and its valves malformed, and there was an aperture in the septum ventriculorum, by which the aorta freely communicated with the right, as well as with the left ventricle; in this instance the heart weighed 10 oz. In three other cases (two of which are not included in the tables), the pulmonary orifice was the seat of obstruction. In one of these, a young man, 20 years of age, it was very greatly contracted from the adhesion and thickening of the valves, and the foramen ovale was largely open, and the heart weighed 12 oz. In another, a female, 19 years of age, the pulmonary orifice was greatly contracted from malformation of the valves, the tricuspid valves were also diseased; the aorta arose from both ventricles, and the ductus arteriorus was still pervious; and the heart weighed 17½ oz. The subject of the third case was an infant nearly 12 months old, in which the pulmonary artery was entirely obliterated, the aorta arose from both ventricles, and the blood was transmitted to the lungs through the ductus arteriosus. The heart in this case weighed 3 oz. 8 dr. The last instance of malformation is one in which, in a female infant aged 5 months, there were two small apertures in the septum ventriculorum, with thickening of the tricuspid valves, causing contraction of the orifice, and the heart weighed 2 oz. 4 dr. A comparison of the weights of the heart in these cases with that of the healthy organ in infancy and early life, as given in Table V., will show how greatly it exceeded the ordinary weight in healthy children at similar ages; indeed, in the female 19 years of age, it very considerably exceeded the extreme limit of health in adults.

General Remarks on the Weight of the Diseased Heart.

The weights of the diseased heart in the above tables are, so far as I am aware, the most extensive series yet published; indeed with the exception of M. Bouillaud, I do not know any writer who has specially collected the weights of diseased organs. M. Bouillaud's observations are, however, very few in number, amounting to only 11 cases in which the heart was hypertrophied, and 7 which he regarded as cases of atrophy. Of the latter, in five males, the weights ranged from 5 oz. 6 dr. in a person 18 years of age, to 7 oz. in one of 20, and the causes of death were marasmus, typhoid, and the effects of having taken nitric acid. The two females were aged 45 and 30, and died respectively of schirrus of the pylorus, and of disease of the liver, and the hearts weighed 4 oz. 5 dr., and 5 oz. 13 dr. Of the observations in the paper, the lightest male heart weighed 5 oz. in a person 53 years of age, who died of cirrhosis hepatis, combined with disease of the kidneys. In a second case the heart weighed 6 oz. in a person 39 years of age, who died of cancer of the pylorus, and in a third, the heart had the same weight in a man 29 years old, the cause of whose death is not recorded. The lightest female hearts weighed 5 oz. 8 dr. in two persons 25 and 35

years of age, and 5 oz. 12 dr. in a third, 21 years old, and all these died of phthisis.

The weight of the heart in the cases of hypertrophy collected by M. Bouillaud, are as follows :—

Hypertrophy apparently without Valvular disease.

1.	Male,	60	years of age,	.	.	12 oz. 15 dr.	avoirdupois.
2.	„	50	„	.	.	13 „ 11 „	„
3.	Female,	75	„	.	.	14 „ 10 „	„
4.	Male,	67	„	.	.	18 „ 3 „	„
.	„	69	„	.	.	17 „ 10 „	„

Aortic Valvular obstruction and probably Regurgitation.

6. Female, 54 years of age, . . 24 oz. 4 dr. avoirdupois.

Mitral Valvular Regurgitation.

7. Male, 47 years of age, . . . 22 oz. 3 dr. avoirdupois.

Mitral Valvular obstruction and Regurgitation.

8.	Female,	56	years of age,	.	.	12 oz. 15 dr.	avoirdupois.
.	Male,	53	„	.	.	16 „ 3 „	„

Combined Mitral and Aortic Valvular disease.

10.	Female,	17 to 18	years,	.	.	11 oz. 14 dr.	avoirdupois.
11.	Male,	40	years of age,	.	.	18 „ 10 „	„

In the tables in the paper the weight of only one heart is given in which there was regurgitation through the left auriculo-ventricular aperture, without disease of the valves, and in this case the patient was a girl only 8 years of age ;—so that no comparison can be instituted between the weight of the heart in M. Bouillaud's 7th observation and my own ; but, with this exception, it will be seen that the weights of the heart, in the different forms of disease in the comparatively small number of observations published by M. Bouillaud, correspond with those deduced from my own much more numerous data ; though in none of his observations did the heart attain so great a weight as in several of my own.[1]

In Dr Clendinning's tables the lightest male heart weighed 6 oz., in three cases, in persons who died of ascites, mania, and phthisis, at the ages of 24, 41, and 43 ; and the lightest female hearts weighed 4 oz. 8 dr., 5 oz., 5 oz. 8 dr., and 5 oz. 4 dr., in persons aged respectively 32, 31, 20, and 26, who died of epilepsy with ulceration of the intestines, chronic bronchitis, typhus, and phthisis.

[1] In M. Bouillaud's treatise the weights are given in grammes, and also in the pound, ounce, and gros. The pound being the poids de marc of 7560·6 grains troy, and the ounce and gros respectively the medicinal weight of 472·5 and 59·1 grains. The weights given above, are avoirdupois, calculated from the gramme, as equal to 15·434 troy grains.

The heaviest hearts in males weighed 24 oz., 24 oz. 8 dr., 26 oz.[1] and 40 oz. 8 dr., in persons 48, 34, 33, and 33 years of age; but, except in reference to the last case, in which there was aneurism of the aorta near the heart, the nature of the disease causing death is not reported. It is curious that the heart in the last case should be precisely the same weight as the heaviest which I have weighed. In females the greatest weights given by Dr Clendinning are 15½ oz. and 17½ oz. in persons 23 and 50 years of age; but though the foramen ovale is stated to have been open in the former case, the precise kind of disease is not mentioned in either.

M. Bouillaud[2] concludes his observations with the remark that, in cases of hypertrophy, the weight of the heart may amount to nearly three times that of the organ of usual dimensions, and five times that of the most atrophied heart. It will, however, be seen, from the data here published, that this estimate is too low. As in males the average weight of the heart is 9 oz. 8 dr. (or, if the cases in which the weight of the organ was 12 oz. be included, 9 oz. 11 dr.), and the extreme weights of the organs examined were 5 oz. and 40 oz. 12 dr., the weight in the most hypertrophied heart was upwards of four times that of the average organ, and eight times that of the lightest heart weighed. In females, the range of weight in the heart, though sufficiently remarkable, is less than in males. The mean weight of the heart in females is 8 oz. 13 dr. (or 8 oz. 15 dr.), and the extreme weights are 5 oz. 8 dr. and 23 oz., so that the most hypertrophied heart was nearly three times the weight of the average organ, and four times that of the lightest heart. The greatest weight recorded was in a case of hypertrophy without material valvular disease; and the lightest hearts were found in cases of cancer of the pylorus and disease of the liver—an observation which accords with the results of M. Louis[3] as to the small size of the heart in cases of this description.

M. Bouillaud, in referring to the statement of Lobstein, that a heart examined by him weighed 2 lbs. (or 34 oz. avoird.), suggests that the organ had most probably been weighed before being deprived of its coagulum and blood; but this weight is so far from being extreme, that there is no reason to doubt the correctness of M. Lobstein's report. Dr Hope[4] mentions having examined, at St George's Hospital, a heart which weighed 2½ lbs., which, if, as is probable, the weight employed was avoirdupois, equals the heaviest organs weighed by Dr Clendinning and myself.

[1] This case is evidently twice inserted in the Tables.
[2] Traité Clinique des Maladies du Cœur 2me Ed. 1841, Tome i. p. 68.
[3] Sydenham's Society's Translation, p. 52.
[4] Diseases of the Heart, 3d Ed., 1839, p. 238.

TABLE X.—Showing the dimensions of the healthy heart *in males*, specifying in separate columns, the cases of phthisis, bronchitis, and of other diseases, unconnected with pulmonary disease, together with the dimensions of the heart in persons dying of chronic diseases *only*, unconnected with disease in the lungs. The table includes the mean of all the observations, together with the dimensions of the lightest and heaviest hearts examined.

	Phthisis.			Bronchitis.			Other.			Chronic cases only.		
	Mean.	Lightest	Heaviest	Mean.	Lightest	Heaviest	Mean.	Lightest	Heaviest	Mean.	Lightest	Heaviest
	lines.	lines.	lines.	lines	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of the heart, . .	94·5	92	97	118·6	116	130	103·1	98	87	104·4	98	120
Girth of right ventricle, . . .	49·5	50	49	67·3	66	76	55·6	54	48	57·2	54	60
„ left ventricle, . . .	45·	42	48	51·3	50	54	47·5	44	39	47·2	44	60
Length of the cavity of right ventricle,	43·3	36	48	50	51	51	42·5	42	51	40	42	34
„ „ left ventricle,	37·3	36	39	40·6	45	36	37·	36	40	34·4	36	32
Thickness of walls of right ventricle base,	2	2	2	2·5	2	3	1.81	2	2	1·8	2	2
„ „ midpoint,	2	2	2	3	2·5	4·5	1 93	2.25	2	1·95	2.25	2
„ „ apex, .	1·58	1·25	1.5	1·3	1·5	1·5	1·37	2	1	1·5	2.	1.5
„ „ of left ventricle base,	5·3	6	4	5·5	5	6	5·1	5	6	4·7	5	5
„ „ midpoint,	6·3	5	7	5.8	5·5	6	5·95	6	8	5·8	6	6
„ „ apex, .	2·3	2·5	2	2·5	2	2·5	2·5	3	2	2·8	3	3
„ of septum, . . .	5·8	5	6	5·08	5	4	5·77	6	6	5·6	6	5
Circumf. of right auriculo-ventric. aper.,	49·3	51	43	58·6	62	60	54·4	57	57	57·7	57	60
„ of pulmonic, . . .	38·6	39	29	46	45	45	40	39	42	40 8	39	42
„ of left auriculo ventricular,	45·3	48	37	54	54	54	44·3	42	45	47·2	42	48
„ of aortic, ...	34·6	36·	26	39	39	36	35.5	33	39	36·4	33	39

The observations in cases of phthisis are three; the ages 20, 31, and 37, and the weights of the heart, 6 oz. 4½ dr., 9 oz. 4 dr., and 11 oz.

The number of cases of bronchitis, etc., is also three, the ages 38, 40. 72, and the weights of the heart 11 oz., 12 oz. 8 dr., and 14 oz.

The number of other cases is twelve, the ages ranging from 23 to 66, and the mean age 32·8; the weights of the heart range from 6 oz., 9⅓ dr. to 12 oz., and the mean weight is 9 oz. 4·7 dr.

The number of cases of chronic disease only are five, the ages 23 to 35, the weights of the heart from 6 oz. 9½ dr. to 9 oz. 1·5 dr.

TABLE XI.—Dimensions of the healthy heart *in females*, specifying separately the dimensions in cases of phthisis, bronchitis, and of all other diseases, unconnected with pulmonary disease.

	Phthisis.			Bronchitis.			Other.		
	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of the heart, . .	102·6	90·	108·	93·	94·	92·	107·8	108.	120·
Girth of the right ventricle, . .	56·	48·	60·	53·	54·	52·	59·	60·	72·
„ left ventricle, . .	46·6	42·	48·	40·	40·	40·	48·8	48·	48·
Length of the cavity of the right ventricle,	45·3	42·	48·	42·6	44·	42·	43·7	42·	48·
„ „ left ventricle,	36·6	27·	42·	34·6	32·	36·	37·5	36·	39·
Thickness of walls of right ventricle, base,	1·6	1·5	1·5	3·	4·	2·	1·81	1·	2·
„ „ midpoint, .	2·3	1·5	3·	2·87	4·	2·	1·87	1·5	1·5
„ „ apex, .	1·3	1·5	1·5	1·87	2·	1·	1·18	·5	1·
„ „ of left ventricle, base,	4·8	6·	3·5	5·2	4·	4·	4·9	4·	5·
„ „ midpoint, .	5·6	7·	5·	6·	4·	7·	5·8	5·	6·
„ „ apex, .	3·16	2·	2·	2·25	2·	2·	2·7	2·5	2·5
„ of septum, ...	3·5						4·7	4·	6·
Circumf. of right auriculo-ventricular apert.	54·	51·	60.	47·5	51·	40·	52·1	54·	51·
„ of pulmonic, ...	42·	42·	45·	37·7	39·	34·	39·	39·	42·
„ of left auriculo-ventricular, .	45·	48·	48·	44·2	45·	42.	45·5	45·	45·
„ of aortic, ...	34·	33·	36·	34·5	39·	39.	34·	33·	36·

The cases of phthisis in the table are three in number, the ages 20, 24, and 66 ; the weights of the heart 7 oz., 9 oz. 5 dr., and 10 oz. 11 dr.

The cases of bronchitis are four, the ages 25 to 60, mean 47, and the weights of the heart 9 oz. to 11 oz., mean 10 oz. 2·7 dr.

The other cases, unconnected with pulmonary disease, are eight in number ; the ages 20 to 64, the mean age 35·3, and the weights of the heart 7 oz. to 9 oz. 10 dr., the mean weight 8 oz. 4 dr.

The cases of chronic disease only have not been separated in this table, as they are only two in number.

PART III.—*Dimensions of the Healthy Heart.*

INFERENCES.

1. In males, the average circumference of the healthy heart, in persons of adult age, dying of diseases unconnected with any pulmonary affection, measured externally, was 103·1 lines, and the extreme circumference in different cases was from 87 to 120 lines.

The girth of the right ventricle averaged 55·6 lines, that of the left ventricle 47·5 lines; but these dimensions varied greatly in different cases, the girth of the right ventricle having a range of from 48 to 60 lines, that of the left ventricle of from 39 to 60 lines.

The mean length of the cavity of the right ventricle was 42·5 lines, that of the left ventricle 37 lines. These measurements also varied—the length of the right ventricle ranging from 34 to 51 lines, that of the left ventricle from 32 to 44 lines.

The parieties of the right ventricle had an average thickness at about the middle of its anterior wall of 1·93 lines; and decreased slightly in width towards the pulmonic orifice, and about half a line towards the apex, having thus a thickness of 1·81 lines in the former situation, and of 1·37 at the apex.

The walls of the left ventricle had a medium width at about the mid-point of 5·95 lines, at the base of 5·1 lines, and at the apex of 2·5 lines. The thickness of the parieties of the ventricles exceeded or fell short of these dimensions, without passing the limit of health, in the right ventricle by half a line, and in the left by about a line and a half. The thickness of the right ventricle ranged from 1·5 to 2·5 lines; that of the left ventricle from 5 to 8 lines.

The septum of the ventricles had an average thickness at the middle of 5·77 lines, and its width ranged from 3·75 to 7 lines, generally corresponding in thickness with the parieties of the left ventricle.

The right auriculo-ventricular aperture had a mean circumference of 54·4 lines; the pulmonic orifice of 40 lines; the left auriculo-ventricular aperture of 44·3 lines, and the aortic orifice of 35·5 lines; but the dimensions of the orifices also varied greatly in different cases, the right auriculo-ventricular aperture having a range of from 45 to 60 lines, the pulmonic of from 34 to 45; the left auriculo-ventricular aperture of from 38 to 51, and the aortic orifice of from 28 to 48.

2. In females the dimensions of the heart are ordinarily less than in males, but the difference is rather apparent in the diminished thickness of the walls of the ventricles and in the circumference of the orifices, than in the capacity of the cavities.

In adult females the circumference of the heart in persons dying of various affections, unconnected with disease in the lungs, had a

mean of 107·8 lines, and in different cases it attained extremes of 87 to 120 lines.

The girth of the right ventricle averaged 59 lines, and varied from 49 to 72 lines in different cases. The girth of the left ventricle averaged 48·8 lines, and ranged from 38 to 60 lines.

The medium length of the cavity of the right ventricle was 43·7 lines, and it ranged from 34 to 48 lines. The left ventricle had a mean length of 37·5 lines, and extremes of 32 and 44 lines.

The thickness of the walls of the right ventricle was near the base 1·81 lines, at the midpoint 1·87 lines, and near the apex 1·18 lines; and the greatest thickness varied in different cases from 1·5 to 2·5 lines. The parieties of the left ventricle had a medium width near the base of 4·9 lines, at the midpoint of 5·8 lines, and at the apex of 2·7 lines, the greatest width ranging in different cases from 5 to 8 lines.

The septum averaged 4·7 lines in thickness, and its width in various cases was from 4 to 6 lines.

The right auriculo-ventricular aperture had an average circumference of 52·1 lines, the pulmonic orifice of 39 lines, the left auriculo-ventricular aperture of 45·5 lines, and the aortic orifice of 34 lines; and the variations in the capacity of the different orifices, was, in the right auriculo-ventricular aperture from 42 to 57 lines, in the pulmonic from 33 to 42, in the left auriculo-ventricular from 37 to 54, and in the aortic orifice from 29 to 36. It will thus be seen that while in the two sexes the dimensions of the whole heart, and the size of the cavities did not differ materially, in females the walls were somewhat thinner, and the orifices less capacious than in males. The comparison is not, however, a just one, for, while a larger proportion of the males the dimensions of whose hearts are given, died of chronic diseases,—the females, with two exceptions, all died after short periods of illness. Had the respective proportions of cases of acute and chronic disease in the two sexes been similar, the dimensions of the heart would most probably have shown a greater difference. It would, however, appear that in the female heart the cavities are somewhat larger, relative to the thickness of the walls, than in males.

3. From these observations it thus appears that the girth of the right ventricle, measured externally, exceeded that of the left, in males by 1-6th, and in females by 1-5th. The length of the cavity of the right ventricle exceeded that of the left ventricle, in males by about 1-7th, and in females by about 1-6th. In both sexes the thickness of the walls of the right ventricle is about 1-3d that of the parieties of the left ventricle. The thickness of the septum is intermediate between that of the external walls of the right and left ventricles.

In males the pulmonic aperture was about 1-8th more in circumference than the aortic; the left auriculo-ventricular orifice about 1-4th more than the aortic; and the right auriculo-ventricular opening one-half larger. In females, the difference between the circumference of the aortic and of the other orifices, is somewhat greater.

4. In the two sexes, the length of the heart measured from the base to the apex was greater than its breadth measured across the broadest part. These dimensions were taken in comparatively few cases: in these the mean length of the organ was 49 lines, and the mean breadth 43 lines.

The walls of the right auricle measured at the middle of the sinus had a medium thickness of half a line to one line, but from the arrangement of the *musculi pectinati*, it is not easy to estimate their width. The walls of the left auricle had an average thickness of 1 to 1½ lines.

The right coronary artery in the cases in which it was measured had a circumference of from 5 to 8 lines; ordinarily this artery gives off the anterior branch immediately after its origin, but not unfrequently that vessel arises separately from the right sinus of Valsalva. The left coronary artery is generally larger than the right. In one case I have seen the two coronary arteries arise by a common trunk, and in another both arteries arose from the same sinus. The orifice of the coronary vein has generally a circumference of about 10 lines.

The fossa of the foramen ovale has a mean size of 8·6 lines in its longest, and 6·8 lines in its shortest diameter, but the dimensions vary considerably. The fold on the left side ordinarily overlaps the isthmus by 2 or 3 lines, and, when a valvular opening remains, it is usually 2 to 3 lines wide.

5. The heart is ordinarily greater in persons who have died of bronchitis and other pulmonary diseases, phthisis excepted, than in persons who have died of affections in which there was no obstruction in the lungs. The enlargement generally consists in hypertrophy and dilatation of both the right ventricle and left ventricles;—the cavity of the right ventricle being enlarged, and its walls increased in thickness, while the left ventricle, though increased in size, retains the usual thickness of its walls.

The circumference of the heart in cases of bronchitis in males, averaged 118·6 lines, and ranged from 110 to 130 lines.

The girth of the right ventricle averaged 67·3 lines, and ranged from 60 to 76 lines; that of the left ventricle averaged 51·3 lines, and ranged from 50 to 54 lines.

The mean length of the cavity of the right ventricle was 50 lines, and it ranged from 48 to 51 lines; that of the left ventricle had a medium of 40·6 lines, and extremes of 36 and 45.

The walls of the right ventricle were on the average 3 lines in width, and ranged from 2 to 4·5 lines, and in one case not included in the tables, of chronic bronchitis,—with curvation of the spine,—in a young man 17 years old, the parieties of the ventricle were fully 5 lines thick. The walls of the left ventricle had an average thickness of 5·8 lines, and ranged from 5·5 to 6 lines at the broadest part. The septum had a medium thickness of 5·08 lines, and a range of from 4 to 6 lines.

The right auriculo-ventricular aperture had a medium circumfer-

ence of 58·6 lines, and extremes of 54 to 62 lines. The pulmonic orifice a medium of 46 lines, and extremes of 45 and 48. The left auriculo-ventricular aperture, in the three cases measured, was 54 lines, and the aortic had a medium of 39 lines, and extremes of 36 and 42 lines. It will thus be seen, that the girth of the right ventricle was $\frac{1}{3\cdot5}$ greater than that of the left, and the length of the cavity of the right ventricle 1-4th greater than that of the left. The capacity of all the orifices was above the average, and the pulmonic exceeded the aortic aperture by 1-6th, and the mitral exceeded the aortic by 1-3d, while the tricuspid orifice was, as usual, one-half larger than the aortic. A comparison of the dimensions of the heart in females in the cases of bronchitis, with those of the healthy organ, show similar results, but in only one case did the walls of the right ventricle attain a thickness of 4 lines, and in this instance there was chronic bronchitis with curvature of the spine, and the subject was 25 years of age.

The fossa of the foramen ovale in cases of chronic bronchitis, becomes considerably enlarged, and the valve is not unfrequently dilated and protruded into the left auricle. In one case, of which I have the preparation, a large sac is thus produced. When the fossa is dilated, the portion of the fold which ordinarily overlaps the isthmus is frequently drawn down, so that it may be found scarcely to reach above the edge of the opening; and when the valve has not become adherent the aperture might so be reopened; but, though I have seen several cases of very enlarged fossa with an unadherent valve, I have never met with one in which the fœtal aperture was restored.

Though the disproportion between the size of the right and left ventricles of the heart, and especially the greater size of the pulmonic than of the aortic aperture, exists at all ages, except in very early life, or before birth, and the experiments of Legallois have shown that is not dependent on the mode in which death takes place, it is certainly much aggravated in all cases in which there exists any obstruction to the transmission of the blood through the lungs. This is most marked in cases in which the obstruction has been of long duration, but I have seen the orifice of the pulmonary artery very considerably expanded in persons who have died of acute bronchitis and pneumonia, after short periods of illness.

6. In persons who have died of phthisis, the heart is usually smaller than in those who have sank from other diseases; but the decrease in size is less than obtains in some other forms of disease, and is chiefly due to the diminution in the size of the cavities and in the capacity of the orifices, while both the right and left ventricles are thicker than in other chronic cases where there is no pulmonary disease.

The average circumference of the heart in males who died of phthisis was 94·5 lines, and the extremes 92 and 97.

The girth of the right ventricle averaged 49·5 lines, and its ex-

tremes were 49 and 50; that of the left ventricle had a mean of 45 lines, and extremes of 42 and 48.

The length of the right ventricle had an average of 43·3 lines, and extremes of 36 and 48; that of the left ventricle averaged 37·3, and its extremes were 36 and 39.

The walls of the right ventricle had a mean thickness of 2 lines; those of the left ventricle, a mean of 6·3 lines, and extremes of 5 and 7 lines.

The right auriculo-ventricular aperture had a medium circumference of 49·3 lines, and extremes of 43 and 51. The pulmonic orifice a mean of 38·6 lines, and extremes of 29 and 48. The left auriculo-ventricular aperture a medium of 45·3, and extremes of 37 and 51; and the aortic aperture a mean of 34·6, and extremes of 26 and 42. It will thus be seen, that while the ventricular walls retain their usual thickness, or exceed it, the length of the cavities, and the capacity of the orifices are much less than usual, and the difference is the more apparent when the dimensions of the heart in phthisis are compared with those of the organ in other chronic affections, unconnected with disease in the lungs. It will also be observed that the proportion between the cavities and orifices on the two sides of the heart is not materially different from that which obtains in the healthy organ; the right side being, however, somewhat less than natural, relatively, to the left. As some of the results here deduced differ from those of M. Bizot, who states the heart in phthisis to be smaller in all its dimensions, it is right to state that the data here analysed are much fewer than those collected by that observer.

7. The observations are too few to warrant definite conclusions as to the different dimensions of the heart in persons who have died of acute and chronic diseases, but they would appear to indicate that the chief difference consists in the diminished thickness of the walls of the left ventricle.

It will be observed, from the annexed statements (Table XII.), that though the series of observations collected by M. Bizot, Dr Reid, Dr Ranking, and myself, correspond to a considerable extent, there are differences between them. M. Bizot's measurements of the width of the walls are generally the least, those of Dr Reid the largest. In the dimensions of the orifices, Dr Ranking's observations show the smallest size, and Dr Reid's the largest. The seat at which M. Bizot took the dimensions of the aortic and pulmonic orifices differed from that selected by Dr Ranking, the former having measured the opening at the level of the free border of the valves, while the latter took the dimensions on the line of their insertions; the precise point measured by Dr Reid is not mentioned. All three observers estimated the circumference of the orifices after they had been laid open, whereas my own measurements were made while the apertures were entire, by graduated balls passed through them, and therefore indicate their absolute capacity. After division the fibrous ring of the arterial orifices,

TABLE XII.—For the purpose of comparison I have prepared the following table, which exhibits the mean dimensions of the *healthy heart* as deduced by M. Bizot, Dr Reid, and Dr Ranking. I have not thought it necessary to include M. Bouillaud's observations, for they are few in number, include persons of different ages and of both sexes, and are very imperfect as to the measurements in each case.

	Bizot [1]		Dr Reid.[2]		Dr Ranking.[3]		Own Cases.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of Heart, ...					113·	99·5	102·7	104·9
Length of cavity of right ventricle, . . .	37·2	34·					43·3	44·
„ „ left „ . . .	34·39	31·03					37·6	37·1
Thickness of walls of right „ base, . .	1·9	1·6					1·85	1·86
„ „ „ „ midpoint, .	1·4	1·29	2	1·75	1·8	1·5	1·98	2·0
„ „ „ „ apex, . .	1·03	·93					1·42	1·3
„ „ left „ base, . .	4 5	4·3					5·15	4·9
„ „ „ „ midpoint, .	5·15	4·8	5·6	4·65	6·25	5·5	6·	6·
„ „ „ „ apex, . .	3·7	3·4					2·4	2·5
Thickness of septum, . . „ . .	4·9	4·4	5·6	4·25	5·2	4·5	5·73	4·7
Circum. of right auriculo-ventricular aperture, .	54·19	48·25	59·7	55·	52·75	47·	53·9	51·
„ pulmonic, ...	32·34	30·1	42·	39·3	30·3	28·1	40·	39·3
„ left auriculo-ventricular aperture, .	45·5	41·33	52·	47·3	44·25	38·75	45·2	45·
„ aortic, ...	31·24	28·8	36·	34·	29·5	27·5	35·6	34·

The measurements of M. Bizot are calculated from the examination of the hearts of 60 males and females, from 16 to 79 years of age. The thickness of the septum refers only to persons between 30 and 50 years of age.—The observations of Dr Reid are based upon the measurement of the hearts of 16 males and 9 females, adults, weighing from 7 to 13½ oz.—Dr Ranking's conclusions are drawn from the weight of the heart in 15 males from 26 to 65 years of age, and 17 females from 18 to 62 years of age.—My own observations include the hearts of 16 males and 13 females, dying of various different forms of disease in persons from 20 to 66 years of age, in whom the hearts weighed from 6 oz. 9½ dr. to 12 oz.—Dr Reid's and Dr Ranking's observations are given in the original papers in fractional parts of an English inch ; I have here reduced them to French lines for facility of comparison.

[1] Mem. de la Soc. Med. d'Observat., t. i., p. 262, Paris, 1837. [2] L. & E. Monthly Journal, 1842 ; and Physiological, Anatomical, and Pathological Researches, Ed. 1848, p. 373. [3] London Medical Gazette, vol. xxix. ; vol. i. 1841–42, p. 903.

TABLE XII. B.—Showing the mean dimensions of the heart according to the observations of M. Bizot, Dr Reid, Dr Ranking, and myself, in fractional parts of English inches.

	Bizot.		Dr Reid.		Dr Ranking.		Own Cases.	
	Males.	Females.	Male	Females.	Males.	Females.	Males.	Females.
	inch.	inch.	inch.	inch.	inch.	inch.	inch.	inch.
Circumference of the heart,					$9\frac{27}{28}$	$8\frac{13}{48}$	$9\frac{2}{24}$	$9\frac{7}{24}$
Girth of the right ventricle,							$4\frac{22}{24}$	$5\frac{2}{24}$
„ left „							$4\frac{5}{24}$	$4\frac{7}{24}$
Length of cavity of right ventricle,	$3\frac{18}{48}$	3·					$3\frac{11}{48}$	$3\frac{13}{48}$
„ „ left „	$3\frac{2}{48}$	$2\frac{36}{48}$					$3\frac{1}{48}$	$3\frac{1}{48}$
Thickness of walls of right ventricle,	$\frac{8}{48}$	$\frac{6}{48}$	8·5	$\frac{7}{48}$	$\frac{8}{48}$	$\frac{6}{48}$	$\frac{8}{48}$	$\frac{8}{48}$
„ „ left „	$\frac{22}{48}$	$\frac{20}{48}$	$\frac{12}{24}$	$\frac{10}{24}$	$\frac{22}{48}$	$\frac{22}{48}$	$\frac{12}{24}$	$\frac{12}{24}$
„ septum,	$\frac{21}{48}$	$\frac{18}{48}$	$\frac{12}{24}$	$\frac{9}{24}$	$\frac{22}{48}$	$\frac{12}{48}$	$\frac{12}{24}$	$\frac{12}{24}$
Circumference of right auriculo-ventricular aperture,	$4\frac{38}{48}$	$4\frac{13}{48}$	$5\frac{3}{10}$	$4\frac{9}{10}$	$4\frac{34}{48}$	$4\frac{8}{48}$	$4\frac{12}{24}$	$4\frac{12}{24}$
„ pulmonic aperture,	$2\frac{42}{48}$	$2\frac{31}{48}$	$3\frac{7}{10}$	$3\frac{5}{10}$	$2\frac{36}{48}$	$2\frac{24}{48}$	$3\frac{27}{48}$	$3\frac{28}{48}$
„ left auriculo-ventricular aperture,	$4\frac{1}{48}$	$3\frac{31}{48}$	$4\frac{6}{10}$	$4\frac{2}{10}$	$3\frac{45}{48}$	$3\frac{22}{48}$	$4\frac{1}{48}$	4·
„ aortic aperture,	$2\frac{37}{48}$	$2\frac{26}{48}$	$3\frac{2}{10}$	3·	$2\frac{31}{48}$	$2\frac{22}{48}$	$3\frac{7}{48}$	3·

According to M. Bouillaud the mean circumference of the healthy heart in 10 males, from 16 to 38 years of age, is 105·37 lines; in 6 cases (4 males and 2 females) from $17\frac{1}{2}$ to 45 years of age, in which he regards the organ as atrophied, 81·5 lines. Thickness of the walls of the left ventricle, $6\frac{1}{2}$ to 7 lines, and $6\frac{1}{8}$ lines; of the right ventricle, $2\frac{1}{2}$ and $2\frac{3}{5}$ths; of the septum, 7 lines; right auricle 1 line; left auricle, $1\frac{1}{2}$ line; right auriculo-ventricular aperture, 46 and 47 lines; pulmonic aperture,[1] $31\frac{3}{4}$ and 28 lines; left auriculo-ventricular aperture, 42 and 40; aortic, $29\frac{1}{2}$ and 28 lines.

Lobstein states the weight of the heart to be from 9 to 10 oz., and the dimensions as follows:—Length, 5 inches and 6 lines; breadth at base, 3 inches; thickness of the walls of the right ventricle, $2\frac{1}{4}$ lines; of left ventricle, 7 lines; the right auricle, 1 line; the left auricle, half a line (French measures).

[1] Traité d' Anatomie Pathologique, tome ii. Paris, 1833, pp. 419 and 449.

TABLE XIII.—Showing the dimensions of the heart in males in cases of hypertrophy, unconnected with marked obstruction, in cases of obstruction at the aortic orifice or in the aorta, and in cases of incompetency of the aortic valves.

	Hypertrophy.			Obstruction at Aortic Orifice or in Aorta.			Incompetency of Aortic Valves.		
	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of heart, . . .	129·	104·	182	119·6	102·	138	124·3	104·	171
Girth of right ventricle, . . .	66·5	54	96	61·3	54	72	60·8	44	90
„ left ventricle, . . .	62·5	50	86	58·3	48	66	63·5	60	81
Length of cavity of right ventricle, .	51·2	48	62	43·	42	48	55·7	38	72
„ „ left ventricle, .	45·7	40	51	38·8	36	42	45·4	38	60
Thickness of walls of right ventricle base,	2·2	2	3	2·3	3	2	2·32	2	2·5
„ „ „ „ midpoint	2·0	1·5	2·5	2·7	3	3	3·25	3	5·
„ „ „ „ apex,	1·7	1·	2·	1·5	2	1·5	1·82	1	2·
„ „ left ventricle base,	5·7	5·	8	8·1	6	10	6·6	7	10
„ „ „ „ midpoint	8·5	7·	11	8·06	8	11	6·7	7	5·o
„ „ „ „ apex,	2·8	2·	3·5	2·47	3	2·5	2·35	2·5	1
Thickness of septum, ...	8·2	7·	10	6·6		8	7·5	9	
Circumference of right auriculo-ventricular aperture, ...	55·7	42	63	57·7	60	60	57 4	48	60
Circumference of pulmonic aperture, .	45·	35	54	44·2	42	42	45·4	39	51
„ of left auriculo-ventricular aperture, ...	51·2	39	60	49·8	48	51	53·5	42	60
Circumference of aortic aperture, .	39·2	34	42	37·7	39	36	39·	36	45

The cases of hypertrophy analysed in this table are four, in persons from 44 to 65 years, and with a mean age of 51 years. The hearts in the cases, weighed from 12 oz. 12 dr. to 40 oz. 12 dr., the mean weight being 21 oz. 4 dr.

The cases of obstruction are eight in number, in persons from 24 to 78 years of age, and with an average of 53·7. The weights of the heart range from 10 oz. 12 dr. to 21 oz., the mean being 15 oz. 2 dr.

The cases of incompetency are seven, the ages of the subjects from 32 to 57, the weights of the heart from 11 oz. 8 dr. to 34 oz., and the mean 19 oz. 3¾ dr.

TABLE XLV.—Showing the dimensions of the heart in females, in cases of obstruction at the aortic orifice, of incompetency of the aortic valves, of mitral valvular disease, and of combined mitral and aortic valvular disease.

	Obstruction at Aortic Orifice.			Incompetency of Aortic Valves.	Mitral Valvular Disease.			Combined Mitral and Aortic Disease.		
	Mean.	Lightest.	Heaviest.		Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of heart,	105·6	104·	120·		111·8	108·	127·	118·	126·	110·
Girth of right ventricle,	56·	60·	60·		62·8	60·	75·	63·5	72·	55·
„ left ventricle,	49·6	44·	60·		49·	48·	52·	54·5	54·	55·
Length of cavity of right ventricle,	42·3	39·	48·	52·	46·	42·	51·			43·
„ „ left ventricle,	37·	39·	42·	52·	39·	36·	42·			42·
Thick. of the walls of the right ventricle base,	2·16	25·	2·	2·	2·2	2·	2·	2·6	2·	2·
„ „ „ midpoint,	2·3	3·	2·	2·	2·2	2·	3·	2·6	2·	2·
„ „ „ apex,	2·	2·	2	1·5	1·3	1·	1·5	2·1	1·	1·5
„ „ left ventricle base,	6·	5·	5·	6·	5·	4·	5·	7·3	6·	7·
„ „ „ midpoint,	7·6	8·	7·	7·	5·4	5·	5·	9·	6·	10·
„ „ „ apex,	2·8	3·	2·5	3·	2·2	2·	2·	2·6	3·	2·
Thickness of septum,	5·5	6·	5·		5·	4·	5·		5·	
Circum. of right auriculo-ventricular aperture,	57·	54·	60·		51·2	57·	54·	48·	42·	51·
„ of pulmonic aperture,	36·5	34·	39·	39·	34·6	39·	39·	35·6	36·	32·
„ of left auriculo-ventricular aperture,	43·5	42·	45·		23·	39·	12·	34·3	42·	48·
„ of aortic aperture,	32·5	32·	33·	29·	29·	30·	33·	30·6	29·	35·

The cases of aortic valvular obstruction are only three in number, the subjects were 75, 75, and 60 years of age, and the hearts weighed 10 oz. 2 dr., 10 oz. 12 dr., and 14 oz. respectively.

In the single case of incompetency of the aortic valves, the subject was 46 years of age, and the heart weighed 16 oz. The cases of mitral valvular disease are five in number, the ages of the subjects ranged from 23 to 68, and the mean age was 39·4, the hearts weighed from 8 oz., 8 dr. to 15 oz. 12 dr., the mean weight being 11 oz. $13\frac{3}{5}$ dr. The cases of combined mitral and aortic valvular disease are three in number, the ages from 18 to 63, the mean being 40 years, the weights of the heart ranged from 13 oz. 8 dr. to 22 oz., and the mean weight was 19 oz. $2\frac{2}{3}$ dr. The dimensions of two cases of combined disease in females, 11 and 12 years of age, included in the general table, are excluded from this table.

especially that of the pulmonary artery, contracts, and it becomes extremely difficult, or impossible, to judge of the size, which should be assigned to the opening. The mode in which the length of the cavity of the right ventricle is estimated by M. Bizot and myself, also differs; his measurements refer to the length of a line from the base to the apex; mine, to that of a line following the course of the ventricle from the orifice of the pulmonary artery to the apex. It will be seen that the relative size of the heart in males and females in my own table differs but little, much less than is the case in the other observations. This is explained, as before mentioned, by the female hearts measured being, almost all of them, those of persons who had died of acute diseases, while the majority of the male hearts were from cases of chronic disease.

Part IV.—*Dimensions of the Diseased Heart.*

INFERENCES.

1st. The heart attained the greatest increase of size in the cases in which the hypertrophy and dilatation were unconnected with any marked disease of the valves or aorta, and the enlargement in these instances was not confined to the left cavities, but involved, though to a less degree, those of the right side also.

2d. In the cases of obstructive disease of the aortic valves, or of the aorta, the heart was also very greatly enlarged, and the enlargement involved both the left and right cavities of the organ, but was not so great as in the cases of hypertrophy unconnected with valvular or aortic disease. The increased size was also by no means proportionate to the amount of impediment to the circulation, being in some cases very great where the obstruction was only trivial, and in others slight where very great obstruction existed.

3d. In the cases of incompetency of the aortic valves, the capacity of the left ventricle was greater than either in the cases of simple hypertrophy or of obstructive disease, but the walls of the ventricle were usually less increased in thickness than in those diseases. The hypertrophy involved both sides of the heart, but the right ventricle attained a greater thickness in this, than in either of the other forms of disease.

It will be seen from the table of dimensions of the diseased heart in males, that the circumference of the organ averaged in the cases of hypertrophy, obstructive disease, and incompetency, respectively, 129, 119, and 124 lines, and attained the extremes of 182 lines, or 15 French inches, or 16 English; 138 lines, or 11½ French inches, or about 12 inches English; and 171 lines, or 14¼ French inches, or about 15 English. The girth of the right ventricle measured externally averaged, in cases of hypertrophy, 66 lines, of obstructive disease 61 lines, and of incompetency 60 lines, and attained the extremes of 96, 72 and 90 lines. The girth of the left ventricle averaged in

three several forms of disease 62, 58 and 63 lines, and attained the extremes of 86, 66 and 81 lines.

The length of the cavity of the right ventricle averaged in the three several forms of disease 51, 43 and 55 lines, and attained extremes of 62, 48 and 72 lines; the length of the left ventricle averaged 45, 38 and 45 lines, and the extreme lengths were 51, 42 and 60 lines. The walls of the right ventricle, measured at the thickest part, were 2·2 lines in width, in cases of hypertrophy, 2·7 lines in cases of obstructive disease, and 3·25 in cases of incompetency; and their minimum and maximum thickness in these several forms of disease were 2 and 3 lines, 3 lines, and 3 and 5 lines. The walls of the left ventricle averaged in width in cases of hypertrophy 8·7 lines, in obstructive disease 8·1 lines, and in incompetency 6·7 lines, and the extremes were respectively 7 and 11 lines, 6 and 11 lines, and 6 and 10 lines.

The septum had an average thickness in the cases of hypertrophy of 8 lines, and its extremes were 5 and 10 lines: in the cases of obstruction of 6·6 lines, and its extremes 6 and 8; and in the cases of incompetency, in the only two cases in which it was measured, it was 6 and 9 lines thick.

The capacity of the orifices in these diseases varied according to the form of the affection. In the cases of hypertrophy they were all above the healthy standard. The aortic orifice averaged 39 lines in circumference, and its extreme dimensions were 34 and 42 lines. The left auriculo-ventricular aperture averaged 51 lines, and its extremes were 39 and 60 lines. The pulmonic aperture averaged 45 lines, and ranged from 35 to 54 lines, and the right auriculo-ventricular aperture averaged 55 lines, and ranged from 42 to 63 lines.

In the instances of obstructive disease the aortic orifice was in some cases greater, in others less, in circumference than natural. Its average capacity was 37 lines, and its extreme dimensions 33 and 42 lines, and all the other apertures exceeded the natural size. The mitral orifice averaged 49, and ranged from 48 to 54 lines, the pulmonic averaged 44 lines, and ranged from 42 to 48, and the tricuspid averaged 57, and ranged from 54 to 60 lines.

In cases of incompetency of the valves the capacity of the aortic orifice also varied with the nature of the disease, in some instances the valves being healthy, but incapable of closing the aperture from the amount of dilatation; while in others the capacity of the aperture was not greater than natural, but the valves being diseased, were incapable of closing it. The mean circumference of the orifice in these cases was 39 lines, and it ranged from 30 to 45 lines; but in the case in which the capacity of the orifice was only 30 lines, the incompetency was the result of laceration of the valves, from violent muscular exertion, and as there was no reason to suspect the existence of any disease of the heart before the occurrence of the accident, it is probable that the aperture had contracted, so as

to afford some compensation for the imperfection of the valves. In these cases the capacity of all the other orifices was above the healthy standard, the mitral had a mean circumference of 53 lines, and its extreme dimensions were 42 and 60 lines, the pulmonic a mean of 45, and extremes of 59 and 51 lines, and the tricuspid a mean of 57, and extremes of 48 and 60 lines.

The auricles were greatly enlarged in all three forms of disease, and their walls generally increased in thickness; the right auricle being from $1\frac{1}{2}$ and to 2 lines thick, the left from $1\frac{1}{2}$ to 2 or $2\frac{1}{2}$ lines. The coronary arteries, when measured, were also found above their natural size.

Though the walls of the left ventricle did not exceed 11 lines or about one English inch in thickness, in any of the cases included in the table; in another instance, in a man 74 years, in which there was some valvular thickening with dilatation of the aorta, the organ attained the weight of 19 oz. 8 dr., and the left ventricle near the base measured 14 lines, or nearly an inch and a quarter English. The cases included in the table, also, do not afford evidences of the extent to which the aortic office may be contracted; in one case, that of a female 75 years of age, which has before been several times alluded to, the aortic aperture was a mere slit 10 lines long, and the thickened and ossified valves did not admit of being separated for more than 3 or 4 lines; yet, though the disease probably originated in a congenital malformation of the valves, and had therefore very slowly advanced, the parieties of the left ventricle were only 8 lines in thickness, and those of the right 2 lines. In a case of obstructive and regurgitant disease of the aortic valves, not included in the tables, the septum had a width of 8·7 lines or $\frac{3}{4}$ of an English inch.

The shape of the heart varies considerably in these different forms of disease, according to the seat and extent of the hypertrophy and dilatation. In cases of obstructive disease, whether the impediment be seated in the aortic valves or in the ascending portion of the aorta, the cavity of the left ventricle becomes peculiarly elongated, and the walls are ordinarily thicker near the base than elsewhere, and hence the whole organ has an acutely triangular form, and is much longer in its longitudinal, than in its transverse diameter. On the other hand, in cases of regurgitation through the aortic valves, while the left ventricle is elongated, the thickening of the parieties is more equably distributed throughout the cavity, and the apex, instead of being pointed, is obtuse, and thus the heart assumes a more oblong form, of which, however, the longest diameter is the longitudinal. In cases of regurgitation through the mitral aperture, and where there is considerable disease both of the mitral and aortic valves, the left ventricle, instead of being elongated, is expanded laterally, and especially at the apex, so that the organ has a more obtusely triangular form, and is generally broader from side to side than longitudinally. In these forms of disease, the right ventricle though implicated to a greater or less degree, is not so much so as

to affect the shape of the heart generally, but in cases of obstructive disease of the mitral valve, the left ventricle, as will be shown more particularly below, is little, if at all, altered in dimensions, while the right ventricle is hypertrophied and very greatly dilated; and hence the organ, though not very greatly larger than natural, is much broader, and is very obtuse at the apex. These differences of form are so striking and peculiar, that in many cases of diseased heart, it is easy to predicate before the cavities are laid open, the nature of the affection which will be found.

4. In cases of contraction of the left auriculo-ventricular aperture, uncomplicated with disease of the other orifices, the heart does not attain so great an increase of size as in the forms of disease before mentioned, for the enlargement is chiefly limited to the right cavities. Indeed, in some cases, more especially in young subjects, the capacity of the left ventricle, the thickness of its walls, and the circumference of the aortic orifice, are found not at all to exceed the natural dimensions, or even to fall below the healthy standard.

It will be observed, that the comparison between the cases of mitral valvular disease and of obstructive disease, in females, would lead to the conclusion, that the heart in the latter disease is smaller than in the former; but this is doubtless an erroneous inference, founded upon the smallness of the number of cases of obstructive disease in females. A more correct impression is most probably to be gained by comparing the dimensions of the heart in cases of mitral valvular disease in females, with its size in the other form of disease in males;—for though the heart does not in any disease attain so great a size in females as in males, the general effect of different forms of disease on its nutrition, may be inferred to be similar in the two sexes. The average circumference of the heart in the cases of mitral valvular disease, given in the table, will be seen to have been only 111 lines, and in no case did the circumference exceed 127 lines or 10½ French, or upwards of 11 English inches. The girth of the right ventricle averaged 62 lines, and in one case amounted to 75 lines. The girth of the left ventricle was very much less than that of the right, or only 49 lines on the average, and in no case more than 52 lines. The length of the right ventricle was on the average 46 lines, and its greatest length was 51 lines. The average length of the left ventricle was 39, and the extreme length 42 lines. The walls of the right ventricle averaged 2·2 lines in thickness, and ranged from 2 to 3 lines; those of the left ventricle had an average thickness of 5 lines, and ranged from 4 to 6 lines.

The mitral aperture varied considerably in the amount of contraction. In two cases, one of which is not included in the tables, it only admitted a cylinder 11 lines in circumference; in a third instance its circumference was only 18 lines, and in three others it was 22, 24, and 39 lines, the mean being 23 lines. The aortic orifice was

also below the natural size, or on the average only 29 lines in circumference, and its dimensions ranged from 25 to 33 lines.

The orifices on the right side of the heart were, on the contrary, ordinarily larger in comparison. The pulmonic aperture averaged $34\frac{1}{2}$ lines in circumference, and ranged from 29 to 39 lines; the tricuspid averaged 51 lines, and ranged from 40 to 57 lines. The left auricle was in all the cases greatly dilated, and its walls varied in thickness from $1\frac{1}{2}$ to 2 lines; the right auricle was generally still more enlarged, and its walls had a width of from 1 to $1\frac{1}{2}$ lines.

It will thus be seen that, while the left ventricle and aortic orifice were below the healthy standard, the dimensions of the other parts of the organ exceeded the usual size, so that it may be inferred that the operation of this form of disease is either to cause atrophy of the left ventricle, or, when the disease commences in early life, to prevent the full development of that cavity. It will also be observed that the hypertrophy of the right ventricle did not, in any cases of mitral valvular disease, equal that which was found in the two cases of chronic bronchitis with curvature of the spine.

5. The tables contain only one case of regurgitation through the left auriculo-ventricular aperture, from dilatation of the orifice without disease of the valves; and in this the subject was a child eight years of age, so that it cannot be compared with the other cases; allowance, however, being made for the age of the patient, the heart was much more enlarged than in the cases of mitral valvular contraction, and the left ventricle also was increased in size.

6. In the cases in which both the aortic and mitral valves were diseased, the dimensions of the heart were intermediate between those in cases of uncomplicated mitral and aortic disease. The enlargement, instead of being chiefly limited to the right ventricle, involved the left also; and the length of the cavities and the thickness of the walls, were greater than in cases of mitral disease alone, but less than in cases of aortic disease.

The girth of the right ventricle in two of the cases in the table was 72 and 55 lines; of the left ventricle 54 and 55 lines. The mean thickness of the parieties of the right ventricle was 2·6 lines, and the extremes 2 lines and 4 lines; the mean width of the walls of the left ventricle was 9 lines, and the extremes 6 and 11 lines. In the only case in which the width of the septum was measured, it was 5 lines. In these instances the subjects were females, but I have notes of other cases of combined aortic and mitral disease in males. The subject of one of these, 36 years of age, and the heart weighed 17 oz. 8 dr., the dimensions are not given in the tables. The aortic valves were incompetent, and the mitral aperture so contracted as only to admit the handle of a scalpel, indicating a circumference of about 12 lines. The cavity of the right ventricle was 42 lines long, the left 37 lines. The walls of the right ventricle had a width of from $4\frac{1}{2}$ to $5\frac{3}{4}$ lines, and the left ventricle of $5\frac{3}{4}$ at the base

and midpoint, and 3½ near the apex. The longitudinal diameter of the heart was 42 lines, the transverse 54 lines. The walls of the right auricle averaged 1½ lines in thickness, those of the left auricle 2 lines. In a second case the subject was a boy 18 years of age, and the disease was the sequence of rheumatism four years before. The organ weighed 16 oz. The pericardium was adherent, and both the aortic and mitral valves were thickened and incompetent. The circumference of the heart measured 122 lines. The cavity of the right ventricle was 27 lines in length, the left 48 lines. The walls of the right ventricle measured 3 lines in thickness, those of the left 8 lines. The aortic orifice had a capacity of 42 lines, the mitral of 45, the pulmonic of 42, and the tricuspid of 54. The longitudinal diameter of the heart was 42 lines, the transverse 56 lines.

7. Only one case of combined obstruction of the right and left auriculo-ventricular apertures is included in the tables; and in this instance the most remarkable feature was the great degree of contraction which existed in both openings, the left auriculo-ventricular aperture having a circumference of only 18 lines, and the right of only 21 lines; yet with this very great amount of obstruction, which had probably commenced before birth, the walls of the right ventricle was only 1·5 lines in width, and those of the left ventricle 4 lines. The right auricle was very greatly dilated, and its walls were from ½ a line to 2 lines in thickness; the right ventricle was somewhat dilated. The left auricle was also dilated, and the left ventricle, though to a less degree. The aortic orifice measured 30 lines in circumference, the pulmonic 35 lines. The aortic valves were also thickened and adherent. The subject of the case was a female 37 years of age.

8. The dimensions of the heart in cases of malformation vary with the kind of the deviation from the natural structure. The most frequent form of irregular development is that in which the pulmonary orifice is contracted, and the septum ventriculorum imperfect, or the foramen ovale open. Three cases of this description are included in the tables. The subjects were males, 15 and 20 years of age, and a female, 19 years old. In the first case the pulmonary artery was so contracted, as only to give passage to a ball 13 lines in circumference, in the second of 12 lines, and in the last of 8 lines. In the first and third cases the aorta arose from both ventricles, in the second the foramen ovale was open. In each of these cases the right ventricle was very greatly hypertrophied, measuring 54, 66, and 84 lines in circumference externally, while the left ventricle had only a girth of 42, 42, and 48 lines. The walls of the right ventricle measured 5½, 7, and 4 lines, those of the left ventricle 4½, 6, and 6 lines. It will thus be seen that, though the enlargement was chiefly on the right side, allowance being made for the age and sex of the subjects, the left ventricle must also have partaken of the hypertrophy; and this is still more obvious in cases of malformation in younger persons. The auricles were also

greatly dilated and hypertrophied, measuring from $1\frac{1}{2}$ to 2 or $2\frac{1}{2}$ lines.

In another instance in which malformation of a similar description was found in a child $6\frac{1}{2}$ years old, the pulmonary orifice only admitted a cylinder $6\frac{1}{2}$ lines in circumference; and in a fifth, in an infant 2 years and 5 months old, it was a mere slit 2 lines long. In these cases the walls of the right ventricle were much hypertrophied, measuring in each case, at the thickest point, 4 lines; while the left ventricle measured in the former case 6 lines, in the latter 3; and the density and resistance of the walls of the right ventricle, as in all the other cases of this kind of malformation, contrasted remarkably with the flaccidity of those of the left ventricle.

The septum is not, in these cases, hypertrophied in proportion to the walls of the right ventricle; indeed, that portion of the heart is more implicated in diseases of the left ventricle than in those of the right. Thus, while in cases of hypertrophy without valvular disease and in others of obstructive disease it had a thickness nearly equal to that of the outer walls of the left ventricle, or of from 9 to 10 and 11 lines;—in the cases of malformation in which the parieties of the right ventricle were from 6 to 7 lines thick, or fully three times the natural width, the septum measured only 6 lines in thickness, or was not materially wider than usual.

Different opinions have prevailed as to the cause of the hypertrophy of the right ventricle in cases of malformation. It has been supposed to be due to the entrance of aerated blood into the right cavities of the heart; but this cannot be the true explanation, for, in the most remarkable cases of hypertrophy, the course of the blood must necessarily be from the right ventricle or auricle into the left. The more correct theory is, doubtless, that which ascribes the hypertrophy to the effort to overcome the obstacle to the flow of blood from the ventricle, through the contracted pulmonary orifice. Another cause, which would have a similar effect, obtains in many cases, and appears to have been overlooked. I allude to the increased action of the right ventricle consequent upon the aorta arising in part from that cavity, and the share which it consequently takes in the maintenance of the systemic circulation. In one of the cases of malformation which I have mentioned, while the parieties of the infundibular portion of the right ventricle, upon which the stress from the contracted pulmonary orifice would chiefly fall, had only an extreme width of 4 lines; those of the sinus, which were chiefly concerned in propelling the blood from the right ventricle into the aorta, were 7 lines in thickness. It is true that the walls of the sinus are ordinarily thicker than those of the infundibular portion of the right ventricle, but the difference is ordinarily much less considerable.

It has been thought that the condition of the right ventricle, in some cases of malformation, affords an exception to the general rule of the non-occurrence of true concentric hypertrophy; and speci-

mens, when first removed from the body, and preparations preserved in museums, not unfrequently present the appearance of increased thickening of the walls of the right ventricle, with diminution of its cavity. These appearances are, however, I believe, deceptive, and depend upon specimens being examined, or the preparations having been immersed in spirit, before the tonic contraction of the muscular fibres had subsided. I have seen cases of malformation in which, when the right ventricle was first laid open, there seemed to be absolutely no cavity, but in which, after maceration, the ventricle proved to be unusually large.

In cases in which the maintainence of the circulation is thrown upon the left ventricle, as when the right auriculo-ventricular aperture is obliterated, or when that opening or the pulmonary orifice is greatly contracted after the complete development of the septum of the ventricles, the left ventricle becomes much hypertrophied and dilated, while the right ventricle undergoes a proportionate decrease in size. In some of these cases, indeed, the right ventricle becomes reduced to a small hollow, about the size of a pea, which is surrounded by thick ventricular walls; but this condition is clearly one of atrophy, not of hypertrophy, and the defective nutrition of the muscular substance of the right ventricle from disuse, is shown by its unusual paleness and flaccidity, and by the looseness of its texture.

General Remarks on the Dimensions of the Diseased Heart.—The changes which the heart undergoes in disease have attracted the attention of most systematic writers, and especially of Laennec, Bertin, Lobstein, Cruveilhier, and Hope; and I may particularly refer to the series of observations published by Bouillaud, and to the incidental allusions to the measurements of the organ in cases of disease, contained in Dr Ranking's valuable paper. It may not be without interest to compare the observations of these writers with the results obtained from the analysis of the cases now published.

The circumference of the largest heart measured by M. Bouillaud was 12 French inches, and of that mentioned by Dr Ranking $12\frac{3}{4}$ English inches—dimensions which very nearly correspond. In my own tables the dimensions are given of the heart of a male in a case of hypertrophy without valvular disease, which measured 15 inches and 2 lines French, or somewhat above 16 English inches in circumference, and one of incompetency of the aortic valves, in which the circumference of the heart was 14 inches and 3 lines, or about 15 English inches. In females the extreme circumference of the heart was 10 inches and 7 lines, or about 11 English inches, in a case of mitral valvular disease, and 10 inches and 6 lines in one of combined aortic and mitral disease, and 10 inches in one of obstructive disease at the aortic orifice.[1]

[1] M. Bouillaud, for purposes of comparison, gives the weight and dimensions

The thickness of the walls of the right ventricle is stated by Laennec[1] rarely to exceed 4 or 5 lines; and in the observations of M. Bouillaud, no case is given in which it measured more than 4½ lines. Bertin,[2] however, describes a case in which there existed a congenital contraction of the orifice of the pulmonary artery from adhesion of the valves, with patency of the foramen ovale, in a female 57 years of age, and, in this instance, the cavity of the right ventricle was less than natural, and its walls from 16 to 11 lines thick. M. Burnet[3] relates a somewhat similar case of congenital disease, in which, however, the foramen ovale was closed, in a girl 7 years of age, in whom the walls of the right ventricle were nearly an inch in width, and the cavity almost obliterated; and Louis[4] has described a case of contraction of the pulmonary orifice with imperfection of the septum ventriculorum in a man 25 years of age, in whom the parieties of the right ventricle had a width of 8 to 10 lines. Hope, in cases of congenital obstruction of the pulmonic aperture with an open foramen ovale, in a girl 8 years of age, and of aneurism of the aorta with regurgitation through the aortic valves, in a man 25 years of age, found the walls of the right ventricle half an English inch, or 5¾ lines thick. Dr Ranking refers to a case in which the pulmonary artery was contracted and the foramen ovale unclosed; and to another, in which the aorta arose from both ventricles, with pulmonic valvular obstruction, in which the parieties of the right ventricle measured 17-48ths (about 4 lines), and 44-48ths (10 lines) of an English inch in thickness. In none of the observations now published were the walls of the right ventricle so extremely hypertrophied as in the cases last named. The greatest thickness of the parieties being 5 lines in a case of chronic bronchitis, with deformed spine, 5 lines in cases of obstructive disease, and of incompetency of the aortic valves, 5¾ lines in a case of combined mitral and aortic valvular disease, and 7 lines in cases of congenital obstruction at the pulmonic orifice, with a patent foramen ovale in one, and deficiency of the septum ventriculorum in the other. In the latter case the subject was a female, while in all the others they were males; with this exception, the walls of the right ventricle did not exceed 4 lines in thickness in females, and the instances in which they attained this width were cases of combined aortic and mitral valvular disease, and of chronic bronchitis.

The parieties of the left ventricle are stated by Laennec to have been seen by him an inch, or even 18 lines thick, or double or triple the size in the sound state. This statement is repeated by Elliotson,[5]

of the heart of an ox; the weight was 66 oz. 7 dr. avoird., and the circumference 18 French inches.

[1] Diseases of the Chest, 4th Ed. Forbes' Translation, 1834, p. 547.

[2] Maladies du Cœur. Paris, 1824, p. 326. Obs. 87. See also Bouillaud, Traité Clinique, 2me Ed., 1841, t. ii., p. 273. Obs. 126.

[3] Bouillaud, p. 281. Obs. 128; and Journal Hebd. de Med., 1831.

[4] Mém. ou Rechérches Anatomico-Pathologiques. Paris 1826, p. 313. Obs. 10. See also Arch. Gen. de Med., 2mo serie, t. iii., 1823.

[5] Lumleyan Lectures.

and Hope mentions, that the walls of the left ventricle may attain a thickness of one, one and a half, or, according to some, of two inches; and he mentions a case in which they were 1½ inch thick in a case of regurgitation through the aortic orifice. The extreme thickness of the parieties of the left ventricle in the cases mentioned by Bouillaud, is 13 lines. Of the hearts examined by Ranking, in one the walls of the left ventricle attained a width of one inch, or about 11¼ lines, and Bertin mentions a case in which they were upwards of one inch in thickness.

The greatest width of the parieties of the left ventricle in the cases given in the present memoir, was in males 11 lines, in cases of hypertrophy without valvular disease, and of aortic disease, and of dilatation of the aorta with chronic bronchitis; but another case is referred to, in which there was slight valvular disease with dilatation of the aorta, and the parieties of the left ventricle measured 14 lines in width. In females the greatest thickness of the walls of the left ventricle was 10 and 11 lines, in cases of combined aortic and mitral valvular disease.

The septum of the ventricles is mentioned by M. Bertin to have been found by him one inch in thickness; the greatest width in M. Bouillaud's observations is 10 lines, and Dr Ranking found it 38-48ths of an English inch (8·75 lines) in one case. In the present observations the greatest width in males is 10 and 11 lines in cases of hypertrophy without valvular disease, and 9 lines in cases of incompetency. In females the greatest width is 6 lines in a case of mitral valvular disease. I regret, that in the observations, the dimensions of the septum were less frequently obtained than would have been desirable.

The parieties of the right auricle were found by M. Bouillaud, in two cases, to measure 3 and 3½ lines in width, but his measurements are taken near the appendix, where ordinarily the walls are thicker than across the middle of the sinus. Bertin also examined a heart in which the walls of the right ventricle had a width of 3 lines; and Dr Hope speaks of a thickness of a quarter of an inch as occasionally seen. In my own observations, the walls of the right auricle attained a width of 3 lines in only one case, that of the female, 19 years of age, in whom congenital contraction of the pulmonary aperture and deficiency of the septum of the ventricles, was combined with some disease of the tricuspid valves. The parieties of the right auricle measured 2½ lines in a case of combined mitral and aortic disease, in a boy of 18 years of age, and attained the same thickness in a case of aneurism of the apex of the left ventricle, with open foramen ovale. They were also 2 lines thick in the case of great contraction of the mitral and tricuspid valves, and in one of combined aortic and mitral valvular disease.

The maximum thickness of the parieties of the left auricle, mentioned by M. Bouillaud, is 2½ lines. In my own observations its greatest width was two lines in cases of regurgitation through the

left auriculo-ventricular aperture, obstructive and regurgitant disease of the mitral valves, combined aortic and mitral valvular disease, and regurgitation through the aortic aperture.

In the data collected by M. Bouillaud, the aortic aperture had a maximum capacity of 41 lines, and a minimum of 11 lines. Dr Hope mentions a case in which it was contracted to the size of a small pea. Its circumference in one case, measured by Dr Ranking, in a boy 18 years of age, was only one inch and 3-48ths (12 lines). In my own observations, its largest size in males is 45 lines, and its least 30 lines in cases of regurgitation through the aperture. In females its greatest circumference was in a case of combined aortic and mitral valvular disease, in which it measured 35 lines, while in another case before referred to, it was reduced to a mere slit 10 lines in length, and the thickened and ossified valves did not admit of being separated for more than three or four lines.

The pulmonic aperture in the hearts measured by M. Bouillaud, had a maximum circumference of 42 lines, and a minimum of 34 lines. The extreme size of the orifice is not mentioned by Dr Ranking in his observations; but he gives two instances in which the aperture was contracted, as is almost always the case, from congenital malformation, and in these the aperture measured one inch, and one inch and 40-48ths (rather more than 11 lines and 20·5 lines) in circumference. In the cases related by Bertin, Louis, and Burnet, the aperture had a diameter of only $2\frac{1}{2}$, $2\frac{1}{2}$, and $1\frac{1}{2}$ lines. In the case quoted by Hope, the pulmonary orifice would only admit a goose quill, and in one related by Dr Hunter, the pulmonary artery would only admit a small probe, though the patient was 13 years old. In the observations published in the paper, the largest size of the pulmonic orifice in males is 54 lines in a case of hypertrophy, unconnected with valvular disease; and in females 39 lines, in a case of mitral valvular disease. In the cases of malformation before mentioned, in males 15 and 20 years of age, and in the female 19 years of age, the aperture had a circumference of only 13, 12, and 8 lines; and in children $6\frac{1}{2}$ years, and 2 years and 5 months old, it was only $6\frac{1}{2}$ lines, and 5 lines in circumference.

The left auriculo-venticular orifice in the cases of M. Bouillaud had an extreme capacity of 51 lines, and of 24 lines; but he mentions another case in which the aperture was so contracted as only to be 6 or 7 lines long and 5 lines wide, and two in which it was only 3 lines in its largest diameter, or 8 or 9 lines in circumference.[1] Dr Ranking measured two hearts, in which the mitral orifice had a circumference of $5\frac{1}{2}$ inches ($61\frac{3}{4}$ lines), and 5 inches and 2-48ths (54 lines), and he found none in which it was less than 2 inches and 19-48ths (27 lines). In the present series of measurements, the extreme capacity of the left auriculo-ventricular aperture is in males 60 lines, in cases of hypertrophy without valvular disease, and incompetency

[1] Observations 112, 116, and 117.

of the aortic valves; and in females 45 lines in a case of obstruction at the aortic orifice. Its smallest size is in males, in a case of combined disease of the aortic and mitral valves, in which it would only admit the handle of a small scalpel, and had a circumference of not more than 12 lines. In females its smallest circumference was 12 and 18 lines, in cases of uncomplicated mitral valvular disease.

The right auriculo-ventricular aperture in the observations of M. Bouillaud had an extreme capacity of 69 lines, and the only case of contraction of this aperture to which he refers, is that of General Whiple,[1] in which there was an opening one inch long and one line wide. Laennec does not refer to any case of extreme contraction of the tricuspid aperture; but Dr Forbes, in a note to his translation,[2] mentions one in which the aperture would only admit the thumb, and in which, like one I have before referred to, the mitral and the aortic apertures were also contracted. In the case related by M. Louis, contraction of the tricuspid aperture was also, as frequently happens, combined with the disease of the pulmonic valves. Dr Ranking measured a heart in which the right auriculo-ventricular aperture had a circumference of $6\frac{1}{2}$ English inches, or somewhat more than 73 lines. He does not refer to any cases of contraction. In the present series, the largest circumference of the tricuspid aperture in males is 63 lines and 60 lines, in cases of hypertrophy without valvular disease, and 60 lines in cases of obstructive disease, and incompetency of the aortic valves. In females the greatest circumference is 60 lines in a case of obstructive disease, and 57 lines in one of uncomplicated mitral valvular disease. The most extreme degree of contraction of this aperture with which I have met, was in a case of combined aortic, mitral, and tricuspid disease, not mentioned in the tables, in a female 32 years of age, in whom the tricuspid valves were adherent, so that the aperture would only admit the point of the forefinger, indicating a circumference of about 16 lines. The case of a similar kind mentioned in the paper, the aperture was 21 lines in circumference. Elsewhere I have expressed the opinion, that the disease of the valves in these cases, like the similar fusion of the valves of the pulmonary artery, is due to intra-uterine disease, and that they are, therefore, to be regarded as malformations.[3]

In the following table are included the weights and dimensions of hearts examined since the first and second parts of the paper were written. They are not, therefore, included in the calculations as to the weight of the heart, but they enter into those of the dimensions of the organ, both healthy and diseased.

[1] Journal de Médicine et de Chir. Par MM. Corvisant, Leroux, etc. Vol. xix, p. 468.

[2] Page 590, 4th Ed. 1834.

[3] See Lectures on Malformations.—London Medical Times and Gazette, 1854.

APPENDIX TO

Sex.	Age.	Weight.	Girth of Right Ventricle.	Girth of Left Ventricle.	Length of Cavity of Right Ventricle.	Length of Cavity of Left Ventricle.	Thickness of Walls of Right Ventricle.			Thickness of Walls of Left Ventricle.		
							Base.	Mid Point.	Apex.	Base.	Mid Point.	Apex.
	Years.	oz. dr.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
M.	14	4 10	41	37	33	34	1½	2	¾	4	5	3
M.	23	6 9½	54	44	42	36	2	2¼	2	5	6	3½
M.	25	11 0			50	44	2	2	2	5	6	3
M.	35	8 0	58	48	44	36	1½	2	1½	4½	5	3
M.	38	11 0	66	50	51	45	2	2½	1½	5	5½	2
F.	19	8 10	54	48	52	42	2	2½	1	4	4	3
F.	21	9 8	60	60	48	44	1½	1½	1½	4½	6	3
F.	21	7 13	58	48	42	36	2	1½	1	6	5	3
F.	20	9 5	60	50	46	41	2	2½	1	5	5	2½
F.	22	9 10	72	48	48	39	2	1½	1	5	6	2½
F.	28	8 8	54	48	48	36	1½	2½	1½	5	6	3
F.	37	7 1	60	48	42	36	1	1½	½	4	5	2½
F.	50	9 4	60	52	48	41	2½	2½	1	4	6	3
F.	56	10 8					2½	3	2½	6	8	2
F.	64	7 0			34	36	2	2	2	5	5	3
M.	18	16 0	66	56	57	48	3	2½	1½	7	8	4
F.	3 mos.	1 0	27	24	17	18	1½	1½	1½	2½	3½	1¼
F.	19 yrs.	17 8	84	48	53	46	4	4	3	5	6	2
F.	75	10 2	60	44	39	39	2½	3	2	5	8	3

GENERAL TABLES.

Thickness of Septum.	Circumference of Orifices.				Cause of Death, etc.
	Tricuspid.	Pulmonic.	Mitral.	Aortic.	
lines.	lines.	lines.	lines.	lines.	
4	42	36	39	30	Fever.
6	57	39	42	33	Peritonitis; waxy liver; morbus renum; weight of body 7 st. 7 lbs.
6	54	45	45	39	Cholera.
		36		32	Hemiplegia; weight of body 88 lbs.
5	62	45	54	39	Secondary abscesses in lungs and liver.
$5\frac{1}{2}$	60	39	48	33	Fever; weight of body 9 st. 7 lbs.
	57	39	51	33	Cholera.
5	45	39	42	36	Chronic peritonitis; meningitis.
$3\frac{1}{2}$	51	39	39	33	Phthisis; weight of body 6 st. 11 lbs.
6	51	42	45	36	Cholera; weight of body 8 st. 10 lbs.
5	57	42	54	36	Cholera.
4	54	39	45	33	Cholera; weight of body 6 st. 2 lbs.
$5\frac{1}{2}$	57	36	45	33	Erysipelas.
	45	39	45	30	Chronic bronchitis.
	54	42	45	36	Morbus renum.
7	54	42	45	42	Aortic valves much thickened and incompetent; mitral valves thickened, adherent and permanently open; pericardium universally adherent, sequence of rheumatism 4 years before.
3	24	18	21	16	Atelektasis pulmonum.
6	51	8	45		Malformation; contraction of pulmonic aperture, aorta arising from both ventricles; posterior wall of right ventricle 7 lines thick.
6	54	34	42	32	Obstruction at aortic orifice, with dilatation of aorta; valves with vegetations, some ath. of mitral and in aorta; 5 valves to Pulmonary artery.

In reviewing the communication, it will be seen that the conclusions drawn from the data collected, correspond generally with those of other observers, so far as relates to the weight and dimensions of the healthy heart; but in reference to the diseased organ, they present several differences which are not without interest. They show that the heart may attain a weight and size much larger than was previously supposed, and that the greatest amount of enlargement may be found when there is no material valvular disease, or any other obvious source of obstruction to which it can be referred. They indicate also, a general relation between the dimensions and weight of the heart, and the different forms of disease of which it may be the seat, or in which it may be indirectly implicated. This connection, so far as I am aware, has not been previously clearly pointed out; but if, as I feel much confidence in believing, the conclusions here arrived at, should prove, on more extended investigation, to be of general application, they must be admitted to be not only of much pathological interest, but also of practical importance.

It was my intention, before concluding this memoir, to have alluded to some of the preparations which illustrate the size and form of the heart in disease, contained in the museums of the metropolis; but the length to which the paper has already extended precludes my doing so.

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CONTRIBUTIONS TO TERATOLOGY.

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THESE CONTRIBUTIONS

ARE AFFECTIONATELY DEDICATED

TO

JAMES Y. SIMPSON, M.D.,

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CONTRIBUTIONS TO TERATOLOGY.

It is the author's intention to lay before the profession, from time to time, a few contributions to our knowledge of fœtal monstrosities and malformations. These will consist, not only of original observations and researches, but also of commentaries on the labours of other investigators; of corroborations of their opinions where he has confirmed them, and corrections of their statements where it seems to him they are fallacious. They are offered with much deference to the opinions of others, in the full consciousness that the subject is one still involved in much doubt and difficulty.

NO. I. OF THE FREQUENCY OF THE OCCURRENCE OF MALFORMATIONS IN GENERAL, AND OF CERTAIN VARIETIES IN PARTICULAR.

Happily for the human race monstrous births are comparatively rare. We are not, as yet, in possession of sufficient data by which to calculate with much exactitude the frequency of their occurrence; for many, doubtless, happen which are either unrecorded through negligence, or are concealed from motives of false shame; and many must escape observation altogether, especially in cases of abortion and premature birth. St Hilaire, in 1836, reckoned the average occurrence of monstrosities in Paris to be 1 in every 3000; and, estimating the annual number of births in the whole of France at one million, he calculated that in about 3300 cases the infants were malformed. According to Riecke 230,939 births occurred in Würtemberg in four years, 50 of which were recorded as monstrous; *i.e.*, in the proportion of 1 in every 4618. The records of maternity hospitals ought to afford us more accurate and valuable statistical information than the returns of Registrars-General, which are well known to be very defective and inaccurate in a medical point of view. Let us, therefore, examine those of the Imperial Lying-in-Hospital at Vienna (the *Gebäranstalt*), and the *Maternité* at Paris. Between the years 1832 and 1840, 23,222 births occurred in the former institution; and of these 68 are reported as being monstrous, or 1 in every 341 children.[1] Among 23,293 infants born at the *Maternité* at Paris, Chaussier found 132 malformed in different ways; that is,

[1] Quoted from Mr Wilde's work on "Austria."

in the ratio of 1 in every 176. Now, it will be seen that considerable discrepancy exists among these statistics. The computations of St Hilaire and Riecke are evidently too low; but it is worthy of remark that their statistics must necessarily be more liable to fallacies than those of maternity hospitals, where the particulars of every birth are noted with accuracy and care, and moreover they probably include merely the graver types, and not the milder and less conspicuous forms of malformations. Hence we are of opinion that the statistics we have cited of the lying-in-hospitals of Paris and Vienna are most to be depended on. If, therefore, we take the mean ratio of both hospitals, we shall find that *malformed children occur in the average proportion of* 1 *in every* 232 *births.*

This computation of mine *may* be fallacious; but it seems to me as near an approximation to the truth as, in the present state of our knowledge, can be obtained. It may be asked with much reason, why it is that our statistics on this point of obstetrics are so very scanty and defective? We apprehend that this is to be accounted for, not by any carelessness on the part of accoucheurs, or by any deficiency of their opportunities of observations, but solely by their defective knowledge of teratology, and by the want of a proper understanding among them as to what is, and what is not, a malformed or monstrous child. As regards this latter cause, however, they are not much to be blamed, for certainly in no department of medicine have we greater need of an accurate and distinctive nomenclature. The word "monster" ought to be entirely abolished from medical literature as indefinite, confusing, and unscientific. It arose in those dark ages, when *lusus naturæ* were regarded as omens of evil import, indicative of the vengeance of an offended Deity, or grimly foreshadowing some impending calamity.[1] The word thus coined unfortunately continued current in after-periods of greater enlightenment, creating no small amount of confusion and misunderstanding. What do we understand by the term "Monster" in these latter days? "We call monsters what things soever are brought forth contrary to the common decree and order of nature." So said worthy old Ambrose Paré; but, according to this definition, the slightest deviations from the specific type, such as the abnormal course of an artery, or the unusual configuration of a limb, might be ranked as monstrosities. Now-a-days this word "monster" (monstrum, *Latin;* Τέρας, *Greek;* missgeburt, *German;* monstre, *French*) seems only to be applied to the graver cases of anomalous formations as Acephalia, Anencephalia, diplogenesis, etc.; and the term "malformation" (lusus

[1] Some have considered the word "monster" to be derived from the Latin verb *monere*. I think, however, that the verb *monstrare* is more probably its radical. The following words of Cicero seem to warrant this etymological hypothesis:—"Monstra, ostenta, portenta, prodigia appellantur quoniant monstrant, ostendunt, portendunt, et prædicunt."—(*De Divinitatione,* Lib. i.) And Isidore von Savilla defines the word thus:—"Quæ aliquid futurum *monstrando* omines monent." –(*De Etymologiis,* ib. ii.)

naturæ, *Latin;* naturspiel or missbildung, *German*) to such *vitia conformationis* as are less defective and conspicuous, *e.g.*, harelip supernumerary extremities, imperforate anus, etc. Now *all* structural abnormalities ought to be called *generically* "malformations" (for a child with harelip or talipes is as truly malformed as one with anencephalia); and these again should be *individualised* under their own proper and specific teratological designations. The word "monster," having so long held a place in our language, cannot well be dispensed with in treating of congenital deformities, but I think that medical men would do well to avoid using a term so very unscientific and confusing. And until obstetricians, paying a little more attention to teratology, carefully record the minor as well as the graver types of malformations, we can never hope to obtain correct statistical information on this subject.

As regards the frequency of certain special varieties of malformations, I may remark that Meckel, long ago, pointed out that those *per defectum* were of more frequent occurrence than those *per excessum*, and that among the former class those defective conditions of the cranium, called Acephalia and Anencephalia, formed a considerable number. These observations have been confirmed by subsequent researches; and the peculiar types specified have been found to be more common than is generally supposed. Among malformations by excess of development, we find cases of diplogenesis (or double-monstrosity) to be very common. Among those *per excessum* the malformation occurs more frequently on the *upper* than on the under half of the body; thus we oftener find two heads joined to one body than two bodies attached to one head. But, as there is some exception to every general rule, supernumerary extremities and consolidations of parts generally occur in the *lower* half of the body. Otto,[1] Isenflamm,[2] Courmette,[3] and other authors assert that the *left* side of the body is weaker, and consequently more prone to disease than the right, and that on this side also malformations most frequently occur. This statement I have frequently tried, but in vain, to confirm, for in the majority of cases where the deformity was unilateral I found it on the *right* side.

NO. II. THE RELATIVE FREQUENCY OF MALFORMATIONS IN THE TWO SEXES.

Malformations occur much more frequently in the female than in the male sex. Haller, Meckel,[4] Tiedemann, Otto, and Burdach

[1] Pathol. Anat. Band i.

[2] Beitrage für die Zergliederungkunst. Band i. Part

[3] Journal de Médécine. Part 85.

[4] Sex est generalis, paucis tantum exceptionibus subjecta, monstra femina longè saepius occurrere masculinis."—MECKEL, *Comment. de Duplic Monstros*, p. 14.

have all observed this fact; and I shall place here, in a tabular form, the results of some of their investigations on the subject:—

		Females.	Males.	Authority.
In	42 cases of Diplogenesis there were	30	9[1]	(Haller)
In	80 do. do. do.	60	20	(Meckel)
In	46 do. of Malformations do.	31	15	(Tiedemann)
In	473 do. do. do.	270	203	(Otto)

In diplogenesis especially the female sex is found to predominate; thus, in 142 cases Otto found only 42 males, and in the 122 cases cited above there were only 29 males. Burdach[2] is very near the truth when he says that in these cases males occur in the ratio of 1 to every 3·2 females; for according to the above statistics the males form only 27 per cent. of the whole, or, in other words, occur in the proportion of 1 to every 3·70 of the other sex. It has, however, been alleged that in some varieties of diplogenesis, the twin-fœtuses are generally of the male sex; but the statistics I have quoted militate against the truth of this assertion. In certain rare species of malformations,—as in those caused by parasitical growths,—males are most frequently found; occurring, according to Burdach,[3] in the ratio of 1 male to 0·40 of the other sex.

It may not, perhaps, be out of place to mention here that in malformations by duplicity the children are generally of the same sex, being either two males or two females. Although this may be, in some measure, due to the symmetrical *lex proprietatis* by which even abnormalities are governed, we shall find, on analysing the statistics of common twin-births, that in most cases the children are of the same sex. In the kingdom of Prussia, during a period of twenty-three years (from 1826 to 1848), there were recorded 141,715 twin births. The particulars of these, as regards sex, are thus tabulated by Dr Meckel:[4]—

STATISTICS OF 141,715 TWIN BIRTHS.

Same Sex.		Different Sexes.	
2 Boys.	2 Girls.	1 Boy.	1 Girl.
47,074	43,413	51,228	
Total, 90,487		51,228	

[1] Of the remaining three, two were hermaphroditic, and in one the sex was unascertainable.—Haller, *Opusc. Anatom. Götting.* 1751. P. 176.

[2] Physiologie. Vol. i. p. 379.

[3] Loc. cit.

[4] *Vide* Müller's Archives. Jahrgang, 1850. p. 236.

From the admirable statistics of Dr Collins, I have drawn up the following table :—

STATISTICS OF 240 CASES OF TWIN BIRTHS.

Same Sex.		Dissimilar Sexes.	
2 Boys.	2 Girls.	1 Boy.	1 Girl.
73	67	100	
Total, 140		100	

These tables sufficiently show the resemblance between normal twins and malformations by duplicity in regard to similarity of sex ;[1] but there is this point of difference to be noted, that while in the latter *females* greatly predominate when the children are of the same sex ; in the former, *males* are most commonly met with in the same circumstances.

How are we to explain this preponderance of the female sex among malformed children ? Meckel accounted for it by supposing that, in early intra-uterine life, the fœtus was, in all cases, of the feminine sex, and that defective development of the genitals was the cause not only of there being so many female monstrosities, but of there being also so many feminine hermaphroditisms. Although Burdach and others justly opposed Meckel's erroneous doctrine as to the primitive sex of the fœtus, Andral and many authors, accepted this hypothesis as explanatory of the facts alluded to ; and probably they inclined to this opinion on account of the assertions of Soemmering, Ruisch, and Autenrieth, that abortions and early embryos were generally of the female sex. I am of opinion that Meckel, and the physiologists who adopted his views, must have mistaken the *sinus urogenitalis*, in the embryo they examined, for the vulva. Be this as it may, it is now well ascertained that their assumptions are incorrect ; that the external genitals are primarily the same in both sexes (as has been shown by Tiedemann, Müller,[2] etc.) appearing first in the form of a small wart-like protuberance in front of the sinus urogenitalis ; and that it is only in the fourth month that the sex is clearly distinguishable. In what manner, then, are we to account for the fact ? Is the female fœtus more weakly, and consequently more susceptible to disease and malformation than the

[1] I may remark that in triplets and quadruplets, the proportion of equal sexes is not so great. Thus Mayer found that of 1594 triplets, 719 were of the same, and 875 of dissimilar sexes ; and of 35 quadruplets 11 were of the same, and 24 of different sexes.

[2] Müller's Physiology, Trans. by Baly, vol. ii. p. 1639.

male? I do not think we are warranted in saying so. The only hypothesis which seems to me at all tenable, is *not* that the sex induces the malformation, but that the *malformation determines the sex*. I am of opinion that it may occur thus:—A higher degree of formative power is probably requisite for the conversion of the embryo into a male than into a female child, for the margins of the sinus urogenitalis need to be developed more largely, and to be united more extensively in the constitution of the male perineum and scrotum. If, therefore, from the fifth to the fourteenth or sixteenth week, anything happens to disturb or arrest the development of the embryo, its *autotrophy*, or inherent formative power, becomes so weakened that it cannot possibly accomplish the perfect closure of the primitive cloacal fissure, and the fœtus consequently becomes a female. This hypothesis may not stand the test of future researches,—I merely offer it because it seems to me capable of explaining the great frequency of females among malformed children.

NO. III. THE RELATIONS BETWEEN TERATOLOGY AND ZOOLOGY.

Transcendental anatomy has shown us that throughout the whole of the animal kingdom *unity of organisation* is the great principle governing the development of the species. After the model of a great archetype or types[1] all animals are fashioned; and although their various species present many distinct generic characters and great structural diversities, the unity of organic formation which has presided over their development gives rise to many very striking and unexpected analogies, existing even between creatures far removed from each other in the scale of being. In the various stages of their development the higher animals present transitory *organic* resemblances, more or less striking, to others which are lower; and the inferior animals again—when regarded in the light of a primitive archetype—are but the permanent embryos of species which are higher. A remembrance of these facts will enable us all the better to understand teratological phenomena in general; and more especially those cases where one species presents, in its malformed condition, a close resemblance or analogy to the normal and perfect development of another.

Bearing in mind this unity of organic type, and symmetry of

[1] *One* type according to Geoffrey St Hilaire: *four* according to Cuvier, etc. The former idea seems at first sight absurd; for how can an invertebrate animal be formed after the model of one having a complex osseous skeleton? Have a polype and a man the same archetype? But both theories are fundamentally correct. Cuvier looked merely to characteristic generic distinctions; Geoffrey's philosophy is more searching and subtle, for he passes over all the superficial differences constantly presented by specific classes, and looks merely to one grand *principle*, in accordance with which they were organised. His doctrine, to quote an able writer in the Westminster Review, is, that "unity of composition is not to be sought in the *form* of animals, nor in their assemblage of parts, but in the *progressive repetition and complication of parts issuing from a common centre*, and formed according to a common plan or process."

design which pervades the animal kingdom, we need not wonder that malformations occur among all living species. All organic processes are liable to mutation and disturbance; and all development, animate or inanimate, is subject to mysterious deviations and abnormalities.[1]

I cannot agree with those writers who assert that, as a general rule, the higher animals are in the scale of being, the more frequent are malformations among them. In the same species, however, we know that their frequency is increased by domestication. St Hilaire has stated that about three-fourths of the malformations in the animal kingdom occur among mammalia, and the remainder among birds.[2] The same author likewise avers that they happen very rarely among fishes, and seldom or never among the articulata, radiata, and mollusca. These observations are obviously incorrect, for among the lower animals structural abnormalities and imperfections occur very frequently,—probably oftener than among mammals. Fishes are very often malformed. I have seen a cyclopic cod, and have frequently examined fishes in which the dorsal fins were deficient or redundant, or in which the tail was bi-pinnate. Gold-fish and carp are very often thus imperfect; shortening or arrested development of the face is said by Otto to be very common among the latter species. Supernumerary toes I have often noticed among frogs; and abnormalities in the number of the thumbs on the claws of crabs are familiar to all.[3] Among snails also anomalies in the windings and shape of their calcareous volutes are far from being uncommon. Malformations are met with among the Reptilia; thus Mitchell relates cases of three rattlesnakes, all of which were bicephalous.[4]

Among Mammalia we find malformations occurring more frequently in certain species than in others. Thus cats are more subject to them than dogs; they occur oftener among oxen than among sheep, and among sheep and oxen than among swine, goats, and horses. Moreover, certain varieties are common to certain species. Diplogenesis and malformations of the cranium occur very frequently in the human species; the former is common among cats, which are multiparous, and also among oxen, which are properly uniparous. Cyclopia is most common among swine, and acrania among birds. I have seen diplogenesis occurring in the domestic fowl by the fusion of a double yolk; and the shells of two eggs may be united together.[5]

[1] "Rien n'est monstrueux dans le nature, parce que les lois qui la regissent émanent d'une intelligence superieure qui met dans toutes ses œuvres la même sagesse et la même harmonie."—*Breschet.*

[2] Hist. des Anom. iii., p. 354.

[3] Otto says:—"There are *a few specimens* of three thumbs on the claw of a crab." Now, with all deference to so great an authority, I think I can pronounce such cases to be by no means rare. I may mention that some may be seen in the Anatomical Museum of the Edinburgh University, where there are many interesting specimens of malformations among fishes and crustacea.

[4] American Journal of Science and Art. 1825. P. 48.

[5] A beautiful specimen of this exists in Professor Simpson's Obstetrical Museum in the Edinburgh University.

Many of the malformations of the human embryo are analogous to the normal conditions of some of the lower animals,—arrest or disturbance of its development having occurred at a period when the fœtus presented some of the specific transitory organic similarities to which I have alluded. The shortened limbs and deficient arms of the malformations called *phocomeles* resemble the condition of the extremities in seals and certain *cetacea;* a duplex uterus, a cloacal fissure, and a small-sized brain approximate the deformed embryo to the characters of the *rodentia;* and diaphragmatic incompetency is analogous to the normal organisation of oviparous animals. These resemblances of the malformed fœtus to some of the lower animals have been, by the vulgar, attributed to the effects of some impressions made on the maternal imagination during pregnancy (*Das Versehen* of the Germans), rather than to accidental and mysterious circumstances arresting the growth of the embryo, and thereby giving permanence to certain of its rudimentary conditions. Thus, it will be seen, to quote the words of Serres, that "L'ordre est dans le desordre;" and that even malformations are governed by certain regular, definite, and symmetrical laws.

> "All nature is but art unknown to thee;
> All chance, direction which thou canst not see;
> All discord, harmony not understood."—Pope.

NO. IV. THE FUNDAMENTAL LAWS OF TERATOLOGY.

"Nature," Soemmering truly remarks, "does nothing undesignedly; for, even in her malformations, the greatest regularity is observable."[1] Modern investigations in teratology, and recent advances in embryology, have clearly shown that malformations are neither the blunders and failures of nature (as was thought by Aristotle), nor yet *lusus naturæ*, her eccentric and inexplicable freaks. To render more intelligible what we may have to consider hereafter, I shall briefly state the three most important of these teratological axioms.

a. Analogous parts have an affinity for each other.

This harmony of union is uniform and constant; it constitutes the "lex proprietatis" of Fleischmann, and the "affinité de soi pour soi," of St Hilaire. Dissimilar parts are only united when (as in the case of the bladder and rectum, or pharynx and larynx) they are originally developed from a common mass. Arteries are never seen coalescing with nerves, nor the alimentary canal with the abdominal aorta; but in malformations by duplicity, the fusion always takes place between similar parts; sternum is united to sternum, vertex to vertex, pelvis to pelvis, and so on. And not only so, but in such cases the corresponding organs of the united twins are joined together; muscle corresponds to muscle with wondrous certitude, the nerves join nerves, and the arteries their fellow-vessels.

[1] Abbildung und Beschreibung einiger Missgeburten, 1791, p. 35

We may here observe that malformations arise, as well from the non-union as from the abnormal junction of several parts. According to Serres'[1] theory of "eccentric" development, the embryo is primarily formed from two lateral halves, and hence all the organs situated centrally, as the heart, uterus, bladder, etc., are originally double. Of the corresponding organs of the two halves, some are intended to be united, and others to remain distinct and double. Now, should embryogenesis at this stage be disturbed, the future appropriate conditions of these organs may be totally disordered; and it is evident that, in such a case, the non-junction of parts which ought to be united, will as surely produce a structural malformation, as will the fusion of others destined to remain separate. We see this exemplified in the uterus, the lateral halves of which, instead of uniting into a symmetrical whole, often remain separate, causing the organ to retain its embryonic type, and remain double or bicornuous throughout life.

The corresponding or homologous organs, which are most frequently abnormally united, are those between which no important texture intervenes. Thus the kidneys, separated by no distinct dividing structures, are oftener amalgamated than the eyes or the ears, between which lie osseous septa; and when in these latter cases union does occur, the partition walls are either found to be in a rudimentary condition, or to have been secondarily destroyed.

b. Malformed organs never wholly lose their normal types.

However deformed a part may be, it never so completely loses its ordinary distinguishing characteristics as to defy recognition. Thus the bones composing the anencephalic cranium, rudimentary and malformed though they be, are all severally distinct and capable of recognition by the anatomist.

c. Malformations seldom occur singly.

This law needs little illustration. How often do we find harelip, talipes, and spina-bifida, co-existent with defective formations of the cranium; and how often do we see anomalies of the fingers, toes, and genital organs, in cases of diplogenesis! Indeed, it would seem as if, when defective development occurred in one part of the body, nature compensated for the atrophy by the increased growth of some other region. This general principle was first noticed by St Hilaire[2] and Breschet,[3] who designated it the "loi de balancement," or "balancement organique." The inference which these authors would deduce from it is, that organic hypertrophy can only occur, in utero, at the expense of the nutrition of some other part. But there are many cases in which no such compensation can be observed; thus,

[1] Memoir on Transcendental Anatomy in Annales des Sciences Natur., vol. xxi.

[2] Hist. des Anomal., vol. iii. p. 401.

[3] Dict. des Medéc. "Deviations Organiques."

for example, the vascular and other systems may be found quite normal, while the nervous is but imperfectly developed; and *vice versâ*. Still, in the majority of instances, the general law holds good, that the malformation of one part affects the development of others in a greater or less degree.

In the simultaneous occurrence of malformations, we generally find that the organs secondarily affected are those which are homologous or repeated in the body. Hence the frequency of anomalous conditions of the teeth, ribs, vertebræ, fingers, and toes—both in regard to number and structure—in all the graver types of fœtal deformity.

NO V. THE HEREDITARY CHARACTER AND FREQUENT RECURRENCE OF MALFORMATIONS.

No fact concerning malformations seems more surely determined than their hereditary character. Let us cite a few illustrative cases:—Naef delivered a woman of a bicephalous child, the father of which had had a brother who was also born with two heads.[1] Anna relates the case of a man who had eleven children by his *first* wife, nine of whom were born dead, and two alive, but all of them with harelip. The first child which this man had by his *second* wife had also harelip; the third, by this marriage, had malformation of the jaw (*Wolfsrachen*); and the fourth harelip again.[2] Two relations of this man had been born similarly malformed. The foregoing, like many analogous cases, proves that a well-formed man may procreate, with different women, deformities of the same kind. I have seen a father and son having each twelve fingers and twelve toes. Congenital luxation of the femur has been known to occur in cases where the mother suffered from that deformity.

Vrolik considers that hereditary transmission exerts a decisive influence over malformations, causing their gradual decrease and final disappearance in the last born. This hypothesis is feasible enough, inasmuch as we know that many hereditary taints become, in the course of time, by healthy intermarriages, completely extinct in succeeding generations.

Malformations, having once appeared, are very prone to recur, and when they do so they are generally of the same species. Many cases are on record proving this fact; we content ourselves with the mention of only one or two. A woman bore three children within three years, and in all of them the forearms and lower parts of the legs were wanting.[3] Another woman who several times bore twins was finally delivered of a double monstrosity.[4] In fact diplogenesis often recurs in the same woman.[5] I delivered a woman, in Edin-

[1] Osiander. neue Denkwürdigkeit, vol. i. p. 193.
[2] Meckel. Pathol. Anatom., vol. i. p. 19.
[3] Flachsland. Observat. Pathol. Anatom. 1800.
[4] Du Vernoi. Act. Petropol. iii. p. 188.
[5] Bischoff: Wagner's Handworterbuch, vol. i. p. 910.

burgh, last summer, of her fifth child, which had harelip. All her former children were similarly deformed, and all of them had been operated on, for this affection, by Professor Syme. Dr Martin[1] relates the case of a lady—who had previously born two perfect and well-developed children, and who enjoyed excellent health—giving birth successively to seven which were malformed. Of these, six were anencephalic (five of them born at the full time, and one an abortion two inches long); the remaining one was a mature fœtus, having a normally formed cranium, but being so defective in the development of its flexor muscles, that its legs, arms, fingers, and toes could not be bent.

NO. VI. THE DURATION AND PHENOMENA OF PREGNANCY WITH MALFORMED CHILDREN.

Obstetrical works take very little notice of pregnancy with malformed children; but, from all the information we possess on the subject, we think it is evident that, in such cases, *the period of utero-gestation may be, and often is, quite normal in its duration and symptoms.* Professor Hohl,[2] of Halle, collected and carefully examined the statistics of 128 cases of pregnancy with deformed children, and the general results he arrived at I shall place here, for the sake of distinctness, in a tabular form.

DURATION OF PREGNANCY IN 128 CASES OF MALFORMATION.

	Nature of Malformations.		
97 attained the normal length,	Per defectum,	28	
	Parasitical,	3	
	Syreniform,	2	
	Diplogenesis,	64	
		—	97
31 were born prematurely,	Per defectum :— Chiefly acephalia and anencephalia,	14	
	Diplogenesis	17	
		—	31
	Total,		128

Of the above 31 cases, in which pregnancy was of abnormal length, the births occurred as follow :—

Before the	1st	month	there were born,	3
„	5th	„	„	3
„	6th	„	„	1
„	7th	„	„	12
„	8th	„	„	6
„	8th and 9th		„	1
„	9th	„	„	5
			Total,	31

[1] New York Journal of Medicine. March 1849.
[2] Die Geburten missgestalteter, kranker, und todter Kinder. Halle, 1850.

The foregoing tables show that, in three-fourths of all cases of malformed children, pregnancy attains the usual length; and that when birth occurs prematurely this happens most frequently in the seventh and eighth months of utero-gestation.

There are, generally speaking, no peculiar *symptoms characterising the period of pregnancy with malformed children*, by which their existence may be suspected by the mother, or diagnosed by the accoucheur. But, as in certain cases, abnormal appearances and sensations have been observed, we consider it our duty to mention them in this place.

a. Great abdominal distension from superabundance of liquor amnii

is the most common of these. Naegele[1] frequently observed it in cases of fœtal malformations; and Hohl[2] remarked it in five cases of anencephalia, and in four of diplogenesis. Ramsbotham[3] mentions the case of a woman who had born six children, three of which were anencephalous, and who had a much greater quantity of liquor amnii with each of the malformed children than with any of the others. My friend, Dr Grieve of this place, informs me that he recently delivered a multiparous woman of a child having defective formation of the cranium, and that there was great superabundance of liquor amnii, more than double the quantity in any of her previous pregnancies. The greatest quantity of liquor amnii I ever witnessed was in a case I attended last autumn, where the child was still-born, having anencephalia and spina-bifida. So great was the superabundance of the fluid, that the whole floor of the apartment was saturated by its discharge. How is this to be accounted for? We know that excess of liquor amnii is not unfrequent when the infant is diseased or dead; and in the case of malformed children we may conjecture that it probably results from some obscure intra-uterine inflammatory action affecting the membranes, or, perchance, in cases of anencephalia, from the bursting of some hydrocephalic sac.

b. Oedema and great weight of the mother's body.

These have been mentioned, as pathognomonic signs of pregnancy with malformed children, by Haller, Detharding, Villeneuve, Klein, etc.; but they seem to be so variable and uncertain as hardly to be depended upon for diagnosis.

c. Peculiarity of the fœtal motions.

This is even a less trustworthy sign than the foregoing; for, as far as I have been able to ascertain, there exists no uniformity in the intra-uterine movements of malformed children. In some cases these are described as being vigorous, in others as very weak. They are often very powerful in anencephalic fœtuses, where the brain is

[1] Lehrbuch der Geburtsh., 7th ed., p. 248.
[2] Op. cit., p. 58.
[3] Principles and Pract. of Obstet. Med., 1841, p. 661.

wanting,—a proof that the motions of the child in utero are not cerebral, but are purely excito-motory or reflex.

In fine, I consider that we have no means of ascertaining, with any degree of certainty, the existence of malformations during the intra-uterine life of the fœtus.

It may perhaps not be out of place here to inquire *whether malformations occur more frequently among primiparous or multiparous women?* The only author I am aware of who has given us any statistical information on the subject is Professor Hohl, the results of whose investigations I shall once more quote.

CONDITIONS OF THE MOTHERS IN 69 CASES OF MALFORMATIONS.

	Nature of Malformation.		
14 were primiparous,	Per defectum	9	
	Diplogenesis	5	
		—	14
55 were multiparous,	Per defectum	17	
	Per excessum, viz.		
	Diplogenesis 35	38	
	Parasites 3		
		—	55
	Total,		69

Hence it would seem that of malformations in general four-fifths occur among multiparæ; that of these the greater number are per excessum,—principally resulting from duplicity; and, on the other hand, that the minority, happening among primiparous women, are chiefly caused by defective development. May we suppose, in explanation of this, that the formative powers of nature become increased, or more fully developed, after they have been, or in proportion as they are called into action? Obstetrical statistics do not warrant any such conjecture; for, although twin births happen oftener among multiparous women, the frequency of their occurrence is not in a ratio corresponding to the number of the mother's pregnancies.[1] It will be seen by the following table (taken from Dr Collins[2]), of 240 cases of twin births, that while 72 occurred in first, and the remainder in subsequent pregnancies, the frequency of their occurrence decreased in proportion to the number of the previous pregnancies.

Statistics of 240 *Twin Births.*

Number of pregnancy,	1,	2,	3,	4,	5,	6,	7,	8,	9,	10,	11,	12,	13,
Number of women,	72,	35,	37,	20,	22,	15,	15,	6,	6,	7,	1,	1,	1.

[1] Dubois and Hardy assert that twin births occur more frequently among primiparæ, but their opinions are proved to be incorrect, alike by the statistics of Collins, cited above, and those of Professor Levy, of Copenhagen, the most recent authority on the subject. According to the latter obstetrician, 7742 births have occurred during the last ten years in the Copenhagen Lying-in-Hospital; and of these 117 were of twins. Of these 117 cases, 51 were born by primiparous, and 66 by multiparous mothers.—*Vide* his paper in *Hosp. Meddelelser., Bd.* 5; and in *Schmidt's Jahrb.* 1854. No. 3, p. 326.

[2] Practical Midwifery, p. 331.

It would seem from these statistics that the formative power (*vis formativa naturæ*) becomes weakened by being frequently called into action; and we think we may safely conclude—reasoning analogically—that, in this respect, malformations by duplicity are governed by the same laws as normal twins.

NO. VII. CONGENITAL LUXATION OF THE FEMUR.

In the beginning of February 1853, Professor Simpson kindly gave me a malformed fœtus for dissection, which had been sent to him by an obstetrical practitioner for minute examination. On inspection I ascertained that congenital dislocation of the femur existed on both sides; and so interesting were the appearances presented by the dissection, that I consider the following account and delineations of them not unworthy of a place among these "Contributions."[1] For, although this congenital displacement is by no means uncommon, opportunities of examining it in the dead body are, as acknowledged by Dupuytren,[2] comparatively rare, inasmuch as it is merely an infirmity, and not a disease necessarily abbreviating human life.

External appearance of the Fœtus.—I learned that the child had been prematurely still-born, and from its size I considered its intra-uterine life to have ended about the beginning of the eighth month of gestation. The head, trunk, and upper extremities were perfectly normal in their development; but the lower part of the body presented all the characteristic marks of congenital luxation of the femur. The limbs—considerably shortened and atrophied—were disproportioned to the size of the trunk, and obliquely placed in relation to it. They were also rotated very much inwards, and both feet were inverted by talipes varus. The femur admitted of very little motion; and on both sides its trochanters formed prominent projections above the site of the cotyloid cavity. The knee-joints were as immobile as if they had been anchylosed. The back could be bent abnormally between the dorsal and lumbar regions.

Dissection.—On both sides the following appearances presented themselves:—

The muscles of the thigh and gluteal region were very much atrophied and contracted, especially where they surrounded the hip-joint. By the retraction of the atrophied and shortened rectus femoris muscle, the patella and head of the tibia were drawn up over the condyles of the femur, thereby rendering flexion of the knee-joint impossible. The head of the femur, displaced upwards and outwards, lay on the dorsum of the ilium, where it had partially

[1] Professor Simpson did me the honour of exhibiting my dissection to the Edinburgh Medico-Chirurgical Society, at their meeting on 16th February 1853. (See *Monthly Journal*, vol. xvi. p. 567) I subsequently exhibited the preparation to the Edinburgh Physiological Society. (See *Transactions of the Society* in *Monthly Journal*, vol. xvi. p. 470).

[2] "Les occasions de determiner, par l'ouverture des corps, la nature de cette singulière espèce de luxation sont fort rares."—*Clinique Chirurgicale*, p. 82.

formed for itself a new cartilaginous acetabulum. The true cotyloid cavity was of the normal size, but slightly irregular in its conforma-

Fig. 1.

Fig. 1.—Dissection showing the displacement of the femur, and the condition of the ligamentum teres.

Fig. 2.

Fig 2.—Dissection of the other limb, showing the atrophied condition of the muscles, and the retraction of the patella.

tion. The axis of the cervix femoris was abnormal, the neck being placed nearly horizontally in relation to the shaft of the bone, instead of in its natural oblique direction. The capsular ligament of the hip-joint was lax; and the ligamentum teres was thicker, longer, and stronger than is usual in fœtal life.

On comparing the fœtus with others of the same age, I found the breadth of the pelvis to exceed the normal standard, and the external surface of the ilium to be more than usually convex.

Between the dorsal and lumbar vertebræ a false joint was formed, strengthened by abnormal fibrous development.[1]

[1] The dissection here described was given by me to Professor Goodsir (to whom I take this opportunity of publicly expressing my warmest thanks for the advice and assistance I received from him during many of my Teratological investigations), and may now be seen in the Anatomical Museum of the University of Edinburgh.

Remarks on the foregoing Case.

In congenital luxation of the femur, the displacement, as in the preceding case, occurs generally on both sides. Baron Dupuytren, who first accurately described the pathology of this affection, remarks that he only observed it confined to *one* side, in two or three out of twenty-six cases he had examined;[1] and, according to Melicher,[2] in four out of nine cases witnessed by Chelius, the luxation was unilateral. The experience of Mr Adams of Dublin[3] coincides with this; and the comparative rarity of one joint alone being implicated, constitutes, in his opinion, an important diagnostic mark of the affection.

The site of the displaced bone in the present case is the one usually met with in congenital dislocation. In malformed children the femur has occasionally been found luxated directly upwards, so as to lie immediately in front of the anterior inferior spinous process of the ilium; and it has also been observed to be congenitally displaced upwards and forwards, so as to rest on the ileo-pectineal eminence. Melicher[4] made several experiments on the dead subject with the view of ascertaining in what direction the femur is most easily displaced in fœtal life. By luxating the bone artificially in infants of various ages he found that the dislocation downwards and backwards into the ischiatic notch was more easily caused than any other variety. He experienced most difficulty in producing luxation upwards and outwards, only succeeding in two cases out of thirty in which he attempted it; and he was quite unable to effect displacement either into the foramen ovale, or upon the pubis. He is of opinion that these experiments warrant the conclusion that in congenital luxations, the head of the femur generally emerges from the cotyloid cavity at its anterior and inner margin, and that, thereafter, by a second dislocation produced by muscular action, it becomes tilted upwards and outwards upon the dorsum ilii. The displacement in the case under notice was, as we shall afterwards show, the one which would be most easily produced, by the influence of the contracted gluteal muscles, in the intra-uterine position of the fœtus.

Another point of great interest in this case, was the condition of the round ligament. It was found to be not only elongated, but thicker and stronger than is usual in fœtal life. These appearances seemed to confirm and illustrate the views entertained of its functions by Professor Goodsir, who regards it as a purely *suspensory ligament*, destined, sling-like, to prevent the whole pressure of the trunk from being thrown upon the head of the femur. The condition of the round ligament in this case is all the more remarkable

[1] Loc. cit.
[2] Dr L. J. Melicher, *Die Angeborne Verrenkungen.* Vienna, 1845.
[3] Cyclopædia of Anatomy and Physiology, vol. ii.
[4] Op. cit.

and interesting, when we reflect that it was observed in a joint which had never been called into action, and that it evidenced an ingenious provision made by nature to counterbalance the effects of the displacement; for, had this child lived, nearly the whole weight of the body, owing to the head of the bone not being in the acetabulum, must have been supported by the ligamentum teres, which, accordingly, was formed of unusual strength, to fit it for this function.

I am not aware of any author who, in treating of congenital luxation of the femur, has specially directed attention to the condition of the ligamentum teres, or who has noticed elongation and thickening of it as peculiar to the displacement. Dupuytren[1] says that occasionally the ligament may be seen, but considerably changed. Elongation of the ligament has certainly been observed in dissections of the *adult* subject. Dr Heine,[2] of Canstatt, in a dissection he made of the lesion in a patient, æt. 40, found the round ligament wanting, and its place of insertion into the femur covered with cartilage. M. Bouvier[3] exhibited to the French Royal Academy of Medicine, a preparation of the congenital dislocation of the femur, taken from a woman, æt. 53. He described the ligamentum teres as "much elongated, flattened, and partly confounded with the capsule of the joint." But the dissections of Heine and Bouvier, as well as most of those executed by Dupuytren, having been made upon *adult*, not upon *fœtal* bodies, the appearances of the joint described by them cannot, with justice, be regarded as demonstrative of the truly *congenital* condition of the structures concerned in this deformity, for it is easy to understand that the parts would undergo considerable alterations during the lifetime of the individual, becoming more and more changed, by spontaneous adaptation to the abnormal circumstances in which they were placed. But appearances of the round ligament, analogous to those I have described have been observed in infancy; although, so far as I am aware, only by one author. Parise,[4] in a case of congenital luxation in an infant ten weeks old, describes the ligament as being "*long* and on the stretch;" and, in another dissection of the displacement, in a child aged six days, as being "*longer than ordinary.*"[5] I am strongly inclined to believe that the increased length and strength of the ligament, observed in cases examined in adult life, are merely its congenital conditions exaggerated by dint of usage; and that more extended future researches will demonstrate these appearances to be present at birth in nearly every case of the displacement.

In order to ascertain how far the present case goes to elucidate

[1] Loc. cit.
[2] Uber Spontane and congenitale Luxationen, Stuttgard, 1842.
[3] Bulletin de l'Academie Royale de Médecine. February 1841.
[4] Archives Générales de Médecine. Tome xiv. p. 439.
[5] Ibid. Loc. cit., p. 546.

the etiology of this congenital luxation, it is necessary, very briefly, to allude to the various views which have been held in regard to its causes. These displacements have been considered due to,

1.—*Mechanical Violence during Gestation.*

Hippocrates and the older authors were of opinion that this luxation occurred *in utero* traumatically, from the operation of external mechanical causes, such as falls, blows, and muscular contractions, or preternatural smallness of the uterus. But we now know that this displacement is very unlikely to result from these causes alone; and, moreover, that luxation is rarely, if ever, found in cases where such injuries have been received by the mother during pregnancy. Pathologists have therefore ceased to regard mechanical violence as a cause of pure congenital dislocations.[1]

2.—*Defective Organization of the Early Embryo.*

Dupuytren and others consider congenital displacements to have existed, as such, from the earliest period of intra-uterine life, and to be due to an aberration of the *autotrophy*, or power of self-development inherent in the fœtus. But this view seems untenable and inconsistent with what we know of embryo-genesis.

3.—*Diseased and Abnormal conditions of the Hip-Joint.*

a. Defective Development of the Acetabulum.

Breschet conceived congenital luxations to be the result of irregular or insufficient formation of the cotyloid cavity. This explanation is by no means satisfactory; for, in many instances (as in the present case), the acetabulum is found to be of normal size. Thus, in three cases examined by Parise, the cavity was found[2] to be of ample size and regular conformation, and in one of the cases (to which allusion has already been made), occurring in a child ten weeks old, the head of the bone, displaced upwards and outwards, rested on the crest of the acetabulum, where it had formed for itself a cartilaginous depression, which communicated with a very large and normal cotyloid cavity.

b. Morbus Coxarius.

This has often been assigned as one of the causes of intra-uterine dislocation of the femur; but the theory is not supported by dissections, which show the disease to be comparatively a rare one in the fœtus. Instances are on record, however, in which congenital luxation is said to have resulted from this affection; but these are very

[1] Undue violence on the part of the accoucheur, during parturition, may undoubtedly occasion luxations in the fœtus, although this rarely occurs; but such obstetric injuries cannot, strictly speaking, be called congenital affections. In the above remarks, therefore, I have avoided making any allusion to mechanical violence exerted by the obstetrician.

[2] Loc. cit.

few in number, and Melicher[1]—the latest and best authority on the subject—says that he never saw a case of truly congenital luxation occasioned by it.

c. Hydrarthrosis.

We have already, more than once, made reference to results stated in a paper written by M. Parise, in volume xiv. of the *Archives Générales de Médecine*. The object of this admirable monograph was to prove that dropsical effusions into the cavity of the acetabulum were the usual and principal causes of spontaneous dislocation of the head of the femur. He found that artificial injections of the hip-joint had the effect of causing this luxation; and he reasoned analogically, that a preternaturally great amount of synovial secretion would produce, pathologically, what he had done experimentally. That hydrarthrosis may be an occasional cause of congenital luxation of the femur, I do not doubt, but that it is by any means a constant or uniform accompaniment of the lesion, appears to me highly improbable. Indeed the testimony of Parise himself seems to destroy the exclusiveness of his theory; for, out of 332 cases, in which he examined the joints of new-born infants, during the time he was house-surgeon to the *Hôpital des Enfans Trouvés*, at Paris, he only found *three* in which he could distinctly trace congenital dislocation to this cause.[2]

d. Hypertrophy of the Haversian Gland.

Parise and others believe that the displacement may also result from an increased growth of those adipose folds, generally found within the synovial membrane of the hip-joint, which have been somewhat erroneously denominated "Haversian *Glands*," but which are, in reality, merely vascular accumulations of fat-cells. In support of this theory, these authors have related cases of congenital luxation, in which there existed visible hypertrophy of these masses; but the assertion that, in these cases, the displacement was caused by the enlargement of the Haversian glands appears to me a most unwarrantable application of the *post hoc ergo propter hoc* line of argument. The increased growth of the adipose substance in these instances seems to be rather the *result* than the *cause* of the luxation; for, had the head of the femur remained *in situ*, there is every likelihood that its presence and pressure would have restrained that hypertrophy, which occurred exuberantly as the sequence of its displacement.

e. Muscular Retraction.

In the great majority of cases I believe congenital luxations to be occasioned by the influence of muscular retraction. Guerin was

[1] Op. cit.

[2] It was in the dissection of one of these cases that he observed the elongation of the round ligament to which I have already alluded.

probably the first who suggested this view of the etiology of the affection; and in 1840 he thus wrote:[1]—"Congenital luxations of the hip are, like talipes, torticollis, and spinal deviations, the product of primitive muscular retraction; and the kind of the dislocation depends upon the direction in which the retraction happens." Melicher and Dr Carnochan of New York—the two most recent writers on the subject—have since adopted the opinions enunciated by Guerin; and the appearances presented by the dissection I have described, seem to corroborate the views entertained by these authors regarding the etiology of the displacement. We know that many pathological conditions are produced by muscular retraction, whether dependent upon spasm or upon shortening and atrophy, both in fœtal and in extra-uterine life. The hip-joint is, of all the articulations, the one most liable to congenital luxations; for the position assumed by the fœtus *in utero*, where it lies with the legs flexed on the abdomen, is the one of all others which predisposes the head of the femur to slip out of the acetabulum, and to mount upwards and backwards, as in this case, upon the dorsum of the ilium. It is easy to understand, therefore, that if muscular force should be exercised upon the head of the femur, while the fœtus lies in this position, that the bone would be displaced from the cotyloid cavity. The atrophy and shortening of the muscles, in this case, sufficiently account for the luxation which occurred. In some cases, however, displacement may exist without any visible abnormality of the muscular system. The retraction, in such cases, is probably dependent upon that super-polarity of the nervous system to which infancy is so prone, which may result from either a perverted condition of the cerebro-spinal centres, or from some obscure irritation, local or remote, communicated to them, and thence transmitted by reflex action, to the parts wherein the morbid action is developed.

NO. VIII.—THE ACEPHALOUS FŒTUS, AND THE PHYSIOLOGY OF ITS CIRCULATION.

Under the term *Acephali*, are ranked all those monstrosities in which the head is entirely absent, and the other parts of the body are more or less imperfectly developed. They are also called *Acardiac;* for in them the heart is invariably wanting. The extent of the co-existing malformations varies in different cases, so that they are divisible into the following five distinct divisions:—*First*, those in which the head alone is absent; *second*, those in which the head and upper extremities are wanting; *third*, those in which the head, thorax, and upper extremities are undeveloped; *fourth*, those in which the fœtus consists of a rounded mass, with rudimentary lower extremities; and *fifth*, those in which there exists only an amorphous mass, possessing no indications of extremities.

[1] Gazette Médicale de Paris, 1840, No. IV.

Acephali almost always occur in multiple births, and are born along with one or more well-formed children—generally with one only—occasionally with two (Superville, Wernher, Göller, etc.); and in some rare cases with three (Soemmering, etc.) Authentic instances of acephali being born singly are very rare. Photenhauer[1] relates a case; and M. Cazeaux, on the 21st February 1851, read to the Imperial Academy of Medicine of Paris, a letter from M. Leroy d'Etiolles, relating to an acephalus having been singly born. Sulsmann, Duneaud, and Vallisneri, mention similar cases; but in these instances, hydatigenous moles were simultaneously expelled from the uterus.

The Placenta.—In most cases, one common placenta supplies the acephalus, and the accompanying children. Occasionally there exist two placentæ united together; and in some rare cases each fœtus possesses a separate placenta. The latter arrangement was found in only three out of seventy-two cases of this malformation described in Elben's admirable treatise.[2] The placenta, when common to both, may contain two cavities, one for the perfect, the other for the malformed child. Sir Astley Cooper describes such a case, and mentions that each twin had a separate funis, the vessels of which anastomosed freely and distinctly in the placenta.[3] This placental anastomosis of the umbilical vessels of both fœtuses is of very frequent occurrence.

The Umbilical cord of the acephalus varies in length from an inch and a half (Clarke),[4] to 7 or 8 inches (Mayer,[5] Hempel).[6] In some cases, as in that of Méry,[7] a single cord, divided into two, is inserted into a common placenta; but ordinarily, each fœtus possesses a distinct and separate funis. The umbilical vessels are often abnormal in number. The cord may contain two arteries and one vein (Tiedemann, Soemmering); one artery and two veins (Busch); or two arteries and two veins (Mayer, Cloquet). It may be attached either to the centre (Clarke), or to the edge of the placenta (Cooper, Hempel). In most cases, it enters the umbilical region of the acephalic fœtus. The two cords are nearly always separate from one another; but Mayer has recorded a case in which they were united.[8]

Osseous System.—Distinct traces of this system are to be found in even the lowest types of acephali. The vertebral column and the ribs exist, in a more or less imperfect condition;—Dr Clarke's[9] being the only case on record in which the spine was entirely absent. The pelvis usually deviates less from the normal type than the other organs, although, in some cases, it is very much deformed.

[1] De Monstr. Acephal. Berlin. 1835. [2] De Acephalis, etc. Berlin, 1821.
[3] Guy's Hospital Reports, vol. i. p. 227. [4] Trans. Roy. Soc., Lond., 1793.
[5] Verhandl. der Gesellschaft. für Geburtsh. Berlin. 1846.
[6] De Monstris Acephalis. By C. F. Hempel. Copenhagen. 1850.
[7] Acad. des Sciences de Paris, 1720. [8] Loc. cit. [9] Loc. cit.

Muscular and Areolar Tissues.—The muscles are pale, soft, and flabby; and they have been found in a state of fatty degeneration (Schelhammer, Gourraigne). Tiedemann, Odhelius,[1] and others, mention cases in which there existed no true muscular system; but as these physiologists were not in the habit of employing the microscope in their examinations of structures, I consider the accuracy of their observations to be very doubtful. The muscles, however, may be very imperfectly developed, and they are often very irregular in their origins and insertions. The areolar tissue in acephali is very abundant, and it is generally very œdematous from the infiltration of effused serum into its texture.

Nervous System.—In the higher varieties of acephali, the spinal cord, with its afferent and efferent nerves, is often found in a tolerably perfect condition; but in the less highly developed forms of these malformations it is either wanting, or in a very rudimentary condition. The number of its nerves varies according to the amount of its development. Hempel found 25 pair in one case. The defective condition of the cerebro-spinal system in the acephalic, and also in the anencephalic fœtuses, shows embryogenesis to be totally independent of the influence of this portion of the nervous system. Not so as regards the ganglionic system of nerves, which seems indispensable for the growth and nutrition of the fœtus. This is invariably present, even in the most imperfectly developed acephali. In a fœtus of this sort dissected by Sir Astley Cooper,[2] the sympathetic began in a large ganglion, and formed several smaller ones, from which filaments were sent to the aorta. Tiedemann and Soemmering found the hypogastric and lumbar flexuses, supplying branches to the principal arterial trunks. In every case branches of the sympathetic follow the course of, and ramify upon the arterial vessels of acephali—a fact of considerable importance in relation to the physiology of their circulation.

Lymphatic System.—The existence of this system in acephali has not as yet been satisfactorily demonstrated. Reasoning analogically, we may believe in its occurrence, since, in a dissection of Munro's,[3] mesenteric and mesocolic glands were discovered.

Genito-Urinary System.—Prochaska's[4] case is the only one on record in which no trace of genital organs could be discovered in an acephalic fœtus. The kidneys are nearly always present, but the supra-renal capsules are often absent. The external genitals may exist while the internal remain undeveloped; and the converse of this may occur, for Atkinson[5] found ovaria and Fallopian tubes present without either uterus or vagina. The uterus is often malformed, and it may be bicornuous (Katzky),[6] arrest of development having occurred at any early period of fœtal life.

[1] Ephem. Acad. Nat. Curios, 1690, Dec. 2.
[2] Loc. cit.
[3] Edin. Phil. Trans., vol. iii.
[4] Elben. Op. cit.
[5] Phys. and Med. Journ., No. 65. 1804.
[6] Act. Med. Berol., Dec. 1, vol. ix., 1721.

Abdominal Viscera.—The stomach is frequently absent; the pancreas, liver, spleen, supra-renal capsules, and thoracic duct, are generally wanting. Schelhammer's[1] seems to be the only case in which the liver and gall-bladder were found in an acephalus. The rectum is occasionally obliterated, but in nearly all cases we find some part of the alimentary canal, and also the internal and external urino-genital organs. How are we to account for the absence of the viscera? Were they never developed, or did they exist until destroyed by some intra-uterine inflammatory action, of which we may regard the serum as evidence? I look upon the serum, effused within the thoracic and abdominal cavities, and infiltrated into the cellular tissue, not as the consequence of inflammation, but as the mechanical result of the sluggish and tardy circulation which necessarily exists in these fœtuses, permitting the liquor sanguinis to exude by exosmose, just as happens in ordinary cases of dropsy arising from some obstruction of the natural current of the blood. I attribute the absence of the viscera solely to defective development. "The connection," says Vrolik,[2] "between the containing and the contained parts, is very distinctly proved by the acephali. The very general presence of the lumbar part of the spine determines the existence of kidneys; that of the pelvis, the existence of the urinary bladder, and the genital organs. The very imperfect condition of the thorax is in relation with the absence of the heart." Although I agree with Vrolik in his hypothesis as to the abdominal viscera, I think his theory concerning the relation between the thorax and its contents is not borne out by facts, for in some acephali the thorax is tolerably perfect, while the heart and lungs are entirely absent.

In the acardiac fœtus we always find more or less trace of the intestinal canal. In the least developed varieties of acephali, the existing portion is generally the lower end of the tube, and this is often attached to a mesentery or meso-colon. In less malformed acephali we find the colon, and in their most perfect varieties, occasionally the small intestines. Malacarne[3] found the intestinal canal ending superiorly in a dilatation, which showed development to have been arrested at the period when a swelling forms on the primitive tube to form the future stomach. In two cases, Tiedemann found it terminating in the substance of the funis, a fact which demonstrated the development of the alimentary canal from the umbilical vesicle.

Physiology of the Circulation in the Acardiac Fœtus.

I have stated that, in the acephalic fœtus, the heart is invariably absent. Gilibert,[4] Katzky,[5] and Vallisneri,[6] have described rudi-

[1] Loc. cit. [2] Article "Teratology," Cyclop. Anat. and Physiology.
[3] Ogetti pice inter di Ostetrica. Padua, 1807.
[4] Adversaria. Medic. Pract. Prim., 1791. [5] Loc. cit.
[6] Elben. op. cit. p. 4.

mentary hearts as existing in acephali, but these very exceptional cases were probably merely dilatations of the main arterial tube. Poujol thought that there are no arteries either in the malformed fœtus or in its funis; and Winslow, on the other hand, believed in the existence of arteries only, and no veins. These learned anatomists must have been in error here, for no other investigator has confirmed their observations.

In an acardiac fœtus (of the second class, according to my division), dissected by Dr Munro in 1792, he found that the umbilical vein, on entering the abdomen, divided into various branches, which were sent to all parts of the body. And it was very remarkable that "vessels everywhere accompanied these branches of the umbilical vein, corresponding with them in size, as well as situation, and joining together formed trunks, from which, at the sides of the pelvis, two vessels were continued, one of them on each side of the vesica urinaria and urachus to the umbilicus, which they perforated, and then went along the umbilical cord towards the placenta, resembling the umbilical arteries."[1] Sir Astley Cooper[2] found the funis containing *one* umbilical artery, and a very large umbilical vein. The latter, on entering the abdomen, divided into two vessels, one of which supplied the abdominal and pelvic viscera, and the lower extremities; while the other, passing to the spinal cord, formed a sinus venosus at its upper end. The aorta begun in two branches at the upper part of the spine; the rudimentary subclavian arteries turned on the ribs and supplied the integuments of the upper half of the body. It "*had no communication with the veins but by means of the extreme branches of each.*" At its lower part it divided into two iliac arteries, from the left of which the umbilical artery proceeded, with the accompanying vein, to the placenta.

In a case recorded by Dr Houston,[3] the umbilical vein opened into the right external iliac vein. The veins were totally devoid of valves. Branches of the arteries "*arose by fine capillary roots out of every texture in the body. These, coalescing, gradually formed larger trunks, which, when united, constituted a sort of aorta or central vessel*, like the aorta in fishes, on the fore part of the spine. . . . The umbilical arteries took origin from the internal iliacs, and ran to the umbilicus, at which point they entered the cord in the usual manner. . . . No communication existed anywhere between the arteries and veins except at the capillary terminations of the vessels."

These dissections, as well as others described by Hempel, Mayer, and Richard,—which limited space precludes me from quoting,—seem to show that the vascular system in the acephalic fœtus consists of two main vessels, one venous, the other arterial; that the former is continuous with the umbilical vein, and the latter with the umbilical artery; and those both are ultimately united to one

[1] Trans. Roy. Soc., vol. iii. p. 217. [2] Loc. cit.
[3] Dub. Journ. of Med. Science, vol. x.

another by means of a fine capillary anastomosis. The portal vein, like the liver, is absent.

Many theories have been advanced to explain the physiology of the circulation in the acardiac fœtus. In discussing this vexed question and advancing some hypotheses of my own in explanation of it, I would do so with great deference to the opinions of the many distinguished physiologists who have written on the subject.

I. Winslow[1] and Poujol denied the existence of any circulation whatever. The incorrectness of this view needs no refutation.

II. Mèry[2] and Le Cat[3] imagined the blood to be propelled by the force of the maternal heart. These authors, however, were unaware that no direct interchange of blood takes place between the mother and the fœtus.

III. Dr Young, aware of the frequent anastomosis of the umbilical vessels in the placenta, ingeniously conjectured that the acardiac circulation might be carried on by the propelling power of the heart of the perfect fœtus. This hypothesis was also adopted by Sir Astley Cooper, who supposed it to be effected by an inverted circulation in the funis of the acephalus, and by a placental anastomosis between the umbilical arteries of both fœtuses, by means of which the blood of the perfect child was conveyed *to* the acephalus by the umbilical *artery*, and *from* it back to the placenta by the umbilical *vein*. But it seems highly improbable, not only that the whole circulation should be thus inverted, but that the heart of the perfect child should possess sufficient power to propel the blood, first through the placental anastomosis, next through the blood-vessels of the acardiac fœtus, and, lastly, from them back to itself through the placenta once more. Moreover, were this theory correct, the normal child, in consequence of its blood passing to and from the acardiac fœtus through the placenta without efficient maternal renovation, would be supplied with very impure blood, and we might therefore expect to find it ill-developed, but this is not its condition, for it is generally plump and well-conditioned.

These objections are also applicable to the more recently enunciated theory of Dr Marshall Hall,[4] that the blood is propelled by the agency of the heart of the normal co-twin "through the capillaries of the placenta into the umbilical vein, and through the umbilical vein and a series of capillaries to the aorta."

IV. "May we not venture to conjecture," asks Dr A. Cooper,[5] "that the peristaltic or living muscular power of the arteries is principally subservient to this important end?" Munro, Clarke, Breschet, Lobstein, Tiedemann, Müller, and other physiologists, have been of this opinion; and I shall presently endeavour to prove that

[1] Mem. de l'Acad. des Sciences, 1740. [2] Ibid., 1790.
[3] Philosoph. Trans, vol. lvii.
[4] Monthly Journ. of Med. Science. 1843. Vol. iii., p. 544.
[5] Philos. Transac., vol. lxv.

the muscular contractility of the vessels plays no important part in the circulation of the acardiac fœtus.

V. Dr Houston[1] regards the placental circulation, in such cases, as normal, but thinks that the course of the blood *through the acephalic fœtus is reversed:* viz., "that that fluid enters by the veins, and returns by the arteries." He considers that the circulation is carried on by the vital powers of attraction inherent in the vessels of the acephalus itself. His views seem to me to be the most correct yet advanced in explanation of this difficult question.

It will be seen that there exists much difference of opinion as to the channels by which the blood is sent to and returns from the acephalic fœtus. Previous to Sir Astley Cooper's time, Breschet, Tiedemann, and Brodie, conceived the circulation in the cord to be inverted, although they did not, like him, attribute the motive power of the circulation to the heart of the co-twin. Munro, long ago, held that the umbilical circulation remained unchanged; and the following circumstances related by Dr Houston seem to demonstrate indisputably that the course of the blood is normal, being conveyed *to* the acephalus *by the vein*, and returned *from* it *by the arteries.* In the acardiac fœtus dissected by him, about an inch from the umbilicus, there lay, in the substance of the cord, a small tumour, which had the effect of rendering *the vein* "dilated, elongated, and tortuous" on its placental aspect, and *the arteries* enlarged between the fœtus and the tumour.

Assuming this to be the order of the circulation in the cord, we have next to inquire what course the blood takes *within* the body of the malformation. "By whatever system of vessels," says Dr Houston, "the blood entered this fœtus, by them it must have been distributed through all the tissues of the body." Seeing, then, that the umbilical vein, in his case, opened into the right external iliac vein, and that all the venous trunks in the body were (as Gurlt affirms is always the case in acephali) *totally devoid of valves*, I think we must, with Dr Houston, conclude that the blood was distributed by the veins to every part of the body, and that it was thence returned to the placenta by the umbilical arteries.

In fine, I am of opinion that, in every case, the blood enters into and is distributed through the body of the acardiac fœtus by means of its venous system. Thence it is conveyed to the main arterial tube or tubes through the medium of the capillary anastomoses which everywhere exist, and by which alone there is communication established between the arteries and the veins; and, finally, from these arteries it is sent once more to the placenta.

Admitting this, then, to be the course of the intra and extra-fœtal circulation in acephali, the question arises, by what agency is it maintained? I consider it to be dependent not on any single propulsive power, but on a combination of vital forces.

[1] Loc. cit.

First, I conjecture that *by means of the inherent muscular contractility of the main arterial vessel or vessels of the acardiac fœtus, the blood is propelled from them to the placenta.*

That arteries *are* possessed of muscular contractility has been placed beyond doubt by the experiments of the Webers, but the amount of influence exerted by this force on the circulation of the blood has been very variously estimated. Some physiologists believe that it aids and supplements the propulsive powers of the heart, and others that no action of this kind is habitually exercised, and that if it were developed to any extent it would tend to retard rather than to accelerate the circulation. We know that in the embryo of the chick, before the foundation of a heart, a free circulation can be perceived in the vascular area; and that the arterial tube, from which the heart is afterwards developed, can then be seen actively pulsating and vigorously propelling the blood. This arterial contractility becomes lost, in a great measure, after the development of the perfect heart, there being no longer the same necessity for its existence. Now, in cases where, at an early period of embryonic life, the vascular system becomes arrested in its development, so as to leave only a main arterial tube instead of a perfect heart, may we not legitimately conjecture that the primitive amount of contractility will remain undiminished to carry on the fœtal circulation in lieu of the absent heart? I think such a hypothesis is quite warrantable, and I fancy that the arteries in the adult body may possess, more or less, a latent power of like contractility, which, however, is never developed, on account of there being no necessity for its manifestation. In the acardiac fœtus the absence of the heart necessitates the arteries to take its place and carry on the circulation; and there seems to me nothing improbable in the supposition that the primitive simple arterial tube, from which the heart is later developed, should continue "in cases of arrested growth" to keep up the flow of blood by means of its contractions. In this supposition I am supported by the weighty authority of Müller,[1] who says in regard to this:—"One point of the arterial stem may have had contractile power, and have thus supplied the place of the heart, which in the embryo at its earliest period had the form of a vessel. If a circulation really did exist, *it could continue any length of time;* and indeed since, in some of these cases, the spinal cord also is deficient, these monsters seem to prove that the circulation of the blood in their double system of vessels can be carried on without the aid of the brain and spinal cord, and consequently that the contractile parts of viscera which are supplied by the sympathetic nerve may be completely independent of the brain and spinal cord." It is remarkable that the sympathetic with its ganglia, the filaments of which probably govern the contractions of the heart and arteries (Valentin having observed them terminating in a plexus in the middle arterial

[1] Physiology, Baly's Translation, vol. i. p. 197.

coat),—is never wholly absent in acephali, even in the lowest types of the malformation, where the spinal cord and its nerves are totally wanting.

The nature of the circulation which we have supposed to exist in the acardiac fœtus, resembles that which is really found in some of the lower animals, as in the annelides, etc. In the earth worm, for example, the circulation is carried on by means of "an elongated, simple, pulsatory, median, dorsal artery, provided with distinct muscular fibres,"[1] to which it is again returned by one, two, or more lateral veins. I conceive the main arterial tube of the acardiac fœtus to act like a dorsal vessel of these lower animals, in maintaining the circulation by its own inherent contractility.

Second. The return of the blood from the placenta to the acardiac fœtus is probably effected by *a vital attractive force inherent in the capillary blood-vessels.*

That such a power resides in the capillaries, capable of carrying on the circulation to a certain extent, quite independently of the influence of the action of the heart, seems to me a well-established fact, proved by the following, among other circumstances :—(*a*), in the vascular area of the chick, long before the appearance of a pulsatile vessel, a distinct current of blood-vessels can be observed ; (*b*), when masses of effused coagulable lymph become vascular, the blood can be perceived circulating in the newly-formed vessels which appear in them, long before these new vessels join the contiguous capillaries ; (*c*), the increased vascularity of inflamed parts, and also of the gravid uterus, argue negatively the existence of some such force, inasmuch as they are not dependent on increased action of the heart.

This vital power has been called by Professor Carpenter, "capillary force," and it is probably dependent on two causes ; *first*, on the peculiar vital attractions and repulsions of the animal solids and fluids, as suggested by Professor Alison ; and *second*, on the laws of exosmose and endosmose as pointed out by Dutrochet, and recently beautifully illustrated by Professor Draper[2] of New York.

The existence of this vital force appears to me to explain satisfactorily the circulation in the capillaries of the human body, and also in those of the acardiac fœtus. In the lungs of the former, in the placenta of the latter, and in all the tissues, of both, this process unceasingly goes on, by exosmose from the blood of the nutritive plasma, and endosmose into it of the effete matters for excretion.

I conjecture that in the acardiac fœtus the circulation is carried on by the two great forces I have mentioned ; that by the contractions of the main arterial tubes, and probably also by the vital attractive force of the placental vessels, the blood is conveyed from it to the placenta ; and that it is thence returned to the acephalus,

[1] Grant. Compar. Anat., p. 440.

[2] On the Forces which produces the Organization of Plants. New York, 1844.

and through its veins distributed to all its tissues, by means of this inherent capillary force.

It may be thought that the free inosculation of the umbilical vessels of the acephalus and its co-twin which I have described as often occurring in a common placenta, militated against the hypotheses I have advanced, and strengthens the theory entertained by some, that the circulation is carried on by the power of the heart of the perfect child. I have already endeavoured to show that the acephalus cannot be thus nourished, but I would, in conclusion, adduce the following facts which seem to me further to disprove this theory of the acardiac circulation :—

1*st*. Instances are on record of acephali having been singly born. Doubtless such cases are very rare, but I have mentioned several carefully observed instances related by trustworthy authors.

2*d*. The acardiac fœtus may possess a separate placenta. This also occurs but seldom; but that it does occasionally happen is undoubted. Thus, in Munro's case[1] the acephalous had "its own proper membranes and a placenta."

3*d*. The placenta of an acardiac fœtus may be quite normal. In Brodie's case[2] there was one placenta common to the malformed fœtus and its co-twin, but there was nothing unusual in its appearance, and the two cords took their origin from it, "about three inches distant from each other."

4*th*. A placental anastomosis of the umbilical vessels occasionally happens in cases of normal twin-births. In ordinary cases of twins and triplets, each fœtus possesses a distinct placenta; these are often marginally united together; but not unfrequently there is only one common placenta, on the surface of which the umbilical vessels freely inosculate. There are one or two placentæ of this kind in the Museum at Berlin, and there is also a cast of one in Professor Simpson's Obstetrical Collection in the University of Edinburgh. Smellie, Chaussier, and other obstetricians, admit the occasional occurrences of double twin-placentæ, having in them a vascular communication between the vessels of the cord. Desormeaux, in 1817, exhibited a placenta of this kind to the Faculty of Medicine of Paris, in which the umbilical arteries freely anastomosed. Guillemeau relates a case of twins, in which the placentæ were united *en raquette*, where the umbilical veins anastomosed freely before entering the placentæ, and the umbilical arteries also inosculated. These cases, I think, render it quite evident that a placental inosculation of the umbilical vessels in multiple births does not necessarily prove either that a common circulation exists between the twins, or that the one fœtus is dependent on the other for its nutriment.

5*th*. A common circulation, instead of aiding, would retard the nutrition of the acephalus. I have already shown that a venosity of the circulating blood would be induced which would be injurious to

[1] Loc. cit.

[2] Phil. Trans., 1809, p. 161.

the growth of both fœtuses. But it can further be proved, on pure mechanical principles, that a common circulation would actually *diminish* the supply of blood sent to the acardiac fœtus. This was first pointed out by Dr Houston,[1] who says, "The suction power of the heart, no matter how feeble it be, will attract to itself the blood received into the capillaries of any veins within the range of its influence, rather than permit it to flow off in a new direction, for the development and growth of another and distinct being; more especially, if the blood-vessels of that being be not, as Dr Marshall Hall's hypothesis teaches, endowed with any innate power of attraction for that fluid. The perfect fœtus would thus, by its heart, take to itself back again all the disposable blood within its reach, and thereby become the instrument of depriving its acardiac twin companion of its supply, rather than of adding to its store."

[1] Loc. cit.

FINIS.

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