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THE MADRAS MEDICAL JOURNAL.

SEPTEMBER 1924.

IMPRESSIONS OF MY WESTERN TOUR.*

(DR. RAO BAHADUR M. KESAVA PAI. M.D. & C.M.,

Superintendent, Government Tuberculosis Hospital, Madras.)

VOYAGE.

I have to thank you all for giving me this opportunity of meeting you soon after my return from Europe and having a talk with you on my experiences in that interesting country during my study leave. I am sure after a long spell of lectures on Medical subjects you will find it a great relief to listen to what is really a lay subject or what I might correctly call the ramblings of a provincial medical man on study leave. Many of you, specially such as have never gone to Europe and are now making your programmes of going there in the immediate future, may easily imagine with what zeal, enthusiasm, and hope one makes his preparations for his departure spending days and nights not only on formulating schemes of study in different institutions and in different countries of Europe, but in arranging for his domestic affairs being conducted during his absence from home.

I do not wish to waste your time by narrating my experiences of the voyage from Colombo to Marseilles. Suffice it to say that after the stormy voyage with

* An address delivered on 20th September 1924, on the occasion of a welcome "At Home" arranged by the Madras Medical Association.

sea sickness in the Arabian Sea, the baking in the Red sea in the hot month of July, it was with a sigh of relief that I found myself after 12 days in the cool air of the Mediterranean and felt so happy after another week's trip to find myself in the metropolis of the British Empire, tasting for the first time after a tiresome voyage of about 3 weeks, a good savoury Indian dinner with rice, curry, kolamboo and mollagatonnie.

The Brompton Hospital.

The morning after reaching London I reported myself at the Brompton Hospital, wherein were centred my hopes of making a deep study of tubercular diseases of the chest. The Brompton Hospital is one of the oldest hospitals of London, opened towards the middle of the last century. It is situated in the midst of the busiest localities of South Kensington close to the bus and underground railway routes of London. In construction there is nothing to indicate from the outside that it is a hospital excepting the large sign board marked "The Brompton Hospital for Consumption and diseases of the Chest." The out-patients department has two consulting rooms which are used in turn on different days of the week by about half a dozen physicians and assistant physicians, each of whom looks after his own set of patients who report themselves on the particular days and hours on which their physicians attend the hospital for the out-patient's work. I might mention in this connection that all the physicians and surgeons of the Brompton Hospital, as of most other hospitals in England, are honorary workers and are given the fullest freedom of working out their own methods of treatment untrammelled, subject of course to the financial limitations of the institution. The resources of the

hospital are kept freely at their disposal and they attend and work as regularly and conscientiously as the paid staff of our hospitals here. The Medical graduate soon after passing has to work as a clinical assistant in such hospitals helping the physicians and assistant physicians in the examination of their cases and later becomes a house physician or house surgeon drawing a small honorarium which barely suffices for his pocket expenses. After a year or so of work and experience as a house physician he can hope to be a resident medical officer, gaining a more practical experience as a clinician which enables him to become later on an honorary assistant physician of the hospital. It is only after several years of honorary service that he can become a physician and at an age when in this country we are expecting to retire from the service, the physician or surgeon in Europe has sufficiently established his reputation to become an honorary consulting physician or surgeon of a hospital. Needless to say that it is the highest ambition of every medical man of repute to be appointed an honorary consulting physician of a recognized medical institution. It raises his status as a consultant in his private practice and as you will find, on the front page of many standard books on medicine, the author never forgets to give a complete list of all the honorary appointments he has held in his professional career.

The in-patients department of the Brompton Hospital consists of about 300 beds affording relief, besides tubercular diseases of the chest, to a number of non-tubercular diseases of the pleura and beings. There are about half a dozen eminent physicians of London between whom these 300 beds are distributed, each physician looking after his

own cases and practising his own methods of treatment with the help of one or two house physicians. Though the premier institution of Great Britain for tubercular diseases of the chest, it cannot be said that either in its construction or in its methods, the Brompton Hospital leaves nothing to be desired. Though fairly well equipped with its X-ray rooms, surgical theatre, pathological museum, bacteriological laboratory, etc., it cannot be said that the hospital is an absolutely ideal one as a clinical school. There is no regular post-graduate course, except a short one of a week or ten days given once a year, meant more for the benefit of undergraduates appearing for their final examination in clinical medicine than for the post graduate who comes from thousands of miles abroad with a thirst for up-to-date knowledge. It cannot be ranked with some other institutions in Great Britain in its clinical methods or with some of the high class hospitals on the Continent.

Frimly Sanatorium.

I might therefore pass on without further comment to the Sanatorium of the Brompton Hospital, situated about 40 miles from London and within 2 hours' distance from the metropolis by train. The air of London is notorious for the vast amount of soot in it, which from the meteorological reports one can see is as much as about half a pound or more in a million cubic yards. As a result, every building in the city looks grey, even black, so that the word 'Black Town' could correctly be applied to London. Though remarkably free from dust on account of the asphalted roads, this large quantity of soot, as well as the organic impurities of the air make London and its immediate surroundings unfit for the location of a sanatorium.

Though there are a number of hospitals for advanced cases in and near the city, the sanatoria are situated 20 or more miles away. The Frimly sanatorium is situated in the midst of a very picturesque sloping country, sheltered by hills from the cold northern winds. There are about 400 acres of grounds attached to the sanatorium with pine forests and plenty of cultivable land for gardening and farming purposes. The sanatorium has accommodation for 150 beds and is constructed in the shape of a cross, each limb of the cross consisting of a row of single rooms facing south. It is very important in the cold northern countries that there should be a free exposure to the south so that the patient can have the maximum of sunshine which is such a luxury in those climates, unlike here where sunshine is an intolerable nuisance. Each room has an open verandah on its southern aspect separated from the room by large glass windows and a door which must be kept open day and night except during very rough and rainy weather, so that free ventilation and the maximum of light, the two great enemies of the tubercle bacillus are let in freely. The door on the northern aspect leads to a corridor connecting all the rooms in each row. The patient spends most of his resting time in the open verandah when he has the full advantage of the open air and sunlight. For the graduated exercises for patients in different stages of the disease and of treatment there are graduated walks and different grades of work including gardening, poultry and pig farming, carpentry, etc. Regarding the system of sanatorium treatment I shall have occasion to speak to you on a later occasion. Suffice it to say at present that the principle of sanatorium treatment is that of graduated auto-inoculation, the patient inoculating himself with the

products of his own bacilli in graduated doses till with the hardest grades he obtains the maximum of anti toxic and anti bacterial immunity against the tubercle bacillus and its toxins and his disease gets arrested. The sanatorium at Frimly is equipped on fairly modern lines with X-ray rooms and artificial pneumothorax apparatus, etc. Without tiring you with details I might just mention that I visited other sanatoria in England of which I shall mention two as types (*a*) of small and private and (*b*) of large and high class sanatoria, the Frimly sanatorium being one for middle and lower class patients.

THE CROOKESBURY SANATORIUM.

The Crookesbury sanatorium under Dr. Walters, an authority on sanatorium treatment is a small modest institution with accommodation for 54 beds situated about 40 miles from London, in the midst of about 20 acres of land partly pine-wood and partly heather with the most picturesque scenery all round. The patients are accommodated in small blocks and in wooden shallies in the midst of the forest, with arrangements for thoroughly open ventilation. There is no X-ray equipment and no artificial pneumothorax treatment. The sanatorium is in fact run on very simple lines and I am sure it will be quite feasible for an ordinary medical man in this country with some capital and a certain amount of training in sanatorium methods of treatment to run such an institution on modest lines to suit Indian social and financial conditions in a Hill station or in the open country.

In the grounds of the Crookesbury Sanatorium are situated the wooden buildings and workshops started by the military authorities during the war for the treatment and training of soldiers invalided for tuberculosis.

[*To be continued.*]

CLINICAL NOTES.

A CASE OF PERFORATED ATHEROMATOUS ULCER OF THE AORTA.

(BY DR. M. N. RAO, L.M.S.)

Body of a salt fish merchant, C, sent for *post-mortem* examination, with the history that while packing fish, he fell down suddenly and died.

No external marks of injury. The man had inguinal hernia and a hydrocele on the right side. Age about 43 years. Fairly well nourished and muscular. Features calm, eyes open, pupils partly dilated. Rigor mortis present. Abdominal fat ½ an inch thick.

Pericardial sac full of fluid and clotted blood, about 12 ozs. Heart weighs 14 ozs. It was flabby and fatty and congested. Chambers empty, valves healthy.

Longitudinal perforation of the aorta on the floor of an atheromatous ulcer, situated on the posterior aspect of the aorta about half an inch in diameter. Perforation was ⅛″ in length. Aorta was thickened in places to 1/6″. Atheromatous patches were seen on the lining membrane. The ulcer was ¼″ from the main thickened patch.

Lungs, liver, kidneys, spleen and membrane of the brain were congested.

MATTERS MEDICO-LEGAL.

A RARE CASE OF GOUGING OUT OF AN EYE-BALL.

(BY DR. A. N. VERGHESE, L.M. & S., *Medical Officer, Palghat.*)

One Gopalakrishna Menon, aged about 27 years, a native of Tharur amsom, Palghat Taluq, was brought to the Municipal Hospital, Palghat, with an injury in the

right eye for admission, treatment and certificate on 28th April 1924. On examination his right eye-ball was found hanging at the outer canthus of the eye. His general condition appeared exhausted and anxious.

The story giver was that, in an altercation that arose over a pack of playing cards, two brothers attacked him. One held the victim tight above the waist keeping the extended arms in the hold, while the other got behind, fixed the victim's head with his left arm, thrust the right index finger in and pulled out the eye. A certificate to the following effect was granted to the party.

"The right eye-ball is pulled out of its socket breaking the optic nerve and tearing asunder the muscles. It hangs out on a few shreds of the external portion of the conjunctiva and the rectus muscle. The socket is filled with blood clots."

The patient was admitted. He was put under chloroform; the remaining portion of the external rectus and outer portion of the conjunctiva was cut and the eye-ball was removed. The socket was cleaned and the soft structures were brought together by a purse string suture. The eye was dressed. The recovery was uneventful and the patient was discharged almost cured on 25th May 1924, but he continued to attend the Hospital for some days more.

The eye-ball was entire. The optic nerve gave way at the optic foramen, thus about an inch of the nerve was found attached to the eye-ball.

The case is extremely interesting from a medico-legal point of view. The only case recorded is in Lyons' Medical Jurisprudence, page 121, case 56 (Fourth edition). Here Lyons quotes from Chevers and Chevers himself is not perfectly clear in his description whether the eye-ball was pulled out *entire*, i.e., without bursting.

In these days of civilisation, to come across a similar case of deliberate pulling out of a man's eye is very unlikely. Perhaps for another half a century another case of this sort may not come to our notice. It is said that in Central Africa such brutal actions are fairly common in brawls and altercations. It is also said that insane patients sometimes gouge out their own eye by ennucleating them with their fingers.

Both Parson's diseases of the eye, and Fuch's ophthalmology give in unmistakable terms that the eye-ball can be gouged out entire with fingers. In this case two expert witnesses were examined. Major Wright, Superintendent of the Ophthalmic Hospital, Madras, gave evidence in unmistakable terms that an eye can be gouged out with fingers and he further stated that in the case under reference, his examination of the eye-ball and the muscles and nerve attached to it both microscopically and macroscopically made him to think that the eye was avulsed or gouged out. Major Gray gave evidence to the contrary.

The First Class Magistrate of Palghat has convicted the two accused, the 1st to a term of two years rigorous imprisonment and Rs..1,000 fine and the 2nd Rs. 400 fine.

The medical evidence given in this case, particularly of the two experts, is interesting.

In connection with the above case, the following Report of the Medical evidence has been sent to us for publication:—

To

The Editor, The Madras Medical Journal.

DEAR SIR,

An interesting case of "Gouging out an eye" is being tried by the Sub-Divisional First Class Magistrate

of Palghat, Mr. D. W. Dodwell, I.C.S. The Medical evidence tendered in the course of the trial will be of great interest to the scientific world. I am sending you under separate post the summary of evidence of the Medical witnesses for publication in your columns. I shall be greatly obliged if you will be pleased to insert them in your valuable columns.

Thanking you in advance.

Yours faithfully,
T. M. VENKATACHELLA AIYER,
Journalist.

Extract from the evidence of Dr. A. N. Verghese, Assistant Surgeon, Palghat Hospital, in Case No. 25 of 1924 of Alathur—325, I.P.C.

I am the Civil Asstistant Surgeon of Palghat Municipal Hospital. I remember having seen P.W. 1 blind in one eye. I granted exibit B, the wound certificate, and it correctly describes the injuries found on his person. I saw him first at the Municipal Hospital on the 28th April 1924. I found only one injury, that to the right eye. He was an in-patient in the hospital for over 20 days. The wound was practically healed when he was discharged. I do not remember whether he attended as an out-patient. I considered the injury to be of a grievous nature. He cannot see now with the right eye as he has lost the eye itself. It is possible to put out an eye with one's finger.

Cross-examinations.—Not desired at present.

8-7-24. *Cross-examinations.*—The injury to the eye-ball was the only injury noted by me in the certificate. The eye-ball itself was intact. I have preserved the eye-ball. It has been brought. It is preserved in spirits

(Ex. as M.O. 1). The optic nerve was torn asunder. The torn end of the optic nerve can be seen. There was no damage to the eye lid proper. The periosteum of the orbit was alright. There would be a fair amount of hæmorrhage. The eye-ball is kept in position by the 4 recti muscles and two oblique muscles, and also by the padding of fat. Every thing was torn asunder except a portion of the external part of the conjunctiva and a few fibres of the lateral rectus muscle. An ordinary piece of cloth had been used to tie the eye with. It was too early to tell whether sepsis would develop in the eye cavity. By "put out" in my examination-in-chief I meant pull out. By poking the eye with a finger it is possible to put out the eye.

Re-examination.—The eye-ball can be gouged out by using the fingers, that is to say, if you press in the finger and then pull out the eye. There are such cases mentioned in Medical Jurisprudence. It is stated in Lyons' Medical Jurisprudence that Chevers gives a case where a man gouged out a woman's two eyes by using his finger. Lyons' Medical Jurisprudence was the book prescribed for use in the Medical College in my time. I have 14 years service. I have been on active service ever since I passed out of college. I have done eye work during that time. I had the first honour in my class in ophthalmology. The year before last I had a special course of training in the Ophthalmic Hospital at Madras. I do eye operations. I have removed eye-balls in the course of those operations. I can claim that I have had special training in eye work. I am quite sure that the eye-ball can be taken out with the fingers.

Extract from evidence of Major W. C. Gray, I.M.S., in Case No. 25 of 1924 of Alathur—325, I.P.C.

I hold the rank of Major in the I.M.S. I have been head of the Ophthalmic Hospital, Madras, for over a year and was also there for another year. I have been Professor of Ophthalmology in the Madras Medical College. I have been for about 5 years chief examiner to the Madras University in the subject. I have seen Ex. B. the wound certificate, granted by Doctor Verghese. I have also seen M.O.1, the preserved eye-ball. It is quite impossible that, as stated in this case if accused 2, caught hold of P.W. 1, from behind around the arms accused 1 could *by taking his right shoulder in his left hand* and poking at the eyes with his right index finger put out P.W. 1's eye. The reason is that if a blunt force is applied to the eye it would *rupture the eye-ball* and it would almost certainly give way at the weakest point, i.e., the junction of the iris and the white portion, that is, called the ciliar body. The second reason why this cannot have taken place as alleged is that the *optic nerve would* have given way at the weakest point, i.e., just close to the eye-ball and not an inch further back. It is rather difficult to say after this lapse of time as the end of the optic nerve is rather ragged, but I should say that it has been clean cut. There are six muscles, four recti and two obliques holding the eye in position ; all these as well as the optic nerve itself which is surrounded by a tough membrane would have to give way before the eye-ball could be removed. I *examined the socket* of P.W. 1's eye. I *should say that the eye-ball was removed* by some sharp instrument or at any rate a comparatively sharp instrument. The examination of the eye socket and the examination of the eye-ball both suggest this. I would say

that it is *impossible judging by the appearance* of the two accused that they would be strong enough to perform the act in the manner alleged by the prosecution. It would require more men to hold P.W. 1 and some instrument other than a finger to put out the eye. A finger would have ruptured the eye-ball.

If a man is able to move his head although his shoulder is held he will not keep still, and let his eye be pushed out, therefore I think the occurrence cannot have taken place in the way described to me and alleged in the prosecution evidence. Some other assistance would be required to keep the head still. The man would also struggle with his legs to get loose even though his arms were held. I have enucleated a number of eye-balls both in the army and in the civil life. In removing the eye you have to cut through six muscles and this is done deliberately with scissors and finally the optic nerve is cut through with a fairly strong pair of curved scissors. The optic nerve is a bundle of nerves. *I do not think that the tip of the finger could be inserted and lever out the eye-ball without rupturing it.* If P.W.1 had been tied by a cloth to a pillar and held by two or three men it would be quite possible to remove his eye-ball with a sharp cutting knife.

From *examining the injured* person and the *preserved eye-ball* I would say that it must have been removed with a *sharp cutting instrument.* Lyons' Medical Jurisprudence is considered a standard work.

Penetration of the Cornea by a finger is quite easy. Behind the Cornea there is a mass of semi-solid matter aqueous humour lens and behind that another mass, viz., vitreous humour. It is *quite easy to rupture eye-ball* by means of the finger and thus take out all these contents

from the eye-ball. What is meant in the case shown to me in *Lyons' Medical Jurisprudence* is gouging out the contents of the eye by the finger not the whole eye entire. That is impossible. The same applies to the instances given in small print below the passage. It is *impossible that P. W. 1 could remain unconscious* for twenty-five and a half hours from this injury. He might have been unconscious for a few minutes from the pain. He would not necessarily be unconscious at all. A few hours after the occurrence he would certainly be in a position to give an account of the occurrence. He ought to know the nature of the vehicle he was carried in certainly.

Major Gray's Cross Examination.

Q.— Do you want the court to believe that you are in a better position to give an accurate opinion than Dr. Verghese was when he examined the raw wound and the eye-ball directly after the occurrence ?

A.—I went on what I was told.

Q.—Not on what you saw ?

A.—Yes, on the conditions of the eye-ball.

Q.—Apart from selective qualifications Dr. Verhese was in a better position to give an accurate opinion ?

A.—Yes. Of course.

Q.—Does it not occur to you that after the lapse of so much time the socket would have regained a uniform appearance and that it would be impossible to say whether the eye had been torn out, or cut out ?

A.—Yes from the socket.

Q.—Regarding the eye-ball you have examined, you say a sharp instrument had been used to sever the optic nerve and the base of the muscle but you admit it is

difficult after the lapse of so much time to give an *accurate opinion* and agree the end of the nerve is ragged ?

A.—Yes. The witness then said that there was nothing incompatible with the prosecution story as regards the eye socket but from his examination of the eye-ball he could not agree for reasons already given.

Q.—Dr. Verghese says it was torn out ?

A.—Yes.

Q.—You therefore suggest that Dr. Verghese is incapable of giving an accurate certificate ?

A.—I don't believe that it would be torn out under the circumstances. My opinion is that the eye could not be taken out with the finger in the manner alleged but prised out with some sharp instrument. In my opinion Dr. Verghese was wrong.

By Court.—What do you say is wrong with Dr. Verghese's certificate ?

Witness.—I do not find anything wrong with the certificate but in my opinion it is improbable considering the whole prosecution story that the eye was pulled out in the way stated. The witness went on to say that he based his opinion of the fact that the eye was quite entire, and that the optic nerve had not been severed at its *weakest point*, i.e., just behind the eye. The nerve and the muscles need not necessarily have been clear cut and he was not suggesting that the assistant surgeon had wilfully given a wrong opinion.

Q.—I put it to you that the fact that a great portion of the optic nerve remained strongly suggests that the eye was pulled out sooner than severed with an instrument ?

A.—It may have been severed out with a comparatively sharp instrument.

Q.—What do you mean by a comparatively sharp instrument ?

The witness explained that the side pieces of spectcles or a piece of melai without a definite edge is what he meant. He agreed that *incidental injuries* to the eye, lids and eye socket might be caused if the victim were struggling.

Q.—There is no obstruction save the conjunctiva to a blunt instrument passing behind the eye ?

A.—It is not impossible.

Q.—You have said it is impossible to extract the eye in this manner with the right index finger ?

A.—*If the man is not struggling, in some circumstances, it might be possible.*

Practical Demonstration.

The prosecutor then gave a practical demonstration as to how the injury is alleged to have been caused and Dr. Gray agreed that it might be possible to have extracted the eye in that way had the victim's head been finely held but the odds were the eye-ball would be ruptured.

Q.—I say it is quite possible in the manner demonstrated ?

A.—Yes. The prosecutor asked Major Gray to examine the 1st accused's finger and described it as thin as pencil. The defence counsel immediately sprang up and objected, quoting a Patna case that an accused cannot be asked to do anything to hurt his case.

The A. S. P. concluded that this was not a parallel incident but he would not press the question.

Q.—You have seen what a weed the complainant is. Do you mean to tell this court that these two accused are not strong enough to hold him ?

A.—I do not think there would be much difficulty in their holding him in the manner demonstrated for the purpose of putting out the eye.

Dr. Gray then explained enucleation and evisceration of the eye.

Q.—Enucleation is taking out the eye entire ?

A.— Yes.

The prosecutor then referred to Fuch's Ophthalmology, para 721, where insane patients have sometimes enucleated their eyes with their thumb.

Q.—That is prising out their eyes entire with a thumb ?

A.— The eye would most likely burst.

A reference was then made to Parson's "Diseases of the eye."

Q.—It says "Insane patients sometimes enucleate their eyes by gouging them out with their fingers ?"

A.— I say this is a misuse of words. Gouging out means gouging out the contents.

Q.— The Oxford Dictionary says Gouging out is forcing out the eye with thumb or finger.

A.— I say that it means the taking out the contents.

A reference was made to Lyons' Medical Jurisprudence, p. 121, where a case was quoted of a person's eyes being gouged out with a needle and a blunt instrument.

Major Gray.—I contend that this does not mean taking out the eye entire.

Prosecutor.—If you dispute the wording in the books I will stop.

Expert Evidence.

Major Gray said he called himself an expert on the eye. He was at the moment in charge of the Maternity Hospital. He lectured on Midwifery and Gynæcology.

He would leave it to the Government to say whether he was an expert in these also.

Q.—You have given expert evidence for the defence before ?

A.—Yes in two cases.

Q.—Were either of these eye cases ?

A.—No. They were murder cases.

DEPOSITION OF ROBERT EARNEST WRIGHT,
1ST COURT WITNESS.

Questions suggested by the prosecution.

I am senior moderator and B.A. of Dublin University. I am a M.D., B.Ch., B.Sc., D.Ph., of Dublin University and L.M.Rt. Dub. I am a member of the Society of Medicine and a fellow of the Royal Society of Tropical Medicine and Hygiene. I am competent to give an opinion on Pathology as I have acted as Professor of Pathology in the University of Madras and I have been Examiner in Pathology there since 1920. When I was Assistant Director of Pasteur Institute, Coonoor, I spent two years in the study of the Histo-Pathology of nervous tissues among other things. I was a member of the Bacteriological Department of the Government of India for 7 years. I am a member of the Royal Ophthalmological Society of England, and Superintendent of the Government Ophthalmic Hospital, Madras, Professor of Ophthalmology in the University of Madras, Examiner in the University of Madras. I am a collaborator on the editorial committee of the leading English and American journals of Ophthalomology. I have contributed to a considerable extent to ophthalmic literature in England, America and India. My work as an ophthalmologist is familiar to the leading specialists in all parts of the world. My annual professional reports

are circulated to the leading schools of ophthalmology throughout the world, and to the editors of the leading ophthalmological journals published in English. They are considered valuable contributions to ophthalmic literature. Patients have been sent to me for my opinion from many parts of the world, such as Soudan, America, Africa, New Zealand, the far East and all parts of India. I am acquainted with the only living Professor of Ophthalmology who has done experimental work on the avulsion of eyeballs, having spent part of last summer in his clinic. I would say that I am better qualified than any one else in S. India to give an expert opinion on the point at issue in the case.

I have examined the eye-ball (M.O. 1) which was sent to me under seal and I have now examined the socket. There is no evidence that the bony wall of the socket has been injured. In my opinion from the examination of the eye-ball it was avulsed. Avulsion consists firstly of a levering of the eye-ball out of the socket which is luxation and secondly a dragging of the eye-ball from the head which completes the avulsion. The two processes take place almost instantaneously. The division of the act of avulsion into two stages is purely technical. There is first a wedge action and then a pull.

The reasons for thinking it was avulsion are as follows :—The first is the length of the optic nerve. In this case the length of the nerve is about 2½ centimetres. The nerve would hardly ever be found to be of that length if the eye were removed by a sharp instrument. Out of about 736 globes in the possession of the ophthalmic hospital which have been enucleated all carefully examined by myself there is only one in which the nerve stump was more than 1 centimetre in length and that was an avulsed eye

produced in court, where the nerve was approximately 28 (twenty-eight) millimetres long. My opinion is that in this case there was avulsion by a finger or a blunt instrument and not by a sharp instrument. There are further reasons besides the above. The muscles were torn. I examined the muscles and the nerve microscopically; in both cases they were torn. That is, today they did not show a sharp line of demarcation. They in no wise resembled the numerous specimens, I had, of eyes removed by sharp instruments. It is unlikely that the ragged end of the optic nerve has been caused by rough handling, after the eye was taken out, or through its having been preserved in spirit.

There was considerable hæmorrhage into the globe which indicates that the globe was thrust upon forcibly. From my examination of the eye-ball I cannot say whether the optic nerve has been torn asunder by pulling out the eye or severed by a blunt instrument: it might be either, but it has certainly not been cut by a sharp instrument. In an avulsion the probabilities are that the nerve will tear and not be severed; that is supposed to be the ordinary mechanical process. There is a point which makes it probable that the nerve was torn by traction; it is this, that the nerve projects beyond the torn end of the sheath. That suggests traction rather than crushing of the nerve by a blunt instrument.

There is no impossibility in the avulsion of an eye by means of a finger. If you try to avulse an eye by means of a finger you might rupture the eye-ball but I believe that it is not commonly ruptured. We know as the result of experience that thrusting a blunt instrument into the eye does not necessarily rupture it. I produce a photograph

of an eye which I luxated myself by pressing; and in that case I was able to close the eye-lids behind the globe and I could easily have dragged the eye out. Some eye-balls are more easily luxated than others. A very sunken eye is much more difficult to luxate than a moderately bulging eye. You could certainly luxate the eye by means of the right index finger only; it would then be very easy to get the fingers behind the eye and drag it out of the socket. I should say that complainant's eye projects a little more than the average but it is not very important; certainly it would be no more difficult than usual to luxate. Parsons' Pathology of the eye, p. 1180, in the last four lines says that it has been proved by experiment that avulsion by a finger is possible but it requires much force; one of the common ways of avulsing an eye is by thrusting in a finger. I have not performed an avulsion myself. I am quite certain that the average person could perform an avulsion by means of the right index finger with some difficulty. That it is done with some difficulty is shown by the fact that in many recorded cases the eye-ball is not completely removed but hangs by one or more muscles, or other tissues.

Ball "Modern Ophthalmology" a standard work, of 1913, p. 648–49, shows that avulsion by a finger is possible and quotes the cases of auto enucleation by the insane and the practice of gouging out in Uganda. By "gouging out" in that passage he means as he actually states; enucleation and not gouging out the contents (prosecution demonstrates the way in which they suggest the act was performed).

I would say that there is nothing impossible in the eye being avulsed in the manner now shown to me. From

my examination of the eye-ball and eye-socket and the demonstration now given to me by the prosecution I form the opinion that there is nothing at all improbable in the prosecution story as to the manner in which the eye-ball was removed.

I have read the certificate given by the Assistant Surgeon when he first examined the complainant. There is nothing improbable in what he has certified and my examination of the eye-ball and socket leads me to conclude that he formed a correct opinion as to the means by which the eye-ball was removed. I do not think that a doctor who has been trained in ophthalmology like Dr. Verghese could have given such a certificate if the muscles of the eye had actually been cut by a sharp instrument. He had the advantage of seeing it soon after the occurrence.

M.O. 2 shows an eye in which the optic nerve has been clean cut by a sharp instrument. M.O. 3 is a section which I took of the optic nerve in the case of M.O. 1 showing that the end of the optic nerve is a blunt cone. Comparing these two I feel certain that the optic nerve in M.O. 1 gave way under strain of the eye being dragged out of the head.

It is possible but not necessary that the victim would lose consciousness when avulsion takes place. It is a temperamental thing and patients differ very much. I would not say that the complainant would be more likely to lose consciousness than any one else. It is possible that the thrusting of a finger or a blunt instrument into the eye might in itself cause a man to lose consciousness, but it is not probable : I can't recollect a case. 6–8–24.

Questions suggested by the Defence, 7–8–24.

I am in a position to say that the eye-ball sent to me was avulsed by a blunt instrument such as a finger. It could not be done with ordinary nail because that is too thin to give the wedge action necessary to the first part of avulsion. I should say it would be difficult to carry out avulsion by means of a blunt knife unless it was assisted by the finger.

I would say unless the victim were under other influences than a mere trauma of this sort, it is unlikely that he would remain unconscious for 24 hours. An avulsion alone would not have this effect in my opinion ; such a thing might happen, and might be aided by extraneous causes such as the influence of drink or drug or concussion caused in a struggle. But a shock of a smaller nature is capable by itself of causing not only to lose consciousness but death ; the whole question of surgical shock is difficult and badly understood. The 736 eye-balls in my museum which I referred to are all either enucleated or eviscerated with the exception of the one produced in Court, which is avulsed. This was avulsed by the horn of a bull. The horn of a bull would give enormous leverage. The thrust of the horn would easily cause luxation. In addition to the luxation either *vis a fronte* or *vis a tergo* is required to remove the eye. A bull's horn is liable to exercise considerably more force than a finger. M.O. 2 is the only case of avulsion we have in the museum. It was preserved as a specially interesting specimen. It is not common except among the insane and in savage countries. In many cases of injury to the eye by bull's horns there is gross trauma such as rupture of the globe, disintegration of

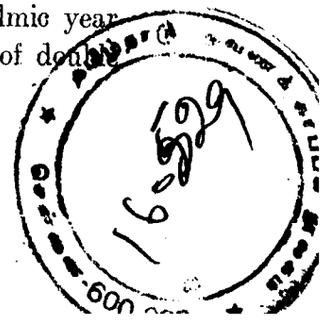
the contents without rupture and tearing of the conjuctiva. Injury to the bony orbit in such cases cannot be regarded as common ; bruising is common but not fracture. The contents of the museum are the result of a collection of several years. They date back to Colonel Elliot's time ; he retired about 1913. I know I have specimens from 1911. Colonel Elliot was in charge for a long time. but I have not many of his specimens not more than 70. In my collection I have no case of avulsion in the course of a fight.

If the operation of avulsion were performed suddenly, I can conceive of its being done in any position of the head in which the finger of the operator can enter the eye from an opposite position. If I were to do it at my leisure. I would prefer to have the subject in the usual position for an operation, that is, lying down face upwards and myself standing facing him at the side. If the man was struggling at the time the chances are much diminished. At the same time in struggling it is very easy for a finger to slip into the eye and cause luxation. If an attempt to injure the eye is going on in the course of a struggle it is likely that there would be injury to the surrounding parts. In the circumstances I would not say that the chances of evisceration would be greater. I have rarely seen an evisceration by a force applied in that way. It is practically impossible to say whether there is any greater likelihood of evisceration in such a case thai when the head is firmly held. I don't think there is much in it one way or the other. It depends entirely where the finger first impinges. The introduction of a foreign body into the orbit of the eye would cause compression of the eye-ball but the line of least resistance tends to be taken

by the globe on the luxation of the eye-ball. It is certainly possible to rupture the eye-ball by the introduction of a foreign body into the orbit. There are certainly some portions of the eye-ball which are weaker than others. For the purpose of trauma one may say that the neighbourhood of the limbus is the side most frequently involved in a rapture. The only specimen I have with me shows that there was a large body introduced into the orbit and the eye-ball came out entire, but there was a good deal of disintegration of the interior of the globe. I have not data from which I can give you a sufficiently valuable opinion as to the frequency of rupture or luxation in cases of trauma.

I cannot quote from the literature any case of luxation caused by the insertion of a finger into the eye of a struggling man. I don't think that any scientist would necessarily record the fact that the man was struggling. My opinion is that most of the cases recorded in the literature as avulsion might be called auto-enucleation ; there is no struggle then as the injury is self-inflicted. I don't think the actual word " struggling " is used in the literature that I have had time to consult but I should have had much more time to say definitely. I don't think it would occur to any medical man to doubt that avulsion could be brought about in a struggling patient. The point as to struggling is not important except that other injuries might be caused.

Presumably in the cases of gouging refered to by Ball in Uganda the act would be done deliberately by a number of men, but I want to make it clear that it was not too difficult an act for one man. The ophthalmic year book Vol. VII, 1910, page 263, quotes a case of double



avulsion in an attack by one insane person on another. Presumably in this case each was bent on attacking the other and not on defending himself. *Fuch's* page 233, I agree that the eye-ball may be ruptured by compression as stated here. The author is dealing with the question of rupture.

I cannot say that the insertion of the thumb would give greater force than the index finger. I have no records of the relative strength of the two ; it is the index finger which is generally *measured* and recorded.

I can't recollect any instance in the literature where index finger is specially mentioned. " Parsons' Pathology of the eye," Vol. IV, in his definition of avulsion (1180, 6th line from bottom) he uses the word " finger." I do not see why the thumb should necessarily be used. I know Sir John Parsons and do not think that he would use the word " finger " if he meant specially " thumb." I do not think he meant to exclude the thumb but obviously the finger was predominant in his mind.

If mechanical force is used and avulsion occurs, the optic nerve curiously does not seem to give way at its weakest point. In the case of the optic nerve and sheath:— when pulled it does not give way at the weakest point of the nerve. If by " optic nerve " is meant the whole bundle of nerves and tissues, then I don't admit that the point just behind the eye-ball is the weakest point. The weakest point of the sheath is where the sheath is attached to the optic foramen and that is where the sheath in the case of the eye-ball sent to me has given way ; it is the natural point of rupture for the sheath of the optic nerve. When subjected to the force of a bull, I say that in this case the sheath gives way there, because its measurement supports this view.

Chevers, page 484, appears to use the term " gouging " loosely to include rupture as well as avulsion. I do not think the application of one finger would be sufficient to pull out the eye after the process of luxation is accomplished ; the natural thing is to close two fingers or a finger and a thumb behind the globe with the optic nerve between them and to get a good grip for pulling out the eye-ball. The two processes are almost synchronous.

It is not at all an uncommon thing in the case of patients of a certain temperament to make the most of their injuries and lie and moan for many hours in an apparently semi-conscious state. A man lying in a cart and moaning with one eye pulled out would not be likely to take much notice of where he was being taken. It is possible that a man whose eye has been pulled out would be in semi-comatose condition for 25 hours ; it is also possible that he might take little notice, and sit up and take his breakfast soon afterwards.

After luxation has taken place it is quite easy to pass a finger behind the eye. The fore-finger would certainly exert more force than is necessary to pull out an eye, but I am unable to say how much more. The amount of struggling that a man could do when held in the manner demonstrated by the Prosecutor yesterday would not, I think, considerably lessen the possibility of avulsion by a finger.

As the index finger has less girth than the thumb, when the latter is inserted into the eye it is more likely to rupture the eye by pressure than the index finger similarly inserted.

Parsons' Text-book of Ophthalmology, page 627, 3rd edition, also says that " insane persons sometimes enucleate their eyes, by gouging them out with their fingers "

using the word finger and not thumb. There is no distinction made here between "gouging out" and "enucleation." The general sense of "gouging out" in modern ophthalmic literature is that the eye-ball is removed entire.

A common way of avulsion by means of a bull's horn is by the bull throwing up its head when being fed. I have not the details of the case to which M.O.2 refers. I feel sure it was an accident and the man was not attacked. Of course in this case much more force was applied to the eye than would be used in the insertion of a finger; the same applies to any case in which a bull's horn is inserted. Avulsion is only the result of an accident or deliberate attack or self-inflicted injury, and so very uncommon, whereas, enucleation is fairly common operation and up-to-date this year 30 enucleations have been done. 258 have been done since 1920.

It is quite possible that if the injury were done in the way discribed to me, that no incidental injuries would be done. If it were done by an amateur with a knife he would be likely to cut the lids and so leave incidental injuries.

I have seen Major Gray's evidence. Presumably he has not the same opportunities as I have, of access to the literature. I don't think he could have given the evidence he has given on the subject if he had been cognizant of the instances of avulsion which I have quoted.

I consider my opinion of more value than Major Gray's and the support I have given to it from authoritative works makes it much more valuable. I refer to Major Gray's original evidence. I was surprised that Major Gray gave such an opinion

GLEANINGS.

THE PASSING OF THE BACK PRESSURE THEORY IN CARDIOLOGY.

Some cynic has said somewhere that, to the making of new theories in Medicine, there is no end; and somebody else has also stated somewhere else that there is nothing new under the sun. Now, in the case of an imperfect and still largely empirical science like Medicine, it may happen that both these apparently contradictory statements are true, according as one is inclined to consider a particular theory as a brand new one or as merely an old one in a new garb. There is much food for thought along these lines in a very instructive and thought-stimulating article on "Changing Standpoints in Cardiac Medicine" which appears in the last June number of that well-known and leading Medical Journal, *The Practitioner*, from the pen of Sir Sydney Russell-Wells. The following extracts from that Article may serve to give such of our readers as have not read the original an idea of the main arguments advanced :—

"In John Hunter's 'Treatise on the Blood, Inflammation, and Gunshot Wounds,' he recounts a case in which we see him in the office of a physician. The following is an abridged version :—

A. B., when a boy, could never use the same exercise that other boys did, he could not run upstairs nor ascend a hill without being out of breath, and had almost throughout his whole life an *irregular pulse*, more especially when he used more exercise than he could well bear. Upon the least increase of motion he had a palpitation at the heart, which was often so strong as to be heard by those near; he soon became fatigued. With all this, he grew to be a well formed common-sized man, but still retained those defects, which indeed rather increased. About the age of thirty he took to violent exercise, such as hunting, and often in the chase would be seized so ill with

alpitations and almost a total suffocation that he was obliged to stop is horse and be held up on the saddle. At such times he became lack in the face, and continued so, as long as the fit lasted. It was ften several days before he perfectly recovered, frequently he could ot lie down in bed, but was obliged to sit up for breath.

When I was a student thirty years ago I was taught, s we all were, that—take mitral stenosis, the stenosed rifice prevented the left auricle properly emptying itself, ence the pressure rose in that chamber; as a result the ulmonary venous pressure rose, consequently the pulmo- ary arterial pressure had to rise, and to bring this about he right ventricle hypertrophied, the right side of the eart thus coming to the assistance of the left, the lesion vas compensated, and all went well for a time. But even- ually the right ventricle dilated and the tricuspid valve ecame incompetent when regurgitation into the right uricle took place, and the right auricular pressure rose vith damming back of blood in the systemic veins and rise in systemic venous pressure; hence a call for in- reased pressure in the systemic arteries, and a rise in eft ventricular pressure, with a further rise in left auricular ressure, and a vicious circle which led to venous engorge- nent of the liver and the systemic veins, and consequent edema and ascites. The raised pulmonary pressure and ulmonary engorgement were held to account for the lyspnœa and cyanosis. With mitral regurgitation a similar rain of events was considered to occur; in aortic stenosis, gain, though the lesion was further along the course of irculation, it was behind the obstruction that the ill- ffects were looked for, and similarly with aortic regur- itation; indeed, back pressure was then held to account or the serious consequences of valvular lesions. Now ote that this theory is a purely mechanical one; the

greater the stenosis the greater the back pressure should be, and the sooner cardiac failure should occur, and all regurgitations should bring about the same results, their severity simply depending on the amount of reflux. An apical systolic murmur was found to be a sign of mitral regurgitation, but numerous cases were observed when an apical systolic murmur was heard, yet no grave consequences ensued, nor was a valvular lesion found after death. Hence a class of functional murmurs had to be erected, and elaborate rules for distinguishing organic from functional apical systolic murmurs were laid down.

Since the amount of back pressure must depend upon the degree of stenosis or regurgitation we should expect to find some sort of inverse relationship between the severity of the valvular lesion and the length of the patient's life after it had been established, but such a relationship is notoriously difficult to establish.

Post-mortem evidence alone should make us dubious whether a mechanical theory such as back pressure by itself can account for the phenomenon of heart failure. Clinical evidence is equally unsatisfactory. We see numerous cases in which a lesion has existed for years unchanged, and the patient has continued in a state of fair health until some intercurrent infectious illness or a time of unusual strain suddenly ushers in the dire symptoms of cardiac failure without there being any fresh damage to the valves, and when the appearance of the heart after death proves that there has been no recent increase of the stenosis or regurgitation as the case may be.

Further, for the telling back of the pressure on the systemic veins there should *always* be tricuspid regurgitation when cardiac failure occurs. He would be a very

bold man who would now declare that there was independent evidence of tricuspid regurgitation in every case of failing heart.

However, one of the most serious objections to the back pressure theory of the origin of cardiac failure is that furnished by Hunter, viz., 'we see every day enlarged hearts where the symptoms have been somewhat similar and yet no visible mechanical cause existed.'

My own faith in what I had been taught was rudely shaken twenty years ago. A gentleman came under my care with dyspnœa, cyanosis, enlarged liver, ascites, and œdema of the legs; he had a rapid and extremely irregular pulse, which I now recognize as having been due to auricular fibrillation. He recovered under digitalis, but a few weeks later relapsed and died. Though constantly examined during life no murmur was ever detected; post-mortem, advanced myocardial degeneration was found, but no valvular lesion, and the mitral and tricuspid rings did not appear to be dilated. Here was a case in which all the symptoms were exactly similar to those of a dying mitral stenotic, yet there were no signs during life or appearances after death which could suggest the way in which back pressure was brought about. This could be paralleled by a large number of myocardial cases.

It may be argued, that when auricular fibrillation is present, pulmonary venous engorgement may occur without valvular lesions and so back pressure be set up, but it is now well known that auricular fibrillation may exist for years without cardiac failure occurring, and we do not find in cases dying after continued auricular fibrillation without valvular lesion that hypertrophy of the right ventricle which the back pressure theory demands.

I need not summarize all the clinical and post-mortem evidence which renders a pure back pressure explanation of cardiac failure untenable, but should like to direct attention to another aspect of its inadequacy. The bluish or

cherry-coloured lips and the slight general cyanosis, with the easily provoked dyspnœa seen in many heart cases, particularly in mitral stenosis, long before a general breakdown, were explained by stating that the damming back of blood in the pulmonary veins and the rise of pulmonary arterial and venous blood pressure caused a pulmonary congestion, which produced insufficient oxygenation of the blood and hence cyanosis and dyspnœa. It seems to me that mitral stenosis is the condition in which the best case for back pressure can be made out; moreover, I am inclined to think, as the late Sir William Broadbent insisted, that it is the gravest of all the valvular lesions. I am, therefore, using it largely for the purposes of my argument. That the pulmonary pressure rises in mitral stenosis is, I think, certain. Indeed, it seems to me that the increased resistance which a stenosed mitral introduces into the pulmonary circulation produces an increased pulmonary arterial pressure and hypertrophy of the right ventricle just as the increased resistance of the arterioles or capillaries in arterio-sclerosis produces an increased pressure in the systemic arteries and hypertrophy of the left ventricle. The accidents which follow the rises of arterial pressure are the same in both circulations, viz., atheroma, bursting of vessels, and the like; the early hæmoptyses of mitral stenosis are the exact analogues of the apoplexes of arterio-sclerosis. But a high systemic arterial pressure does not in itself give rise to œdema of the legs or systemic venous congestions, so why should a high pulmonary arterial pressure give rise to œdema of the lungs or pulmonary venous congestions; indeed, the high arterial pressures are in both cases Nature's means of preventing these events occurring. Can the back pressure through the pulmonary circulation and the

consequent rise of pulmonary blood pressure and right-sided hypertrophy account for the cyanosis and easily provoked dyspnœa so frequently seen for years in 'compensated' mitral stenotics? These phenomena are the signs of diminished oxygenation of the blood. Now the essential factor to secure adequate aeration of the blood is the free exposure of a sufficient quantity of healthy blood in the pulmonary capillaries to a sufficient quantity of oxygen in the alveoli of the lungs. If it could be shown that a rise in pulmonary blood-pressure brought about a chronic condition of pulmonary œdema, which, by effusion into the alveoli, prevented the access of air to the blood, the question would be answered in the affirmative; have we, however, any evidence of this? Many patients go on for years with well-marked mitral stenosis and show no signs of moisture in the alveoli, yet they are slightly cyanosed. Even supposing their lungs contain more residual blood than normal, *provided there is the normal amount* flowing in and out at each heart beat, is there any reason why their blood as a whole should be more venous than usual? Would not the existence of a larger quantity of blood in the lungs mean that more blood than usual was exposed to the air in the alveoli, and therefore had a better chance of absorbing oxygen and getting rid of carbon dioxide, always assuming that no exudation into the alveoli interfered with aeration? With regard to a simple venous engorgement of the lungs producing chronic œdema, we have to bear in mind that there is a fundamental difference between this and a venous engorgement on the systemic side. The systemic venules and the capillaries adjacent to them contain blood poor in oxygen and laden with carbon dioxide and other waste products, and therefore likely to be noxious

to the capillary endothelium, while the pulmonary venules contain revivified blood ready to be poured into the systemic arteries. A lagging of blood in the systemic capillaries where the blood becomes venous might cause more venous blood in the gneneral circulation and hence cyanosis, if the pulmonary circulatory and aerative factors remained the same, but it is difficult to see how a moderate increase in the amount of residual blood in the lungs could do so. I hope I have said enough to show that a purely mechanical theory of back pressure is inadequate; this inadequacy has long been generally felt and attention concentrated more on the cardiac muscle. We were told that the right ventricular muscle failed in mitral cases when compensation broke down, the tricuspid ring being stretched by the ventricles dilating, that in myocardial cases the ventricular muscle gave way and dilatation occurred, that in arteriosclerosis all went well so far as the heart was concerned, until the left ventricular muscle was exhausted; but though homage was done to the myocardium, the old back pressure theory with all its inconsistencies was trotted out to explain the phenomena of cardiac failure. Is there no other theory on which we can explain the phenomena of cardiac failure? Let us look in front of the lesion in a case of mitral stenosis instead of, like Lot's wife, perpetually looking backwards. The sole function of the heart is to deliver into the systemic arteries a certain quantity of blood at a certain pressure in any unit of time and to force at an appropriate pressure exactly the same amount in the same time into the pulmonary arteries. The quantity and the pressure naturally vary according to the needs of the organism at the moment. The heart may be regarded as a pump and motor combined; the blood reaching the heart

by the veins is at low or even negative pressure, leaves blood plus energy. It has been said that the secretion (the heart is energy. This energy is furnished by the coı traction of the ventricles ; the valves are purely an accessoı mechanism.

The amount of energy the blood leaving the heart hı acquired by its sojourn there may be divided into kinet and potential energy. The kinetic energy is such a smı proportion of the total that it may be disregarded, and w may concentrate our attention on the potential energy this may be measured for each side of the heart by mult plying together the amount of blood put out at each beı by the mean pressure at which it leaves the heart and tl number of beats per minute ; so we have three factors mass, pressure, rate, each of which may vary independentl The energy imparted to the blood by the heart is used u mainly in overcoming the resistance in the arteries, arteri les, capillaries, and venules, while its return to the heaı by the systemic veins is chiefly due to the movements (the diaphragm and those of the skeletal muscles, its flo back on to the venules being stopped by the venous valve Since, however, there are no valves in the pulmonary veir or those of the splanchnoportal area, the return of the bloo to the heart in these two systems depends upon the energ imparted to it by the ventricles. Nature is economical (effort, and it is interesting to note, under normal conditior only imparts enough energy to the blood to carry it to tl commencement of the great veins, and leaves the movement of the diaphragm and skeletal muscles to propel it throug the last part of its course.

The amount of energy available to propel the bloo along the vascular pathway depends on three independentl

variable factors : (1) the output per beat of the ventricle ; (2) the mean pressure at which it leaves the ventricle ; (3) the ventricular rate. This energy is used up in overcoming the resistance the vessels oppose to it, that is, mainly by friction. Normally, the left ventricle endues the blood with sufficient energy to take it to the commencement of the great veins. If the vessels all remained in the same condition of contraction or relaxation, and the left ventricle only gave the blood half the energy, it would come to a standstill long before the great veins were reached ; the vaso-motor system by altering the resistances in various parts of the circulation, however, profoundly modifies this.

Let us now take the three factors independently and consider first the effect of diminished output, the mean aortic pressure and heart-rate continuing constant. As an instance of this we will take mitral stenosis, and will first deal with the left side of the heart and the great circulation.

What are the proofs that in mitral stenosis there is a diminished output of the left ventricle ? *A priori* we should be led to expect that the extreme narrowing of the mitral orifice would lead to the left ventricle being insufficiently filled during diastole, for it is hard to conceive that as much blood can in the same time get through the narrow chink we often see as through the normal orifice admitting three fingers, and we have no reason to suppose that the diastole is lengthened in mitral stenosis. The left ventricle receiving less during diastole would put out less during systole.

It has been found by many observers that the pulse pressure, that is, the interval between the systolic and diastolic pressures, in the brachials and radials is much less than normal in cases of mitral stenosis. This statement

I can fully confirm from a large number of observations made over many years by various methods. It is generally agreed that a small pulse-presssure points to a small systolic output. A diminished output might be compensated, so far as total energy is concerned, by an increased average blood-pressure, but, in fact, the systolic pressure tends to be lower rather than higher than normal in mitral stenosis. An increased heart-rate also might compensate for a diminished output, and it is not uncommon to find the pulse-rate somewhat raised in mitral stenotics. Probably the organism does attempt to compensate for the lesion by increasing the pulmonary venous pressure, quickening the heart-rate, and endeavouring to raise the mean blood-plessure, hence the slight hypertrophy of the left ventricle without appreciable dilatation frequently seen post-mortem in these cases. But that these efforts do not entirely countervail the stenosis is shown, I think, not only by the diminished pulse-pressure, but also by the condition of the systemic arteries.

When the total energy that is to be distributed to any capillary area is diminished, more and more capillaries will contain blood at a standstill so far as motion is concerned, but active chemical changes will be taking place; it will steadily be losing oxygen to the living cells which surround it, steadily taking up carbon dioxide from them, be losing susbtances they require for their nourishment and receiving in exchange their waste products, until it ceases to be a suitable nutrient medium for them and their vitality begins to decline. As the capillary endothelial cells become ill-nourished, and their vitality is impaired, they will allow the fluid of the blood to pass through them more readily at the same time that the waste products of

the surrounding cells not flowing so freely to the capillaries will accumulate in and about them and raise the endosmotic equivalent of the intercellular fluids, and so œdema of the tissues will be produced. Thus if the energy supplied to any particular capillary area falls below a certain point œdema will ensue. There is another and grosser mechanical result of diminished output. Quite apart from any vasomotor control, which determines the relative amount of blood which shall go to any particular area, the arteries as a whole are highly elastic tubes, while the veins are distensible rather than elastic; consequently, when the cardiac output is stopped, as at death, the elastic recoil of the arterial walls forces all the blood out of the arteries into the veins. Similarly, when the cardiac output is diminished the less well-filled arteries pour the excess they previously contained into the veins, so that a diminished output quite easily accounts for the engorgement of the venules and systemic veins in mitral stenosis without having recourse to any back pressure theory with its correlated tricuspid regurgitation. Incidentally, it may here be remarked that for venule engorgement to take place as the result of back pressure all the venous valves would have to be incompetent, for it is hardly likely the back pressure could be so high as to counterbalance the contractions of the skeletal muscles and the movements of diaphragm, which are mainly responsible for the return of the blood along the great veins. A little consideration will show that practically all the phenomena of the failing heart on the systemic side can be rationally accounted for by diminished output of the left ventricle, and consequent lessened available energy without calling in any back pressure assistance. Now what will be the effect of diminished left ventricular output

on the pulmonary circulation? Diminished output from the left ventricle necessarily entails exactly the same diminution in output of the right ventricle: the two ventricles keep step as regards the time of their contractions, and they must keep step also as to the amount of their effective output—I say effective output, because aortic or pulmonary regurgitation might make the apparent output of one ventricle seem larger than the other. If the effective output of one ventricle was only a single drop per beat larger than the other, within a few hours all the blood in the body would accumulate on one side of the circulation; therefore the output of the two ventricles cannot for any length of time be different.

A diminished left ventricular output, as the right ventricle has to follow the left ventricle, both as to rate and amount, means that in unit time less blood will flow through the lungs, consequently, a smaller quantity will be exposed to the air in the alveoli, and, taking the arterial and venous blood as a whole, it will become less oxygenated than normal; hence the easily provoked dyspnœa and cyanosis of patients with mitral stenosis.

To summarize. The lessened output of the left ventricle in mitral stenosis leads to a slowing of the systemic and pulmonary circulations, the latter causes cyanosis and dyspnœa, the former the systemic venous and venule dilatation. If the lessening of output is moderate, and the patient learns to live within the necessary limitations, all may go well for years; if, however, the output becomes from any cause below a certain amount, engorgement of the liver, œdema, ascites, and the other symptoms of cardiac failure will occur, because the energy furnished by the heart is not sufficient to maintain the circulation at an adequate rate

through all the peripheral areas; further, this can occur without any general fall of blood-pressure, diminished output being alone sufficient to produce it.

A similar line of argument will show that in mitral regurgitation, aortic stenosis, auricular fibrillation, and myocardial degeneration we have primarily to deal with a lessened left ventricular output, and so a general slowing of the whole circulation, pulmonary as well as systemic. Indeed, in auricular fibrillation, and even better, with myocardial degeneration without fibrillation, the sequence of events is easier to trace because they are uncomplicated by the hypertrophy of the right ventricle and the rise of of pulmonary blood-pressure.

Let us now consider the effect of lowering the mean pressure at which the blood leaves the ventricle, the ventricular output being of normal or more than normal amount.

Since the needs of the cerebral circulation fix an irreducible minimum below which the mean blood-pressure must not fall, it is only just before death that we usually see a definite diminution in the accustomed level. In arterio-sclerosis, however, we have a condition in which the blood-pressure is set at a higher level than normal.

Yet in these cases all goes well so far as the circulation is concerned. Sooner or later, however, people whom we have watched for years with a systolic pressure of 200 or more come before us complaining of loss of energy and of feeling ill, and we find that they have a lowered systolic blood-pressure of, say, 160 or 150, with possibly slight œdema of the ankles, a little enlargement of the liver, some dyspnœa on effort. Experience teaches us that such symptoms in these cases are not infrequently the beginning of the end. Now, what is the explanation?

In arterio-sclerosis we have an increased resistance in the systemic arteries and arterioles, and it may be also in the capillaries. It matters not, so far as the left ventricle is concerned, whether the increased resistance is in the peripheral circulation or is due to aortic stenosis; more work is called for, a greater amount of energy has to be generated if the circulation is to be efficiently carried on. There is this difference, however, that in aortic stenosis the tendency is to diminish the output into the systemic arteries, while in arterio-sclerosis the output remains normal; therefore, to provide the increased energy required the mean pressure must rise. Speaking generally, we may say that when there is an obstruction in any part of the circulatory system the pressure tends to rise on the cardiac side of the obstruction, and the output tends to fall on the peripheral side.

Now Starling has shown that the way the ventricles respond to a call for increased energy is by dilating, the force with which the individual muscle fibre contracts being directly proportional to its length. As the healthy heart muscle can, on emergency, contract with something like ten times its usual force, one can understand how greatly increased blood-pressures can be maintained over long periods. Therefore, in arterio sclerosis on account of the demand for increased energy to overcome the raised resistance in the systemic arterioles, the left ventricle dilates and hypertrophies, the systemic arterial pressure being set, as it were, at a higher normal, the output of blood remains normal, the extra energy being used up in overcoming the resistance in the arterioles, the rate of flow through the systemic capillaries being thus kept normal as is the flow through the pulmonary arteries and the capillaries

of the lungs, so neither œdema, dyspnœa, nor cyanosis occur. It may even be that in some cases the output is slightly increased, and so the whole circulation quickened; the concomitant enlargement of the right ventricle seen in many cases of arterio-sclerosis points in this direction. The higher systemic arterial pressure results in the systemic arteries being more distended than normal, so that while in mitral stenosis there is a shifting of the mass of the blood on to the venous side, in arterio-sclerosis there is a tendency for the blood to accumulate on the arterial side of the systemic circulation. So long as the left ventricular muscle can continue to generate the increased amount of energy required, the circulation is efficiently maintained, but a continued high blood-pressure entails changes in the arterial walls, and the vessels of the heart itself are not exempt from these changes, so ultimately the nutrition of the dilated and hypertrophied left ventricle is impaired. Moreover, whatever is the fundamental cause of the arterio-sclerosis it has a tendency to be progressive, and an ever-increasing demand for energy is made upon the heart; ultimately, the ventricular muscle becomes unable to supply the high quantum of energy required, either because the demand has risen too high, or in most cases more probably because its nutrition for some reason is beginning to be impaired. Frequently, a toxæmia due to an intercurrent infection or some period of extra exertion with an increase of waste products in excess of the rate of elimination, or the like, is the last straw that determines the moment of the failure signalized by a fall of systemic blood-pressure.

While the output is normal, or possibly even more than normal, the moment that the fall of pressure occurs, the fact that the total energy available to force the blood

past the peripheral obstructions is diminished, soon results in less blood getting through to the veins; hence a diminished filling of the right auricle, a lessened output of the right ventricle, and less blood for the lett ventricle to deal with. This may for a short period relieve the tired and overworked left ventricle, but the result of the diminished left ventricular output is a slowing of the systemic capillary circulation, which produces all the signs and symptoms, such as swelling of the liver, œdema, and ascites, already discussed when we were considering cardiac failure in mitral stenosis.

Aortic regurgitation presents many points of resemblance to arterio-sclerosis; space does not permit my analysing the factors in aortic regurgitation, but I may say that in both the ventricular output is not diminished until near the end—indeed, in aortic regurgitation there is good reason for believing that it is increased; in both there is a tendency for the blood to accumulate in the arteries and arterioles rather than in the veins, and in both cardiac failure is initiated by a fall of average blood-pressure rather than a diminished output, though this necessarily follows; in both the immediate cause of failure is the inability of the left ventricular muscle to put out the abnormal amount of energy required.

We see that cases of cardiac failure fall into two groups:—

1. Those in which the essential factor is the inability of the ventricles to obtain a sufficient quantity of blood to maintain a normal output. To this group belong cases of mitral stenosis and mitral regurgitation, with certain reservations to be mentioned later, aortic stenosis, auricular fibrillation, and in the main, cases of myocardial degeneration. This group of cases we are accustomed to think of as the mitral group.

2. Those in which the essential factor is the ultimate inability of the ventricular muscle to furnish the abnormally large amount of energy required. To this group belong arterio-sclerosis, chronic interstitial nephritis, and aortic regurgitation. As aortic regurgitation was the earliest of these conditions to be studied, we may call this the aortic group.

When considering mitral stenosis we confined ourselves for the time being to discussing how the lesion affected the working of the left ventricle and its effects on the systemic circulation. We are now in a position to consider its results on the right ventricle and the pulmonary circulation. They are precisely analogous to those of arterio-sclerosis on the left ventricle and the systemic circulation. In both cases there is a distant obstruction to the circulation, in arterio-sclerosis in the peripheral arterioles, in mitral stenosis at the end of the veins in the mitral orifice, in both cases the ventricle behind the obstruction dilates and becomes hypertrophied in order to raise the arterial blood-pressure and so furnish the energy necessary to overcome the increased resistance and maintain the normal rate of flow. This raised blood-pressure is indicated by the accentuation of the aortic second sound and the pulmonary second sound respectively, in both cases the raised blood-pressure leads to atheroma and hæmorrhage from ruptured vessels in the respective circulation, and in both cases the ultimate end is failure of the muscle of the ventricle concerned. All that has been said concerning the left ventricle in arterio-sclerosis can be repeated regarding the right in

mitral stenosis. We see, therefore, that cardiac failure in mitral stenosis can be brought about in two different ways. First, purely as the result of a deficient output and a general slowing of the circulation; second, by a fall of pulmonary systolic pressure from failing right ventricle. Can we in any individual case differentiate during life between these two conditions? I believe we can, but the discussion would take me too far afield. The temporary failures so common in mitral cases are in the main due to diminished output; the final fatal failure, though by no means always, is not infrequently due to the right ventricle giving out. But whether a mitral case fails in one or the other way, in both cases it is due to insufficient *vis-a-tergo*, not to increased resistance *a-fronte;* it is due to an insufficient 'secretion' of energy, and back-pressure has nothing to do with it.

Having dealt at some length with two of the factors which determine the total energy given out by the ventricles, viz., amount of output of blood and average pressure, the third factor, rate of beat, can be dismissed in a few words.

Fall of rate is very rarely the cause of cardiac failure. Indeed it is only when the slowing is extreme as in the 'fits' of Adam Stokes's disease that we see grave effects on the circulation from bradycardia alone. It is easy to understand why this should be so, for a slow heart-beat means a lengthened diastole, and in consequence a better filling of the ventricles. An accelerated heart-beat, on the other hand, is a frequent occurrence and is a common phenomenon in cardiac disease; it is probably Nature's first line of defence when circumstances occur which either demand a greater output of energy by the

heart than normal, or when there is any tendency for either of the other factors to fail.

It is not wonderful that the older observers fell into error with regard to back-pressure; the decided dilatation and hypertrophy of the right ventricle in mitral cases and the accentuation of the pulmonary second sound directed their attention to what went on behind the lesion rather than in front of it; they lacked our present accurate means of observation, graphic methods were unknown, any pulse in the veins meant to them tricuspid regurgitation, there were no means of clinically measuring systemic blood-pressure. Hence many of the lessons to be learned from arterio-sclerosis were unappreciated, and the back pressure theory was seductively complete in its explanation, but it is now time that we definitely relegated it to books of medical history in the same way that the chemists have relegated the once popular theory of phlogiston to the position of an interesting episode in the history of their science.

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