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MEDICINE AND LAW.

M. K. Lo.

I regard it as a great privilege to be invited to read a Paper before the Chinese Medical Association.

I have always taken a very keen interest in Medicine, and have been much fascinated by the history of the wonderful and triumphant advance of the science of Medicine in recent years, especially in relation to Preventive Medicine. It is therefore a great pleasure to me to meet so many representatives of the Medical Profession, for which I entertain a great admiration, and to discuss with them a few topics which concern both their profession and mine.

The subject on which I have been asked to address you this evening is Medicine and Law. It is so vast that even an expert would find it difficult to deal adequately with its barest outline within the time at my disposal. And no one can be more conscious of his lack of qualification for this task than I am, for although I may know a little law, and am interested as a layman in Medicine, I do not pretend to have any extensive medico-legal practice, or to have devoted a special study to this subject. At the outset, therefore, I must apologise to you for a very sketchy and imperfect presentation of this subject.

I have said that the subject is a vast one, for it includes at least the subject of forensic or legal medicine, otherwise known as Medical Jurisprudence, which Taylor, in his celebrated work, defines as "the science of the application of every branch of medical knowledge to the purposes of the law; hence its limits are, on the one hand, the requirements of the law, and on the other the whole range of Medicine: —Anatomy, Physiology, Medicine, Surgery, Midwifery, Gynaecology, and the sciences ancillary thereto, viz., Bacteriology, Chemistry, Physics and Botany, etc. all lend their aid as necessity arises; and in some cases all these branches of science are required in the same case to enable a Court of Law to arrive at a proper conclusion on a contested question affecting life or property."

The security of a civilised State depends on its ability to protect life and property, and it is obvious that in the steps taken for the detection and punishment of crimes against life and property, and in proceedings for enforcing rights relating to property, such "a contested question affecting life and property" must be of frequent occurrence. Was the testator of unsound mind when he executed his last will? Was he of such a mental disposition as to be easily susceptible to undue influence? Is a certain person insane? Did A. die a natural death or was he poisoned, and, if so, how? In the case of the death of B. was it homicidal or accidental? These and innumerable questions of a similar kind cannot be satisfactorily answered by the law without the help which the Sciences, continuously advancing as the result of unremitting researches and improved technique, alone can give.

It is nowadays more or less a truism that all of us should have some knowledge of the general principles of the Law of the place in which we live, and this point has been very recently stated in rather picturesque language by the Lord Chancellor of England, who says:—

> "Some elementary knowledge of the law, and above all of the duty of obeying it, should be part of the education of every citizen. Amid the shifting sands of politics and the clouds of rival economic theories, the law is a rock upon which a man in troublous times may set his foot and be safe. Its greatest task is to establish justice between man and man and to see that obligations, both private and public, whether between States or individuals, are ascertained and obeyed."

I have no doubt that some knowledge of the guiding principles of the Law is essential to every citizen, but I suggest that it is of vital necessity to the medical practitioner if he is to be the efficient helpmate to Law. He can help, in the truest sense of the word, in the impartial administration of justice. By placing his expert knowledge at the service of the Law, he materially assists, not only in bringing the guilty to book, but in the triumphant vindication of the innocent.

Law is really synonymous with civilised life, and the absence of law is synonymous with savagery. Once this idea is kept clearly in mind it is obvious how much law must depend on medicine. Now I need not remind you, who are masters in your own profession, of the wonderful strides made by the science of Medicine. And if the law is to be also a progressive science, which it claims to be, and if the Courts are to mould and apply the laws in accordance with the ever-changing conditions of scientific thought and outlook, it must receive continuous assistance from your profession. Medicine is ever working steadily in the direction of health or rather the prevention of ill health, and the law in the direction of justice. But in order

that the essential partnership between Medicine and Law should produce the fruitful beneficial results to the public it is absolutely necessary that they should understand each other's methods of technique. Both professions require the best available evidence in order that the truth ascertainable under any given stage in human knowledge may be realised.

This aspect of the subject is a very interesting one, but I must now leave this and proceed to the discussion of a few isolated points which may be of interest to your profession. I take it that you would like me to say a few words on the responsibilities of your profession in law. I can do this very simply by quoting to you from that very indispensable work, Halsbury's Laws of England:—

Halsbury, Volume 20, page 330 et seq.

Sect. 2.—Responsibility.

Sub-Sect. 1.—Civil Responsibility.

814. A medical practitioner, whether he be registered or not, impliedly undertakes that he is possessed of a reasonable amount of knowledge and skill necessary for the performance of any professional task upon which he enters, and any such person who for reward or in the performance of a duty, either through negligence or ignorance, causes injury to the patient, is liable in damages for the consequences resulting therefrom.

815. No general rule as to the degree of skill or knowledge so required can be laid down, and the question in each case must depend upon the particular circumstances which surround it. The practitioner need not, however, bring to the performance of his duties the highest degree of skill, and where it can be shown that he exercised reasonable care and average skill his duties have been sufficiently discharged. Accordingly, he is not responsible merely because some other practitioner of greater skill and superior knowledge might have prescribed a different treatment or operated in a different manner; nor would a mere charge of unskilfulness without negligence, nor the fact that the injuries happened through some variation from the normal in the particular patient, render the practitioner liable.

816. Where during an operation a practitioner forms an opinion that it is necessary, in order to save the patient's life, to remove some organ or limb, and accordingly removes the organ or limb whereby the patient is injured, the practitioner cannot be charged with negligence for having taken that step, unless there is evidence that express instructions were given by the patient that no organ nor limb should be so removed, and that the operation was performed negligently, and it is for the jury to consider whether such instructions were communicated or not. Nor will negligence be admitted in an operation involving

possibilities of danger to the patient, if it can be shown that the nature of the operation and the possible dangers were communicated to the patient who expressly or tacitly signified his assent thereto. Further, it is submitted that, where an operation of a new or unusual nature is performed, no negligence is to be imputed to the practitioner if the operation be performed in accordance with modern science, and the peculiarities and the possible results of the operation have been previously communicated to the patient who has submitted himself thereto.

Sub-Sect. 2.—Criminal Responsibility.

820. At common law, when death or injury arises from treatment, there is no distinction either in civil or criminal proceedings between those who are qualified or regular practitioners and those who are not, for the gist of the proceedings is the actual maltreatment, whether arising through ignorance or negligence. Ignorance, therefore, is no excuse in the case of an unqualified practitioner, just as qualification is no excuse in the case of a qualified practitioner.

821. Any person, whether a registered medical practitioner or not, who deals with life or health, is bound to have competent skill, and, if a patient under his charge dies for want of such skill, he is guilty of manslaughter. Similarly, a person, whether he has received a medical education or not, who is guilty of gross carelessness in the application of a remedy, is liable to be convicted of manslaughter if death ensues in consequence of his act; but he can only be convicted if he has been guilty of the grossest ignorance or of criminal inattention.

822. To justify a charge of criminal negligence it is not sufficient to show mere want of care and caution; there must be gross negligence and want of that degree of skill which everyone, who undertakes the exercise of any particular art or profession, is bound to bring in each particular case; thus an injudicious and indiscreet administering of medicine will not make a man guilty of manslaughter; there must at least be gross negligence on his part. So, on a charge of manslaughter by administering poison in mistake for some other drug, the prosecution must show that the poison got into the mixture in consequence of the gross negligence of the accused: it is not sufficient to show that the accused, who supplied his own drugs, supplied a mixture which contained a large quantity of strychnia.

The fact that qualified medical assistance is available is an element to be taken into consideration when a charge of negligence is made against an unqualified person.

The question of legal responsibility is perhaps more of a legal matter than of a medico-legal matter, and yet in ascertaining the questions of fact on which legal liability rests, expert medical evidence is absolutely necessary.

The next question which I should like to touch upon as being of some interest to your profession is as regards the liability of a medical practitioner to be compelled to give evidence in a Court of Law. The law is quite clear and emphatic on this point:—

> "A medical practitioner, when called as a witness, is bound if asked, and if the question is pressed and allowed, to disclose every communication, however private and confidential, which has been made to him while attending a patient in his professional character."
>
> (Hals. Vol. 20, p. 337, para. 828).

Mr. Justice Hawkins, in his summing up to the Jury in a case in 1896, made the following observation:—

> "I can quite understand a case, especially in a civil cause, where a doctor is quite justified in refusing to divulge questions of professional secrecy. . . . The judge might in some cases refuse to commit a medical man for contempt in refusing to reveal confidences. Every case must be governed by particular circumstances, and the ruling of the judge will be the test."

But there seems to be absolutely no authority which can be cited in support of this learned Judge's ruling. The origin of this rule of law is usually traced to the ruling of the House of Lords in the trial of the Duchess of Kingston. In this case the Duchess was indicted for bigamy, and in the course of this trial a surgeon, Mr. Caesar Hawkins, was called. He was asked whether he knew from the parties of any marriage between them. He objected to answer this question but was over-ruled. There are of course subsequent authorities in which this rule of law is clearly laid down, but as Kingston's case is of historic interest I propose to read to you an extract from the report which is to be found in 20. State Trials, and consisting of 218 closely printed pages:—

The trial of Elizabeth calling herself Duchess Dowager of Kingston, for Bigamy: Before the Right Honourable the House of Peers in Westminster Hall in Full Parliament assembled 15th, 16th, 19th, 20th and 22nd April, 16 George III, A.D. 1776.

Mr. Caesar Hawkins sworn.

Examined by Dunning. Do you know from the parties of any marriage between them?—Mr. Hawkins. I do not know how far anything that has come before me in a confidential trust in my profession should be disclosed, consistent with my professional honour. . . . . .

Lord Mansfield: I suppose Mr. Hawkins means to demur to the question upon the ground that it came to his knowledge some way from his being employed as a surgeon for one or both of the parties; and I take for granted, if Mr. Hawkins understands that it is Your Lordships' opinion that he has no privilege on that occount to excuse himself from giving the answer, that then, under the authority of Your Lordships' judgment he will submit to answer it: therefore to save Your Lordships the trouble of an adjournment, if no Lord differs in opinion but thinks that a surgeon has no privilege to avoid giving evidence in a Court of Justice, he is bound by the law of the land to do it: (If any of Your Lordships thinks he has such a privilege, it will be a matter to be debated elsewhere, but) if all Your Lordships acquiesce, Mr. Hawkins will understand, that it is your judgment and opinion that a surgeon has no privilege, where it is a material question, in a civil or criminal cause, to know whether the parties married, or whether a child was born, to say that his introduction to the parties was in the course of his profession and in that way he came to the knowledge of it. I take it for granted, that if Mr. Hawkins understands that, it is a satisfaction to him, and a clear justification to all the world. If a surgeon was voluntarily to reveal these secrets to be sure he would be guilty of a breach of honour, and of great indiscretion; but, to give information in a Court of Justice, which by the law of the land he is bound to do, will never be imputed to him as any indiscretion whatever.

Taylor states:—"The law of England on this important subject undoubtedly conflicts with the law of honour observed by medical men (from a breach of which more than one has suffered severely), viz., that information obtained in the consulting-room from patients relative to their ailments must be held to be inviolably secret."

The Medico-Legal Society, in March 1933, devoted two evenings to the reading and discussion of a Paper on this subject by Lord Dawson of Penn. Reports appeared in the Lancet on April 1st, 1922, and in the British Medical Journal on March 25th and April 1st, 1922. In this connection I should like to conclude with an observation by Lord Justice Atkin:—"He was bound to say, speaking as a lawyer, that there was no protection for a doctor in respect of such matters, and he had no doubt the law would so remain until some medical man, when he was put into the witness box, said that the confidence of his patient was as sacred to him as that of the penitent to the priest, and the client to the lawyer, that nothing would induce him to disclose it, and that he was prepared to go to prison for the remainder of his life rather than violate it. The question might then arise as to whether or not the conflicting claims of public health and public justice could not be ratified by something else than the special obligation of the doctor to make a disclosure when called upon to do so."

The next question I would like to refer to is the question of insanity in relation to the law. Here again the inter-dependence between the law and medicine is emphasised.

The leading authority in English law is of course what is known as M'Naughten's Case. In this case 15 Judges were consulted by the House of Lords, and they were asked, and answered, five questions, and the guilding principle of the law is expressed in these terms:—

> ". . . . every man is presumed to be sane, and to possess a sufficient degree of reason to be responsible for his crimes, until the contrary be proved to their (the Jury's) satisfaction: that, to establish a defence on the ground of insanity, it must be clearly proved that, at the time of committing the act, the accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing, or, if he did know it, that he did not know that he was doing what was wrong."

A very interesting paper was read before the Medico-Legal Society in 1925 by Dr. Lionel A. Weatherly. He pointed out that while the law had more or less stood still for generations (the law being still the law enunciated by the Judges in 1843), Medicine was a science which was ever advancing and was able to show how disease could affect the intelligense, the imagination, the reasoning power, and also the effective side of what was called mind, viz., the emotion and the will; and affect it in such a way as to make the ruling by which criminals were judged at the present time, when insanity was the defence, unsound and unfair.

In connection with this subject I should like again to quote Lord Justice Atkins, who is really a great authority on medico-legal matters:—

> ". . . . He had presided not very long ago over a committee which accepted some theories, he thought rightly, which were put before it by the most representative member of the medical profession, but he was sorry to say the report of the committee in that respect had not been accepted with very great enthusiasm. One of the great difficulties which existed was that, up to the present, medical men had not been able to define what they meant by insanity. He was awaiting the time when some legal authority would explain the true position in such cases, for instance, those where a mother had several children to whom she was passionately devoted, and found herself to have an irresistible desire to kill them, a desire which she resisted for months, knowing well that it was wrong for her to kill them, but to which she eventually succumbed. He was not satisfied that at present there was any legal principle which held her to be irresponsible; though juries, departing from strictly legal principles often, he was glad

to say, found such a woman "guilty, but insane." (L. T., Vol. 159—413).

One branch of the law with which the Medical Profession must frequently come into contact is the law of evidence. Whole books have been devoted to the study of this branch of the law, and it is impossible at the time at my disposal to deal with it. But I would like to say one word on "dying declarations." A doctor may be suddenly called in to attend a patient who, for instance, had been shot and who was obviously in a dying state without any possibility of recovery. If this man had been shot by another person his evidence may be vital because, indeed, there might be no other evidence to connect the person who had fired the shot. What should the doctor do in such a case?

You will remember that in 1916 both the Royal College of Physicians and the Council of the British Medical Association took a lot of pains in order to elucidate the position of a medical practitioner on attending a case of abortion, and that resolutions were passed by both bodies in regard to this matter. The Royal College of Physicians was advised by eminent Counsel to this effect:—

1. That the medical practitioner is under no legal obligation either to urge the patient to make a statement, or, if she refuses to do so, to take any further action.

2. That when a patient who is dangerously ill consents to give evidence, her statement may be taken in one of the following ways:—

(*a*) A magistrate may visit her to receive her deposition on oath or affirmation. Even if criminal proceedings have not already been instituted, her deposition will be admissible in evidence in the event of her death, provided that reasonable written notice of the intention to take her statement was served on the accused person and he or his legal adviser had full opportunity of cross-examining.

(*b*) If the patient has an unqualified belief that she will shortly die, and only in these circumstances, her dying declaration will be admissible. Such a declaration may be made to the medical practioner, or to any other person. It need not be in writing, and if reduced into writing it need not be signed by the patient nor witnessed by any other person, though it is desirable that both should be done, or that, if the patient is unable to sign, she should make her mark. If possible, the declaration should be in the actual words of the patient, and if questions are put, the questions and answers should both be given, but this is not essential.

> If the declaration cannot there and then be reduced into writing, it is desirable that the person to whom it is made should make a complete note of it as soon as possible.

The law as to taking a dying deposition by a magistrate need not concern you. In Hong Kong it is taken under sect. 32 of the Evidence Ordinance, 1889, and this section provides for reasonable notice being given to the accused person so that he might have full opportunity of cross-examining the person who made the dying declaration. The second part dealing with a dying declaration applies to all persons, and should be borne in mind by medical practitioners.

It must be remembered that a dying declaration is a statement made by a person who believes himself to be on the point of death. It is only admissible if the death of such a person is the subject of the charge, and further if the cause of the death is the subject matter of the dying declaration. Therefore the statement should contain and express hopeless expectation of imminent death; he must say so, and if on account of delicacy or kindliness of feeling, the doctor were to give him some hope that he may survive, and he entertains such a hope, the declaration is clearly not admissible.

As a matter of precaution a doctor who has attended to the making of a dying declaration should always immediately inform the Police, so that the Police may report the matter to the Magistrate. The reason is that if the declarant were to live on for sufficient time to enable a formal deposition being taken under sect. 32 of the Ordinance, this is always in practice done, as it is unsafe to rely on a dying declaration alone in a case in which there is sufficient time subsequently to take a formal dying deposition.

There are so many other topics which I would like to mention, but I have already taken up too much of your time.

In conclusion I should like merely to refer to another subject which illustrates and illumines the interdependence of law and medicine in elucidating questions of fact, namely, evidence in maternity cases:—

How far is physical resemblance evidence of consanguinty? Is such evidence admissible in the English Courts? Logically the Courts should not accept evidence that a child resembles a certain person as proof that such child was the child of such person unless it had already been proved in court that, as a matter of science, children are apt to inherit the features or general appearance of their parents. But so far as the English courts are concerned they seem to have taken judicial notice of such scientific testimony and do receive evidence of physical resemblance upon the assumption that "like is apt to beget like."

There is a very interesting article on this subject in a Law Quarterly Review of 1923, and I wish to pursue this subject for a few more

moments in order to emphasise the potent effect of science—and in this case chiefly of physiology—upon law.

At the present time it seems quite clear that, in accordance with a long line of legal auhorities, our courts will admit evidence of resemblance between the child and one (or both) of the persons in support or in denial of paternity. But just as the gradations of resemblance are infinite the probative value of any given resemblance varies accordingly, and the great need for caution in not placing an undue weight upon such evidence is recognised. In particular it is recognised that facial resemblance in a child of not more than one or two years old, by reason of its peculiar immaturity of feature, has practically no evidential value. But apparently the English rule is not universally followed. The article says: "The continental jurists of the sixteenth and seventeenth centuries set a much more limited value upon resemblance. . . . But the conclusions of all these jurists were deeply influenced by the old physiological belief, which prevailed universally until challenged in England in 1724 by Dr. Blondel, that a pregnant mother's mental impressions can produce an effect upon the physical frame of her child. When a white husband accused his wife of adultery, because she gave birth to a child of Ethiopian colour and features, Quintilian had defended her by showing that, in the room she occupied, there had hung a picture of some Ethiop. Similarly a woman's ardent thoughts of some lover might, it was supposed, stamp his features even upon a child lawfully begotten by her husband. Hence these jurists agreed that resemblance could never afford any conclusive proof of paternity."

"At the present day, however, the physiological supposition which led these jurists to distrust the probative value of resemblance is generally discarded by men of science. There is no known means of communication by which ideas or emotions can be conveyed from a woman. . . . to an embryo' (Sir J. G. Frazer's 'Totemism and Exogamy,' IV. 64-71; of. Dr. J. W. Ballantyne's 'Teratogenesis,' p. 45; and Heape's 'Sex Antagonism,' p. 140)."

It seems to be universally accepted that "whenever the controversy lies between two alleged parents who belong to different Races of mankind . . . . and the resemblance of the offspring to one of them is so comprehensive and so precise as to embody the racial peculiarities which differentiate these two Races," evidence of such semblance is of great weight and is accepted.

The author of the article gives as his considered opinion that it was perhaps the hesitation which the old civilians had learned from the primitive physiology mentioned above that led Scottish lawyers to distrust the probative effect of resemblance, and at the present day evidence as to likeness (except in the case of difference of colour) has in Scotland for more than a century been rejected.

Dealing with the position in the United States the author proceeds as follows:—"Professor Wigmore, in his monumental work on 'Evidence,' cites (paragraph 166) thirty cases decided in eighteen different jurisdictions; and, since he wrote, another (in favour of recognizing resemblance) has been reported (Overseer v. Eason, 1 A. L. R. A. 631). These decisions fall into as many as four groups. Some follow the wide English rule. Others, whilst admitting resemblance, require it to be proved by actual exhibition in Court; regarding it as a matter of mere Opinion, to which ordinary witnesses accordingly must not testify. Others, however, regard it as a Fact, and admit witnesses; but actually forbid exhibition in Court, for the technical reason that this would be a form of proof that could not be put upon the record and sent to a Court of Appeal. Finally, a fourth group take the Scottish view and reject resemblance, except in the extreme case of a contrast between different races of mankind."

I have dealt with this question somewhat fully in order to show that the view of the law has been coloured by the theories of physiology, and I have no doubt that future advances in physiology will re-act on the evidential value of this class of evidence. As a lawyer, and therefore a layman in regard to Medicine, I cannot but suppose that this question is intimately connected with the subject of bloodstains. Dr. G. Roche Lynch, Senior Official Analyst to the Home Office, in the course of his lecture before the Royal Institute of Public Health, Russell Square, in March 1928, entitled "Some Problems in Medico-Legal Practice," showed by means of diagrams how modern scientific research had advanced so far that it was not difficult, where a sample of the blood of the victim of murder and also of the person accused could be subjected to examination, to say definitely whether dried stains were those of either. He proceeded: "A common defence in murder cases where bloodstains were found upon the clothing of the accused was that they were the result of some accident to himself and not those of the murdered person: but it could now be shown quite clearly whether the blood had come from the accused or from the victim. A further result of the analysis of the blood was that, in paternity cases, it was possible to say whether the child was of the same blood as the presumed father or not. In eighty unions in various parts of the country 350 children had been observed carefully, with the result that their blood had been found strictly to conform to tables that had been drawn up."

As a layman I was under the impression that, apart from the exceptional cases, there were no means of distinguishing human blood of different individuals. I find in the 1928 edition of Taylor the following:—

> "It is obvious that there can be here no possible scientific evidence, except in extraordinary circumstances in which there

is disease of the blood in one or the other, and with opportunities for examining the stains while fresh. It is just possible that in the future tests may be able to be applied for different diseases. Wassermann has developed one for syphilis, and others may follow based upon similar lines."

Then as regards Group Blood Tests for paternity Taylor says:—

"Our knowledge of the Blood Groups has not yet reached the stage when the results are likely to be implicitly accepted by the Courts."

If Dr. Lynch's statement proves to be correct, it is obvious that Science has forged yet another potent weapon for Law in its unceasing fight against deliberate falsehoods and perjured denials.

In this connection I was rather interested to see the following statement by Professor J. B. S. Haldane quoted in the South China Morning Post of Friday, May 18th, 1934:—

"It is as unscientific and out of date to-day to brand a man as the father of an illegitimate child without blood tests as to hang him for poisoning without chemical analysis."

ARHYTHMIA AND CARDIALGIA IN HEART DISEASE.

M. O. PFISTER, HONG KONG.

In my student days the teaching of heart affections concentrated upon the valvular diseases and very little was taught about the irregularities of the heart's action, an irregular pulse being considered as a more or less common accompanying sign of the failing heart. With the advancing knowledge of the anatomical structures of the heart, especially since the discovery of the conductive system and with the improved methods of clinical examination, foremost of all since the introduction of the electrocardigraph our conceptions of the irregularities have undergone essential changes; the various forms of primary *arhythmia*, which of course is only a symptomatic term, have been separated as a group of their own, which the general practitioner has learned to consider to be of equal importance to any kind of the valvular forms of heart disease.

The term *cardialgia* comprises various forms of pains in the region of the heart, among which angina pectoris—also a symptomatic diagnosis—is the most typical example. Arhythmia and angina pectoris often stand out in the foreground of a clinical picture as such striking symptoms that these terms are used as diagnostical denominations for the convenience in clinical teaching; yet we always have to realise that behind these clinical symptoms there are anatomical changes, for which we have to search.

We should not feel satisfied with a symptomatic diagnosis of a heart affection, only a thorough understanding of the underlying anatomical alteration and pathology with a classification according to the nature and localisation of the process will help us to get a clear picture of the disease and will provide the necessary basis for a rational therapy.

Arhythmia and cardialgia are not seldom the only symptoms—either one of them alone or both combined—which indicate conditions which within a limited period of time may lead to a sudden primary heart death (Sekundenherztod of the German literature).

In reviewing the reports in recent medical literature on primary disturbances of the circulatory mechanism in middle aged men, followed by sudden death (other than from cerebral artery troubles) the increase of such conditions seems to be striking; those cases, in which even without having experienced any symptom of heart trouble before, a person had died suddenly without warning at his desk or during the night while quietly resting in bed.

This kind of death appears to be remarkably frequent among members of the medical profession and quite recently I have lost three medical friends from similar conditions. American statistics record 40

per cent. death from heart disease, with 10 per cent. from angina pectoris among physicians.

I therefore think that this subject should interest medical men also from the personal point of view especially with regard to prophylactic measurements which could be taken to avoid the factors, which may lead to the development of cardiac troubles.

There is another point which for many years has attracted my attention, it is the *fate of the heart in men who in their youth had indulged in various kinds of strenous physical exercise* as rowing, ski-ing, mountaineering, long distance runs and similar sports. There is no doubt that these activities develop the heart, as they do with other muscles to a much more vigorous organ than a sedentary life would have done.

Do these *"athletic" hearts* under similar other conditions guarantee better general health and greater efficiency for the strains of later life than the hearts of persons who never took an active part in any kind of sports, or will the "athletic" heart later on show effects of the wear and tear of former days and be more liable to develop pathological cardiac conditions?

This is an interesting problem which needs further investigation. As an example of this category of "athletic" hearts I should like to contribute one of the cases which I recently had the opportunity of studying carefully and which at the same time well illustrates the problems to be discussed in his paper.

A man, 59 years old, in his college days very active in rowing, football and other athletics, until recently still very keen on various kinds of physical exercise, never seriously ill before, one day after a comparatively moderate physical strain lifting some heavy weights suddenly experienced a feeling of discomfort underneath the sternum. Soon afterwards when walking up hill he noticed a great irregularity of his pulse, he had to pause several times on account of a choking feeling in the chest and palpitation of the heart. When I saw the patient on the same day I found as practically the only morbid sign an absolutely irregular and unequal pulse of a rather poor quality, so that it was difficult to count the beats. The apex beat was felt somewhat outside of the nipple line, and on percussion a slight increase of the cardiac dullness to the left was found; no murmurs were heard, but the second sound at the base seemed accentuated, the action of the heart was rather slow, 60—64, but accelerated quickly up to 90 and more on the slightest exertion. Blood pressure was 150—160 mm Hg systolic. In the urine a slight trace of albumen, but otherwise no abnormal findings. No drugs were given, rest was advised.

A few months before this sudden onset of arhythmia the heart of the patient had been examined by a specialist and no pathological

signs whatever had been found, the blood pressure having been 160 mm and the pulse rate 72—74 and absolutely regular. In his younger days, the patient further stated, a slightly enlarged "athletic" heart had been found.

Course and Further Development.

The main sign, the irregularity of the pulse, persised during the next 3—4 months without essential change. It was an absolutely irregular pulse, larger and smaller beats following each other in shorter or longer intervals without any regularity, but the number of beats in the minute was no higher than 58—62 at rest; ascending 20 steps of a staircase within half a minute quickly raised the rate to over a hundred. On auscultation it could be ascertained that each heart beat was followed by a wave at the radial pulse. The quality of the pulse was varying in strength, the impulse of the wave short and rather abrupt, conveying a somewhat quivering sensation to the palpating finger; the vessel wall showed no signs of sclerosis.

During the first two weeks after the onset the patient, when at complete rest, felt very little discomfort, but afterwards *cardial sensations* gradually developed which increased not only with the slightest physical effort but also made their appearance after psychical emotions. The patient described these symptoms as sensations of varying nature and localisation. Nearly continously there was a more or less heavy pressure underneath the sternum at its lower half, a weight which rested upon the lungs making breathing uneasy. On effort this feeling had a more "wobbling," vibrating character, extending upwards to the neck along the course of the large bloodvessels.

Different from this the patient experienced a sensation of another character, which he localised to the area of the heart itself (its ventricular region!), a cramp like pain as if the heart was held in a tight grip, occasionally also, even at night when resting in bed, a sudden sharp pain like the shooting pain of muscular rheumatism was felt. Pain never spread out towards the arm. Ascending the stairs or going up hill as well as lifting some heavy articles, like a chair, invariably increased the substernal discomfort, a cold bath had the same bad effect; in times of warmer weather the patient felt easier.

At first no relation between the irregularities of the pulse and the patient's subjective seansations seemed to exist, but after repeated examinations I came to the conclusion, that a *very irregular, unequal and weak pulse always meant increased subjective discomfort.* Later in the course of the disease the patient had no difficulty in keeping the two sensations—the substernal and the cardiac pain as I may call them—apart from each other. The cardiac pain seemed the more serious trouble which regularly was accompanied by a poorer condition of the pulse.

Except for an occasional slight cyanosis of the lips there were no evident signs of a circulatory stasis. The trace of albumen in the urine which persisted during the first few weeks after the onset, gradually disappeared. The blood pressure remained at about the same level, the examination of the blood, including the Wassermann test, did not reveal any morbid findings, no latent focal infection or toxic conditions could be detected. The temperature had always been normal. Teleradiographic examination showed, what could be interpreted as a slight enlargement of the left side of the heart, but viewed from the oblique diameter no obvious changes of the heart or the great blood vessels could be made out. Unfortunately an electrocardiograph which could have given valuable diagnostic help in this case, was not available and I am quite aware that a report of a heart case without an accompanying electrocardiogram cannot be considered as complete.

Effects of Drugs.

As means which further may assist in the diagnosis of circulatory disturbances the reaction on drugs is often a quite useful guide. There are certain drugs, the effects of which on the heart and circulatory system in healthy and pathological conditions are quite specific, and as my patient proved to be an intelligent and scientifically interested observer and quite willing to co-operate with me in trying out the effect of various drugs on him, in doing this I was able to perform a kind of pharmacological experiment.

Keeping in mind that I had to deal with a very severe kind of arhythmia cordis, the pathological nature, seat and extention of which were still objects of speculation and further that the pharmacological effect of some of the drugs, I was going to use, was not yet sufficiently known, I started with small doses, in order to avoid any possible harm, gradually increasing them so that after 3—4 days the dose of the strength, usually given, was reached. For the same reason of preventing sudden drastic effects which to a highly sensitive mechanism such as the circulatory system, could prove a dangerous shock, no injections were given, but all drugs were taken by mouth. After a substance had been tried for 8—10 days, the patient was given no medicine at all for a week and then a new drug was started. Combinations of drugs were avoided as far as was possible.

Quininum hydrochloricum (0.1 gram as starting and 3 x 0.5 as final dose) and its stereoisomeric compound *quinidinum* basicum (0.1—3 x 0.2) were first given a trial.

Quinine and its derivates act as depressors of the heart, retarding the initiation of the impulse, lowering the tonus of the sinus and prolonging the speed of conduction. Quinidin is the more effective but also more dangerous drug; it lately has been strongly recommended, especially by German and French clinicians for the regulation of

arhythmic actions of the heart. Cases are reported of arhythmia of 6 months and longer duration in which the rhythm had become regular after only one week's treatment with quinidine. The present case seemed quite suitable to be benefitted by this drug, therefore after a short but unsuccessful trial with quinine, quinidine was given at three different periods, each extending over one week. As effect a sinking of the blood pressure was noticed (140—145), the pulse became smaller in volume, the cardiac sensations of the patient however seemed rather to increase and the irregularity showed no essential changes.

To counteract the depressing effect on the circulation digitalis was used, first separately, later combined with quinidin. The drug was given in the form of folia digitalis in standard doses of one grain of powdered leaves, equivalent to approximately one cat unit, in hermetically sealed capsules (Kapseals Digifortis, Parke, Davis & Co.), which I have found very reliable in their effect in the tropical climate. Already after the third dose the pulse had slowed down to below 60, the wave having become stronger and fuller in volume, but the irregular rhythm remained unchanged even after the heart had been kept under the effect of digitalis for some time. Also in *combination with quinidin* the desired, regulating effect failed to appear, on the contrary the patient's disagreeable sensations grew decidedly worse so that the medication had to be given up. There are cases reported in which regulation took place only after the administration of large doses of quinidin, up to 3 x 1.0, but in a case in which degenerative changes of the myocard are suspected there is always danger of doing harm with high doses; I therefore refrained from such forceful treatment.

A substance of the *adrenalin group, ephetonin* 1—3 x 0.05, which predominantly has a depressor effect on the vascular tone was tried next. The pulse remained irregular but became slightly quicker and somewhat fuller, a moderate diuresis set in, occasionally headache and shoulder pains appeared, of which the patient had never complained before; the cardiac sensations remained the same or even seemed to increase.

Then *iodides* were given in various forms as sodium iodide (0.5—3 x 0.5), sajodin (0.5—4 x 0.5), iodotropen and iodocasein (2—5 tablets) with the effect that the patient soon complained of a feeling of heaviness in the chest especially in recumbent position with a tendency to cough, the quality of the pulse showed no alterations. *Jod-* and *rhodan calcium diuretin* and *calcium diuretin* alone, acting as vasodilatators, had all a prompt diuretic effect, but otherwise did not essentially influence the condition.

A similar result was obtained with *coffeinum natriobenzoicum* (0.2 1—3 x daily) which, contra acting angiospastic tendencies appeared to ease the pains for a certain time, but later as a kind of reaction the discomfort seemed even to become more severe.

The experiments with atropinum sulfuricum (1—3 x 0.005) and papaverin (1—3 x 0.02—0.04) gave also no satisfactory results.

During the last 10 years *organ extracts,* especially muscular extracts, have been recommended in cases of myocardial degeneration, and Schwarzmann (Odessa) has repeatedly reported good results with his own extracts in cases of angina pectoris. I had two kinds of these extracts at my disposal, *Padutin* (formerly named Kallikrein), a substance which in inactive form circulates in the blood and is obtained from pancreas tissue, where it is found in particularly high concentration, and *Lacarnol,* extracted from the cow's heart, which contains adenylic acid (which is absent in Padutin) and adenosin, a substance of importance in muscular chemistry. Both substances increase the capillarisation in the muscle. On Lacarnol (5 gtt—3 x 20 gtt) the pulse rate increased, the impulse of the wave became shorter and the irregularity even more pronounced, at the same time the patient complained of an uncomfortable feeling of tightness, sometimes increasing to a sensation of pain in the region of the heart and towards the diaphragm. From Padutin (10 gtt—3 x 20 gtt) a similar but not so outspoken effect was noticed.

At times when the cardiac pain adopted a more anginal character nitrates were used, nitroglycerin in alcoholic solution, 10 gtt of a 0.01 : 10 solution, or nitrolingual tablets. The effect was of course only temporary as it was with sedatives such as bromium, luminal, allonal, ortal and similar drugs.

After this thorough trial with more than 12 drugs of various nature and action I was satisfied to learn from the experimental point of view that each drug, principally those of an outspoken effect on the heart, had in a striking way produced the same reaction at each time of the different periods it had been given. As a further result I had learnt that the arhythmic action of the heart had not been essentially influenced by any of the drugs given and that also the subjective sensations of the patient had only temporarily been relieved. The patient always felt best in the intervals when at rest and no drug at all was given.

It was apparent that every agent, be it of a physical, psychical or chemical nature which tended to disturb the mechanism of the present circulatory equilibrium caused increasing discomfort.

For a better understanding of this very complicated mechanism and of the pathogenesis of our case it might be well first to recall some of the *anatomical* and *physiological* facts.

Anatomical and Physiological Notes.

The heart's action is primarily regulated by an autonomous system of myogenetic nature, which is intercalated into the whole regulating

apparatus of the vegetative nervous system. Hence the organ is largely dependent on extracardial nervous stimuli; but, when cut off in the animal experiment from all external nerve supply, the heart in a short time will resume its regular automatic action.

The elements which are responsible for the autonomous action of the heart are special muscular fibres; an accumulation of these fibres, known as *sinus*—or *sino auricular*—or *Keith Flack's node,* is found in the right auricle near the entrance of the superior vena cava. A similar accumulation, first described 1906 by Aschoff and Tawara is situated in the wall of the right auricle between the attachment of the tricuspid valve and the entrance of the coronary sinus, this is the *auriculo—ventricular node* (a. v. node). Whereas there is no visible trace of a conductive tissue connection between the two nodes, a bundle of fibres, the only muscular connection between auricle and ventricle, runs from the a. v. node towards the top of the ventricular septum, there it divides into two branches of Purkinje fibres which find their endings in the left and right ventricular walls. This a. v. *bundle of His* is the conductive system, of which the a. v. node is the head. As an early part of the embryonic heart and traceable down to the lower stages of animal life the bundle of His is the most conservative element of the heart. The sinus node in which the generation of the impulse takes place is the pace-maker of the rhythm, through the walls of the auricle the impulse is transmitted in regular intervals to the a. v. node and from there further to the ventricles. Pathological stimulation of a purely functional nature or from organic lesions may occur at any place along the course of the conductive system and interfere with the regular rhythm; at the place of disturbance a local centre of stimulation will then be formed with its own automatic rhythm. This heterogenetic stimulation interfering with the original homogenetic rhythm naturally will lead to irregular contractions of the heart,—ectopic beats.

The latter are clinically known as *extrasystoles* and by the characteristical feature of each variety in the Electrocardiogram they have been classified according to their site of origin as *auricular, ventricular and nodal extrasystoles.* In dealing with extrasystoles as clinical diagnoses it must be understood that to this group belong only cases in which a dominating fundamental rhythm exists, into which premature beats are interposed in more or less regular intervals. In other types of disturbances of the regular rhythm, in which lesions at the a. v. node or at other parts of the conductive bundle cause partial or complete block of further transmission of the impulse, an entire dissociation of the auricular and ventricular action will take place (ventricular anarchy). Without graphic records however it is impossible to differentiate between the types arising from the lesions at the various places.

Alterations in the heart's action are due not only to pathological conditions in the heart itself. As we have seen the heart is to a great extent also under the *influence of extracardial nerves.* Vagus and sympathicus—or accelerans as it is often called—continously control the tonus, the former having an inhibiting, the latter a stimulating effect. but their action must be understood to take place in an absolutely synergic manner like the steering of a sculler, who will keep his boat on a straight course by regulating the muscular power of his arms which he transfers to the oars on each side. Therefore when the accelerans tonus increases the tone of the vagus automatically decreases and vice versa. Both nerves as centrifugal nerves constantly receive stimulations from their centres in the medulla, where chemical agents, hormons or products of metabolism continuously are at work, such as e.g. potassium and calcium, the former stimulating the vagus, the latter the sympathetic nerve. Transmitters of centripetal stimuli are the elements of the nerve plexus around the ascending aorta and sinus caroticus, from these also arise the fibres which convey pain sensations to the centre. According to H. E. Hering these nerve plexus receive stimulations by changes in the blood pressure—hence called pressor receptory nerves. They convey this impulse as a reflex action through the medulla and back through vagus or accelerans to the heart, with either an inhibiting or accelerating effect; in this way the reflectory selfregulation of the blood pressure takes place. Disturbance of this reflectory mechanism of selfregulation leads to arterial hypertension and increase of pulse rate; the same effect has complete elimination of the aortic and carotic sinus nerves. The so called vagus pressure phenomenon—decrease of pulse rate and blood pressure—is in fact produced by a pressure on the carotic sinus and not on the 10th nerve itself. In sclerosis of the carotic sinus this reflex is more pronounced.

Ultimately thus we see that *the blood pressure automatically regulates the action of the heart,* its rate of frequency, systolic output and minute volume.

This *regulating effect* however is not only exercised on the heart, but *on the whole circulatory system,* of which the heart is only its starting central motor. Heart and blood vessels—arteries, capillaries and veins are a functional unity and in cases, in which disturbances of the heart itself seem to be in the foreground, the primary cause may often be looked for in the periphery, for the peripheral system,—the arterioli and capillaries,—with its tonic elasticity has an important motor function of its own, which when failing, may lead to a purely peripheral circulatory failure.

An important *function of the elements at the pressor receptory areas* at the ascending aorta and carotic sinus is also the *regulation of the arterial tonus in the splanchnic area* (liver, spleen, kidney, mesentery) which serves as a reservoir in case of a sudden afflux from the periphery.

Decrease of pressure at the aortic arch will increase the rate of the heart beat and the minute volume and cause arterial and venous constriction in the periphery as well as in the splanchnic area; from these places the blood stream is then directed towards the heart and central nervous system. *Hormonal regulations and the tonus of veins* are *also directed from the pressor receptory areas.* Contraction of the veins increases the tonus and systolic output of the heart and a low tonus in the venous system results in inefficient function of the heart. The *proper function of the dynamic mechanism of the venous system is therefore equally necessary to the efficiency of the heart* as the sound working of the arteries.

Among the agents which have a *haemodynamic effect, muscular effort* certainly is one of the most potent factors, but we should not underestimate the effect of *psychical stimuli* which in a labile condition of the circulation and especially of its central motor may cause just as much harm as physical strains. Cases in which sudden death occurred after the patient had been upset by violent emotion are not rare. Experimenally the effect of psychical excitement on the circulation has been tested and in aviators an increase of blood pressure from 21 to 32 mm. Hg. could be demonstrated immediately before they ascend.

The *constant,* synergetic *regulation* of the constrictor and dilatator mechanism is *kept up by chemical substances,* produced and circulating continuously in our body, among these CO_2 plays the most prominent part. Introduced into the arterial blood stream CO_2 increases blood pressure and pulse rate; hyperventilation of the vasomotor centre on the other hand lowers the pressure. Elderly people are said to react quicker on the stimulation with CO_2. Next to CO_2 *products of internal secretion* are instrumental in regulating the circulation. *Adrenalin* under certain conditions, such as increased destruction of tissues in burns or infections, actually is secreted from the adrenal glands into the blood and leads to contraction in the splanchnic system as well as of the vessels of the resting muscle, whereas those of the active muscle remain dilated.

Histamin, a metabolic product, present everywhere in the body and closely related substances like *acetylcholin and adenosin,* isolated from urine bile and milk, have a dilating effect on the whole vascular system, thus lowering the peripheral tonus; contracting in a vaso constrictor sense is *vassopressin* a component of the posterior lobe of the hypophysis, the substance entering the circulation by way of the spinal fluid.

It would unduly lengthen this paper to go into further details of the physiology of the circulation; these few main facts may suffice to illustrate the wonderfully ingenious and exact working, yet very complicated arrangement of the normal circulation. The astonishing efficiency and rapid accommodation of the heart to tests of utmost

physical strains as in boat racing, Marathon runs, etcetra belong to the most wonderful physiological phenomena.

On the other hand it is evident that like in a very complicated clock work a small dust particle is able to upset the working of the whole mechanism so even a small lesion, situated in a vital part of the heart may cause serious disturbance of its function. The *normal action of the heart,* as we have seen, *depends on the healthy condition of all those organs which are intimately connected with the circulation and any morbid alteration in one of them may bring disorder into the whole system.* Primary lesions of the heart itself in most cases present pictures of a well defined clinical character, affections of the valves and the myocard belong to those groups of heart disease.' As in our case no valvular signs could be detected I shall confine myself to the discussion of a few topics out of the *pathology of the myocard* in relation to our case.

Pathological Notes.

Recent experimental work on animals hearts, in which systematic lesions were produced and registered by the electrocardiograph (E.C.G.) and later controlled by histological examination, has added much new material to increase our knowledge in cardiology. I have stated above that in many cases of heart affections an exact local diagnosis of the lesion is impossible without the help of graphical records. In the E.C.G. we possess an instrument which produces curves, characteristic for a certain localisation of a lesion. As the E.C.Gm. of a rabbit is not essentially different from that obtained from a human heart, the E.C.G. has become also a valuable means in experimental cardiological research. Deviations from the normal E.C.Gm. resulting from experimentally produced lesions in the rabbit's heart, when obtained in similar form from a human heart will indicate lesions of a similar localisation. By letting rabbits run for a certain period of time in a revolving drum F. Buechner, a pupil of Aschoff, succeeded to produce *disseminated necroses* in the heart muscle, which he considers as *the effect of an anoxaemia* through inefficient blood supply by the coronary arteries. These histological changes, situated mainly in the subendocardial layer of certain areas in the left ventricle and papillary muscles, probably the parts which were exposed to the greatest strain, or were somewhat handicapped by the distribution of the blood supply corresponded in nature and localisation exactly to the alterations found in the hearts of patients who had suffered from attacks of typical angina pectoris. The similarity of the two conditions extended also to the graphic phenomena. The rabbit's heart with the disseminated "*exhaustion necrosis*" rendered a E.C.Gm. which although only transitory, showed the *same alterations which are typical for the anginal attack.* By systematic histological examination of serial cuttings of the myocard and especially of the papillary muscles Buechner could further demon-

strate that the various stages of regressive metamorphosis found in hearts of patients who died of angina pectoris, corresponded chronically with the various attacks of anginal pain which had been graphically registered. Regeneartion of diseased muscular elements does not seem to take place.

These experiments and anatomical findings have revealed important facts which in my opinion help to elucidate certain conditions and phenomena in the pathology of the heart which up to the present time have not yet found satisfactory explanation.

I am referring here to the *problem of pain,* the cardialgia in its widest sense. Sir James Mackenzie, some 20 years ago already emphasized the great valne of the due appreciation of the patient's sensations in estimating the condition of his heart and circulatory system generally; but it seems to me that still very little attention is paid by most men of the medical profession to this kind of investigation which seems so simple as it does not require the use of any instrument; but it certainly demands a good deal of patience, criticism and skill to extract from the patient such information which may characterize certain pathological conditions. Having learned by sifting the chaff from the wheat, to interprete the patient's sensations correctly and to bring them in convincing relation to the objective findings such information will often be found to be of great diagnostic value.

Most certainly a continuously persistent dull pain underneath the sternum will indicate a different pathological condition than a sharp, cramp like sensation localised at the apex of the heart. An occasional pain in the heart region after an effort in an elderly individual which otherwise has no physical signs indicating heart trouble, may be a signal, warning that there is danger lurking. In such a case if the danger is recognised in time, by adequate prophylactic measurements life can often be prolonged. The medical practitioner to a greater extent than the hospital physician has the opportunity of observing a case over a longer period of time in different conditions and stages of a disease, it is in this field of study where he can do his bit of research: in *careful investigation of the nature and localisation of the patient's sensations in correlation with the objective findings.*

How little we still know about this subject is best illustrated by the different conceptions and explanations given with regard to the nature and origin of pain in that form of heart affection, in which this symptom is so prominent that the whole clinical picture was named after it: the angina pectoris. Since Heberden in 1768 had given a well defined description of this disease, angina pectoris has since figured in textbooks as clinical entity such as the various forms of valvular disease, but with our present knowledge in pathology we are no longer justified to consider the disease as a clinical unit; *angina pectoris is only a complex of symptoms without uniform pathological back-*

ground. Lesions of various nature, sclerosis, thrombosis and embolism of the coronary arteries, degenerative changes in the myocardium, infarct and aneurysms all may produce anginal pain and lastly a patient may die in an attack of "angina pectoris" and no lesions at all neither in the myocardium nor in its bloodvessels are to be found. Just recently I have seen a case with typical attacks of anginal pectoris in which the post mortem examination revealed a pericarditis.

At least we have to differentiate between *various clinical types of cardialgias.* The classification can of course be only symptomatic with the pain as leading principle but our endeavour should always be to find out the pathological background for each symptom. Then with the typical anatomical changes at our hand we will still more vividly realize that *a morbid process in an organism does not necessarily produce a condition or disease with definite outlines, which will exactly fit into a clinical scheme; all activities and development in nature are continuously moving and changing* and boundaries which we artificially erect for didactic purposes are often forced upon our mined to such an extent that they are apt to create false conceptions.

Types of Cardialgia.

Clinically the *types of painful sensations* arising from pathological cardiac conditions manifest themselves in the following forms:

1. Attacks of pain mostly felt below and somewhat outside the nipple, usually accompanied by palpitation, chocked breathing, pallor and a feeling of cold all over the body. This kind of, as it is also called, *phrenocardiac pain* is the mildest form and of a transitory, *psychogenetic* character; it is not rarely found in young women, the "Liebesschmerzen" (love pain) of the German writers.

2. Similar in origin but displaying more marked physical signs and symptoms is a condition which is known as *angina pectoris vasomotoria or neurotica.* Severe pain in the cardiac region with symptoms of vasomotor disturbances. Like the former this form is represented mostly in women of a nervous constitution.

3. Slight cardialgic pain, coming and going in irregular intervals.

4. More or less constant dull pain in the substernal or cardiac area which is felt when the patient is at rest as well as when he is moving about.

The latter two forms mostly occur in elderly people, predominantly in men and point to a more or less advanced myocardial degeneration; a persistent, dull, choking sensation below the lower half of the sternum is in my experience highly suggestive of an affection of auricles, as e.g., auricular fibrillation.

5. Persisting painful sensations of a varying degree confined to the region of the aortic arch, the *aortalgia in cases of syphilitic arteritis*. Very likely the same condition in the coronaries will also lead to a typical pain sensation but with different localisation—*coronaralgia*.

6. Conditions, described as *angina ambulatoria* (Wenckebach), angine de poitrine d' effort (Vaquez), formerly as angina pectoris chronica, intermittens or minor, in which every increased effort, psychical emotion or also a heavy meal (post coenal angina) produce more or less severe attacks of cardialgia, which may soon disappear when the patient has regained his physical or mental equilibrium, or in other cases may last for a considerable length of time.

7. The *typical angina pectoris* which is an acute attack of very severe pain accompanied by cold perspiration, and a feeling of great anxiety and distress, there is often some vomiting; the pain usually spreads from the cardiac region towards the shoulder into the left arm and may last for minutes only or through several hours.

8. *Status anginosus,* a picture like the last described, but con-continuously persisting for a period of days, usually ending in death.

There is still a conflict of opinions with regard to the *origin and nature of pain* in cardiac affections and in particular in the typical form of angina pectoris. Sir Clifford Allbut put forward the *theory* that *distention of the ascending aorta* was the source of the pain, Vaquez and Wenckebach have later supported this view, but their theory can no longer be maintained since experiments have demonstrated that the sympathetic nerves in the adventitia of the ascending aorta as well as those of the coronary arteries respond with pain sensations only on chemical (e.g., anoxaemia) and not on mechanical stimuli. Besides increased pressure in a patient, suffering from angina pectoris, does not always produce an attack; the aortalgia proper, the typical symptom in syphilitic mesaortitis in which distension of the aorta has been observed on the X-ray screen is an altogether different sensation.

Another theory considers the pain due to a *paroxysmal vasomotor spasm* of the coronaries, analogous to the pain in claudicatio intermittens, but this explanation does not seem very convincing as the vasomotors of the coronary arteries do not play an important part such as those of the lower extremities and e.g., the splanchnic system in abdominal pain.

R. Schmidt (Praha) locates the origin of the pain into the nerve itself and assumes structural changes in the substance of the cardial nerves and plexus which he holds responsible for the pain, a kind of *visceral neuralgia*. He further claims that this condition is usually associated with a certain status of vaso motor allergy. This neurogen theory however does also not explain all the phenomena satisfactorily.

The *muscular theory* at last, brought forward some 30 years ago and of which Mackenzie was its strongest exponent, through recent experiments, especially those of Buechner has gained great support.

The clinical phenomenon of an *anginal attack* can now definitely be *linked up with constant changes in the E. C. Gm. and also with certain histological findings.* Suffocation and lack of oxygen in the inhaled air produce the same characteristical features—alteration, even disappearance of the T wave—in the E. C. Gm. as seen during an anginal attack. These changes are due to *anoxaemia* in the heart muscle. E. C. Gm. taken by Hausner and Scherf in Eppingers Clinic in Vienna, further show that the changes in the S-T complex occur at each attack of anginal pain which was produced by a certain amount of physical effort and that they gradually disappear after the pain has subsided with the patient at rest. From these experiments we learn that changes in the E. C. Gm. hitherto thought to indicate a complete coronary block, may also temporarily take place in cases in which the blood supply was only temporarily insufficient. *Anoxaemia in the myocardium, producing clinical symptoms,* is *most liable to occur in hearts in which atheromatous and sclerotic lesions, and probably also spastic changes of the coronaries impair the latter's function of sufficient blood supply to the heart* (cardiopathia arteriosclerotica), but the anoxaemia may also be due to other causes, thus the somewhat epidemical outbreak of angina pectoris attacks among sailors on board the French ship "Embuscade" has been explained by the effect of lead poisoning.

Anoxaemia of the heart muscle through insufficient blood supply will also appear as *plausible cause in other forms of cardialgias.* The transient "neurotic" pain is probably due to a temporary spasm of the heart vessels. In elderly people the arteries gradually undergo anatomical changes in the sense of fibrosis and collagenosis, thickening of the intima and splitting up of elastic elements, alterations which especially at *periods of increased strain to the circulatory apparatus lead to ischaemic and anoxaemic degeneration.* These degenerated and later fibrosed areas may exist without producing symptoms during lifetime especially if the foci are situated in the so-called *"silent" areas* of the heart muscle. It is astonishing to find at autopsies quite extensive degenerative processes in the myocardium in cases where sudden death had occurred and during life no heart symptoms had been complained of.

On the other hand a comparatively small focus of degeneration, if it happens to involve the more vital parts of the heart e.g. the junctional tissue, is able to disturb the harmonic action of the whole organ and cause painful sensations.

The *appearance and character of pain thus largely depends on the nature, extention and localisation of the affected muscular elements.*

The sensory nerve fibres of the heart take their course through the nervi cardiaci inferiores to the sympathicus and from there through the rami communicantes to the spinal cord; a part of the sensory tracks probably has also communications with the vagus nerve. According to Dale and others chemical substances are the agents in the exchange of mutual stimulation between nerve and muscle; thus *anoxaemia* will *act as a causative agent for the sensation of pain.* If a great part of the heart muscle is affected as e.g. in a case of infarct, following coronary embolism a condition of a severe pain, suddenly setting in and continuing for a long period of time will result (status anginosus). Smaller disseminated necroses may cause cardialgic discomfort of a greater or lesser degree, acerbating with the change in the circulatory equilibrium and muscular activity.

We further have to assume that with *increased sensibilisation* of the pain receptory elements painful sensations will gradually become more easily released.

After these excursions into general physiology and pathology it will be easier to understand the condition of our case. In an elderly person perpetual absolute arhythmia, lacking any trace of a leading basic rhythm and increasing in irregularity after physical effort, excludes the diagnosis of extrasystole, but is highly suspicious of auricular fibrillation under *those circumstances which* we have found in our case.

Importance of Careful Examination of the Pulse.

I might on this occasion say a few words on the importance of a careful *examination of the pulse,* inasmuch as the method of *pulse feeling* has been valued *in China as a great art* since ancient times. I must confess that in this particular point I have learnt a good deal from China; first of all that pulse feeling can be developed to a real art. The *little cushion* which invariably is used by the Chinese doctor of the old style and which custom I have met in no other country is certainly more than a mere symbol, it gives the performance that *atmosphere of solemn rite and restfulness* which indeed is required for the necessary mental concentration at such a delicate examination. The many qualities the old Chinese pulse expert claims to be able to discriminate will of course not stand the tests of medical science but a very careful observation of the pulse wave may often reveal minute alterations of its quality, which will be sufficient indication for some disturbance of the circulatory mechanism. To discover irregular beats the *pulse has to be controlled for a sufficiently long period,* an irregularity may make its appearance occasionaly only after a period of a dozen or more regular beats.

Pulse anomalies may further become evident only after the heart had been put under a certain amount of strain. A *functional test* should therefore never be omitted in the cardiological examination. Letting the

patient mount 20—40 steps of a staircase without rest provides a very simple and quick method of testing the heart.

Since nowadays more attention seems to be paid to laboratory and instrumental tests than to the simple bedside methods of examination, which threaten to become more and more neglected it seems necessary to stress the great value of the clinical observations of the finer changes in the quality of the pulse wave; the full appreciation of their diagnostic meanings will only be gained through long experience. A pulse may be quite regular and equal, of normal rate and filling and yet the *character of the wave will convey to the trained finger* of the expert *the impression* that the heart is working under a certain strain. In these cases the E. C. G. will clearly show that the finger has felt rightly. We have an excellent means to improve our abilities in pulse examination by the control with this instrument which should belong to the equipment of every modern hospital as indispensable an outfit as a X-ray apparatus.

Besides the somewhat characteristic type of arhythmia the pulse in our case gave the impression of a slightly oscillatory sensation which we may observe in cases in which the E. C. G. shows a prolonged phase of auricular fibrillary waves. As in the case described no signs of arteriosclerosis could be detected, with regard to the possible origin of the arhythmia we may reasonably assume that the many and various physical strains, occasionally exceeding the safety limits, drawn by the age of the patient, had gradually marked their effects of "wear and tear" on the myocardium of apparently principally the auricles until a last, not necessarily exceptionally heavy, strain had attacked and outbalanced the last healthy portion of the muscular elements, which up to then had still been able to maintain the regular transmission of the rhythm. Thus the cup was suddenly brought to an overflow, the contractions of the degenerated myocardium of the auricles turned into irregular fibrillations which prohibited the transmission of the normal impulse. Whereas the ventricles still seem to possess a fairly good working power the auricles constantly stand under a certain amount of strain, enough to cause a sensation of discomfort increasing to pain with increasing muscular effort. The degenerated areas however are not likely to be confined to the auricles only, the painful sensations localized by our patient to the left side of the heart and usually appearing after a comparatively strong effort as well as the then poorer quality of the pulse indicate that also the ventricular myocardium is not free from degenerated and thus inefficient elements. The negative results with the various remedies given further seems to indicate that the perpetual arhythmia is due to myocardial degeneration rather than to purely dynamic disturbances.

The *prognosis* in the case will depend on the reserve strength of the heart, on the amount of healthy tissue left especially in the

ventricles and on the degree of work the patient will allow his heart to do. As the condition of the patient kept stationary during the last few months of my observation, the outlook seems to be quite favourable as long as the patient avoids every extra strain.

With regard to treatment it is evident from the nature of the process that drugs are of little or no value in our case and that prolonged rest in bed will also serve no purpose unless signs of beginning insufficiency make their appearance. The main object has to be the strengthening of the circulation by systematic exercise upon the lines of Oertel's treatment ("Terrain kuren," well supervised, graduated walks on more or less level roads), together with carbonic acid baths. There is no doubt, that the *Nauheim baths* deserve their reputation and Sir James Mackenzie who considered the claims of the baths as more or less absurd, would probably have changed his opinion if he had seen and studied the effects of the whole Nauheim treatment at the place itself and in its present time. On my last visit to Nauheim in 1933 I visied the Kerckhoff cardiological station there; the work done at this place and the many improvements in the health of patients after a few months treatment in Nauheim controlled by regular cardiographic examinations convinced me of the great value of the Nauheim scheme in the treatment of heart diseases.

It is certainly absurd to believe that an organic heart affection can be cured in Nauheim, but many patients return from there greatly benefitted, at least they have learnt how to prolong their lives by a changed method of living adapted to the altered condition of their hearts.

Excrcises in deep breathing act in the way of massage of the heart and aorta and should be performed regularly to promote an easier circulation.

Careful attention has further to be paid to the *diet;* most of the patients, suffering from heart trouble, know from their own experience that a heavy meal or an indigestion has an aggravating influence on the condition of their heart—a phenomenon which is known as the *gastro-cardiac symptom complex*—therefore a light easily digestible and not too bulky food is an essential factor in the treatment.

Psychical effects as we have seen influence the circulation to a great extent, the patient therefore should learn to control his emotions by avoiding occasions which are apt to unbalance his temper.

A few words have at last to be said with regard to the *attitude towards the pain.* Attacks of violent anginal pain demand the immediate administration of drugs, of which amyl nitrite is unsurpassed in its quick effect, conditions of minor degrees of cardialgias as a rule readily respond to the various kinds of sedatives,

but there is another important side to the question: *shall we,* generally speaking, *in all cases try to combat the pain?* I have known patients suffering from cardialgia who had become so accustomed to their pain that they considered it as a sort of a good friend and *guide* which they would not like to miss; they had learned to understand and follow its warnings and since then by strictly avoiding to overstep the limits of suitable physical activities had managed to live a quiet but comfortable life, their heart giving scarcely any trouble at all. From this point of view *operative measurements* like the *sympathectomy* must be consideed to be of a very *dubious value. Manifestations of discomfort and pain are the red lamp, the signal which warns the patient of the immediate danger which threatens his heart during an effort,* indicating that the strain is too great for the circulation. In some cases the warning comes too late and the patient succumbs to the attack, more often lighter attacks had preceded but had not received adequate attention, which otherwise might have saved the patient for a longer life. Thus *by operation we deprive the patient of a very useful guide,* besides, these operations, which are directed towards one symptom only and entirely neglect the primary cause of it, mean a violation of our first principle in therapeutics: Nil nocere. In trying to cut or remove pain conducting nerve fibres we destroy parts of an immensely complicated system which controls the most vital parts of our body. The pain may be relieved, but the damage otherwise done to the heart will soon bring the tragedy to an end. The result of most of these operations always tells the same story.

A much milder procedure will give the same, although only temporary relief in cases of prolonged severe anginal pains, namely the *block anaesthesia with novocain* of the rami communicantes from C 1 to D 4, eventually also from C 2—4, the anaesthesia usually lasts for a sufficiently long period to overcome the attack.

Conclusions.

In concluding this study it remains to draw some *conclusions* out of the facts and experiences given in the paper. We have seen in a perfectly healthy individual in advanced age, but still being very active and physically fit, following an overdose of physical exertion suddenly appearing a cardiac disorder which renders him a permanent invalid. This is not a rare case and belongs to the same category as those sudden and unexpected deaths from an acute "heart attack" during a heavy meal, on the golf course or at other similar occasions. I have already pointed out that this form of death is particularly frequent among the brain workers. No doubt the mental strain and unrest, the "hectic" life of modern days, are to a great extent responsible for the quicker wear and tear of the heart, very likely also an excessive amount of physical exercise may under certain conditions

even in a healthy individual prepare the foundation for myocardiac changes in future life.

I was as I mentioned in the beginning always very much interested in the following up of the behaviour of hearts of those men, who in their youth had gone in for expecially strenous exercise. Let us take e.g., the classical Oxford and Cambridge boat race which certainly means a tremendous strain on the heart. Many of these old blues have kept fit until old age without symptoms of any damage to their hearts; others have early developed signs of heart troubles. The same experience can be made with other vigorous sporting activities as e.g., in mountaineering. I have known guides in Switzerland, who when over 70 years of age were still able to climb 3—4,000 feet without difficulties, others, good climbers in their youth, developed heart symptoms in middle age suddenly after an altogether not too strenous climbing tour. Certainly in these latter cases other factors, like nicotin and alcohol, previous infectious diseases or similar agents of a damaging nature may have caused an additional strain to the heart, but the question is still open as to whether in a healthy youth strenous physical exercise predisposes to an earlier weakening of the heart muscle. Systematic inquiries into the histories and repeated examinations at certain intervals of individuals who in their youth indulged in a vigorous sporting life are required to clear up this important question. In my opinion the *danger does not lie in the exercise done in the youth, however strenous it may be*, provided of course that the individual is strong and healthy and the exercise done under proper training, but that *later in life the former athlete will find it difficult to restrict his physical activities within the limits which nature i.e. age imposes upon him*. A person who never played any outdoor game and whose physic is underdeveloped will soon be warned by signs of exhaustion to overexert his body beyond the danger line but an old sportsman will not get tired so quickly and will not so easily give way to these warning signals, for he is used to force his physical as well as mental capacities up to the highest possible limit.

Much could indeed be done *in the way of preventing* such suddenly arising manifestations of heart troubles if individuals in whose lives physical and mental strains have played a prominent role as predisposing factors for the development of heart affections had in time *regularly undergone careful physical examinations of their hearts, particularly with the electrocardiograph*. In analysing 27 cases of sudden death during golf playing C. W. Lieb in a recent article on the effect of golf on heart troubles points out that if those players had been warned before of their cardio-vascular handicaps and advised to play a game, consistent with their physical limitations, many of them would still be alive. Similarly many fatal accidents in elderly mountaineers, due to acute heart failure could have been averted, had there been made an electrocardiogram before!

ON THE ANTHROPOLOGICAL ASPECT OF BLOOD GROUPING. *

by

Lindsay T. Ride.

(Professor of Physiology, The University, Hong Kong).

In his paper, 'The Value of Blood Grouping in Anthropology,' read before the Anthropology Section of the Vth Pacific Science Congress in 1933, Furuhata says: "As to the value of blood grouping for application to Anthropology we must point out in the first place, the heredity of the blood groups; secondly, the stability of the groups throughout life; thirdly, the constancy of the proportions of the group of a population from generation to generation in the absence of crossing, and definite change in the blood group distribution due to racial crossing." There can be no doubt that the scientific establishment of the truth of these four points must precede any serious evaluation of Blood Grouping data in Anthropology. With regard to the first point we are already in a position to assert that the agglutinogens and the agglutinins are inherited according to genetical laws, nor is proof wanting to establish the fact that once the haemotype of the individual has developed, it remains unchanged and can be ascertained as long as fragments of body cells or samples of body fluid can be obtained. (The word haemotype is here used to describe the blood phenotype of the individual with regard to the A & B agglutinogens. Such a word is necessary because we have no other term to describe an individual in terms of the Blood Group to which he belongs. While it is perfectly correct to say a person belongs to Group A or Group B it is not litterally accurate to use the common loose method of describing him as being "a Group A." This term has the additional advantage that it can also be used in connection with any sub-group or with M & N groups also. Thus a person belonging to Group B is referred to as a haemotype B individual, one belonging to Group O as haemotype O and so on.) Snyder's work on the American Indians (which has been amply verified by later investigators) has definitely shown that the crossing of races with different blood grouping distribution leads, in the resultant new race, to a distribution different from that found in either of the parent races, but differing in a manner consistent with our expectation from genetic principles. With regard to the third point, the position is not so clear. Are the group percentages in a population constant from generation to generation in the absence of racial crossing? In other words is racial crossing the only method of altering the established percentages peculiar to a people? The haemotype of an individual has proved to be independent of environ-

* Read before the Section of Anatomy and Physical Anthropology at the 1st Session of the Congrès International des Sciences Anthropologiques et Ethnologiques at London on August 1st, 1934.

mental changes brought about by climate, diet, age, disease etc., but the frequencies of haemotypes in a population must be subjected to variation just as those of any other hereditary characters are.

Before we can accept the statement that the frequency can be altered by racial crossing alone, we must show that other factors such as selection or mutation can not, and do not, do this. So far it has not been possible to demonstrate linkage between the haemotype and any other character, and this has led to the acceptance of the belief that the non-selective value of the blood groups has been established. Considering the number of chromosomes in the human and the small number of characters that have been thoroughly investigated genetiacllly, it is, as yet, hardly scientific to accept this belief as true. The number of factors influencing or capable of influencing selection must be large, and even amongst any one race these factors must change from time to time; thus it must be well within the realms of possibility that under the action of these many selective forces, there must have been in almost every race an interference with random mating as far as the haemotypes are concerned. In large populations, not only are the isolates—a term used by Dahlberg to denote that portion of a population from which one individual has the chance of choosing a mate—large, but the isolate of each individual differs from that of his neighbour. The large size and variety of isolates has this result, that the distribution of genetical characters is as even as though mating had occurred in a purely random manner throughout the whole population. This explains why in countries with large and more or less homogeneous populations, (homogeneous here being used in the sense of an even distribution of various characters throughout the population) different investigators generally find the blood group percentages to be more or less constant.

There is another reason for this and that is that their data are taken in a manner which assures as far as possible, that they should be pure random samples; they test, for example, each patient who enters a hospital or an institution, and the inclusion of members of the same family or of near relatives is thus reduced to a minimum and governed by pure chance. When we come to the method used amongst native tribes, however, we find it somewhat different. An expedition visits a village, or a group of villages which constitute a tribe, and every individual of attending batches is grouped. Such batches do not constitute random samples of the population for the people always tend to present themselves in family groups and hence the data thus taken suffer the same family grouping. That is one factor which tends to make results obtained by expeditions less constant; another factor is that when a tribe is small and cut off from easy contact with other tribes, the isolates of individuals in that tribe are small, and what is more important, coincident. This must inevitably result in inbreeding with its opportunity for selection, and if this selection effects the haemotypes it will result

in a change from a homogeneous to a heterogeneous distribution of agglutinogens among the people as a whole.

This heterogeneity of the population does not ensure that each character has an equal chance of being included in the sample chosen, nor do the social conditions of the natives ensure that the isolate is of a size and variety sufficient to prevent selective factors from operating. These statements should show how erroneous it may be to deduce anthropological conclusions from serologicial data unless methods of collection of data and local social conditions are known and taken into account.

Let us now see how this idea affects the generally accepted explanations of some of the peculiar blood grouping distributions. Its most important result is that any found blood grouping distribution *may* be but the expression of racial isolation and the consequent opportunity it gives for the action of selection factors; in other words the peculiar group frequencies found among the Australian aborigines may not be due to the supposed fact that the original inhabitants of that continent were cut off from Asia before the B mutation had infiltrated there, *but may simply be evidence of lengthy isolation of small communities during their migration.* As their forebears emigrated by gradual steps along the narrow corridors connecting Asia with Australia, this narrow human stream may easily have been split up into numerous small isolated groups and gradually colonies consisting mainly of haemotypes O & A may have been formed in much the same way as the modern bacteriologist's technique isolates pure colonies of bacteria. Then once having arrived in the large Australian continent, no longer limited by the difficulties of narrow confines and thick jungle, the population increased and spread, its isolates again becoming large and various enough to permit of random mating and the consequent establishment of fixed group frequencies now found to be peculiar to these aborigines. It is worthy of note here that the other peoples showing a similar unusual type of group frequency, namely the North American Indians, are generally believed to have crossed from Asia by means of a similar narrow land corridor, and it is significant that again it is the O & A groups that are to be found.

When we visualise such a method of migration, we realise that it may not even be necessary to call in the aid of selection to explain changes in group percentages. Such migrations as we are now considering could not possibly have been simultaneous migrations of large masses of people, but rather must they have been of a type of a gradual budding out from the periphery of the parent population. Each new community must have been drawn not from the old population as a whole, but from one special peripheral part of it and whether the new community will start with a similar distribution of characters to that of the parent community will depend on whether these characters are evenly

distributed throughout all parts of the parent community, and this as we have seen above is unlikely where the community is composed of isolated tribes.

But even if the peripheral part did have the same character distribution as the whole parent community, it is obvious that a random sample taken from the former will not have exactly the same character distribution as the whole of the parent body, and the chance variation will depend on the frequency of the character in the parent body and the size of the sample chosen. The smaller the sample the greater is its variation liable to be on the grounds of chance alone. And this is just the type of the migrations which must have resulted in the peopling of both Australia and America from Asia. Small groups of people moved from the periphery of their parent community along the narrow land corridors, and thus, setting up new communities with blood grouping percentages already varying from the prime stock, they became the new periphery. As this new periphery gave rise to a further migration, a further variation took place, and as at no time in the narrow land corridor was wide mixing with other outgrowths possible, each variation was preserved until the last migration, arriving at a new continental land mass, was able to established a stable character distribution by means of the panmixie possible there.

We have already seen that the perculiar percentages of the Australian aborigines and the North American Indians may be due to selection factors acting on small communities during the process of migration. Now we have just shown how they may also be due to random emigration of small groups along narrow land masses in a way which prevents the general mixture of these groups with other off shoots: and there seems to be no reason why both these methods did not occur, each aiding the other. So much for the existence and effect of these possible causes of blood group variation in peoples. And now what of mutation? The theory accepted at present assumes that the agglutinogens A & B arose as two independent and solitary mutations, the A preceding the B. There is no proof at all of this theory, the only evidence being that it would account in a general way for the known geographical distribution of the agglutinogens. But can we in the light of modern genetical knowledge accept this theory without more scientific evidence? Recent work in the lower animals has shown that each mutation has a frequency of occurrence—known as a mutation rate—and this frequency is a constant for the mutant factor, or if it changes, it does so only gradually with evolution. We can hardly believe that the factors causing the appearance of human agglutinogens are exceptions to this rule. Furthermore Fisher has shown that "a mutation, even if favourable, will have only a very small chance of establishing itself in the species if it occurs once only." On scientific grounds therefore one would expect the mutation to have occurred more than once, an expectation which, if true, would undermine

the whole basis of the present theory. To add to the difficulties of the situation we now have to take into consideration the existence of sub-groups and the M, N & P substances of Landsteiner; they must also have arisen as mutations. Were they too, solitary mutations? As yet we know very little of their racial distribution, but we may confidently look forward to the next few years throwing a flood of light on this important aspect of the case and judgment must be suspended till then. But on this general point, if we admit the probability of the survival of a character, we must also be prepared to discuss the probability of its extinction, and if we admit the possibility of a mutation resulting in the appearance of dominant characters such as the A & B agglutinogens, we surely must consider the possibility of a mutation resulting in the appearance of a recessive character such as haemotype O. Such a mutation is surely no new thing in genetics! It will thus be seen that from this aspect we are nowhere near a final scientific judgment of the case.

The outcome of all this is that we cannot assess the real anthropological value of serological data unless and until we can discover the cause of existing similar and disimilar agglutinogen distributionns, *and the real value of such investigations at present is not the discovery and tabulation of these racial differences or similarities, but the ascertainment of the manner in which these distributions arose.* This will only be achieved by an accurate investigation not only of the frequency of the A & B agglutinogens, but of the sub-groups and M & N substances as well, in other words by a thorough genetic and serological examination of races wherever possible. These investigations demand expert knowledge, special facilities and training, and it is therefore highly desirable that the energies of a Congress such as this should be directed in promoting and encouraging the utmost co-operation between anthropologists and serologists in accumulation of accurate and complete —rather than prolific—data. Racial blood group frequencies are still of anthropological value, and Snyder's four laws may still be considered true in general, as long as we realise they are only applicable where random mating with regard to haemotypes is certain i.e., in large communities; in cases where the communities are small and where there is not continual mixing from larger sources, the data should be subjected to genetical scrutiny to test their value, *and in any case must only be used as racial criteria when considered along with other characters of known anthropological and ethnological value.*

SUMMARY.

Before blood grouping can be said to have a definite and useful place in Anthropological investigations the four points mentioned by Furuhata must be scientifically established as true. They are (a) the heredity of blood groups (b) the stability of the groups throughout life (c) the constancy of the proportions of the groups of a population from

generation to generation in the absence of crossing and (d) a definite change in the blood grouping distribution due to racial crossing. Most people will agree that (a) (b) and (d) have already been proved but the position with regard to (c) is very different. If (c) were true, racial crossing would be the only method of altering the established percentage peculiar to a people. It is not only doubtful whether this is true, but it is certain that it has not been proved. The fact that we have not demonstrated linkage involving blood groups is no evidence that it does not exist and we therefore cannot assume that selection does not influence these percentages. In small isolated communities inbreeding must exist, and these selective factors may operate in large communities. Where panmixie operates the chance selection due to inbreeding is reduced to a minimum and hence the blood grouping percentages are more or less constant.

The distribution found amongst Australian aborigines may therefore be due to the change from the original brought about by selective factors operating amongst small isolated communities during their slow migration to Australia from Asia. When the migration takes place from the periphery of a population one would expect, by pure chance, the migrating people to have a different grouping from the whole of the parent body.

There must also be a definite rate of mutation of the A and B factors which is strong evidence against the solitary mutation theory, and the possibility of a mutation causing the appearance of the recessive haemotype O condition must be seriously considered.

We cannot therefore, at the present, assess the real anthropological value of serological investigations, and we will not be able to do so until we ascertain the real manner in which the various different distributions arose. This should be the real aim of the work at the moment and in any case data thus collected should only be used as racial criteria when considered along with other characters of known ethnological and anthropological value.

PSYCHOSIS IN PERNICIOUS ANAEMIA.

By L. H. TAN, Central Hospital, Patavia.

Psychosis and subacute combined degeneration of the spinal cord are two know complications of pernicious anaemia, a rare disease in the tropics.

The patient whom I am going to discuss was a European woman of 40. She began to show delusions of persecution 2 years ago. She was then working in military hospital and had to come up as a witness in a theft case, which happened in the hospital. After that incident she acquired a morbid idea that the persons who were involved in the case, always followed her and tried to murder her. Then she was taken ill, thinking that she was poisoned. She suffered from dizziness confusion. During the period of 2 years these symptoms varied.

One day she was brought up to our psychiatry clinic in a desperate and furious condition. She looked round anxiously and tried to catch hold of anyone that came near her. She did not recognise her surroundings any longer (disorientation). She also showed symptoms of exhibitionism. The above clinical picture shows distinct amentia. But peculiar enough hat the next moment she became sensible again and her orientation became normal. Her mood was one of apathic depressive, while conciousness was much above the amentia stage and yet below normal (the Germans call it Benommenheit). In the next few days the psychical picture became one of aggresiveness. She tried to beat any one near by. She was artificially fed.

We have here, therefore, a woman, who was mentally fit up to the age of 38, and who at this age began to show form gradually delusions of persecution and to sufter from mental confusion. During this phase, which lasted for two years she still could get on with the community, until suddenly she came to a serious condition of desperation and fury.

We see here changing symptoms of (*a*) period of amentia—disorientation, desparation and fear—(*b*) period of the so-called Benommemheit with apathic depressive mood and vague delusions of persecution, and (*c*) period of irritability and aggressiveness.

This kind of psychosis which hardly belongs to any particular disease in psychiatry points to the presence of a toxic factor, and a careful physical examination immediately leads us in the right direction.

Patient had a yellowish pale appearance. Her tongue was red and somewhat atrophied. The heart was not enlarged but haemic murmurs were heard in he pulmonary and mitral areas. The spleen and liver were palpable. All reflexes were normal.

The blood picture was as follows:—

Haemoglobin 30%; Erythrocytes 1,550,000;

Leucocytes 2900; Eosinophils 0%; Polymorphs 79%;

Band-forms 7%; Monocytes 0.5%

47% of the erythrocytes were normoblasts. There were poikilocytosis, anisocytosis, and marked macrocytosis.

Two megaloblasts were found.

The blood sedimentation test was:—after 1 hour 35.
after 2 hours 70.
after 24 hours 150.

W. R. of the blood and cerebro-spinal fluid were negative.

Van den Bergh test: indirect +; billrubin serum 24E.

We have thus here to do with a case of pernicious anaemia, and the question is whether there is a relation between this disease and the accompanying psychosis. Psychosis in pernicious anaemia has been repeatedly described. The sympatoms of this psychosis have few characteristic points. They may be devided into two groups:—(*a*) explosive-depressive alternating with atitude of indifference. (*b*) manic excitement, dreamy situation, amentia and paranoid pictures. Dementia of light degree is always present.

In our case, to answer the question whether there exists a relation between the psychosis and the pernicious anaemia, we have to rely on the effect of therapy.

The patient was treated for a month with liver injections, and hand in hand with the improvement of the blood picture, the psychic condition also improved.

A month after the beginning of the treatment, the blood picture was as follows:

Haemoglobin 50%; Erythrocytes 3,450,000; Leucocytes 6,400.

Eosinophils 5%; Lymphocytes 40%; Polymorphs 46 per cent. Band-Forms 9 per cent.

Megaloblasts and normoblosts disappeared. Weight increased from 44 to 47 kilograms.

In the first week of the treatment, patient began to be restful in the daytime, though at night frequently there were still attacks of fury, which, however, diminished gradually and after a month's time all mental disorders disappeared. She could again get on with the society as a normal individual.

Although here there is a sure relation between the psychosis and the pernicious anaemia, we must not too readily come to a conclusion that the pernicious anaemia has caused the psychosis. In the first place she may have a pre-psychotic personality.

We can see here an analogy with the subacute combined degeneration of the spinal cord that often accompanies pernicious anaemia, with the difference that here herapy gives no improvement. Probably we have here an irreversible degeneration of the white matter of the cord caused by local fooding defficiency as a direct result of the qualitative changes in the blood.

Recent publications have shown that a long continued organ therapy (liver, stomach) with acidol-pepsin sometime leads to complete or partial cure.

The importance of the case described lies in the fact that in every case of psychosis a thorough psychical and physical examination should be performed. Without this performance our patient, who is a normal individual, might have been sent to a lunatic asylum and doomed for her life-time.

ABSENCE OF COMMON CAROTID PULSATION OPPOSITE A NON-MALIGNANT THYREOID TUMOUR.

W. H., Male, Age 58, Chinese U.S.C. No. 155/34.

Patient was first seen in the Out-Patient Department with a large thyreoid tumour affecting chiefly the right lobe. He first noticed a swelling in the right side of his neck 4 years before. This enlarged slowly and caused him some pressure discomfort, and until 2 weeks prior to admission was about 4" diameter in size, when he noticed that it suddenly began enlarging rapidly, causing him some dyspnoea and discomfort, some hoarseness of voice, slight headache and general malaise. It then measured about 9" across and 4½" vertically, with enlarged superficial veins running downwards. He had no history of trauma. It was stony hard to the touch, and no carotid pulsation could be felt. There were no enlarged glands. The vocal cords were unimpaired although the larynx was displaced very much to the left and somewhat posteriorly. The general condition was fair. The hardness, the recent rapid increase in size, the age of the patient and chiefly the fact that the line of carotid pulsation was interrupted opposite the broadest part of the tumour induced us to diagnose the tumour as malignant. Operation seemed fruitless, so radium was tried instead, and 20 two milligram needles on paste were applied at 1 cm. distance from the skin all around the tumour for 286.5 hours in May 1934, a total dose of 11,460 milligram-hours being given. Barring some itching of the skin over the tumour, the patient had no untoward symptoms during irradiation. He felt better after irradiation, as the tumour seemed to have decreased in size. A week after irradiation, a definite reduction in size could be noticed, the measurement then being about 1" less, and the dyspnoea was also less. The skin was darker, but the erythema so often seen after irradiation was, fortunately, absent. The swelling then became softer and some fluctuation seemed present. This raised the question of it being cystic, a suspicion which was entertained before, but which was discarded in favour of malignancy as mentioned above. A month later, a definite decrease in size of about 2" occurred, and as the skin appeared healthy, an operation was advised. Under the influence of sodium amytal orally, the tumour was removed under local anaesthesia, Barker's Solution (0.2% benzylamine lactate) being used. The connective tissue around was rather adherent perhaps largely due to the effects of radium. The tumour was found to be a large cyst with a moderately thick wall containing about one pint of old blood and grumous material, and another smaller portion containing much colloid material. The cyst at one point wrapped around the carotid sheath so as to lie well posterior and lateral as well as anterior to the common carotid artery. (Diagram 1). The case was clearly a cyst-adenoma with sudden haemorrhage into it. The histological section of

the wall returned was that of a cyst-adenoma. The patient made an uneventful recovery and was discharged nine days after the operation.

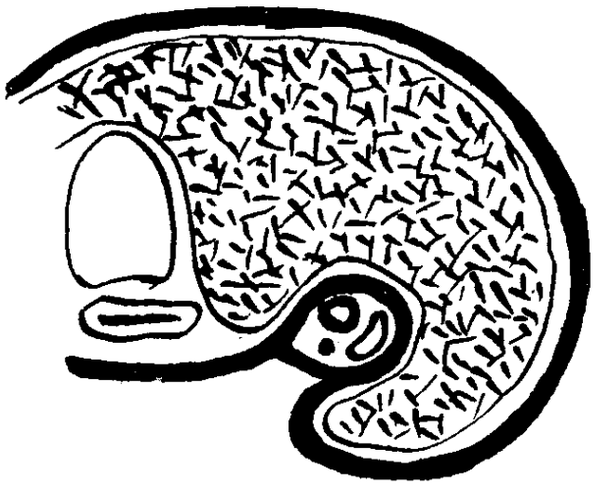

Diagram 1.

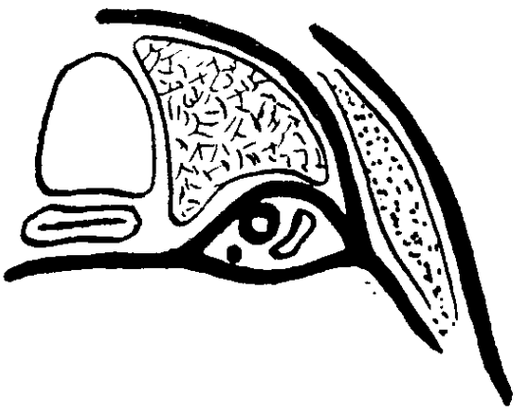

Diagram 2.

This case is recorded to show that reliance cannot always be placed on the absence of carotid pulastion as indicative of malignancy in a thyreoid tumour. The thyreoid gland lies in the middle fascial compartment of the neck and the pretracheal and prevertebral layers bounding this compartment meet laterally at the carotid sheath. (Diagram 2). As a consequence of this innocent tumours of the thyreoid may be expected to push the carotid sheath laterally and posteriorly but not to overlap it. (Diagram 3). Hence the usual

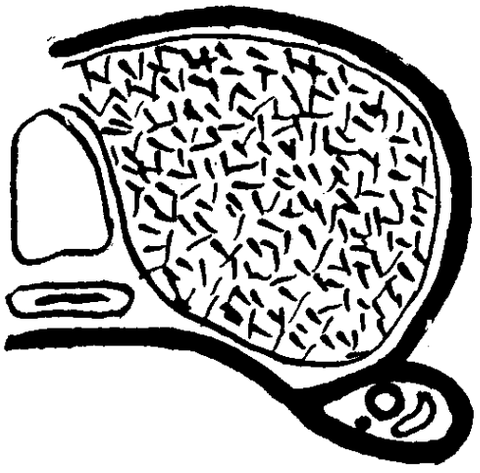

Diagram 3.

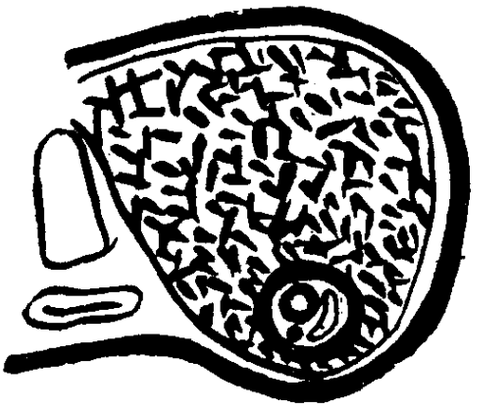

Diagram 4.

teaching that in innocent thyroid tumours the pulsation of the common carotid artery may be traced all the way up the back of the tumour however large it may be. Malignant growths infiltrate beyond the fascial limits of the middle compartments of the neck and in time the pulsation of the common carotid appears more or less interrupted in the region of the tumour. (Diagram 4).

The case recorded here was an exception to the first part of the above. One must imagine that the pretracheal fascia was so stretched as to wrap itself round the common carotid artery. (Diagram 1).

Thanks are due to Prof. K. H. Digby for his help in preparing this note and for his permission to record this case.

F. Y. KHOO, M.B. B.S.

Hong Kong University Surgical Clinic.

Review of Books

"The Essentials of Histology, Descriptive and Practical for the use of Students." By Sir E. Sharpey-Shafer, F.R.S., Thirteen edition. Edited by H. M. Carleton, M.A., B.Sc., D.Phil. Pp. x x 618, with 721 Illustrations, Lonon, Longmans, Green and Co. Ltd. 1934. Price 15s. net.

The first edition of this well known text-book was published in 1885. All succeeding editions until this one have been edited solely by the original author, Sir Sharpey-Shafer who has recently retired from the chair of Physiology at Edinburgh after a long and distinguished career. The revision of this edition of his histological text-book has been deputed to Dr. H. M. Carleton, the lecturer on histology at Oxford. The size of the book has been increased and includes new sections on the development of blood corpuscles and on ossification. Many new illustrations have been added, including numerous excellent micro-photographs.

The book continues to deserve the reputation previous editions have won for it among students of medicine and their teachers. It also serves as a reference book of considerable value to senior students and graduates who have occasion, as it is to be hoped most of them do, to revise or extend their knowledge of histological love.

This volume then, is a better edition of an excellent text-book, which needs no further recommendation.

L. J. D.

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