1900, to January, 1902, except for six weeks in the summer, namely, during the hottest weather (July and August), and he omitted it then because wearing the linen skull cap, which I recommended him, made his head uncomfortably hot, at all events at night.

In using the ointment he has persistently done as directed—that is to say, he divided the hairy scalp into eight imaginary divisions or regions, and has applied the ointment to one of these divisions on one day, to another the next day, thus going over the whole of the hairy scalp in a period of eight days, and then reverting to the division or region first treated, and so on so that the whole of the scalp in this manner had ointment applied to it once in eight days with undeviating regularity. This ointment, of course, produced a sensation of heat with redness of the skin, followed by desquamation, so that during the whole period of treatment some part of his head was tender. He was not directed to have any internal treatment, but only ointment.

It was quite six months (after I first saw him in December, 1900) before any hair showed itself—that is, before he saw any difference at all, and his hair has only made anything like the show it now makes about three months ago—that is, dating back from January 28th, 1902.

January 28th, 1902. Now his head is patchily furnished with dark brown hair, some of which is quite 3 in. long—namely, as seen in the illustration. His hair is about the same on both sides of the head. It may be said that pretty well half the total superficies of the scalp is furnished with dark brown hair, and that at no part is the scalp absolutely hairless. His

eyebrows and eyelashes, which when he was first seen by me were entirely wanting, are now complete, although somewhat scanty in general appearance. The hair of his face is more abundant, although, because of his shaving his whiskers and beard, the condition of these does not show in the illustration.

THE CAUSATION OF DEATH DURING THE ADMINISTRATION OF CHLOROFORM.

By E. H. EMBLEY, M.D.,

Honorary Anaesthetist to the Melbourne Hospital.

(From the Physiological Laboratory of the University of Melbourne.)

(Continued from page 821.)

II.—VAGUS INHIBITION AS A CAUSE OF CIRCULATORY FAILURE.

As experience showed that circulatory failure was most prone to occur in the early stage of chloroform administration, it was essential to be able to study the phenomena occurring during this period. To attain this end two methods were employed:

(a) Connecting the carotid artery with the manometer under morphine anaesthesia only. The air containing the chloroform was administered through a trachea tube, and therefore did not come into contact with the nose or larynx.

(b) Preparing the dog for connexion with the manometer under ether anaesthesia eight or ten hours previously, so that, when the effects of the anaesthetic had passed off, the carotid artery had only to be connected with the manometer. By this method the nervous system of the dog was in its normal condition when the record of the induction was started.

(1) Results of Experiments with Morphinized Dogs.

One hundred and twenty experiments were performed. It was found that when air containing more than 2 per cent. of chloroform vapour was administered, slowing of the heart ensued, and that when higher percentages were employed the degree of the inhibition was rapidly intensified.

The slowing of the heart was accompanied by a steady fall of blood pressure, which rose rapidly on section of the vagi. (See Tracing 2, pp. 886-887).

When the percentage was raised to 2.5 per cent. the inhibition of the heart was so marked as frequently to stop it suddenly and completely. With this percentage strength, however, the heart always recommenced.

With 3 per cent. or upwards the recovery of the animal was not constant, for the inhibition lasted several minutes (as long as seven and a-half minutes), and although the heart escaped ultimately to some extent, in these prolonged inhibitions such escape was often too late. The circulatory system was too nearly paralysed by chloroform and the asphyxia consequent upon cessation of circulation to admit of recovery being possible. Artificial respiration was maintained till after the heart had permanently ceased. (See Tracings 3, pp. 888, 889, and 4, p. 890).

This slowing or cessation of the heart beat was due to stimuli passing down the vagi, for:

(a) It never occurred in animals in which the vagi had been divided.

(b) When occurring in animals with intact vagi, it immediately disappeared upon section of both of them. (See Tracings 2 and 4).

In non-fatal cases, when the heart recovered from the inhibition, the blood pressure usually rose rapidly, the pulse often being quick as though the vagi had been cut.

The possibility of the heart's recovery appears to depend upon the extent of the damage that it has sustained from the chloroform ere the inhibition came on.

The effects of a persistent vagus arrest of the circulation is seen in Tracing 3. In this experiment, in which artificial respiration was employed throughout, it is seen that the vagi have arrested the heart's action at a time when the blood pressure has been brought down to 40 mm. of mercury pressure by the chloroform. The vagal inhibition was fatal. The

Fig. 2.

rise of pressure in the first temporary escapes of the heart shows the asphyxia consequent on cessation of circulation has brought on vaso-constriction. The vasomotor system was obviously not paralysed when syncope occurred. The vagus control was exhausted after 6 min. 45 sec., but the circulatory mechanism was by this time so much impaired that the heart did not recover.

The following is an analysis of 54 cases in which vagus inhibition was sufficiently pronounced to stop the heart; 2.3 per cent. and upwards of chloroform vapour in the air was employed. The proportion of fatal to non-fatal administrations was:

	Non-Fatal.	Fatal.	Total.
With artificial respiration... ..	18	8	26
With natural respiration	4	24	28
	22	32	54

In the twenty-six experiments with artificial respiration this was performed throughout the inhibition period also, so that the animals were getting chloroformed air throughout.

Fig. 2 (Continued.)

In the natural respiration experiments no artificial respiration was performed when the respiration ceased. In the four recoveries the blood pressure rose in consequence of the exhaustion of the vagi and the asphyxial rise. As the blood pressure rose the respiration returned.

Experiments with the Vagi Previously Divided or in which these Nerves were Cut when Slowing of the Heart Ensued.

Forty-nine experiments were made with the vagi cut. It was found that sudden circulatory failure never occurred from inhalation of chloroform after the vagi had been divided. The respiration ultimately ceased, and this was followed by no

sudden fall of blood pressure. A rise in pressure usually occurred from vaso-constriction due to asphyxia.

In other experiments the vagi were divided after the administration of chloroform had brought on arrest or slowing of the heart. The consequence was the same in every instance: the heart recommenced and the blood pressure rose at once. Tracing 4 shows the type of blood-pressure curve obtained by such experiment.

Intravenous injection of atropine before the administration of chloroform began was found to have the same effect in preventing inhibition of the heart as section of the vagi. Absolute arrest of the heart by vagus inhibition in mor-

phnized dogs could be obtained at will by causing them to inhale 4 per cent. chloroform vapour in the air, and the heart could be released and the circulation restored by vagotomy, providing there was not too long a delay before the section of the vagi.

The results of six experiments in which the same strength of chloroform vapour in air was administered by artificial respiration, first with the vagi intact, and subsequently with the vagi cut, are collected below in tabular form. It contrasts the interval before circulatory failure ensued under these two conditions.

Experiment	Percentage of Strength of Chloroform used.	Blood Pressure before the Administration began.	Time of Administration to cause Syncope.	Blood Pressure when Syncope occurred.	Recovered by Vagotomy	Blood Pressure upon Readministration.	Duration of Administration before Death occurred.
101	4%	120 mm.	2' 25"	40 mm.	—	140 mm.	20'
" 140	4%	130 mm.	2' 15"	40 mm.	—	140 mm.	13' 45"
" 149	3.5%	120 mm.	2' 30"	40 mm.	—	130 mm.	30' 20"
" 162	3%	130 mm.	3' 10"	30 mm.	—	130 mm.	13' 15"
" 181	3.8%	140 mm.	2' 54"	56 mm.	—	160 mm.	28' 44"
" 202	4.2%	160 mm.	3' 30"	20 mm.	—	170 mm.	15' 20"

In some experiments the vagi have been found to completely arrest the heart in as short a period as 1 min. 40 sec. In morphinized dogs the vagi are capable of arresting the heart, upon raising the strength of chloroform vapour to 4 per cent. in the air inhaled, after 39 min. 30 sec. of light anaesthesia.

It has been stated by the Hyderabad Commission,⁸ MacWilliam,⁷ Wood,⁹ and Richet,¹⁰ that it is impossible to permanently arrest a chloroformed heart by vagus excitation. This is absolutely opposed to my experience. I have never succeeded in doing this with a heart that has not been

chloroformed; but, on the other hand, I have never failed (5 cases) to occasion fatal syncope in a sufficiently chloroformed animal by excitation of one or both vagi with the interrupted current.

Upon faradizing one vagus only without chloroform, the inhibition usually lasted about sixty seconds, and when both nerves were stimulated simultaneously the inhibition lasted about 100 seconds. Recovery was complete, notwithstanding continued stimulation of both vagi. If chloroform was then administered until the blood pressure had fallen to 40 mm. of mercury, and then both vagi faradized with a stimulus of the same strength as before, the heart was always completely and permanently arrested. There was no escape. Artificial respiration was maintained throughout in these experiments.

Tracing 5 (p. 890) furnishes further evidence of the necessary conditions of fatal inhibition. In this experiment chloroform was administered until the blood pressure fell to 80 mm. of mercury pressure, then the right vagus was faradized and the heart arrested. The heart escaped from the inhibition (chloroform was being administered by artificial respiration meanwhile). When the blood pressure had fallen to 40 mm. mercury pressure, the peripheral ends of both vagi were stimulated and the heart arrested permanently.

(B).—RESULTS OF EXPERIMENTS WITH DOGS NOT MORPHINIZED.

The dog's carotid was connected with the manometer by a thick-walled flexible tube. The animal was supported in the air by an assistant, so that struggling might not kink the tubes connecting it with the manometer and the respiration tambour.

Twelve experiments were performed, of which Tracing 6 (p. 892) is typical.

The amount of chloroform administered in this experiment was uncertain, as it was given on a cloth, but it was considerable. Thirty seconds after the chloroform was given the pulse-rate was greatly diminished, and after one minute the pulse became intermittent, with short stoppages and slowing, due probably to reflex inhibition. The longer intermissions were followed by struggling, which resulted in a rise of blood pressure. Following this rise the blood pressure began to permanently fall with a rapid heart beat free from vagus

Fig. 3(Continued.)

control. After two and a-half minutes the anaesthetic was discontinued.

Tracing 7 (p. 891) is another experiment of the same kind. It will be seen that the excitement, etc., of the induction has resulted in a great increase in the pulse-rate, which was maintained till the blood pressure began to fall, when the inhibitory action began to come on, causing slowing of the heart-rate. This slowing increased till the heart's action was suddenly arrested. This occurred after three minutes thirty-five seconds of the administration, when the blood pressure was at 25 mm. of mercury. It will be noticed that the heart was arrested fifteen seconds before the respiration ceased. The respiration became deeper and more gasping with an expiratory pause after the inhibition occurred. The heart was arrested for one minute thirteen seconds, after which the inhibitory mechanism failed and the heart recommenced. The heart was able to raise the blood pressure to 40 mm. of mercury, but this rise was unsustained, and the blood pressure gradually fell to zero.

THE CAUSE OF VAGAL INHIBITION IN CHLOROFORM ANAESTHESIA.

Inhibition of the heart might be brought about by chloroform inhalation:

1. As a reflex excitation by the chloroform vapour of the sensory terminals in the respiratory passages—nose, larynx, and lung.

2. By the chloroform in the blood occasioning a preliminary increase in the excitability of the vagus mechanism, so that the usual control exercised by the vagi is increased.

It is conceivable that the paralytic effect of chloroform upon cardiac muscle, which has been shown to be so considerable, is such that stimuli reaching the heart through the vagi are more effective than in the case of a heart not poisoned by chloroform.

Reflex inhibition of the heart from the stimulus of strong chloroform vapour upon the nasal mucous membrane, the larynx, and lungs, has been shown to occur by Dastre and Morat,²¹ Morat and Doyen,¹¹ and by Brodie and Russell.¹⁷ In most of my experiments the chloroform was delivered through a tracheal tube, so that it did not come into contact with the nose or larynx.

To ascertain whether reflexes from the lower respiratory

passages or the alveoli of the lungs were alone responsible for cardiac inhibition, which I have shown to be such a constant occurrence in the administration of strong chloroform vapour to dogs, two series of experiments were performed.

1. Chloroform was administered by inhalation to a morphinized dog through a tube secured low down in the trachea. The vagi were then divided below the heart branches. The pulmonary branches being thus eliminated, all sources of reflex from the lungs and respiratory passages were cut out. Upon the administration of chloroform the heart was inhibited in seven out of ten experiments. The inhibition was fatal!

2. Slowly injecting normal saline containing 0.7 per cent. (by weight) chloroform at a temperature at 40° C. into a vein in continuity. The 0.7 per cent. chloroform solution was injected from a pressure bottle slowly and continuously into the external jugular vein (in continuity) of a morphinized dog.

The resulting records were close representations of blood-pressure tracings from chloroform inhalation experiments under the same conditions.

Six experiments were performed. The blood pressure in each experiment fell slowly and regularly. When the pressure had fallen to an average of 45 mm. of mercury pressure, the heart was arrested and blood pressure fell to zero. When section of the vagi was performed the heart always recovered.

In all my experiments the *dangerous* stoppage of the heart by the inhibitory mechanism, occurred, not at the very commencement of administration, but after the effect of chloroform upon the cardiac muscle had been sufficient to reduce the pressure to about 40 to 50 mm. of mercury pressure. Extreme slowing or arrest of the heart came on sooner and with higher blood pressure the stronger the tension of chloroform in the air breathed.

In those experiments in which no morphine was given, but in which the animal was prepared eight or ten hours before under ether, intermissions and slowing occur quite early. The heart would suddenly stop and blood pressure fall, upon which struggles ensued with a great rise of blood pressure, followed again and again by further intermissions (see Tracing 6).

Whether the inhibition was in this case brought about re-

Fig. 4.

Fig. 5.

Fig 2

flexly from the nasal or respiratory surfaces I am unable to say, but it is very probable. The later and often fatal inhibitions are, from the evidence adduced above, apparently not reflex, but appear to be caused by an increased excitability of vagus nervous mechanism acting upon a heart, the spontaneous excitability of which is diminished by the action of chloroform. When the heart is poisoned by chloroform it undoubtedly has less power of escape from the restraining influence of these nerves, for, as mentioned above, stimulation of both vagi never failed permanently to arrest a chloroformed heart.

Summary of Conclusions of the Effect of Chloroform upon the Cardiac Inhibitory Mechanism.

It was soon found during the course of the work that the sensitiveness of the inhibitory mechanism varied greatly with the methods employed for preparing the animals for connexion with the manometer, etc., and that if the animal had been anaesthetized by chloroform without morphine in order to perform the surgical work, normal vagus control was more or less completely abolished by the time the record was started. On the other hand, when morphine alone was used prior to the establishment of the connexion with the manometer, the vagus control was found to be unimpaired.

One hundred and twenty experiments were made to ascertain the part played by vagus inhibition in chloroform poisoning. In 54 cases vagus inhibition embarrassed the circulation to a more or less dangerous extent, and in 33 experiments was the immediate cause of death. To sum up:

1. A heart which has been poisoned by inhalations of chloroform of strength 2 per cent. and upwards can always be permanently inhibited by stimulation of the vagi with the faradic current when the blood pressure has fallen to about 40 to 50 mm. of mercury pressure.

2. Chloroform raises the excitability of the vagus mechanism, particularly in the early part of the administration.

3. The increased excitability of the vagus mechanism is due to the action of chloroform on the vagus centres, and the inhibitory action is more intense from being exercised upon a heart whose spontaneous excitability is diminished by the action of the chloroform upon it.

4. Chloroform administered to morphinized dogs in air containing not more than 1.5 per cent. of the vapour, after a period of mild excitation, slowly depresses vagus excitability. The excitability may again be raised with more or less readiness according to the duration of the administration and the endurance of the vagi, by increasing the percentage strength of the chloroform, or by asphyxia.

5. Vagus inhibition is, in dogs, the great factor in the causation of sudden death from chloroform.

6. Dangerous inhibition is liable to occur whenever the strength of chloroform in the air inhaled rises above 2 per cent.

EXPLANATION OF TRACINGS.

Tracing 2.—(Effects of slowing of heart-rate by vagus action.) Blood-pressure tracing from Von Frey manometer (1 mm. equals 4 mm. mercury pressure). Dog, weight 16 lbs. Morphine, 0.25 gram. Artificial respiration. Time line, 3 mm. below zero. Chloroform vapour, 2.2 per cent. in the air administered throughout the whole period of the tracing. 5', 5', 5', 15' indicate the minutes the recording drum was stopped at intervals. Circulation nearly failed at 25 min. 50 sec. of administration. The vagi were then divided at *, whereupon blood pressure recovered, and the heart continued for 69 min. 30 sec. longer.

Tracing 3.—(Vagal effects.) Dog, 24 lb. weight. Morphine, 0.5 gram. Artificial respiration throughout. Blood-pressure tracing. Blood pressure, 120 mm. mercury at starting. Five per cent. chloroform vapour in the air inspired at "on" and continued to "off." The vagi arrested the heart in 2 min. 49 sec. when the blood pressure was equal to 58 mm. of mercury pressure. The inhibition lasted 2 min., then the heart was released for 22 sec. and the blood pressure was higher than at the start of the inhibition, that is, to 92 mm. mercury pressure. The heart was then again inhibited for 2 min. and again released, the blood pressure only rising to 60 mm. of Hg. A third time the heart was inhibited for 1 min., the blood pressure rising to 52 mm. of mercury on release. A fourth time the heart was inhibited for 45 sec. On release, the blood pressure rose to 40 mm. mercury pressure. The blood pressure was at zero in 13 min. 35 sec. after the first inhibition.

Tracing 4.—(Vagi cut after inhibition.) Dog, weight 16 lb. Morphine, 0.4 gram. Natural respiration. Blood-pressure tracing only. Chloroform, 6 per cent. in air administered. Chloroform started at "on" stopped when inhibition occurred. Gasping liberated the heart twice. Respiration failed after the inhibition occurred. Vagi were then cut. Respiration returned 20 sec. after the vagi were cut, being 190 sec. in abeyance. Blood pressure had risen again to 74 mm. of mercury pressure.

Tracing 5.—(Faradizing vagi.) Dog, weight 26 lb. Morphine, 0.5 gram. Artificial respiration. Blood-pressure tracing. Chloroform, 4.5 per cent. vapour in air administered throughout. Time line at zero pressure. Right vagus stimulated with blood pressure at

80 mm. mercury pressure, heart recovering. Both vagi stimulated, heart permanently arrested.

Tracing 6.—(Process of vagus exhaustion during induction with chloroform alone.) Dog, weight 24 lb. Blood-pressure record. Chloroform administered on a cloth. Period of administration indicated between vertical lines. Natural respiration. Dog was supported off the table to prevent kinking of manometer connections by the struggling. Struggling ensued upon the sudden short falls of blood pressure. Chloroform administration was stopped while corneal reflex was still present.

Tracing 7.—(Vagus inhibition in non-morphinized dog, including the induction period.) Weight 15 lb. Blood-pressure and respiration tracing. Chloroform administered on a towel from "started" till cessation of respiration. Much struggling with intermittent heart beats in early part of induction. Respiration continued 15 sec. after the heart was arrested.

(To be continued.)

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

URTICARIA ACUTA.

At this season the young stalks of the rhubarb plant are in very general use for tarts and other dishes. It is common knowledge that the ingestion of large or even moderate quantities of this vegetable in susceptible persons will give rise to severe attacks of intestinal colic; but I am not aware that any skin rashes have been described as due to this form of toxæmia. Two cases of urticaria acuta in children have recently been under my observation, which certainly point in that direction. In both the type was severe and lasted three or four days. The convex elevations, with their pink areolæ, were scattered all over the body, but chiefly on the limbs. The irritation caused by the rash was intense. Both the children had marked facial erythema, with a decided rise of temperature on the third day, and in each the distressing symptoms were promptly relieved by a full dose of castor oil, after other more elegant but less efficient aperients had failed to sweep away the irritant from the alimentary canal. Careful inquiries into previous errors of diet and other known causes of indirect irritation met with a negative reply; but it has been since ascertained that the nurse in each instance has admitted giving the children some stewed rhubarb on the day previous to the attacks. An examination of the urine for albumen was made in both the cases without result, but not for any excess of oxalates, which would be suggested on another occasion.

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KAFFIR THERAPEUTICS.

On reading in the *BRITISH MEDICAL JOURNAL* of November 23rd, 1901, page 1529, Dr. W. A. Densham's letter on "Kaffir Treatment of Gunshot Wounds," I thought a few notes I have made on native treatment of disease might prove interesting. The observations extend over five years' residence in Rhodesia, and for the greater part of the time in close relationship with native life.

CASE I. *Pneumonia*.—The witch doctor treated this by making incisions on the outer sides of the calves, and inserting the tips of cows' horns, about 2 inches long; there were three stuck in each leg. The patient died from blood poisoning.

CASE II. *Wound of Palm*.—I saw this, caused by a native axe, about ten days after the injury. The hand had been dressed by a Mashona with a plug of cowdung. The hand was swollen, and pitted on pressure; and, on removing the cowdung plug, the blood squirted out in jets, showing that most probably the palmar arch had been injured. I replugged with boracic lint, gave chloroform, and then enlarged the wound, endeavouring to find the wounded artery; I was unable to do so, and therefore tied the brachial. I cleaned the wound of the hand thoroughly, dressed it antiseptically, and the patient recovered rapidly, with a perfectly useful hand.

CASE III. *Droopy of Legs and Feet*.—Here incisions were made in both legs, and goats' horns inserted, having had the tips sawn off; these seemed to act fairly well as drainage.

Cowdung is also used for "smearing out" (applied like paint) the mud floors and walls of huts. This is done weekly and is very beneficial, as it destroys the fleas and other pests that much abound.

A bracelet or anklet made from the hair of a cow's tail is an infallible Mashona remedy for rheumatism. The operculum

of a shell to be found only on the Zambesi is worn by those natives who can afford them as a charm against disease and death. It is almost impossible to buy these charms. I offered a Shuna a sovereign for one the other day, and gradually increased the offer to two pounds, but it was ineffectual. I ascertained the history, and though the native had inherited it from his father who had died, the confidence of the son in its virtues was not lessened.

The Dutch settlers in Rhodesia are not much more advanced in medical knowledge than their Mashona brethren, inasmuch as they give a decoction of goat's dung in water as an internal remedy in any abdominal complaint from stomachache to peritonitis. Also in convulsions they give children blood from a dog's ear; this is caught fresh in a spoon, and the unfortunate youngster has to drink it, willy-nilly. For bronchitis, pneumonia, and kindred complaints they kill and skin a goat, often in the bedroom of the patient, and clap the hot skin on the inflamed part; this is left on about half an hour, and the hair or wool on the skin is pulled; if this comes off easily another unfortunate animal is killed and the fresh skin applied, and so on until the hair remains firm on the skin after it has been applied to the sick person. Fresh cowdung is universally used amongst the Dutch as a poultice to "draw" an abscess.

These are absolute facts.

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TORTUOSITY OF INTERNAL CAROTID ARTERIES.

SEVERAL papers have appeared in the *BRITISH MEDICAL JOURNAL* of late describing the above condition. I may state that tortuosity of these vessels is of not infrequent occurrence in the subjects examined in the anatomy department of Glasgow University. We find, as Dr. Moorhead has stated, that a condition of atheroma or arterio-sclerosis of the vessel wall is very frequently present.

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MIXED SCARLET FEVER AND MEASLES INFECTION.—INSTANCES of mixed infection—scarlet fever and measles—perhaps sufficiently rare to make the record of the following cases interesting.

Mrs. B. has three children; a girl, M., aged 8 years, and two boys, A. and J., aged 6 and 4 years respectively. The girl and the elder boy are at school, while the younger boy is at home.

On January 18th, 1902, the two elder children became suddenly sick and vomited, and, according to their mother, were "feverish" then and on the 19th, when the girl complained of sore throat. Mrs. B. then examined her daughter's skin, and found a slight rash on the chest and back. On the 20th the boy A. appeared all right, and was sent back to school; but, as the girl was no better, I was called in. On examining M. I found her temperature to be 102° F. She had a typical sore throat and tongue; her skin was dry, and covered on the chest, abdomen, back, and limbs, with a well-marked scarlet fever rash. I had no doubt in my mind as to the diagnosis, and at once reported the case as scarlet fever, and ordered the brother A. to be brought back from school, and kept under observation. My next visit was paid on January 22nd, when I found the lad A. to be suffering from a typical attack of measles, the measles rash having first shown itself on the 21st. I was also surprised to find the girl M. had now all the symptoms of measles (her measles rash also appeared on the 21st) in addition to those of scarlet fever. Her attack of measles was quite as typical as her scarlet fever, the measles rash on the face being unmistakable, as were also the cough, coryza, etc. On the body the two rashes were commingled, so that the measles papules were there somewhat lighter in colour than usual, and tended to be confluent in patches.

The younger boy, J., had slept with his sister on the nights of January 18th and 19th, and thus constituted himself an involuntary control experiment. Accordingly, as I was prepared to find, he became suddenly ill on January 23rd, and when I saw him on the 24th he had all the symptoms of scarlet fever, including a very definite rash. It only remained for him to develop a measles rash on February 1st to make my case complete. This he did in due time. His attack of,