THE STAPHYLOCOCCUS AUREUS AS THE PROBABLE CAUSE OF A FATAL DISEASE SIMULATING LARYNGEAL DIPHTHERIA.

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THE following notes of four cases which clinically resembled laryngeal diphtheria, are recorded as being of interest owing to the evidence pointing to the *Staphylococcus aureus* as the ætiological agent. These cases occurred in a district in which diphtheria was prevalent. The evidence in favour of primary laryngeal diphtheria, that is, croup with gradually increasing obstruction, seems to be more than balanced by the evidence against it, the absence of response to injections of diphtheria antitoxic serum and the absence of visible membrane in the trachea. Further, the higher temperature recorded, the repeated failures to find *Bacillus diphtheriæ* in cultures from the throats and material from tracheotomy tube, the repeated findings of *Staphylococcus aureus* in these cultures and the culturing of the same organism from the patch of bronchopneumonia and the pericardial fluid at *post mortem* examination strongly support the view that the conditions were primarily an infection of the larynx with highly virulent and highly toxigenic staphylococci.

Inquiries have been made of medical practitioners in surrounding towns on the Darling Downs as to whether similar cases have been met with recently. From the replies received it is evident that there has been a number of cases in the district. They have resembled those described above, in that there was no evidence of membrane formation in the throat or trachea or of *Bacillus diphtheriæ* on culture, no response to diphtheria antitoxic serum. In twelve patients the gradually increasing obstruction rendered tracheotomy necessary and the temporary relief afforded was followed in nine by evidence of further obstruction lower down in the respiratory tract and death.

During July, 1929, three children of one family were affected in succession with an illness closely resembling laryngeal diphtheria. Two required tracheotomy and died, the third one recovered without tracheotomy. No *Bacilli diphtheriæ* were observed in cultures from these patients. The clinical histories were as follows:

CASE I. Male child, aged four years, was admitted to Toowoomba General Hospital at 3.20 in the afternoon of July 5, 1929. The temperature was 39·4° C. (103° F.), the pulse rate 160 and the respiratory rate 40. The child was well nourished and had no history of previous attacks of croup or of other illness. He had been croupy for two days. The child was very distressed and there was much laryngeal obstruction and recession. The colour was good. On admission he was given 20,000 units of diphtheria antitoxic serum subcutaneously and put in a steam tent. The signs of obstruction increased, with definite suprasternal, infrasternal and supraclavicular recession and cyanosis. Tracheotomy was performed at one o'clock in the morning of July 6, 1929. No definite evidence of membrane formation was observed in the trachea. Relief was obtained, but signs of obstruction again appeared; the child's condition became worse and death occurred a few hours later, apparently from suffocation and cardiac failure. No *Bacilli diphtheriæ* grew in the culture tubes inoculated with the throat secretion.

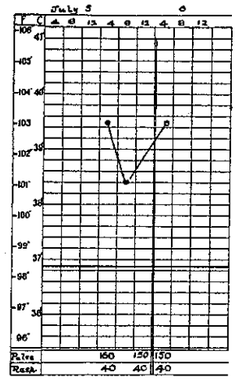

CHART I.

CASE II. Male child, aged two years, was admitted to Toowoomba General Hospital at three o'clock in the morning of July 7, 1929. The temperature was 37·6° C. (99·8° F.), the pulse rate 120 and the respiratory rate 30. The child was playing about at five o'clock in the afternoon of July 6, when it was noticed that his voice was a little hoarse. He awakened with croup at eleven o'clock. Medical advice was obtained and the child was given 5,000 units of diphtheria antitoxic serum and sent to hospital. The child was well nourished and there was no history of previous attacks of croup or other illness. At the time of admission the throat was clean, but there was some evidence of laryngeal obstruction. He was given 25,000 units of diphtheria antitoxic serum subcutaneously and put in a steam tent. The serum was repeated until in two days 60,000 units had been given. The child was fairly comfortable with less evidence of obstruction at eleven o'clock in the morning of July 7, but the evidence of obstruction increased and the child's condition gradually became worse; tracheotomy was performed at eleven at night. No diphtheritic membrane was observed in the trachea. The child was relieved at the time, but the relief did not last long. Attacks of acute obstruction with convulsive spasms occurred in the next two days and the child died early in the morning of July 10, 1929. Cultures from the throat on July 8 and 9 contained *Staphylococcus aureus*, but no *Bacilli diphtheriæ* were observed. Mucopurulent material unlike diphtheritic membrane was coughed through the tube on July 9. *Staphylococcus aureus* grew on culture, but no *Bacilli diphtheriæ*.

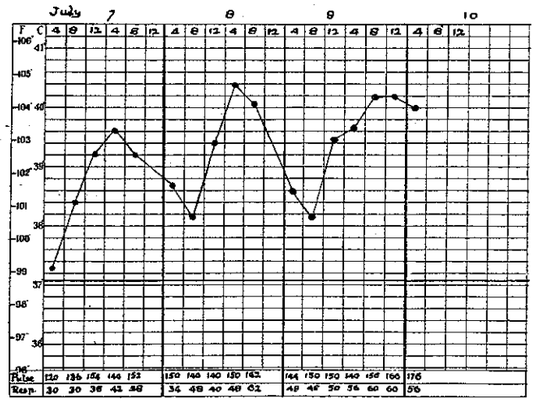

CHART II.

CASE III. Female child, aged six years, was under observation and although she was well, her throat was swabbed on July 8, 1929. Her throat was clean and no *Bacilli diphtheriæ* were recovered. She was admitted to Towoomba General Hospital at half past seven in the evening of July 10, 1929. Her temperature was 38·8° C. (102° F.), her pulse rate 144 and her respiratory rate 44. She had loss of voice and croup for twelve hours. Her throat was clean. She had slight evidence of laryngeal obstruction with recession. Her colour was good. She was well nourished and there was no history of previous attacks of croup. On admission 30,000 units of diphtheria antitoxic serum and ten cubic centimetres of antistaphylococcal serum were given subcutaneously. The same dose of antistaphylococcal serum was repeated on the morning of July 11. The child's condition was then much improved. The fauces were slightly inflamed, but no evidence of membrane formation was present. There was still some croup, but no evidence of respiratory embarrassment. The child's condition steadily improved and she was discharged relieved on July 17, 1929. Throat cultures on July 10, 1929, contained *Staphylococcus aureus*, but no *Bacillus diphtheriæ*. A culture tube inoculated with blood on July 11 remained sterile, while in culture tubes inoculated from the throat *Staphylococcus aureus* but no *Bacillus diphtheriæ* was grown.

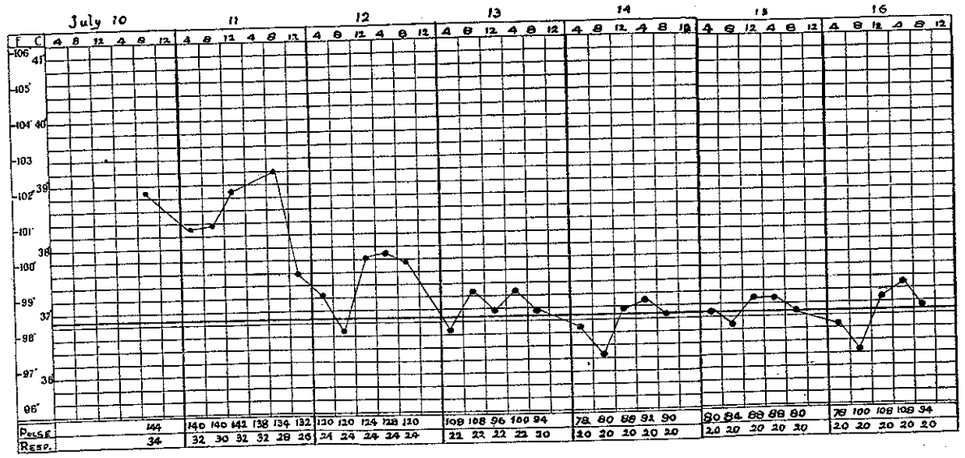

CHART III.

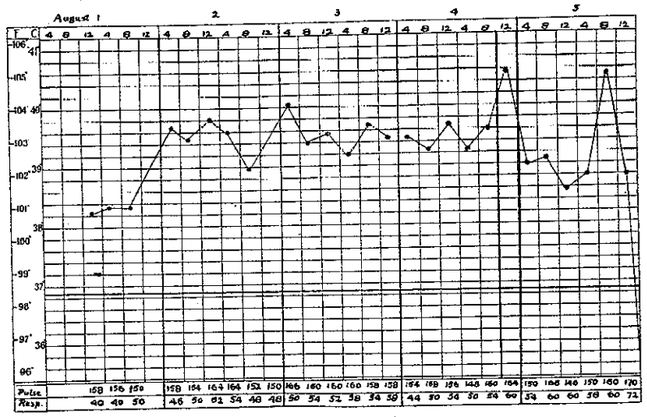

CHART IV.

On August 1, 1929, another child, not associated with the other three, was admitted to Toowoomba General Hospital in a condition similar to those just described. The clinical history was as follows: CASE IV. Male child, aged fifteen months, was admitted at half past one in the afternoon of August 1, 1929. His temperature was 38·1° C. (100·8° F.), his pulse rate was 158 and his respiratory rate was 40. He was well when he went to bed on July 31, 1929, but awakened at four o'clock in the morning with croup. He improved during the morning, but on admission to hospital there was a good deal of laryngeal obstruction and recession. The child was given 30,000 units of diphtheria antitoxic serum and twenty cubic centimetres of antistaphylococcal serum, both subcutaneously; he was put in a steam tent. The child became more distressed with definite evidence of obstruction, and at five o'clock in the afternoon tracheotomy was performed. No evidence of membrane was observed in the trachea. Relief was obtained for a few hours, when breathing again became obstructed. These attacks of obstruction with convulsive spasms, which were apparently relieved by cleaning the trachea with an oiled feather, came on at intervals until death occurred at six o'clock in the morning of August 7, 1929. Some bright blood was coughed up through the tube on the morning of August 4, 1929, and the tube was removed. On August 5 the child was breathing through the tracheotomy wound in the neck with a certain amount of difficulty and occasionally coughing up some purulent material of offensive odour. Ten cubic centimetres of antistaphylococcal serum were injected subcutaneously each day until August 5, 1929. Cultures from trachea immediately after tracheotomy had been performed on August 1, from the throat on August 2 and from material coughed through the tube on August 5 revealed *Staphylococcus aureus* only, no *Bacillus diphtheriæ* being observed.

The following observations were made at a *post mortem* examination of this child's body. It was

a well nourished, pot bellied male child. There was sero-purulent fluid evident round the tracheotomy wound in the neck.

On opening the peritoneal cavity the colon and loops of the small bowel bulged out and were much distended with gas, but were devoid of liquid or solid matter. No evidence of any bowel lesion was observed. The liver was dark red in colour and engorged. Blood dripped from the cut surface. The spleen was enlarged and engorged. No abnormality was observed in the stomach, kidneys, suprarenals, pancreas or bladder.

The pericardial sac contained half a teaspoonful of slightly turbid fluid from which a few colonies of *Staphylococcus aureus* were cultured. The right auricle and ventricle of the heart were distended with coagulated blood. On the surface of both lungs were areas in which the pleura was dark red in colour and mottled. These areas were situated at the base of the left upper lobe, at the base and lower half of the anterior surface of the left lower lobe, at the base and anterior surface of the right lower lobe, in a portion of the right middle lobe and at the base of the right upper lobe.

These areas when sectioned appeared to be confluent patches of bronchopneumonia producing a pseudolobar effect. Profuse growth of *Staphylococcus aureus* was obtained on culturing the purulent fluid expressed from one of these patches. The bronchial glands on both sides were enlarged and dark red in colour. The trachea and bronchi which contained some sero-purulent fluid, were dissected down to the bronchioles. No evidence of membrane formation or gross inflammatory changes were observed. The air passages above the tracheotomy wound were clear and clean.

I am indebted to the late Dr. Freshney, then Medical Superintendent, and the members of the honorary medical staff of the Toowoomba General Hospital and to the medical practitioners of the surrounding country towns for their assistance in collecting the information and for their permission to publish the notes of these cases.

COMPLICATION OF INJURIES ABOUT THE ELBOW.[1]

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I WISH to call your attention to the various complications that may be associated with injuries about the elbow since it has been my experience that they are not rare and it is common knowledge that some of them have grave consequences.

The series of thirteen cases I have tabulated is in no sense statistical, for I have no record of the total number of injuries about the elbow I have seen during the last two or three years, the period over which I commenced to collect the cases that form the basis of this communication. In all probability they would be found to constitute a very small percentage, but the fact that these complications exist at all, is sufficient justification to call our attention to them, to their gravity and to the best means to avoid and overcome them.

To analyse them in detail would require an amount of time which can ill be spared this evening and whilst I shall elaborate several of the case histories, I must content myself otherwise by drawing your attention to one or two general points which arise from their consideration. It will be noted that each main nerve trunk and the brachial or radial artery may be damaged and that all types of injury about the elbow region may produce the complications in question. All the injuries with two exceptions occurred in children and the parents of eight of the patients did not seek advice for the various disabilities arising from the elbow injury for periods varying from one to fifteen months. In four instances the complications were discovered very shortly after the injury, at periods ranging from fourteen hours to three days and in three of these patients the discovery predated any attempt at treatment by myself. In the fourth child the supracondylar fracture had already been reduced when she came under my care. In none of these four did the child concerned make any complaint likely to call attention to the complication existing. If there is one lesson to be learnt from studying these cases, it is the need of careful clinical examination and in these days of dependence on extraclinical methods for diagnosis, I have no hesitation in laying heavy stress on the necessity for making clinical examination our first line of attack in these as in all other fractures. The modern tendency to turn at once to radiography when faced with a fracture cannot, in my opinion, be too strongly condemned. I yield to no one in my appreciation of the value and importance of radiography, but I use it as a supplement to clinical methods.

It has to be remembered when dealing with children that the only subjective symptom with which the child concerns itself is pain. Loss of sensation and of muscular power, the presence of deformity, the diminution or cessation of the radial pulse are not to be detected by any dependence on the small patient's complaint.

Since all the grave complications following injury about this region exhibit all or some of these symptoms, it follows, if we are to detect them, that we must examine for them clinically. Radiograms will not yield evidence of injuries to nerves or vessels nor will they reveal the symptoms of such injuries. In tiny children it is not easy to elicit positive evidence of early nerve injuries and care and patience have to be used and frequent examinations made, if we are to detect these symptoms early. An examination of each radial pulse on the other hand is easily carried out and should never be omitted.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association in conjunction with the Section of Pædiatrics, the Section of Radiology and the Section of Orthopædics on July 25, 1929.

My own routine in dealing with all injuries about the elbow is to examine carefully first the hand and forearm for any signs of interference with any of the nerve trunks or vessels about the elbow. It is disquieting to find after an attempt at reduction that the radial pulse is absent or that there is loss of power in some of the hand muscles. To detect these signs beforehand is to arm oneself with a most valuable piece of knowledge in formulating treatment and prognosis.

Let me now quote three of the cases to illustrate this point.

One, a young boy, Case 11, presented with a supracondylar fracture with the usual backward displacement sustained some hours previously. Examination showed absence of radial pulse on the affected side, but no other sign of vascular disturbance and no sign of involvement of the median or other nerve trunk. His fracture was reduced under an anæsthetic, the forearm flexed on the arm and he was admitted to hospital for continuous observation over twenty-four hours. The reduction was completed without difficulty, no untoward symptoms followed and no radial pulse could be detected at the time of his discharge with complete function.

The most probable explanation is that the radial artery was absent on this side, but the early discovery saved much heart burning after reduction and made us carefully alive to the possibility of any permanent damage resulting from injury to the radial or brachial vessel.

Case 3 was that of a small girl seen by myself within three days of injury. She had had a classical supracondylar fracture which at the time I saw her had been fully reduced. Examination showed a diminished radial pulse and loss of power in the thenar group of muscles with anæsthesia in the median distribution. Treatment was at once commenced by splinting, since it was thought at the time that the condition was a median nerve injury mainly. The child developed within the course of several weeks typical signs of ischæmic palsy. These, however, ultimately cleared up very satisfactorily to the point of practically full function. If the symptoms had not been detected early, the ultimate outcome might have been very different.

Case 5 was that of a small girl referred to me three days after injury resulting in a classical supracondylar fracture. One attempt at least had been made to reduce it, but without success. The forearm was secured in full flexion by bandages. Examination revealed absence of radial pulse and definite involvement of the median nerve. The forearm was at once extended. The pulse had returned in about twenty hours, fracture was reduced by open operation and secured at right angles in plaster. Open operation was carried out in this case in the presence of a good deal of persistent swelling about the elbow and it was hoped that the free opening of the tissues would have the effect of lessening this. It also allowed reduction to be carried out without the use of full flexion which we found immediately blocked the radial pulse.

There is yet another case I wish to describe a little in detail. It is the first of the series and one of the two adult cases. It illustrates one of the delayed complications of elbow joint injuries and raises the interesting question of the best method of dealing with injuries to the head and neck of the radius.

This patient was a woman of thirty-seven years. In childhood she had dislocated the head of one radius forward. For many years it had given her no trouble. She had perfect function at the false joint and disregarded the moderately developed *cubitus valgus* which naturally resulted from her mechanical discrepancy. Sixteen months before she was referred to me she had commenced to feel numbness in the little and ring fingers. Her symptoms progressed and at the time of my examination she had a complete picture of ulnar nerve irritation. At operation to transfer the nerve to the front of the elbow and forearm, I found about two and a half centimetres (one inch) of the trunk reddened and sclerosed. The increase of the fibrous tissue elements was well demonstrated when the branch of supply to the *flexor carpa ulnaris* and *flexor profundus digitorum* was stripped to lengthen it and to allow the nerve trunk to come to the front. The nerve twig stripped as readily as usual until I came to the inflamed area, when the greatest care had to be exercised to preserve it intact, so firmly was it bound by scar tissue. The trunk was quite tightly stretched over the medial epicondyle. Sensation began to return about a week after operation and she has made a steady improvement to the point you have seen.

This and similar cases reported from time to time show that *cubitus valgus* following an injury is liable to be followed by this distressing sequela.

Brickner reports a case following an injury to the elbow in a woman of fifty-two years, occurring fifteen months after injury, but not associated with *cubitus valgus*. In the majority of the reported cases the typical picture follows the onset of the injury in from three to forty years.

The question of treatment of injuries about the elbow is being dealt with by Dr. Humphries and I do not wish to detain you by covering the same ground, except to stress one or two points. In the first place, it must be recognized that reduction of a supracondylar fracture is not effected by flexing the elbow. The essential factor in reduction is realignment by steady traction in slight hyperextension at the site of fracture and then while traction is being maintained, the fragment is pushed forward into position as the forearm is drawn up into the flexed position. The rôle of flexion is to maintain reduction. The fragment can usually be felt to slip forward into position, but if I do not so feel it, I have no hesitation in gently undoing my previous movements, when, if reduction has been effected, it will be undone and the fragment will be seen and felt to slip off the humerus. Lateral displacement should be corrected while the preliminary traction is being maintained.

In the second place, I wish to point out that flexion without reduction having been secured is a dangerous measure and if the surgeon is not quite sure that reduction has been secured, he should maintain the limb at not more than the right angle position.

The third point is that it is not necessary for the maintenance of reduction to keep the forearm acutely or forcibly flexed. The correct degree can be maintained by using a bandage clove hitched about the wrist and tied about the neck with the ends of the bandage left long and the hand held well up to the chin or as close to it as swelling will permit. Under no circumstances should the forearm be secured to the arm by bandages and particularly not by adhesive strapping. The pad in the elbow I regard as not only unnecessary, but as a menace. A little methylated spirit dropped