becoming thickened and rigid, will interfere with the collapse of the chest wall, and the thoracoplastic operation will also be a failure.

There is one type of case for which I consider that thoracoplasty should always be preferred. I refer to these cases of advanced chronic unilateral fibrotic tuberculosis in which there are extensive changes, part cavitation, part great thickening and dilatation of the bronchi and bronchioles. For these cases artificial pneumothorax is only rarely feasible; even where it is, the principal advantage is the building up of the patient's general condition, if necessary, as a preliminary to, and control of the sputum during, the major operation.

The contraindications for thoracoplasty are much more precise than they are for artificial pneumothorax. In the latter the presence of active disease in the other lung is, in itself, no bar; it is rather the anatomical extent of the disease that is the deciding factor. When considering the suitability of a case for thoracoplasty the presence of an active tuberculous lesion in the opposite lung must be regarded as a complete contraindication—with one exception, to be noted directly. It is sometimes difficult, however, to determine whether fine crepitations heard at the apex of the better lung, in which there is a certain amount of fibrosis, are due to active tuberculous disease or to a catarrhal condition. If these signs have been watched over a period of some months and show little variation in area or quality, and the indications in the more seriously diseased lung are very strong, and if, further, after a preliminary phrenic evulsion these crepitations are not deleteriously altered, then a thoracoplasty need not be considered as contraindicated.

The single exception referred to above is in the case of haemorrhage. Bleeding from the more damaged lung, when it is periodically recurring and is of sufficient quantity to endanger the patient's life, and when the fear of it has become an ever-present terror, justifies certain additional risks. In such cases it is permissible, in my opinion, to attempt to control the bleeding by thoracoplasty, when no other means have been found practicable, even if there is a small area of activity in the less involved lung.

Tuberculous enteritis seems to me to be an absolute contraindication. Cachexia, a failing heart, albuminuria, unless transitory, and diabetes, unless well controlled, are also contraindications. Tuberculosis of the larynx is not necessarily a bar unless there is considerable oedema interfering with the airway, or involvement of the epiglottis interfering with nutrition.

Multiple intercostal neurectomy has been tried by J. Alexander in certain cases in which there are contraindications to thoracoplasty. The nerves are resected under local anaesthesia. Slight collapse of the chest wall and immobility are obtained. There has been apparent benefit in some cases, but the number so treated is too small as yet for any definite conclusions to be drawn.

Extrapleural pneumolysis is the local collapse of the lung, which is secured by separating the parietal pleural membrane from the chest wall and by filling the space thus formed with a foreign body to prevent its return. This operation, which may be performed as an independent one, is generally used to induce collapse of the apex of the lung, and it is then called apicolysis. It is often supplemented by hemidiaphragmatic paralysis, and may be used to increase the collapse obtained by a thoracoplasty, especially in cases with thick-walled upper lobe cavities.

Various substances have been employed to fill the cavity between the parietal pleura and the chest wall, so that the collapse of the underlying lung may be maintained. Paraffin, however, may produce an extrapleural effusion, which, finding its way through the divided tissues, escapes and carries the paraffin with it. Fat and fatty tumours may degenerate and form an abscess. I have found a pedunculated graft of the pectoralis major in man, or the breast in woman, the most satisfactory.

As already mentioned, however, when an apical collapse is needed, a very extensive resection of the upper four or five ribs gives a more certain and satisfactory result.

It has been possible to touch the fringe only of the subject in this article. No reference has been made to technique or to the rarer operations.

EFFECTS OF AIR IN THE VASCULAR SYSTEM AND THE MECHANISM OF DEATH.*

BY

D. T. BARRY, M.D., D.Sc., F.R.C.S.,

PROFESSOR OF PHYSIOLOGY, UNIVERSITY OF CORK.

THOUGH some surgeons profess to entertain no fears concerning the evil results of the introduction of air into a vein, most operators believe that such an accident is one to be carefully avoided. Experimental work on the subject was pretty extensive before modern apparatus like the electro-cardiograph could be used for interpretation, but not in recent times. The conclusions arrived at by experiment, though presenting general agreement with regard to salient effects, show certain discrepancies. The literature on the subject is not very extensive. Laborde and Muron¹ injected large quantities of air (500 c.cm. and more) into the jugular vein of the dog at a slow rate without fatal results, whereas rapid introduction of 100 to 200 c.cm. caused paralysis of the heart by distension. Couty² described most of the symptoms observed in the present experiments. The lack of an electro-cardiograph in his and other researches did not permit correct observation and interpretation of the cardiac phenomena. Begouin³ allowed air into the jugular vein in cats and dogs; he also blew it into the vein by the mouth. Withdrawal of air from the ventricle by means of an aspirator was practised with apparent benefit. Depage⁴ says that the entrance of air into veins is not so dangerous as is generally believed. He cites two cases of its occurrence during operation which were cured by artificial respiration, and quotes instances from the work of other surgeons. The three main theories of the mechanism of death—the nervous theory (Morgagni, Bichat), the pulmonary theory (Poisseeule, Bernard, et al.), and the cardiac theory (Magendie, Amussat, et al.)—are discussed. Jung and Auger, in a fairly recent paper,⁵ sum up the general views concerning the mechanism of death, the chief factors being rapid fall of arterial pressure, the entrance of air into the coronary vessels, and ventricular fibrillation. There is no reference to any influence of the respiratory mechanism, except in so far as disturbance of function may be implied as a result of the fall of blood pressure, and in no work up to the present account apparently is there a reference to the use of the electro-cardiograph. Little evidence is provided by the experiments done by myself to favour the view that ventricular fibrillation is a common cause of death after injection of air. The results of these experiments on dogs weighing 10 to 15 kilos are here considered in two chief categories.

A. Thorax Closed.

If 50 c.cm. of air or more be pretty rapidly admitted into the jugular or saphenous vein of a dog with closed thorax and natural respiration a fairly definite train of symptoms is produced—namely, respiratory distress, muscular agitation, polypnoea, marked fall of arterial pressure, and irregular action of the heart. The heart sounds are sometimes audible at a distance from the animal, and have a gurgling character. The electro-cardiogram may exhibit arrhythmia, extrasystoles, inversion of the T wave, etc. A dog thus treated does not necessarily die as a result; in fact, it usually recovers quite fully in the course of twelve to twenty-four hours. If a second large quantity of air be introduced before the effects of the first have passed off the animal may succumb to its influence, and death is generally preceded by a period of apnoea.

In deep anaesthesia the symptoms may be somewhat different from the above. Especially in the case of two dogs under chloralose, there was no marked polypnoea, and apnoea set in early, the effects of one injection being fatal. These two experiments were performed at the Institut Marey, in conjunction with Mr. L. Bull, to whom I am greatly indebted for help and counsel in the taking and interpretation of electro-cardiograms.

The most constant symptom, observed in practically all cases, and varying in degree with the amount of air admitted, is the fall of arterial pressure. When large

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quantities of air are introduced into the venous circulation the sudden drop in arterial pressure is due to mechanical obstruction to the venous flow through the pulmonary artery. The obstruction is soon overcome by an immediate process of breaking up of the air into small particles, so that the frothy blood passes to the lungs. It is remarkable how the pulmonary artery rapidly becomes free; post mortem it shows but few particles of air in the lumen either of the trunk or of the large branches. These appearances give little support to the view that the pulmonary factor is of importance in the mechanism of death.

Does anaemia of the bulbar centres account for the polypnoea? Not fully; polypnoea is sometimes developed when small amounts of air are introduced into the veins which are not sufficient, either in amount or in rate of entrance, to cause an appreciable lowering of arterial pressure. A reflex factor in the causation of the polypnoea is not unlikely, the afferent impulses arising within the cardiac area or in the carotid sinus, which, as shown by the work of Hering,⁵ Heymans,⁶ and others, is the seat of important reflex control of the vascular system in general, and of the encephalic blood pressure in particular.

In a paper on this subject read at a meeting of the Société de Biologie in Paris¹ I drew attention to apnoea as a prominent feature in the effects of injection of air. It is maintained in some cases for quite long periods before cardiac death. The respiratory centre fails, and the anomalous condition is seen of a heart beating, however slowly and irregularly, with no air entering or leaving the lungs, and a very low arterial pressure. The role of apnoea in the mechanism of death seems to be an important consideration, though it has not been given prominence as a factor in this. When sufficient air is injected to cause death apnoea is usually seen before the end, but it needs a larger amount of air to cause it than in deep anaesthesia. In animals with cut vagi the symptoms were like those of deep narcosis with intact vagi. Probably the vagus is an afferent path, not necessarily the only one, for the impulses to the respiratory centre, a path which is deleteriously affected in deep chloralose anaesthesia. In this condition and in vagotomized animals the polypnoeic disturbance is much less than in other cases, and the polypnoea is to be regarded as a mode of defence of the organism. Apnoea of short duration does not necessarily portend a fatal result. But if the apnoeic phase lasts more than half a minute or so it is a certain sign of failure of further resistance, and indicates a fatal termination.

B. Open Thorax.

When the chest is opened and artificial respiration performed the symptoms caused by the introduction of 50 to 100 c.cm. of air into the jugular vein are different from those mentioned in the case of natural respiration. The blood pressure falls as before, but of course there is no evidence of interference with the respiratory mechanism and no indication of the reactions of the respiratory centre in response to reflex or other influence. The heart is the organ which succumbs, and fibrillation is common in these conditions. In case of closed thorax the nervous centres die for lack of blood, consequent on the mechanical obstruction of the circulation, while the heart remains active for a time and is apparently capable of recovery if conditions can be made favourable. The duration of the period of starvation of the centres by deprivation of blood is a factor in causing the death of the centres. In some cases this period is fairly short.

How does the heart maintain its activity without an efficient coronary circulation and in conditions where one would expect serious anoxaemia? Probably a small supply of oxygen is sufficient to keep up the discharge of impulses from the sino-auricular node, and this supply may be available for a time by direct diffusion of oxygen from the arterial blood in the right auricle, which remains bright red for a long time. Removal of the products of combustion in the absence of a proper venous circulation may be also effected by diffusion, aided by contraction of the muscle.

Conclusions.

Dogs can recover from the effects of introducing about 100 c.cm. of air into the jugular vein.

Resistance to the effects is diminished in deep anaesthesia and after section of the vagi.

The heart in some conditions is the organ which fails in fatal cases, but failure of the respiratory centre is the cause of death in others. Apnoea is an important symptom indicating the loss of reaction in this centre. Ventricular fibrillation is not a common cause of death.

With regard to treatment, the inhalation of oxygen while the respiratory centre is active is indicated. Artificial respiration should be tried when it shows signs of failure. As for bleeding and aspiration, they inspire little faith. Animals in experiment exhibit marked powers of natural recovery, and one is very prone to attribute to some remedial measure an undue beneficial effect.

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THE TREATMENT OF DESPERATE CASES OF DIABETIC COMA.

BY

R. D. LAWRENCE, M.D., M.R.C.P.,*
CHEMICAL PATHOLOGIST, KING'S COLLEGE HOSPITAL.

MANY physicians must have had the disappointing experience of losing a certain proportion of cases of diabetic coma even since the introduction of insulin. From all countries records and statistics show a varying number of patients who die in spite of energetic treatment with insulin. Such has been my experience. It therefore seems worth emphasizing an aspect of the treatment by means of which recovery has followed in two desperate cases of coma—in-patients who I am sure would have died under my treatment of a year ago. The deciding factor in their recovery was, in my opinion, the administration of *extremely large* quantities of intravenous fluid, hypertonic saline and gum acacia solution.

The most dangerous cases of coma requiring such treatment are easily differentiated by one or two simple clinical observations from the less severe cases which respond readily to adequate doses of insulin alone. Typical air hunger and varying degrees of unconsciousness are present in both types, and chemical tests show a large excess of sugar and ketone bodies in the urine and blood. In addition the desperate cases show evidence of dehydration, collapse, and circulatory failure. The pulse is rapid and feeble, the blood pressure very low, the veins are empty, the tissues drained of water, and the most striking sign is the *low eye tension*. This may be so extreme and the eyeball so flat that one has almost to lift the lid to make sure the eye has not previously been removed. There is a real "melting of the flesh into water"—an early description of diabetes—or, more exactly, a melting of the water from the flesh, dehydration. This dehydration is produced by the intense polyuria of the preceding period and the failure to absorb fluid because of vomiting and unconsciousness. The volume of the blood is reduced and the blood is concentrated, of which a raised haemoglobin content is evidence.

Another feature of these desperate cases is a very scanty secretion of urine, sometimes actual anuria. It has been suggested that kidney failure is the cause of death in such cases, and that death is really a uraemic one. In my opinion, however, oliguria or anuria is due to dehydration and a low blood pressure. An exactly similar

*Working on insulin for the Medical Research Council.