THE STATE OF THE CAPILLARIES IN CERTAIN PATHOLOGICAL LESIONS AND THEIR CONDITION IN CASES OF SUDDEN DEATH.

by

Jean W. Symington, M.B.,Ch.B.

Muirhead Scholar, Clinical Laboratory,

Victoria Infirmary, Glasgow.

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The material on which this thesis is founded was obtained from the investigation of cases of sudden and unforeseen death occurring in the wards of the Victoria Infirmary, Glasgow, and of cases which were reported as deaths on arrival at the Infirmary. It includes also certain cases of a similar nature in the medico-legal work entrusted to Dr. Anderson by the Procurator-Fiscal.

The works of Krogh, Dale, Lewis and others reveal how extensively these authors have investigated the changes found in the capillaries; and these changes are considered from the anatomical, physiological, and clinical aspects of research. Pathologists, however, have not contributed largely to the literature on this subject, and it is from the pathological view-point that this article treats the changes produced in the capillary vessels.

The Role of the Capillary Circulation.

The capillaries are the small vessels interposed between the finest branches of the arteries and the commencement of the veins; and they form a distributive system whereby the exchange of substances between the blood and the tissues can be carried out. Their function is regulated and adapted according/

ing to the varying needs of the body, and although their mechanism is to a certain extent independent, it is necessarily also co-ordinated with that of the circulatory system in general.

Distribution of Capillaries.

The capillary vessels are of fairly uniform size and do not sub-divide or coalesce, being arranged in the form of a network, which varies in size of mesh in different tissues. This variation modifies the amount of blood supply to the separate parts of the body: for example, the lungs have a very close reticular formation, while ligaments and tendons have a wide network of few vessels. In infants all the tissues are relatively more vascular.

In the heart the capillary loops run parallel to the long axis of the columns of muscle cells, and the network has comparatively close meshes, two or three capillaries being found to each column. The vessels lie moreover in very close proximity to the contractile elements.

In the lung the meshes are round or polygonal in shape and in such close formation do the capillaries lie that the vessels are apparently in direct contact. In the septa between contiguous alveoli they run singly and wind from one alveolus to another projecting superficially into the lumina.

The liver has instead of true capillaries a sinusoidal arrangement, of which the endothelial covering is in many places incomplete, being represented only by the irregularly spaced cells/

cells of Kupffer. Thus the blood comes into direct contact with the liver cells, although according to V. Kupffer an adventitial layer, perhaps constituting a lymph space, intervenes. The canaliculi, however, inside the liver cells communicate directly with the lumina of the capillaries.

The splenic circulation is so peculiar and complex that a histological description in all its details is almost impossible. This is admitted by McNee, who with others believes in the hypothesis of a two-way circulation, one route avoiding and the other traversing the pulp. In the former the arterial blood traverses a venous sinus before entering the veins, in the latter it passes out of the ellipsoid-walls into the pulp and from the pulp back to the veins. In sections of the spleen the appearances presented are those of capillary endothelial cells separated from each other and united by processes to the reticulum cells which form the ground work of the splenic pulp. The gaps thus formed in the capillary walls vary in size with the state of distension of the organ.

In the kidney there is a double arrangement whereby the larger vessels are split up into capillaries. The afferent arteriole to a Malpighian body, on entering, divides into capillary loops to supply each lobule of the glomerulus. These loops, on/

on issuing from the tuft, join with each other to form the efferent vessel, which in structure resembles a small vein: and this venule almost immediately breaks up into a second set of capillaries running between the convoluted tubules.

The cortex of the suprarenal shows that its capillary vessels run in the connective tissue septa only, and do not penetrate between the cortical cells. The endothelial walls are, however, somewhat incomplete, and are probably of sinusoidal nature; and this form of arrangement is more definitely represented in the medulla. In this region the sinuses are larger than those of the cortex and a single layer of endothelium forms the only separation between the blood constituents and the medullary cells. Indeed this endothelial wall may even have gaps which allow the contained blood to bathe the chromaffin cells directly.

In the case of the pituitary body the pars anterior is the richest in blood supply, the pars intermedia being poorer, and the pars nervosa the poorest of all. The capillaries of the pars anterior are of large sinusoidal appearance and are separated from the clumps of epithelial cells only by a very thin covering of connective tissue and by lymphatic spaces.

The arterioles of the skin communicate with their corresponding venules by breaking into capillary loops which project into each dermal papilla. These loops lie at right angles/

angles to the subpapillary arterial and venous plexuses of which they are branches and which lie parallel to the papillary ridges. According to Lewis there are capillaries from these plexuses which intercommunicate in the subpapillary layer without ever having traversed the superficial papillae.

Structure of Capillaries.

The walls of the smallest arteries and veins are composed of three layers - an outer of connective tissue, an intermediate of muscle cells, and an inner of endothelial cells. As the capillaries are approached the connective tissue layer first disappears. Then the muscular layer becomes gradually diminished in size, until finally it is represented in the capillaries by the irregularly spaced Rouget cells which have been clearly demonstrated in the superficial vessels. The endothelial lining is seen throughout all the vessels and the individual cells are of polygonal shape, being pointed at both ends. These cells vary in appearance with the state of contraction or dilatation of the vessels. In the latter condition the cells are large, their edges straight, and their nuclei are thin oval discs which only project very slightly, if at all, into the cavity of the vessel. In the former the cells appear to be smaller, their edges tortuous, and their nuclei more rounded, projecting/

ing into the lumen. The smallest veins differ from the capillaries only in size.

Nervous Control.

The capillary vessels are tonically innervated like the arteries through the sympathetic nervous system. Thus, on stimulation of a sympathetic ganglion the corresponding capillaries contract: on removal of the ganglion they dilate, and are extremely slow to recover their original tonus, their regulation ever afterwards being imperfect. The vaso-dilator fibres appear to be identical in every way with the sensory nerves which are able to carry anti-dromic impulses and they act quite independently of the dilator nervous control of the arteries. This vaso-motor mechanism has both a central and a peripheral control; and a reflex arc has been demonstrated to pass from the skin along the posterior root fibres to the posterior root ganglion, and back to the vessels along the sympathetic fibres (Krogh).

This nervous control does not apply to the cerebral vessels, although histologically nerve plexuses can be seen around the arterioles. Here, if the blood supply be inadequate the vaso-motor centre is stimulated to contract the splanchnic vessels and to raise the arterial pressure. This produces an alteration in calibre of the cerebral vessels and an increase in velocity of the blood, at the expense of loss

of/

of cerebro-spinal fluid which forces its exit from the skull. The cerebral sinuses are then compressed until their pressure equals that of the brain substance, but the escape of blood is not prevented. Thus the amount of blood in the cerebral circulation is uniformly maintained.

The lungs also either lack or are very scantily supplied by vasomotor nerves. When the left heart fails suddenly and the right heart continues to force blood into the pulmonary vessels the absence of vasoconstrictors allows engorgement of the lungs with the production of oedema. As Cohnheim has said "A man does not die because he gets oedema of the lungs; he gets oedema of the lungs because he is dying." The lack of vaso-dilator control also is demonstrated by the beneficial effects of treating haemoptysis by amyl-nitrite, when the resultant widespread dilatation leaves the lungs unaffected.

Neither are the coronary vessels under the control of the vasomotor system, and the explanation is simple. When general vaso-constriction with rise of blood pressure takes place, the heart must perform more work and therefore requires more nourishment. If the coronary vessels shared this constriction their blood supply would be diminished. Similarly when the general blood pressure falls the heart performs less work and the coronary vessels automatically receive less blood.

Thus/

Thus the organs composing the 'tripod' of life - brain, lungs and heart - because of their great importance are independent of vaso-motor control, for this control causes centralisation of impulses and administers for the general needs of the body before the local requirements of a particular organ.

Some Physiological Observations.

Normally the capillaries of the skin continuously open and close, and so they deliver an adequate but economical supply of blood. Prolonged contraction is rendered impossible by the lack of oxygen so produced, and the consequent loss of tone involves dilatation. Many factors influence this variation in capacity. The same stimulus may cause either constriction or relaxation of vessels, depending entirely on its strength: for example, a weak mechanical stimulus will cause dilatation of a capillary while the same stimulus applied with greater force will cause contraction . Variations in temperature also cause reactions in the capillaries: heat causes dilatation of vessels and stasis of their contents: cold may induce constriction of the lumina with increased viscosity of plasma, or, if more severe, it may cause relaxation of the capillary walls to their greatest extent. This latter effect is independent of any nervous control and is probably due to a paralysing effect on the Rouget cells. Light rays also/

also can be made to produce local reactions. Normally the capillaries of the skin vary in tone in adjacent areas. It is a general rule that heightened tone is found in these areas to which the blood flows most freely, and this decides the mottling of the skin which occurs at times, the dark areas corresponding to the more atonic vessels.

The formation of lymph is not a secretary process, but is brought about by the purely physical action of filtration and osmosis. Three factors are concerned in its formation:- (1) capillary pressure, (2) metabolic activity of the tissues, and (3) under abnormal conditions the increased permeability of the capillary walls.

The permeability of capillaries varies in different organs and also in the same set of vessels directly with their state of dilatation. The most permeable capillaries are found in the liver where the blood pressure is very low: and in this organ as well as in the intestinal mucosa the capillary walls are apparently permeable to a fraction of the blood protein. Laqueur has demonstrated this phenomenon also in the pulmonary capillaries.

The two processes of absorption into and filtration from the capillary vessels balance each other under normal conditions. No dilatation, however, which involves mechanical stretching of the endothelium can take place without being accompanied by an increase in permeability. If this dilatation results/

results in the production of a slight degree of filtration oedema, the transudate can be removed by the lymph channels, but it cannot be reabsorbed into the blood. The explanation is that with the recovery of their normal tone the capillaries become impermeable to the protein in the transudate and the osmotic pressure of the latter will also prevent the reabsorption of water and of crystalloids.

Four substances have an important and almost selective action on the capillary circulation - adrenalin, vasopressin, acetyl-choline and histamine.

Adrenalin, produced by the medulla of the suprarenals, is poured out into the blood in response to stimulation of the splanchnic nerve. Its action constricts the capillaries and also the arteries, and causes a rise in blood pressure. Dale states that "It is probable that the normal function of adrenalin as continuously secreted into the blood is the maintenance of healthy capillary tone against the dilator effects of metabolic products". Any sudden emotion will greatly intensify this capillary tone by causing a gush of adrenalin into the venous blood which conveys it in equal concentration to all parts; but the responses are modified in different situations, the vessels of the alimentary canal being much more constricted than those of the skeletal muscles. The blood thus redistributed gives the opportune physical response to any external force which calls for increased energy for fight/

fight or flight.

The capillaries are also constricted, with a resultant rise in blood pressure, by the action of vasopressin, produced by the posterior part of the pituitary gland. Vasopressin differs somewhat from adrenalin in its action: firstly by acting directly on muscle fibres while adrenalin stimulates the sympathetic nerve endings; secondly, by stimulating all unstriped muscle to contract, while adrenalin has an inhibitory action on that of the alimentary tract: and thirdly by being more lasting in its effects than adrenalin. According to Krogh the pituitary hormone is the chief regulator of capillary tone.

Acetyl-choline is liberated in heart muscle under the influence of the vagus nerve and its first action is the production of a rapid fall in blood pressure. The suprarenals then become reflexly stimulated, adrenalin is produced and a rapid rise in pressure follows.

Histamine, or histidine, is a cleavage product of protein and is liberated from all tissues on injury. It causes capillary dilatation with resultant increased permeability of the walls, stasis, and oedema. The vicious circle thus produced leads by a failure in blood supply to lack of oxygen and of tonic hormones, and these aggravate the condition until failure of heart occurs. Histamine causes also a fall in systemic blood pressure by constricting the pulmonary vessels and/

and thus reducing the output of blood from the left ventricle. Contraction of plain muscle therefore and constriction of arterioles are produced, so that at some point in the circulatory system the reaction of the smaller vessels to histamine changes from contraction to dilatation. Once peripheral stagnation has occurred, the constriction of arterioles and of venules will obviously only intensify the condition.

The Explanation of Shock.

Krogh has stated that the capacity of the capillaries of resting muscle becomes increased by 750 times when those capillaries are dilated to their fullest extent. Therefore should all the capillaries in the body be simultaneously influenced to relax, they would be perfectly capable of holding the total quantity of circulating blood. This state of affairs actually does occur; it has been described as a "bleeding into the capillaries", and the clinical picture is labelled shock. Two varieties are described: primary shock occurring immediately on receipt of an injury, and secondary shock which develops some hours later. The primary type is caused by temporary failure in the central nervous system control over the circulatory system, and is of the nature of a reflex. Stimulation of sensory nerves causes temporary paralysis of the vasomotor centre with dilatation of abdominal veins and cerebral anaemia. Secondary shock is due to the liberation of some toxic substance which induces/

induces a fall in blood pressure brought about by the dilatation of the capillaries.

Dale has stated that the effects produced on these capillaries by nervous impulses and by chemical stimuli are closely similar. Examples of these chemicals are histamine and the poisons causing anaphylactic shock, and their results are almost identical - dilatation, increased permeability, and oedema. This loss of capillary tone accompanied by contraction of arterioles in an endeavour to maintain the blood pressure, reduces the volume of blood in circulation and heart failure supervenes.

Shock and the effects of histamine and similarly acting poisons are aggravated by haemorrhage, cold, and the administration of an anaesthetic. It is obvious that after a severe haemorrhage the blood pressure will be greatly reduced. The immediate effect of cold is to constrict arterioles, capillaries and venules; but the prolonged effect is to cause dilatation of the capillaries and of the venules and to produce cyanosis. Most anaesthetics have in themselves a dilator action on the capillaries, and it follows therefore that the deeper the anaesthesia induced the greater will be the liability to shock.

Many bacterial products can produce a shock-like death. For example, inflammation has the local effect of dilating the capillaries and arterioles through stimulation of the sensory nerve/

nerve fibres; but the poisons causing inflammation may be present in such quantities that a general effect is produced, as in septicaemia, when the capillary vessels simultaneously lose their tone and cause the death of the victim by vasomotor collapse. Other examples of the primary failure of the vasomotor mechanism are seen in the state of cholera, where cyanotic collapse occurs after enormous dehydration, and in the state of diphtheria before the appearance of respiratory obstruction or of the toxic effect on the heart.

The results due to paralysis of the capillary tone may be closely imitated by other toxins which act by directly poisoning the endothelium and by disorganising its structure. Salvarsan acts in this way as well as by its precipitation in the blood. According to Dale "When a specific sensitiveness is "produced to a normally innocuous substance this is accompanied "apparently by a minor degree of enhanced sensitiveness to the "naturally poisonous substances producing these effects. This "corresponds with the clinical experience that the victim of a "toxic idiopathy has capillaries or bronchial plain muscle which "are to some degree abnormally sensitive to naturally poisonous "substances of this class." These statements help very considerably to explain the tragic suddenness of the deaths which occasionally follow the injection of a regulation dose of salvarsan.

Anaphylactic/

Anaphylactic Shock.

Anaphylaxis is defined as an "acquired specific sensitiveness to foreign protein". It is assumed that there is located within idiopathic sensitive cells an antibody which reacts intracellularly with any substance which can function as an antigen. The effect is the same as that produced by the reaction of normal cells to histamine, namely, the plain muscle of hollow viscera and of bronchioles is stimulated to contract while the capillary blood vessels are relaxed to their fullest extent. Death may thus take place either by asphyxia or by circulatory collapse.

The Condition of the Capillaries in Certain Pathological Lesions.

Active Hyperaemia. This condition is essentially a physiological process, being concerned with the supply of increased nutritive fluid to tissues which are temporarily more active than usual. Such tissues with heightened metabolism produce an increased amount of acid and Gaskell has shown that the action of acid causes dilatation of capillary vessels. Krogh too has demonstrated that local erythema is brought about by a spinal reflex, and Dale states that both nervous impulses and chemical stimuli produce this condition. Dilatation of vessels is probably a reaction on the part of the tissues to protect themselves from injury, or it may be induced by the local production of a substance of histamine-like character.

Passive/

Passive Hyperaemia. This is more commonly a pathological process, and is the result of interference with the outflow of blood. It may be general as in heart failure, or local because the veins are more easily compressed than the arteries. The tissues become congested and distended; the capillaries pulsate with the heart beats, and later the blood in them becomes stagnant; the nutrition of the capillary walls becomes impaired, increased permeability results and oedema takes place. When gravity affects the tissues in this way the condition is called hypostasis. Oedema occurs whenever the capillary blood pressure exceeds the osmotic pressure, but it is doubtful if the capillary pressure alone ever becomes high enough to accomplish this without being accompanied by an appreciable increase in the venous pressure.

Inflammation. In many cases the vascular changes develop slowly and long after the application of the responsible stimulus, as in the effects of sunburn. In others the process follows immediately on injury and appears to be adapted to hold in check the injurious agent and to destroy it.

The capillary vessels react firstly by a momentary contraction, but this is soon followed by an extensive and uniform widening of the capillaries, arterioles, and venules of the affected area. Klemensiewicz views this widening as being due to complete paralysis of their walls, for during the state of inflammation/

inflammation the affected vessels cannot be stimulated by any means to contract. It is possible that the local injury of tissue may liberate enough of a histamine-like substance to cause this paralysis. If this view be accepted the reaction of the capillaries in the state of inflammation cannot be regarded solely as of beneficial purpose, but it is apparently more of the nature of a passive role.

Coincidently with their widening, the rate of flow in the capillary vessels becomes greatly increased until it almost equalises that in the arteries. The affected area feels hot, for the blood is hurried on so rapidly that there is no time allowed for the process of cooling which normally occurs in the superficial capillaries. Pain is experienced because the sensory nerves are rendered more sensitive by the hyperaemia, a fact which can be demonstrated even when the cause is non-inflammatory.

After a time the blood stream slows down although the capillary bed remains wide. The corpuscles are seen to float in the centre of the lumina, and to be separated from the walls by a layer of clear plasma. Gradually with the slowing of the stream the leucocytes appear to be thrown out to the margins and rolled along the walls to which they eventually adhere. Finally they pierce the walls with their protoplasmic processes and escape away into the surrounding tissues. Red blood corpuscles escape/

escape passively and follow the movements of the leucocytes; and simultaneously with this escape of cells fluid has been exuding from the vessels. A coagulable inflammatory oedema now surrounds the capillaries and causes swelling and increased tension in the affected part.

The current of blood may come to a complete stop with the development of stasis. This condition is, however, quite different from a simple retardation or stoppage of flow, which leaves the quantitative relationship between the corpuscles and plasma unaltered. Nor is it similar to agglutination which may occur with stoppage of flow, and which is recognised by the interspacing of clumps of corpuscles and spaces of plasma. In stasis the concentration of corpuscles is increased, but if it develops rapidly the resulting oedema is slight. Emigration of leucocytes ceases and the capillary endothelium is damaged, as Cohnheim has shown. Histologically at this point the endothelial cells appear more prominent than normally.

In other cases although the corpuscles pass along very slowly no complete stoppage of flow occurs. Eventually the irritant is removed, the blood vessels regain their normal calibre, their walls appear healthy and contract, and the circulation resumes its normal rate.

A persistent chronic inflammation differs from an acute reaction chiefly in the arrival of the mononuclear forms of leucocytes. The congestion of vessels and the oedema may subside/

subside for the most part and persist solely along the margins. There is usually associated marked overgrowth of interstitial connective tissue of which the vascularity varies greatly. Very frequently, however, considerable formation of new blood vessels is noted as in cirrhosis of the liver and kidney.

"An almost universal result associated with injurious effects is the new formation of connective tissue in congested and cyanotic organs."

Cirrhosis of the Liver. In this condition the capillary circulation of the liver is influenced by the extent and distribution of the connective tissue. Such forms as pericellular, monolobular, and coarse atrophic cirrhosis are described. In the pericellular type the connective tissue spread occurs along the course of the hepatic vessels, and a new formation of capillaries, in connection with the terminations of the hepatic artery, is an accompanying feature. In the early stages of the coarse cirrhosis the new formed connective tissue is cellular and highly vascular; later when portal congestion is an obvious feature, the isolated groups of liver cells are interspaced with capillaries which have sprung up in every direction and grown into a complete labyrinth.

The capillaries offer obstruction to the blood flow because of their tortuosity or obliteration, and because the rigidity of the scar tissue prevents their normal distensibility. Sometimes very small communications between the portal system and/

and the branches of the vena cava are opened up in an attempt to relieve the congestion. An interesting theory is advanced by Herrick:- that the communications between the hepatic artery and the portal vein become much wider in cirrhosis of the liver and so the arterial pressure is directly communicated to the veins whose outflow it blocks as in arterio-venous aneurysm.

Because of the portal obstruction there ensues a passive hyperaemia of the mucosa of stomach and of intestines, of peritoneum, and of spleen. Capillary haemorrhages and haemorrhages of larger size occur in the walls of stomach and intestine, while in the case of the rectum the dilatation of the internal haemorrhoidal veins leads to the formation of piles.

Nephritis. The feature of acute inflammation of the kidney, is, as in inflammation generally, an active hyperaemia with exudation of serous fluid, leucocytes, and red corpuscles. These find their way into the tubules and interstitial tissue. In the case of glomerulo-nephritis the toxic agencies attack especially the capillaries of the glomerular tufts. Their walls may be so damaged that the corpuscles agglutinate and form hyaline thrombi, which completely occlude the lumina. In other cases the blockage of capillaries is apparently due to increase of the endothelial cells which become swollen, proliferate, and form syncytium-like masses. These capillaries are mostly empty of blood and the obliteration is completed by a spongy mass of protoplasm. In others, leucocytic infiltration is/

is the marked feature and the injured walls allow penetration of these cells even into the Bowman's capsules and tubules. Occasionally small haemorrhages are abundant and almost confluent. In all cases these obliterated capillary loops exert pressure on the glomerular capsules to which they become adherent, and finally the whole glomerulus becomes distorted into a mass of connective tissue.

In the arterio-sclerotic type there occurs gradual diminution of the blood supply due to thickening of the vessel walls and narrowing of their lumina. This is not due to thrombosis but to a sclerotic change of the walls. These may show fatty or hyaline degeneration, or endarteritis obliterans may even be present. The capillaries may become impermeable to blood primarily through the cutting off of the supply of the afferent vessels, or they may collapse because of the increased pressure of obstructed tubules and distension of the capsules. Unless they collapse completely their lumina are seen to be filled by structureless material, the origin of which may be the remaining red blood corpuscles or proliferation of their own endothelial lining. Eventually the capillary loops fuse together, their walls disappear, and the whole mass of a glomerular tuft may be enclosed by a layer of continuous epithelium.

Thus in nephritis the stream bed of the capillary vessels is narrowed, and the high blood pressure which accompanies this disease may be of a compensatory nature with the purpose of increasing/

increasing the blood flow. On the other hand it may be due to the toxic action of the retained products of metabolism.

Degeneration of capillaries. In cases of severe anaemia and of phosphorus poisoning fatty degeneration of the capillary vessels may be found. The fat is deposited in the form of small or medium sized droplets in the endothelial cells, and their function is then impaired. Necrosis may supervene. Degeneration of the hyaline type has been described in the capillaries of the central nervous system (Mallory). This material is most frequently deposited as minute droplets external to the endothelial cells and these droplets enlarge and fuse together to form a homogeneous sheath for the vessel. The cause of this degeneration is unknown.

Haemorrhages. Minute haemorrhages, occurring by rupture of the capillary vessels and termed petechiae, are frequently attributed to toxaemia. The toxin may act by the production of an acidosis which dilates the peripheral vessels, and, as Langdon Brown has suggested, increases the viscosity of the blood by actually increasing the size of the corpuscles. In most cases the weakening of the wall and the haemorrhages occur so quickly that little else is seen histologically but the extravasated blood around a vessel (Fig. 1.). In septicaemia, however, petechiae are due to the actual presence of organisms which plug the capillary vessels. Emboli from cardiac vegetations also may rupture a normal vessel by inducing a sudden excessive/

excessive distension, and in both conditions a central necrotic spot surrounded by haemorrhage may be found on examination. There is usually associated with the presence of petechiae an increased tendency to bruising on slight injury, and Bedson has concluded that a lesion of the vascular endothelium is produced by the serum. Other writers, however, attribute this condition primarily to the accompanying deficiency of the blood platelets.

Eruptions of the Skin. The reactions of the cutaneous vessels to any form of stimulation are threefold, as Lewis has shown. (1) Local vasodilatation occurs, (2) with resulting local increased permeability of the vessel walls, and (3) the development of a widespread flare. The various eruptions thus produced owe their form to the preponderance of one of these factors, the other two being comparatively insignificant.

In the macule the localised active dilatation is the most pronounced feature; when oedema is superadded by increased permeability of the vessels a papule is formed; the surrounding flare is not seen because a contraction of the arterioles results when a stimulus of reflex origin is excessively prolonged. The stimulus in this instance is the liberation of the H-substance slowly and with diminished potency, as Lewis has described, and the consequent response is a prolonged one. After the disappearance of a macule by cutting off its blood supply, its reappearance is striking whenever the flow of blood is resumed. The relaxed condition of the capillary vessels is shown by their lack/

lack of response to adrenalin and to histamine, and it is not surprising therefore that this type of lesion is very commonly followed by pigmentation.

When the arterioles are similarly relaxed, the widespread flare of a scarlatiniform rash is produced. When both the capillary and arterial vessels are affected by liberation of the H-substance the appearances of urticaria arise. The acuteness of onset and of defervescence of this rash are explained by the rapid discharge of a dose of this poison and by the abrupt onset of a refractory period. A wheal is formed when the increased permeability of vessels allows the production of oedema; and most substances which produce whealing will, on stronger concentration, cause blistering, and this condition is also found when the application is prolonged. In the condition of herpes zoster, inflammation of the posterior root ganglion is responsible both for the sensations of pain and for the anti-dromic vaso-dilator impulses to the skin. A creeping oedematous lesion frequently shows central umbilication; this is not due to the exhaustion of the poison in the central area but to the exhaustion of the power of the vessels to remain dilated for prolonged periods; and so contraction accompanied by diminished permeability takes place and the oedema subsides. Similarly are the serpentine markings, so commonly found in skin lesions, produced; namely, by the fusion of two advancing hyperaemic circles with subsidence of their walls at the point of contact where/

where the responsiveness of the capillary vessels becomes exhausted. In the same manner can infections of the skin radiate without showing any sign of their presence, provided that the superficial capillaries have already experienced the noxious stimuli; but as soon as the infection arrives at a fresh area of skin the corresponding capillaries dilate and react as in the original focus.

The trophic changes in the skin associated with various nervous disorders are essentially due to some abnormality in the vasomotor mechanism of the capillaries. Thus in tabes dorsalis, where the sensory nerve fibres are affected, one is not surprised to find vaso-dilatation in certain areas of skin. This leads on to oedema and ulceration from impaired vitality. In the same way in cases of peripheral sensory paralysis no local inflammatory reaction occurs when an irritant is applied.

Telangiectasis. This is a condition of the capillary vessels resembling a simple tumour, for it develops independently of the general circulation. A capillary naevus so formed very frequently anastomoses freely with the vessels of the surrounding tissues although it is supplied with an afferent artery and an efferent vein. The abundant capillaries maintain their original form as a rule and histologically their endothelium is seen to be composed of cells rather more cubical in shape than those of normal vessels. The surrounding stroma is composed of fully formed fibrous tissue. Very rarely these tumours take/

take on a malignant character.

The Condition of the Vascular System and of the Capillaries in Cases of Sudden Death.

The literature references to the modes of death are to be found chiefly in the works of medico-legal authorities who alone make a distinction between death and sudden death.

Bronardil and Benham define the latter condition as "the rapid and unforeseen termination of an acute or chronic disease which has in most cases developed in a latent manner". They state that "No one dies suddenly apart from the effects of violence, as long as all the organs are sound; but there are some diseases which develop slowly and secretly, without the attention of the patients having been called to them by any pain or by any feeling of illness, and without a physician having ever been called in, and which terminate naturally by a rapid death."

Thus a man may die suddenly without experiencing at all or only for a very short time any alarming symptoms or signs; and the victims of arterio-sclerosis, coronary artery disease, and myocarditis frequently die in this manner. A sudden emotion or a chill can precipitate the end. In such cases, however, the knowledge of accurate observations made prior to death might elicit symptoms to which no attention had been paid, and which were indications of the true state of affairs.

It is intended in this article not to refer to cases of sudden/

sudden death occurring in older people, but to deal with those cases in which it affects the young adult or child in whom the organs are expected to be in a healthy condition.

It is customary to follow Bichat's classification of the modes of death, namely, death beginning at the lungs, at the heart, and at the brain. Clinically, those three types are described as deaths by asphyxia, syncope and coma. "If the respiration and circulation persist, the remaining functions, even those of the brain, may be in abeyance and yet the person may survive." (Coats).

Asphyxia. Failure of the respiratory movements is described as follows: "This may occur suddenly by paralysis of the respiratory centre in the medulla oblongata as in haemorrhages in the brain, but for the most part it occurs gradually from exhaustion of the respiratory centre and is then called asphyxia." (Coats).

Whether asphyxia is produced by mechanical obstruction or by lack of oxygen in the circulating blood the sequence of events is as follows.

1. Hypernoeic stage. When the accumulated carbon dioxide becomes excessive it stimulates the respiratory centre and both the expiratory and inspiratory movements are greatly exaggerated especially the former. The vascular tension increases, the superficial veins dilate, the skin becomes livid, and the eyeballs protrude. The symptoms may be sensory illusions such as ringing in the ears or a sense of fulness in the head. The/

The accompanying deficiency of oxygen induces loss of consciousness at the end of this stage.

2. Expiratory convulsive stage. The lack of oxygen renders the central nervous system more exciteable with the production of convulsive movements of the whole body and of the extremities. The sphincters are forced and excretions voided.

3. Stage of exhaustion. This is due to paralysis from the prolonged want of oxygen. An occasional deep inspiration takes place but the muscles are flaccid, the pupils dilated and immoveable, and the reflexes absent. As death approaches the inspirations become more shallow and more irregular; the back is straightened, the head is thrown back, the mouth gapes and the nostrils dilate. The heart continues to beat after all the other movements cease, for the medullary centres retain their vitality even after the brain and spinal cord have lost their excitability. Ultimately the heart stops in diastole, and its failure is attributed to the asphyxial character of the blood supplied to the cardiac muscle and to the resistance of a heightened blood pressure.

This high blood pressure in asphyxia is caused by the changes in composition of the blood which stimulate the vasomotor centre and this in turn causes general constriction of arterioles. The impulse passes along the splanchnic nerve and results in constriction of abdominal vessels and in outpouring of adrenalin which further increases the constriction.

Should/

Should the blood supply to the brain be inadequate the respiratory centre assists the vaso-motor centre to raise the blood pressure which it does by increasing the respiratory movements. A graph of the blood pressure taken during asphyxia will show those variations in vasomotor control; they are described as the Traube-Hering curves and are slower in rhythm and larger in amplitude than the waves corresponding to heart beats and to respiratory movements.

The resistance of both the systemic and the pulmonary capillaries is increased by this great rise in arterial pressure and it is intensified by the convulsive movements. The heart's action becomes laboured, the diastolic intervals longer, and the left side first becomes distended with blood. This is closely followed by distension of the right heart, for the inefficiency of the left side causes a backward pressure. When death occurs in cases of asphyxia all four chambers are distended with blood and immediately afterwards the blood pressure can be demonstrated to be equal in both sides. The right side contains a greater amount of blood than the left because of its greater distensibility. With the incidence of rigor mortis the left ventricle contracts firmly and expels its contents, while the various fibres in the right side do not coincide in their contraction with the result that the expulsive power is very limited. This gives the characteristic post-mortem appearance, namely, great distension and engorgement of the right side/

side of the heart and of the veins opening into it, associated with contraction and an empty state of the left side.

Syncope. This is described as the sudden loss of consciousness due to diminished blood pressure in the cerebral vessels. "For an efficient circulation it is necessary that there should be a sufficient quantity of blood or vascular tension and a differential tension in the arteries as compared with the veins. The circulation will be brought to a standstill by any cause which greatly lowers the vascular tension or annihilates the differential pressure in the arterial system." (Guy and Ferrier). Thus syncope may be produced by lesions of the heart or of the vessels or of both together.

When the heart is affected by organic or structural disease, the blood tends to accumulate and stagnate in the capillaries and veins, and congestion and dropsy and extravasation of blood are apt to take place. If heart failure should affect the right side suddenly,or blockage of the pulmonary artery ensue,the prominent symptom is extreme dyspnoea. On the other hand, if the left side be affected suddenly, insensibility and convulsions will be the earliest consequences.

The heart may be inhibited temporarily or finally through the influence of the central nervous system and via the vagus nerve. A sudden emotion may cause this condition or it may be produced by a reflex action through stimulation of a sensory nerve. Thus syncope and shock may co-exist, both having been produced/

produced by the same cause: when the condition of syncope passes off with return of consciousness, the symptoms of shock alone remain. In syncope the skin becomes deadly pale and cold to the touch, and life may become extinct without the supervention of any other symptom.

J. A. MacWilliam has expressed the opinion that cases of sudden death are due to the occurrence of fibrillation in the ventricles. Certainly in cases of asphyxia it has been demonstrated that the conductivity of the auriculc-ventricular bundle is diminished and that heart block is produced. The reflex action of the vagus nerve in slowing the heart in response to the vaso-constrictor stimulus has also been proved, and this would appear to be a safety valve for lessening the strain and for prolonging life at least temporarily.

When death occurs from cardiac failure the heart is found to have stopped in diastole and the post-mortem findings may be of three types: 1) all four chambers may be distended, the amount of blood in the right side being greater than that in the left because of its greater distensibility; 2) the condition associated with asphyxia may be found; 3) both ventricles may be found contracted and empty while the left auricle contains a very small amount of blood. When death occurs from shock the heart is found to be empty of blood. Failure of the heart has an immediate effect on the circulation in the lungs, and oedema of the lungs is one of the most constant effects of this condition./

condition.

Coma. The state of coma may be produced by the presence of a grave cerebral lesion which causes pressure on the brain, or in consequence of a lesion in some other part of the body. In coma there is profound unconsciousness and the patient cannot be roused by any external stimulus; the breathing is slow, irregular and stertorous; the saliva and other secretions from the mouth, throat and air-tubes accumulate and are not expelled; and gradually, partly from this cause, partly from failure of the respiratory muscles, the patient dies of asphyxia. Death may occur quietly or in a convulsion.

The post-mortem appearances in such a death are essentially those of asphyxia associated with oedema of the brain. Glaister uses the term comato-asphyxia to indicate this condition, for it represents a combination of both proximate forms of death.

Thus, death from coma resolves itself eventually into death from asphyxia, which in turn resolves itself into death from arrest of the circulation. The cause of arrest of the circulation may be a spasmodic and insuperable contraction of the pulmonary arterioles, and this condition in its turn can be referred to the influence of the vaso-motor centre. It is obvious, therefore, in a comparison of the several modes of dying, that there is much in common in the different processes which induce death, and that these processes tend to blend into one another.

As a rule the heart does not cease to beat because its muscular/

Fig. 1. Cerebral cortex from a case of asphyxia showing congested venule with perivascular haemorrhage.

Fig. 2. Cerebral cortex from a case of asphyxia showing a punctiform haemorrhage in the white matter. The capillaries in the neighbourhood are much engorged. The brain at post-mortem examination showed a puncta vasculosa character.

Fig. 1. Cerebral Cortex.

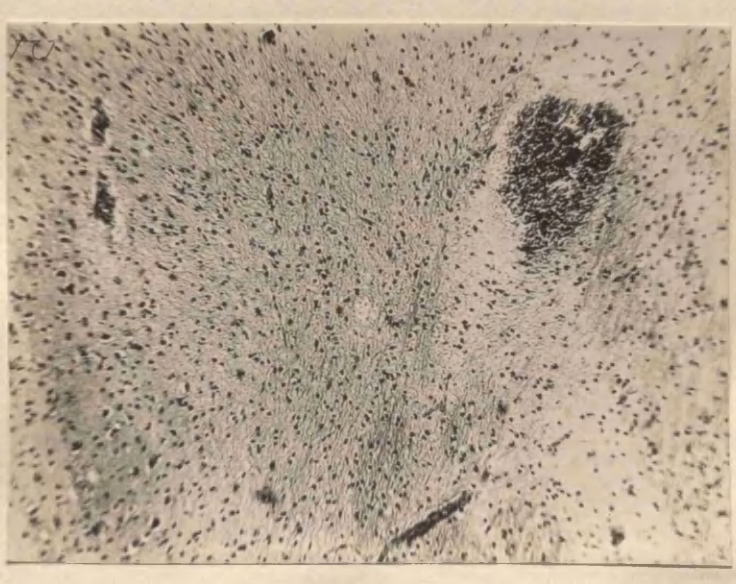

Fig. 2. Cerebral Cortex.

Fig. 3. Lung from a case of syncope. Note the engorgement of the vessels in the interalveolar septa and the occasional extravasations of red blood corpuscles.

Fig. 4. Lung from a case of syncope, showing engorgement of the capillaries and extravasations of red cells. Note the rupture of the interalveolar septa (enphysema).

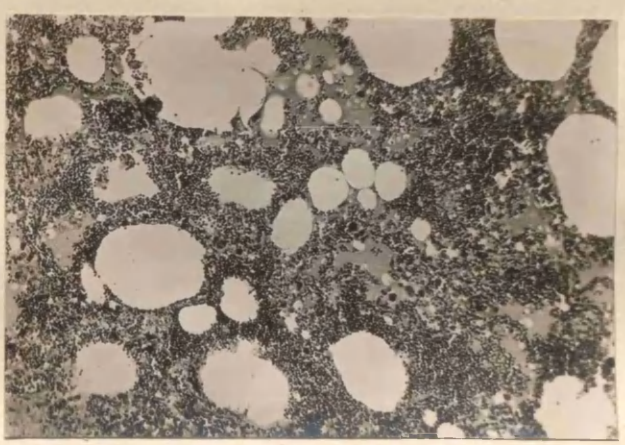

Fig. 3. Lung.

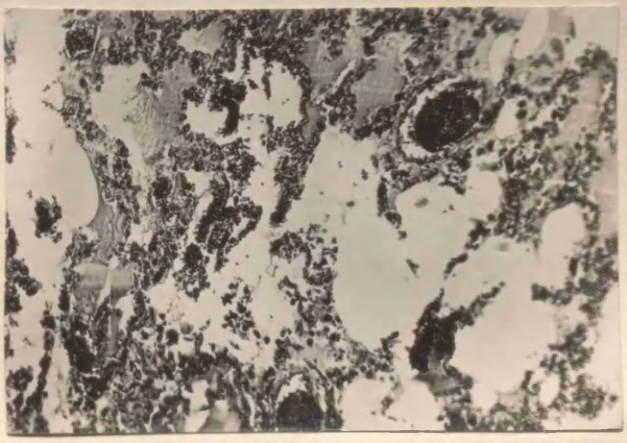

Fig. 4. Lung.

Figs. 5 & 6. Liver from a case of electrocution, showing marked engorgement of the capillaries between the liver cells. Note a small area of leucocytic infiltration.

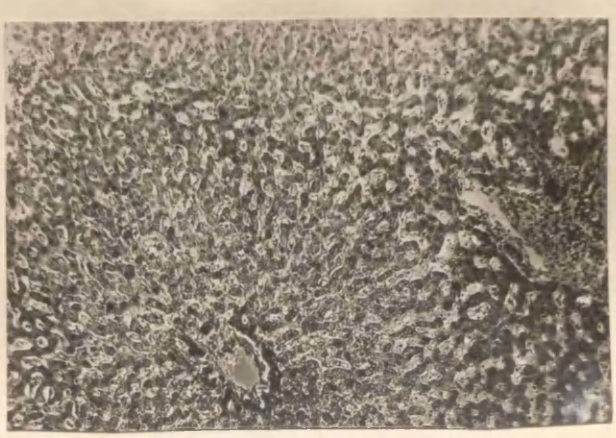

Fig. 5. Liver.

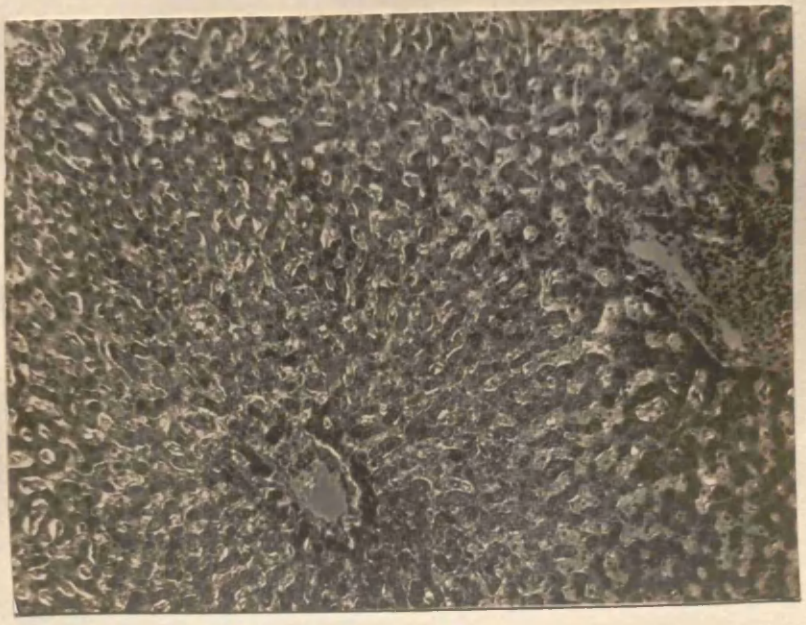

Fig. 6. Liver. Enlargement of above photograph.

Fig. 7. Suprarenal from a case of comato-asphyxia following on the injection of neo-kharsivan. Note the marked engorgement of cortex and part of medulla.

Fig. 8. Suprarenal cortex from the same case. Note the engorgement and lipoid appearance of the cortex.

Fig. 7. Suprarenal. Low power.

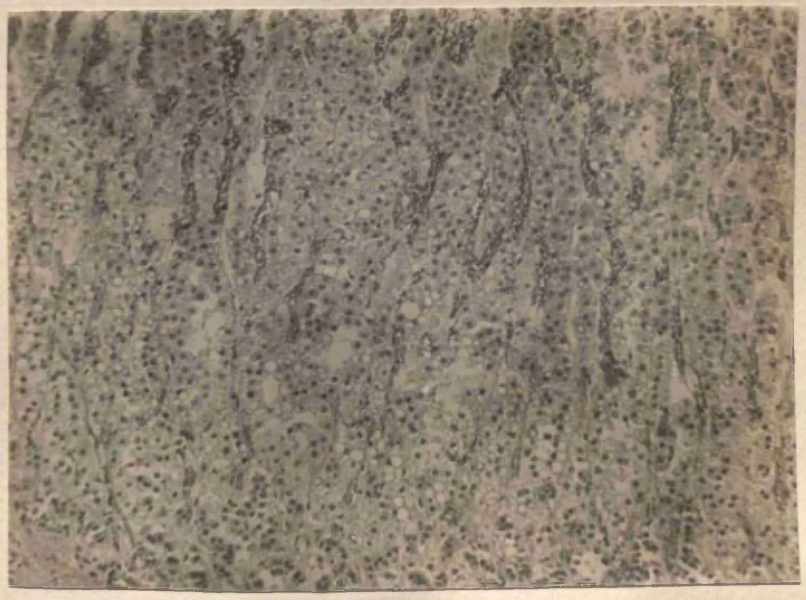

Fig. 8. Suprarenal Cortex. Enlarged photograph.

Fig. 9. Spleen from a case of syncope. Note the full character and engorgement of the pulp. The artery in the photograph shows an empty lumen.

Fig. 10. Kidney from a case of syncope showing congestion of the intertubular and glomerular capillaries and slight cloudy swelling of the epithelium.

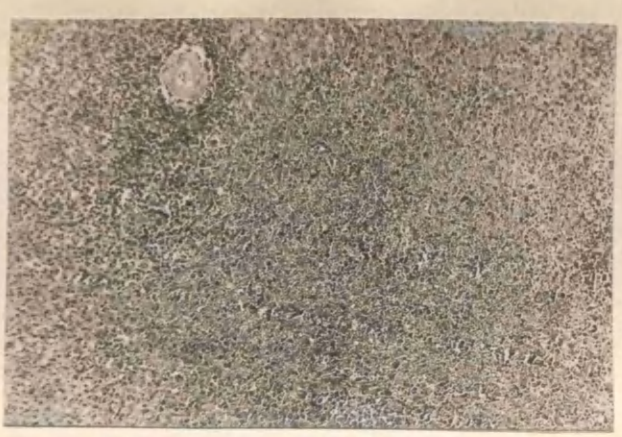

Fig. 9. Spleen. Low power.

Fig. 10. Kidney. Enlarged photograph.

Fig. 11. Heart muscle from a case of comato-asphyxia following on the injection of neo-kharsivan. Note the engorged venule and the red corpuscles in single file in the capillaries between the columns of muscle cells.

Fig. 12. Cerebellum from the same case showing chromatolysis of the Purkinje cells. A congested capillary with perivascular oedema is present.

Fig. 11. Heart muscle. Enlarged photograph.

Fig. 12. Cerebellum. Enlarged photograph.

muscular fibres have wholly lost their power of contracting, for after death the muscular tissue may still contract under the influence of artificial stimulation. The failure is due to the debility of the ganglionic centres and nerves which do not send forth their accustomed stimuli.

Illustrative Cases.

The following cases are cited as examples of sudden death. Cases of Asphyxia. In these, the salient features which are common to all are the presence of marked lividity, the engorgement of the right side of the heart, and the presence of petechial haemorrhages.

Case No. 1. A. McC. Male. Aged 3 months. The social status of the parents was that of the poorer working class. The child was healthy from birth, bottle-fed, and slept in a cot by himself. One morning his mother found him in an almost lifeless condition, being livid in appearance. No history of a convulsion could be obtained. Death occurred prior to the arrival of a doctor.

The post-mortem examination revealed a well developed and well nourished child of 25½" long. The lips and finger-nails showed well-marked lividity. The pupils were equal and of small size. On the surface of the heart numerous petechial haemorrhages were found, and several of these were of fairly large size. The right side of the heart was engorged with dark coloured fluid blood. The heart was of normal size and the cardiac muscle and valves were healthy in appearance. Both lungs were engorged, and,/

and, on the surface of each, numerous petechial haemorrhages were noted. Similar haemorrhages were also present in the pleura, and on the surface of the thymus gland which was enlarged and engorged, weighing 27.5 grammes. There was no obstruction in the throat, and larynx, trachea, and oesophagus were normal. The stomach and intestines were normal, a small quantity of clotted milk being found in the former viscus. The liver, spleen, and kidneys were all in a congested state, as was shown on section. The meninges were healthy and the brain on section had a normal appearance, no haemorrhages being noted.

On histological examination the organs showed intense venous and capillary congestion, the latter being the marked feature; minute haemorrhages were also prominent. Sections of the heart showed the muscle bundles to be somewhat widely spaced and to be regular in outline; the individual cells stained well and there was no fatty change. In several areas congestion was noted, the capillaries being in a state of distension and the venules also engorged and dilated. These areas, however, were always found in close proximity to actual extravasations of blood from the capillary vessels. In other areas no congestion was noted. Sections of the lung showed marked distension of vessels with red blood corpuscles, the capillary vessels in the alveolar septa being especially engorged. Some desquamation of alveolar epithelium was noted, but there was no pneumonic consolidation nor/

nor extravasation of red blood corpuscles into the alveoli. The thymus gland showed marked congestion of capillary vessels, and presented appearances as of red blood corpuscles collected into sinusoidal areas, or extravasated between the lymphoid cells. These aggregations of red corpuscles were closely similar to minute haemorrhages. The small veins were also distended. The medulla oblongata was sectioned at different levels and examined serially; congestion of capillary vessels and of venules was noted in the region of the floor of fourth ventricle, but the specific nuclei were not involved; no gross haemorrhage was found, but occasionally the vessel wall was ill-defined, and there was diapedesis of the blood cells into the spaces around.

Case No.2. B.L. Male. Aged 7 days. Weight 7½ lbs. Length 20½". This child was born in a nursing home and the confinement was normal in every respect. On the evening prior to death the child, with four others, was put to bedon a couch. In the early morning a nurse discovered him lying dead.

The post-mortem examination showed a well-developed and well-nourished body. There was marked lividity of the lips and of the finger nails, and this change was present to some extent on the left side of the face. There was no suffusion of the eyes. Internally some petechial haemorrhages were found in the pericardium and on the surface of the heart, and these were specially abundant at the auriculo-ventricular junction and at the orifices of the great veins. The right auricle and right ventricle/

ventricle were distended with dark coloured blood, and the cardiac muscle and valves were apparently healthy. On the surface of the lungs a large number of petechial haemorrhages were present and both lungs on section showed marked congestion especially at their bases. The larynx, trachea, and oesophagus were normal; and the thymus was not enlarged. The stomach and intestines were normal. The liver and spleen were in a state of congestion; and the kidneys were also deeply congested and showed the presence of small haemorrhages immediately beneath their capsules. The meninges were healthy and the brain, apart from evidence of congestion, was normal.

Histological investigation showed that congestion was the prominent feature of the sections. The lungs presented appearances somewhat resembling consolidation, for the septa were broadened and many alveoli were obliterated and unrecognisable. Others were seen as clear distended spaces, however, without proliferation or desquamation of their lining epithelium. Extravasated blood increased the inter-alveolar septa to an enormous extent but no rupture took place into the alveoli, thus differing in appearance from an infarct. Capillaries, venules, and arterioles showed marked engorgement and in one area at least the occurrence of haemorrhage into the adventitial coat of a large artery was noted. The sections of the medulla showed intense congestion, almost resembling those of an infected brain; distension of the capillaries and of the smaller vessels was generalised, but this condition was especially obvious around the/

the nuclei of the cranial nerves; a number of small haemorrhages were present and in many places spilling of red blood corpuscles had occurred into the perivascular spaces. Some ganglionic nerve cells showed a ghost-like appearance of their cytoplasm due to loss of their staining properties; others failed to reveal their Nissl's granules and many showed the variations of hypo-chromatism, of hyper-chromatism, and of chromatolysis.

Case No. 3. B. McK. Male. Aged 7 days. Weight 6¼ lbs. Length 21". No doctor was in attendance at the birth. The child appeared to be healthy, cried lustily, and slept in a cot with three other children in a separate room from his mother. One morning he was observed at 4.30 a.m. and was apparently well; at 8 a.m. he was found to be semi-conscious. A doctor was summoned, but the child was dead on his arrival.

The post-mortem examination revealed a somewhat poorly-nourished child. The lips showed the presence of slight lividity, and this condition was seen to affect the toe nails somewhat and the finger nails to a very marked degree. The heart showed the right auricle and right ventricle to be engorged with dark coloured fluid blood, while the chambers of the left side contained only a small quantity. The heart muscle and all the valves were apparently healthy. The lungs were markedly congested at their bases, but were otherwise normal for neither patches of consolidation nor evidence of petechial haemorrhages were/

were obtainable. The structures of the mouth, pharynx, larynx, trachea and oesophagus were normal. The stomach and intestines were examined and found to be healthy. The liver, spleen and kidneys were all in a state of venous congestion. The bladder was empty. Examination of the brain showed the presence of an area of haemorrhage at the base in the region of pons and medulla. This haemorrhage extended outwards to the surface into the canal of the spinal cord. On dissection of the brain a quantity of blood clot was found to fill each of the ventricles, but there was no evidence of haemorrhage into its substance.

Death was ascribed to cerebral haemorrhage. If this be accepted as the full explanation a weak condition of the child's vessels must be blamed, and this is a most unusual state of affairs. The lividity of the lips, fingers and nails suggests a diagnosis of asphyxia even in the absence of haemorrhages on the heart or lungs, and this conclusion includes the probability that the rupture of a vessel occurred during an asphyxial convulsion.

Case No. 4. J.W. Male. Young adult. This man met his death as the result of an explosion in a coal pit.

The post-mortem examination showed a well-developed and a well-nourished body. Rigor mortis was marked. The pupils were of medium size. There was marked lividity of the finger nails, and the post-mortem lividity on the back and on the sides of the body and neck was well shown. The front of the abdomen showed/

showed greenish discolouration. There was no rose-red colouration of the skin or of the muscles. A few slight bruises of a superficial nature were noted on the left side of the chest outside the cardiac area, on the left eyebrow, and on the front of the right ankle. The heart was of normal size and its chambers and valves were normal, the right auricle and right ventricle containing a large amount of dark coloured blood. A few small haemorrhages were present on the surface of right auricle near its base and on the outer aspect of the aorta near its commencement. The cardiac muscle and the coronary arteries were healthy. Both lungs were deeply engorged with dark coloured blood but were otherwise normal. Larynx and trachea were also congested. The stomach and intestines showed congestion of their mucous membranes, and the liver, spleen, and kidneys were also deeply engorged with dark coloured blood. Apart from the congestion the viscera were apparently healthy. The bladder contained four ounces of urine. The brain was intensely congested but no gross lesion was found. A spectroscopic and a chemical analysis of the blood proved the absence of carbon-monoxide.

The microscopical examination of this case showed the conditions which one would expect to find in a case of asphyxia from lack of oxygen. The sections showed marked chromatolysis of the motor and ganglionic nerve cells. In the cerebellum many of the Purkinje cells showed ghost-like appearances, while others/

others showed signs of hyper-chromatism. Congestion of capillaries and of venules was a marked feature of all the sections, and peri-vascular oedema was also prominent.

Cases of Comato-asphyxia. The post-mortem findings are invariably those of asphyxia but the appearances are not of such a marked degree as in cases of suffocation. The right side of the heart is generally deeply engorged and the lungs also and the solid organs show marked engorgement. The brain shows congestion and in some cases the presence of oedema. Clinically, unconsciousness exists prior to death.

Case No. 5. J. M. Female. Aged 38 years. This woman, apart from an occasional attack of "asthma", enjoyed good health until two weeks before her admission to hospital when her husband died of pneumonia. For eleven days she continued to perform her household duties, all the while being worried about her financial position. Then she retired to bed, her breathing became laboured as if after strenuous exertion, and she gradually became comatose. The blood sugar was found to be within normal limits; and no albumen was detected in the urine, which, however, contained 1.09% of sugar and abundant acetone. She died on the day after admission. Her social status was good, her parents and sisters were in good health, and she had no children.

The post-mortem examination revealed a well-developed and well nourished body. An old scar was present in the right iliac/

iliac fossa. The lips and the finger tips were livid in colour, and there was well marked post-mortem lividity along the back of the neck and shoulders. The pupils were contracted. The pericardium was normal. On the surface of the heart numerous asphyxial petechial haemorrhages were present at the auriculo-ventricular junction. The heart was of normal size and the chambers of the right side were engorged with blood. The cardiac muscle was soft and flabby in consistence but showed no condition of fibrosis; the coronary arteries were apparently healthy; and the valves were normal. The right lung was slightly adherent to the chest wall and the left lung was free; both showed the absence of any emphysema of their margins or of consolidation or oedema; the presence of congestion, in character resembling a terminal engorgement, alone was found. The bronchi appeared to be chronically inflamed and coated with a layer of muco-pus.

In the abdomen it was found that a colopexy had been performed on the ascending colon, and adhesions united the omentum to the abdominal scar. The stomach, intestines, liver, gall-bladder, spleen, pancreas, suprarenals, and urinary bladder were apparently in a healthy condition. Both kidneys were in a state of congestion. The uterus was shrivelled and affected with small fibroids; the ovaries were somewhat fibrosed. The thyroid gland had a small oedematous nodule on the left side but was otherwise normal in appearance; the larynx, trachea and oesophagus showed no variation from the normal. There was no evidence of/

of meningitis. The brain was wet on the surface and on section oedema was noted; congestion was especially marked in the region of the basal ganglia but no haemorrhages were observed on naked-eye examination.

Microscopical examination showed congestion of the vessels in the medulla. The nerve cells stained well but many showed loss of outline and degeneration of their Nissl's granules. There was also noted some pigmentary degeneration. The thyroid gland showed congestion of its vessels; the colloid material was abundant and there was some heaping up of the epithelial cells around the alveoli. The nodule showed the characters of a simple fibrous growth. The pancreas showed the presence of areas of fibrosis; its blood-vessels showed thickening of their walls; and the islets of Langerhans could not be demonstrated.

Clinically this was regarded as a case of diabetes, the terminal phase of comato-asphyxia having been precipitated by the depression and disturbance following on the husband's death. The only apparently diseased organ was the pancreas, and microscopically the vascular condition was shown to be that of a terminal engorgement, the veins and capillaries in the various organs being deeply congested.

Case No. 6. H. B. Female. Aged 4 months. Length 24". The child was healthy at birth, and shortly afterwards commenced to attend a Baby Clinic where the records show a steady increase in/

in weight. She was breast fed. Five days prior to her death she was vaccinated at the Clinic, but this did not appear to hinder her progress. Four days later there was slight sickness with vomiting after a feed and the vomitus consisted of curdled milk and had a slimy appearance. The vaccination marks were present. During the following night the child did not waken as usual for a feed. The next morning at 6.15 a.m. the mother found the child snoring heavily and with a glazed appearance of the eyes which were wide open. Death occurred two hours later.

The post-mortem examination showed a child of normal appearances. Rigor mortis was slightly marked. The finger nails and the fingers showed distinct lividity, but there was no lividity of the lips. The left arm showed the presence of two small dried up vaccination vesicles each about $\frac{1}{4}$" in diameter. The heart was of normal size and its chambers and valves were normal. The right auricle and right ventricle contained a fair quantity of fluid blood but they were not engorged. Both lungs showed the presence of a few punctiform haemorrhages on the surface, and, in addition, the right lung showed congestion of its base. The pharynx, larynx, trachea, and oesophagus were all normal and the thyroid gland was of small size. The thymus gland was enlarged and weighed 20.5 grams. The stomach contained only a small quantity of mucous fluid and this organ and the intestines were normal in appearance/

ance. The liver and kidneys were somewhat pale in appearance but otherwise showed no abnormality. The spleen was of normal size and the lymphoid tissue of its substance was not increased in amount. There was no enlargement of the mesenteric glands. The other abdominal organs were examined and found to be normal in character. The meninges were healthy. The brain externally and on section presented a distinctly congested appearance but it was not oedematous.

On histological examination the heart showed neither any obvious congestion nor the presence of haemorrhages. The muscle cells were well-stained; there was no loss of striation; and neither fatty degeneration nor infiltration was present. The lungs showed a prominent emphysematous condition. The venules were greatly distended, one in particular showing a marked haemorrhage around its walls; and the capillaries were also congested. The liver columns of cells were apparently normal and well separated from each other, the intervening capillaries being distended with blood corpuscles. There was well-marked congestion of the portal veins and in these zones a few slight aggregations of lymphocytes were noted. The spleen showed intense engorgement and the Malpighian bodies were numerous and closely approximated, many of these being of large size. The stroma cells were well represented but the lymphocytic type of cell was not greatly in evidence. The kidneys showed congestion to a slight extent in the cortical areas/

areas and more markedly in the medullary regions; some cloudy swelling was also noted. The suprarenals showed congestion of the veins at the hilum and venous and capillary engorgement in the medulla. The thyroid gland had a normal appearance apart from congestion of the larger vessels; but there was very little congestion noted between the acini. The thymus gland was enormously congested, and the engorgement of the inter-lobular veins was a marked feature. The cerebral cortex showed venous and capillary congestion and some escape of blood corpuscles was recognised, but there were no gross haemorrhages. The ganglionic cells stained well and there was no chromatolysis. There was no evidence of encephalitis. The medulla was sectioned at two levels:- at the upper level congestion was a more prominent feature than at the lower level, and no haemorrhages were recognised. The nerve cells showed definite chromatolysis. The cerebellum showed congestion to a marked degree and this was present also in the basal ganglia which showed in addition loss of Nissl's granulation. The choroid plexus and the cerebral meningeal vessels were congested but no rupture had taken place.

There was no evidence on examination that death was connected with vaccination, and the animals which were innoculated with an emulsion of the brain tissue were unaffected. There was undoubtedly an element of asphyxia in this case, but the findings were not those of a case of overlying. The congested brain/

brain on the other hand was such as is found after an infantile convulsion but no such history could be elicited, nor was any cause discoverable which would occasion such a phenomenon.

Case No. 7. J. McD. Male. Aged 36 years. This man contracted syphilis and was treated by a course of injections of neo-kharsivan. Six months afterwards, as the Wassermann reaction was still positive another dose, similar in quantity to his first one, was given. Five hours later he began to take epileptiform convulsions and did not regain consciousness prior to death.

The post-mortem examination revealed the body of a sparely-nourished man. In the head the meningeal vessels were found to be much engorged and the brain was congested and oedematous. In the thorax, the heart showed over-distension of the right auricle with dark blood but the other chambers were empty and somewhat dilated. The valves, coronary arteries, and heart muscle were in a healthy condition. The commencing aorta showed several slight areas of degeneration. Both lungs were free from adhesions; the left one showed marked emphysema of its margins and both were congested and oedematous. The larynx, trachea, and oesophagus were normal in appearance. In the abdomen some fatty change was found in the liver, but no destruction of tissue had taken place. All the other organs were examined in detail and were apparently healthy.

Histological examination of the heart muscle revealed some dissociation/

dissociation of the muscular fibres. The individual cells were somewhat swollen and showed loss of striation, but they were stained distinctly and showed neither fatty infiltration nor degeneration. ' Distributed throughout the heart muscle were areas of intense congestion which even resembled in appearance minute haemorrhages. Microscopical sections of the lung showed markedly congested vessels; and catarrhal desquamation of epithelial cells without exudation had occurred into the alveoli. In one part of those sections the appearances of a recent pneumonic patch were noted; the alveoli were packed with cells of the polymorph type and epithelial cells, many of which were pigmented, were also in evidence. The liver showed intense congestion of all its capillaries; well marked fatty degeneration was seen in a number of its cells in addition to some cloudy swelling; a few areas of lymphocytic infiltration were present around the portal zones. In the spleen the feature of the sections was the intense congestion. The lymphocytic and stroma cells were in the proportion of a full cellular but congested pulp and the Malpighian bodies were of good size,and many showed germinal areas. Both kidneys were intensely congested. The tubular structure showed marked evidence of cloudy swelling, but the glomeruli were of good size and shape and were evenly spaced and there was no increase of the fibrous stroma. The suprarenal gland showed evidence of congestion and the cortex had definitely a lipoid appearance./

appearance. Examination of the aorta confirmed the atheromatous ulceration found on naked-eye examination. The cerebral cortex, cerebellum, medulla oblongata and spinal cord were all examined and showed congestion of their vessels and peri-vascular oedema. The ganglionic cells were swollen and ill-defined and their staining properties showed poor differentiation.

Cases of Syncope. In the syncopal and shock deaths the findings are practically the same. The feature of these cases is the failure of the heart muscle and both sides of the heart coincide in this. The fall of blood pressure is the clinical manifestation, and engorgement of veins and capillaries is the histological result. The systemic capillaries show engorgement as markedly as do the pulmonic vessels.

Case No. 8. J. S. Male. Aged 22 years. This young man was apparently in good health and he ran some distance to overtake a comrade who was entering a picture house. Shortly afterwards he collapsed and was brought to the Victoria Infirmary, but life was extinct on arrival.

The post-mortem examination revealed a well-developed and well-nourished man. The pupils were of medium size. Rigor mortis was present. On opening up the thorax a normal pericardium was found. Slight distension of the right ventricle of the heart was noted and both right auricle and right ventricle contained a large quantity of fluid blood. The left ventricle was in a state of contraction, and cardiac muscle and valves/

valves were apparently healthy. No haemorrhages were present. Both lungs were non-adherent and voluminous, and on section showed marked congestion and oedema. The bronchi contained a quantity of blood-stained frothy sputum. On opening the abdomen enlarged mesenteric glands were found. The liver was of normal size, and apart from being acutely congested was otherwise normal. Gall-bladder presented normal appearances. The spleen was slightly enlarged, of purplish colour and congested appearance, and the Malpighian bodies were apparent. The kidneys showed congestion but were otherwise normal. The stomach, intestines, pancreas, suprarenals, and urinary bladder were normal. In the neck a large healthy looking thyroid gland was found. The tonsils were definitely enlarged, and, at the back of the tongue, shedding of the mucous membrane and enlarged lymphoid follicles were noted. No oedema or congestion was found in the larynx, trachea or oesophagus, which all presented normal appearances. On opening up the cranium a slightly congested and oedematous brain was found.

Microscopical examination revealed a practically normal condition of the heart muscle. The muscle bundles were of good size and shape, and there was neither dissociation nor loss of striation. Pigmentation and congestion were negligible and neither extravasated red blood corpuscles nor haemorrhages were apparent. In the lungs the alveoli were patent and no consolidation was found; and the inter-alveolar septa were broken in many/

many places giving an emphysematous appearance. The feature of these sections was the enormous distension of the inter-alveolar septa caused by congestion of the capillary vessels and the presence of haemorrhages. On account of this congestion the capillary endothelial cells were very indistinct, and indeed were only recognised with difficulty. There was no apparent lesion of the epithelial cells, but the pigment carrier cells were fairly abundant. The liver, under the low power of the microscope, presented normal appearances. The columns of cells were widely separated and in some parts a slight fatty change was noted, but this change was definitely absent in other areas examined. The spleen showed congestion of the loose tissue of its pulp. There was no apparent increase of the splenic tissue, but the Malpighian bodies were fairly numerous and of good size. The arterioles were neither dilated nor congested. The kidneys showed marked engorgement chiefly of a capillary nature, but the veins also showed this condition. Congestion of the glomeruli was marked, and that of the intertubular vessels was of an even greater degree. The medullary portion was enormously engorged and diapedesis of the red cells was recognised. Some cloudy swelling and some desquamation of the epithelial cells was noted, but the feature of the section was the glomerular and intertubular congestion, without the escape of blood into the tubules, and without the presence of inflammatory foci. The thyroid gland presented normal/

normal appearances apart from some congestion of vessels between the acini. The thymus gland showed the presence of small haemorrhages in the lymphoid tissue and the veins and capillaries were in a state of congestion both in the cortical and in the medullary areas. In the medulla oblongata congested veins were found throughout. There was no marked capillary congestion however, nor gross haemorrhage. Peri-vascular oedema was present and the meningeal vessels were congested. The ganglionic cells of the cerebral nuclei showed hyperchromatism.

Case No. 9. J. B. Male. Aged 34 years. Investigation reveals that this man had been in poor health for two years during which time he was unemployed. The illness was labelled neurasthenia. After a holiday in the country of two months duration he left his house one morning and did not return. When a search was made, his body was found on an adjacent hillside.

Post-mortem examination revealed a well-developed and well nourished man. There were no marks of injury upon the body. The pupils were of medium size. Rigor mortis was well marked. The heart was of normal size and both the chambers of the right side were dilated and contained a quantity of fluid blood. Those of the left side were normal. The heart muscle was of good consistence and the valves were all healthy. There was no disease of the coronary arteries. The left lung was free; the right/

right lung was adherent to the chest wall by a few old-standing adhesions. On section, both lungs showed the congested appearance of a case of sudden death, being also somewhat oedematous. The bronchi also were congested. The structures of the mouth and throat were examined; and well marked enlargement of the lymphoid tissue at the base of the tongue and some enlargement of the tonsils was noted. The larynx, trachea, and oesophagus were congested but otherwise normal. The thyroid gland was of larger size than normal, but on section presented a healthy appearance. The thymus gland was considerably enlarged, cellular in character, weighing 43 grammes. The stomach was of normal size and it contained a fair quantity of well digested food, with a marked odour of alcohol. Some congestion of its mucosa was noted in one area, but there were no changes suggestive of irritant poisoning. The intestines were normal. Several of the mesenteric glands were definitely enlarged. The spleen was of normal size and its lymphoid bodies were distinctly evident. The liver was in a state of congestion but was otherwise normal. The kidneys also were deeply congested and showed no other abnormality. The suprarenal glands were of small size; the pancreas was healthy. The urinary bladder was normal in every respect. Dissection of the brain revealed no gross lesion but congestion and oedema were in evidence. The meninges were normal in character.

Histological examination of the heart revealed the presence of/

of enormous dilatation of the capillary vessels between the columns of muscle cells. These cells stained well and had a healthy appearance. In the lungs the prominent feature was broadening of the interalveolar septa by congestion of vessels and by haemorrhages. There was also an emphysematous condition with rupture of the septa. The liver showed some congestion which was especially marked in the region of the central veins of the lobules. The spleen showed enormous engorgement of its pulp with red blood corpuscles. The Malpighian bodies were apparently very numerous and the arterioles associated with them showed no dilatation. The kidneys were enormously engorged, the intertubular and glomerular capillaries being dilated to their fullest extent. Some desquamation of the lining epithelial cells of the tubules was noted, but there was very little diapedesis of blood corpuscles, and no haemorrhages were found. The suprarenals showed marked congestion of both cortex and medulla, and this condition was well seen immediately below the capsules. In a portion of the bowel wall which was examined histologically enormous congestion of the subepithelial veins and capillaries was found, and there was marked shedding of its mucosa. The thyroid gland showed the presence of some venous congestion, but the interalveolar capillaries were not dilated. The thymus gland showed marked congestion of its capillaries and venules and even of its arterioles. There was, however, only very slight diapedesis of blood corpuscles among/

among the lymphocytes of the pulp. In the medulla a few small haemorrhages were noted and congestion and perivascular oedema were present. In addition the nerve cells showed marked chromatolysis.

This was regarded as a syncopal death in a person who had the condition known as persistent status lymphaticus.

Case No. 10. B. McK. Female. Aged 32 years. Electrocution was the cause of this woman's death. While working as a charwoman she stood on a wet sink with a damp cloth in her hand for the purpose of cleaning a window. Accidentally she established contact with a defectively insulated electric lamp suspended above her head. She was discovered in a rigid condition and fell down after the current was switched off. On admission to hospital life was pronounced extinct.

Post-mortem examination showed the body of a well-developed and well-nourished woman. Rigor mortis was passing off. The pupils of the eyes were of medium size. There was lividity of the lips and marked lividity of the left ear and left side of the head, as well as post-mortem lividity on the dependent parts. A small amount of blood was present at the orifice of the left ear. A number of wounds and abrasions were present on the face and arms and these were consistent with the lesions produced by an electric shock. All the chambers of the heart were of normal size, those of the right side being engorged with dark coloured fluid blood. The cardiac muscle, the valves, and the coronary arteries/

arteries were normal. The lungs were free within the pleural cavities and a few small sized haemorrhages were present on the surface of each lung. Both were in a state of engorgement with dark coloured blood. The bronchi were deeply congested. The thyroid gland was of normal size and appearance. The thymus gland weighed 15 grammes. The stomach and intestines were normal. The liver was of large size and was engorged with dark coloured blood. The kidneys also were in a state of engorgement but were otherwise normal. Spleen, pancreas, suprarenals and other organs were examined and found to be healthy. The aorta and blood vessels generally were in a healthy condition. There was no injury to the bones of the skull and the meninges and the brain substance showed no gross lesion but were in a state of congestion.

On histological examination, the heart showed congestion of its capillaries and venules; the muscle cells showed well marked striation and there was no fatty change. The arterioles were not dilated. The lungs showed marked congestion of arterioles, venules, and capillaries; and blood corpuscles and desquamated epithelial cells were frequently seen in the spaces of the alveoli. The liver showed distension of the central and portal veins, and the columns of liver cells were well separated from each other by red blood corpuscles. There was distinct leucocytic infiltration around many of the portal tracts, and within the lobules many small aggregations of leucocytes were also/

also noted. The liver cells were apparently healthy. In the kidneys, areas of congestion alternated with areas in which there was no congestion. A few small haemorrhages were noted and there was some cloudy swelling of the uriniferous tubules. The suprarenals showed a normal relationship of cortex to medulla. Both regions were markedly congested and the surrounding fat showed engorgement of arterioles and venules. The aorta showed congestion of the vessels in its adventitial coat. In the thyroid gland there was marked congestion of arterioles, venules and capillaries between the acini, where some heaping up of the epithelial cells was also noted. No rupture of capillaries or diapedesis of blood cells had taken place. In the medulla oblongata the marked feature was congestion of the capillaries within as well as around the nuclei of the cranial nerves. Certain of the ganglionic cells stained well, while others had an ill-defined character presenting the features of a well defined chromatolysis.

Conclusions.

The investigation of this subject clearly demonstrates that the role of the capillaries is an active rather than a passive one. These vessels are controlled, under physiological conditions, by nervous impulses on the one hand and by chemical stimuli on the other. In the case of the latter there is the influence of secretions of the suprarenal and pituitary glands; and/

and certain substances of a metabolic character also influence capillary action whenever liberated into the circulation. The administration of various drugs gives a like result.

In the local skin reactions a close similarity in effect is given by the action of appropriate nervous and chemical stimuli. Both can lead to loss of capillary tone with excessive permeability and resulting oedema or extravasation of blood. The latter takes the form of diapedesis of blood cells and is directly the result of the congested state of the capillaries. Actual ruptures in the endothelial walls are only very occasionally discovered. The coarser haemorrhages of the asphyxial cases can be accounted for by the strain of a convulsive fit.

In the case of shock Crile has demonstrated that the essential pathology is the same whatever the cause, namely, severe physical injury, intense emotional excitation, toxic, anaphylactic or drug influence. These agents induce a sudden fall in blood pressure and this is not rectified for the mechanism of compensation has also failed. Yandell Henderson asserts that intense pain alone is sufficient to produce this condition for the pain stimulates the lungs to prolonged and excessive ventilation, with depression of the vaso-motor centres and the consequent fall in blood pressure. On the other hand in severe cases of shock pain may be the only stimulus to the respiratory centre, and when the pain is removed on the administration of an anaesthetic/

anaesthetic a collapse ensues. The emotional factor influences to a great extent the course of many diseases whether a decline be accentuated by worry and depression or a sudden collapse be induced by a fright. It is difficult to disprove that a sudden shock induces a reflex cardiac paralysis: but the impression remains that the atony of the vascular system is the responsible factor for the collapse.

In the sudden deaths from various causes quoted in this paper there was found a marked similarity both in the post-mortem findings and in the histological examinations. In all cases the indication of the sudden fall in blood pressure was the marked capillary congestion, and this was a common feature and of fairly uniform extent in the solid organs, namely, liver, spleen, and kidneys. The muscular action of the heart and the tone of the arteries were good but failure occurred because of the accumulation of blood in dilated capillaries and venules.

The distribution, size, and shape of the capillaries demonstrates their capacity to Hold the entire quantity of circulating blood, and its withdrawal from the circulation is the primary cause of failure of the heart or vaso-motor centre.

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