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Chronic subdural hematoma : with special reference to etiology, diagnosis, and treatment

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CHRONIC SUBDURAL HEMATOMA

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INTRODUCTION

Chronic subdural hematoma is a clinical entity which until relatively recently has been considered quite a rarety. Eighty three years ago Virchow described in the literature a pathological entity to which he gave the name "pachymeningitis hemorrhagica interna". The pathology of these two lesions is so nearly identical that some investigators say that there is no difference while still othersmaintain that the histo-pathological picture does vary somewhat. An interpretation of the above relationship will be fully discussed in the body of this paper. It is placed in this introductory paragraph merely as an example of clinical pathology which has been given little consideration by anyone until within the last twenty years when it has come especially into its own as a definite diagnostic problem in the life of the neurosurgeon.

It would appear from a cursory glance at the term, "chronic subdural hematoma", that the definition is perfectly obvious. Either chronic hemorrhage beneath the dura or the formation of an irritating hematoma which over a length of time might be expected to react in some manner to its environment, seem rather simple definitions of such a lesion. Yet in the last twenty-

six years, since Trotter's article on "Chronic subdural hemorrhage of traumatic origin and its relation to pachymeningitis haemorrhagica interna" in 1914, many differences of opinion concerning the etiology and the pathology of a lesion which Virchow thought rather completely settled many years previously have been displayed.

After a somewhat limited review of the literature it is the plan of this paper to attempt to present a reasonable explanation for the above pathology as it is given by the various authorities in this field. With the disposal of development history of this process, which for so many years went completely undiagnosed causing the death of a great number of individuals, this paper will take up a discussion of those diagnostic points by means of which this condition has come to be recognized. It is of course only after a careful analysis of all signs and symptoms presented that it is at all possible to arrive at some sort of treatment. A brief although it is believed adequate review of the surgical and medical treatment will also be included.

Chronic subdural hematoma is a condition which when diagnosed yields such beautiful results under correct surgical care, and ends so hopelessly when not treated that an understanding of such a lesion is an important

step in the career of every neurosurgeon.

I know not whether I should defend my choice of a topic for a senior thesis or not, but I do feel that there is some justification. Partly because of interest in the field of neurology and especially in the field of neurosurgery, and partly because of a case I saw but recently lead me to this choice. At the Mid-West Medical Clinic held in the year 1939 at Omaha, Nebraska, there was presented to the group a case of chronic subdural hematoma. Seated on the platform was the patient, a man in the sixth decade of life. The only sounds which he emitted were mumbled jabberings, and his movements were vague and meaningless. The history of several months told of a rather slowly progressing change in personality, mental changes, and sensory and motor upsets which had continued until the pitiable picture we saw that day presented itself. I remember so distinctly the neurologist who presented him saying that if this patient had been taken to a surgeon and correctly diagnosed, with proper treatment there would have been no need for him to be in his present condition. He also stated that with the signs and symptoms then present, treatment would avail but little except to prolong his life, a not too kind procedure at this time.

With this as introduction it is now my purpose to

present a study of chronic subdural hematoma and attempt to arrive at some definite conclusions as given in the literature concerning etiology, diagnosis and treatment.

REVIEW OF THE LITERATURE

The first case that has been found in the literature is one recorded by Ambroise Pare (Sherwood 61, Putnam 55). He gives the history of one Henry the second of France who, wounded in a tourney in 1559, presents a lesion described as a subdural hematoma. Johannes Wepfer in 1657 (Putnam 55) after an autopsy examination on a patient seventy years of age, who died after an "apoplectic stroke" with the symptoms and signs of an aphasia and hemiplegia, wrote concerning a "blood cyst the size of an egg" which he found beneath the dura of this particular patient. Wepfer believed that in this case he was able to demonstrate numerous, multiple ruptures of the meningeal artery. It was Morgagni in 1747 in a report of a similar case (Putnam 55) who first described a sac containing the blood and expressed a doubt as to its formation. He, however, finally came to the conclusion that it was composed of arachnoid. At this same time there came to be many cases of the same sort reported, mostly by French and Englist pathologists. Thubert in 1822 (Putnam 55) was the first apparantly to describe a lesion in which symptoms had not occurred until nine years after the trauma which was probably the main factor in the production of the collection of blood beneath the

dura. Bayle in 1826 has described twelve cases and in them he has pointed out the similarity between the organized membranes on the inner surface of the dura and the membraneous envelops containing the fluid or clotted blood and the skin. Bayle believes that the laminated appearance of the lesion is due to repeated hemorrhages and he places the hemorrhage between the parietal layer of the arachnoid, a layer which was first postulated by Bichat, and the dura. (Putnam 55) Hewitt (Robertson 57) in an article entitled "On extravasations of blood into the cavity of the arachnoid" published in 1845 believed that the membrane which covered the clot was the direct result of the effusion of blood into the space and not a part of the arachnoid or dura. He was also of the opinion that the extravasation might well be caused by traúma. Heschl in 1855 (Huegenin 32) very definitely opposed the idea that the hemorrhage was the primary factory in the formation of this pathologic picture but believed instead that an inflammatory membrane preceeded the hemorrhage.

It was Virchow, (Huegenin 32, Robertson 57, Putnam 55) the great German pathologist, who first accurately described the pathology after post mortem examinations of many patients almost all of whom died in asylums of one sort or another for patients with nervous disorders.

He believed that the primary process was the exudation of a thin film of fibrin upon a chronically inflamed dura. With the continued formation of the exudate, capillaries and fibrous connective tissue were stimulated to grow out into it and so organize the film of fibrin. This in time became a very vascular irritative membrane or pseudo-membrane. Under the influence of this hyperemic condition many of these capillaries or small vessels may rupture producing small ecchymoses which may absorb leaving behind them a certain amount of pigment, or they may not absorb and a clot of blood will be found on the autopsy table. He also postulated the occurance of large hemorrhages between this false membrane and the dura giving rise to his pachymeningitis hemorrhagica interna. The fibrinous exudate with its organization he called merely "pachymeningitis interna chronica" and blood sacs or cysts he called "hematomas of the dura". Although he recognized the possibility of trauma playing an active part he considered it quite minor and definitely believed the primary pathology was one of organization of a fibrinous exudate.

Kremiansky in his article in 1868 (Putnam 55) believed the middle meningeal artery to be always engorged in these cases "due probably to a generalized

diseased condition". The engorgment he stated was the chief etiologic factor in the production of the fibrinous exudate and vascular organization, agreeing with Virchow as to the primary condition of a lesion which is to puzzle investigators for a number of years to come. Kremiansky also recognized the possibility of local irritation playing a part as well as a systemic disease.

In 1872 Sperling (Nothnagel 53) in a dissertation on "pachymeningitis hemorrhagica" (so-called) readopted the earlier view that the hemorrhage was the primary factory and the formation of the pachymeningitic pseudomembrane was due to the organization of the extravasated material. This work was based on experimental evidence gained from injecting whole blood beneath the dura of dogs. After such a procedure, Sterling was able to demonstrate the existence of pseudo-membranes, as described by Virchow, in his experimental dogs. Huegenin (32) also refused to accept the inflammatory theory as to the development of the pachymeningitis but believed rather it was the rupture of veins as they passed from the cortex of the brain to the superior longitudinal sinus, a theory at that time which is now more or less generally accepted as a fact. He also stated that pachymeningitis had been found in healthy individ-

uals with a history of some previous traume. Huegenin writes that due to atrophy of the brain, a condition commonly found in those patients autopsied because most were from the insane asylums, there was a tendency toward the formation of a vacuum which was compensated for by an increase in the serous fluid of the meshes of the pia and in the cerebral cavity with possible rupture of the small capillaries.

Wiglesworth (66) in 1892 is firmly convinced that the primary pathology is one of hemorrhage, following which organization and membrane formation takes place. He feels that if the membranes were primary it would be possible to get the membrane without the blood but never the blood without the membrane and he makes the statement that such cases as the latter have been found. He also refutes the idea of a chronic inflammation of the dura, there being no inflammation there in the cases he has seen. Wiglesworth feels, as does Huegenin, that brain degeneration and atrophy play a large part in the process. The former states that the main factors leading to the pathology of the above condition is loss of support experienced by meningeal vessels coupled with a localized or generalized congestion of the meninges. Wiglesworth believed as did Bayle that the laminated appearance was due at times to fresh hemorrhagic films

of fibrin being superimposed upon those of a preceeding date.

Ford Robertson (57) describes in a beautiful manner the histologic process in the formation of the membrane.. A description which Putnam in 1925 (55) still calls one of the finest descriptions to date. He describes the blood as extravasated from a pial artery. Active proliferation of the cells of the endothelium or mesothelium, as it is often called, lining the subdural cavity caught in the mesh of the clotting blood begins an organization, forming the membraneous sac so often referred to as the false membrane.. He, that is Robertson, believed that the increase in size of the sac is entirely the result of hemorrhage and hemorrhage alone. Ford Robertson quotes G. M. Robertson who describes a thoery of hematoma formation known as the "dry cupping theory". He states that the relatively negative pressure which might possibly occur in the surface of the dura as a result of discrepancies between the cerebral and the systemic blood pressures could cause engorgment and rupture of the delicate subendothelial capillaries with an extravasation of blood into the subdural cavity.

Some rather interesting experiemental investigation has been done by Barrett (3) in 1902 concerning the etiology of the lesion under discussion. He took smears,

sections, and cultures from subdural hematomas in an effort to establish a bacterial etiology. All such laboratory procedures were quite negative. He also attempted innoculation of cats but without any positive results. Barrett often found intravascular clots in some of his cases within the large thin walled spaces and believed these causing an obstruction were the main factors in eventually bringing about the rupture of the vessel with increased hemorrhage.

Trotter's article (63) mentioned in the introduction was rather epoch making in the history of chronic subdural hematoma. In the first place Trotter believed that internal hemorrhagic pachymeningitis was a term which "involves an unjustified hypothesis", that of chronic inflammation of the dura with exudative lesions as the primary step in the formation of the pseudo-membrane, and he felt that it should be definitely discarded. The explanation which Trotter gives for comparatively uncommon first or irritative symptoms is one which has to do with a physiological compensation for one of the three causes of symptoms in cerebral compression. He believes these symptoms are due to first physical compensation on the part of brain tissue to account for increase in intracranial pressure; second, venous congestion; and thirdly, a capillary anemia with a re-

sultant ischemic condition of the nervous tissue. Taking for granted then that this condition develops slowly, there is a physiological compensation for the above three thus allowing for the time interval between the causation and the symptoms. Trotter definitely places the points of hemorrhage in the cerebral veins passing from the cortex of the brain to the tributaries of the superior longitudinal sinus. This work of Trotters was more or less overlooked until the next decade due probably to the advent of the first world war which held most of the interest of all the world at that time.

Sachs (59) in a review of several years experience with brain tumor suggested in 1920 that injuries at the birth of infants who survive may in some manner form the foundation of future chronic subdural hematomas. In 1924 Stephenson (62) reported a case which clinically resembled the so-called pachymeningitis in a patient who was a known syphilitic at the time. Stephenson believes that the condition was directly due to his syphilis.

An excellent article was published in 1925 by Putnam and Cushing (55) which consists of a complete review of the literature, etiologic and pathologic pictures, and a review of several cases with a careful analysis of each. Putnam arrives at the conclusion that there

are two types of the lesion both, however, which occur primarily as hemorrhage which later becomes organized. He describes a spontaneous and a traumatic type and is able to separate them on the basis of their pathological picture as seen in the microscopic study. The main difference Putnam believes is the presence of giant capillaries which are connected with one another in the spontaneous type, in contradistinction to the finding of large mesothelial-lined spaces, which are connected with capillaries but which contain few, if any, erythrocytes, in the traumatic type. Although Putnam emphasizes the above description, other authors do not report the same as found in their histological findings but merely quote Putnam as their authority and consider that quite enough.

Surgeons have noticed, upon trephining, the presence of a peculiar yellowish green tinge beneath the dura in the region of the hematoma. Craig (10) in an editorial in Surgery, Gynecology, and Obstetrics in 1928 makes the statement that the mesothelial cells found lining the spaces have been classified as a part of the reticulo-endothelial system and as a result have the power to react on the hemoglobin present. This inter-reaction breaks down the pigments of the blood in much the same manner as do the Kupfer cells of the liver and the result-

ant constituents are the biliverdin and bilirubin thus giving to the contained fluid the above described color.

In 1932 Gardner (16) wrote concerning the latent interval. He believes that there are two possible explanations of this phenomenon. First that the granulation tissue which constitutes the outer wall of the sac may be the seat of repeated hemorrhages from the capillary source or from the large mesothelial-lined spaces, and secondly that the encapsulated clot undergoes partial liquefaction with a resultant fluid high in protein molecular content, thus increasing the osmotic pressure of this fluid. An osmotic interchange taking place across the membrane with enlargement of the fluid contents increases the intrcranial pressure and gives the symptoms which come after the latent period. Merely a brief review of the conclusions are here shown and will be discussed at some length in a discussion of etiologic factors.

An exceedingly important paper by Munro and Merritt (50) was released in 1936. They gave a pathological description of one hundred and five cases as found at surgery and grouped them according to time after injury or trauma. Their carefully worked out pathology and experimental studies give us a rather fine understanding

of the progressiveness of the lesion.

Recent work by Hannah (28) and Kaump (40) would place the initial hemorrhage within the dura and a secondary formation of a false membrane on the inner surface of the dura not in contact with the clot. Secondary hemorrhage into the membrane they believe gives the classic picture of the "pachymeningitis hemorrhagica interna chronica" or chronic subdural hematoma.

With this review of the literature giving some understanding of the history of the lesion and a bit of the present concept, it is now the purpose of this paper to discuss at some length etiology, diagnosis, and treatment as it is now thought of by the majority of authorities.

ETIOLOGIC FACTORS

There has been a great variance of opinion as concerns the progressive pathologic picture, that is from the initial hemorrhage on through to the final hematoma, in contradistinction to the rather well described pathology met with at surgery and on the post mortem table. Although, as has been stated before, Virchow was somewhat inaccurate in his etiology of the lesion, his description of the histo-pathology as he found it at autopsy has well stood the test of time and but little has been added or taken away from his original description.

Under some discussion has been the source of the bleeding, the formation of the membranes surrounding the hematoma, the organization of the lesion, the failure of clot formation in some instances, and an explanation of the latent interval during which time few or no symptoms may be recognized. It is the plan of this portion of the paper to present the views as given by the men who are working on the problem and then an attempt will be made to sum up somewhat the points on which there is the greatest agreement, if any.

Primarily we are of course interested in the initial factor which provoked the hemorrhage. On two ab-

solutely opposed positions we have first the attitude that the lesion is primarily an inflammatory lesion as described, again by Virchow (Huegenin 32) and by Heschl (Huegenin 32) and has its beginning in an inflammatory exudation followed later by hemorrhage due to diseased blood vessels. On the other side are the majority of writers and especially of the present day authorities (Putnam 55, Gardner 16, 17, Munro 50, Gross 23, Keegan 42) and others who believe that the hemorrhage is the initial step followed by organization of the clot. As to just what brings on the hemorrhage will now be taken into consideration.

Trotter (63) makes the statement that internal hemorrhagic pachymeningitis is a term which "involves an unjustified hypothesis" and should be discarded. He is willing to account for the presence of chronic subdural hematomata on a traumatic basis and on a traumatic basis alone. He also believes that the lesion which Virchow has described is the same lesion which he calls chronic subdural hematoma and is in no way related to any primary or chronic inflammation of the dura. There have been, however, and are now many men who believe other factors are the provoking factors which bring about the hemorrhage. Gilman (18) reports seven cases of subdural hemorrhage the origin of which was

proved to be scurvy. The weakened condition of the capillary bed due to the avitaminotic process caused their rupture and the extravasation of the blood subdurally, thus promoting the formation of the organized subdural hematoma in a scorbutic patient. Ingalls (34) also reports a case of chronic subdural hematoma in a patient with scurvy and makes the statement that in scurvy a "hemorrhagic diathesis" is present and much less trauma is needed to precipitate a hemorrhagic attack then in a normal individual. In his article he states that chronic subdural hematoma, especially in children, is associated with poor hygiene and disturbance of nutrition in one form or another. He quotes from Doehle and Rosenberg. The former states that in fifty-seven cases he noted the presence of rickets and felt that it was an etiologic factor. Rosenberg after a long experience says he has never seen the lesion occur in a healthy, normally-developed, breast-fed child.

In 1934 Russel (58) published a paper in which he describes a case seen by him where the primary factor was a carcinomatose process. He states that the veins in the dense layer of the dura contained masses of tumor cells and engorgement followed, associated with edema, thus causing a rupture of the capillaries and a diffuse

extravasation of blood in the areolar layer of the dura and into the subdural space. Clein (6) writes concerning an infectious theory which was first described by Finkelstein. The process as discussed in his article apparently occurs more frequently between the ages of three to five months than it does after the age of one year. Finkelstein believes that following a rhinitis, these infants develop a retrograde thrombosis of nasal and ophthalmic vessels, eventually leading to a partial thrombosis of the cavernous sinus with a resulting rupture of the small capillaries in the dura due to passive congestion.

A great deal of interest was aroused in 1925 by the report of Putnam and Cushing (55). In their work they have divided the chronic subdural hematomata into two groups, and differentiate them on the basis of the histological picture. Their first group is the traumatic type in which they describe large mesothelial-lined spaces which connect with the capillaries but contain few or no red blood cells. Their second group is the spontaneous type in which they found giant capillaries connected with one another, within which were numerous blood elements. Griswold and Jelsma (22) two years later make the statement that features such as the development of endothelium, which has been consider-

ed as differentiating the chronic subdural hematoma of traumatic origin from the pachymeningitic or spontaneous type are rather differences in degree of organization than differences in type.

Most authorities feel that the majority of the chronic subdural hematomata are the result of trauma and usually of very slight trauma (Coblentz[7]). Furlow (14) places the history of a trauma as the most important factor in the final pre-operative diagnosis of a hematoma. Just why such lesions should follow very slight injury has been most reasonably explained by Keegan (42). He states that injuries which are severe enough to produce even a transient unconciousness are quite likely to be followed by a very appreciable amount of edema of the brain as seen by the signs of increased intracranial pressure. Such edema would tend to compress the subarachnoid and subdural spaces and would undoubtedly favor closure of the torn veins long enough for a relatively firm thrombus to form and prevent extensive or recurrent bleeding. Likewise, the absence of edema of the brain following mild injury of the head might presumably permit some subdural venous bleeding. Griswold and Jelsma (22) say that it is probable that most if not all cases of so-called pachymeningitis hemorrhagica of the spontaneous type have a

beginning in a forgotten injury. They also feel that the presence of so many of these in psychotic individuals in asylums may very likely be due to the fact that these people undergo more trauma because of their abnormal mental state. The condition need not be associated necessarily with brain atrophy as Oppenheim (Trotter 63) has suggested.

Of interest to investigators for some time has been the question of the point of bleeding. From just which vessels does the hemorrhage occur and if always from the same, why? Although this problem may be considered as quite well answered there are one or two recent men who are not at all in accord with the idea which shall be presented here. However, the work of these men will also be presented making it somewhat difficult for us to arrive at too definite conclusions.

Trotter (63) gives the site of the initial bleeding as the cerebral veins which pass from the brain to the tributaries of the superior longitudinal sinus. Gross (23) and Grant (20) are also in accordance with Trotter's view. They report that these veins running from the cortex to the longitudinal sinus are straight and extend unsupported across the subdural space at right angles to the cortex and sinus. One end fixed to

a rigid dural sinus and another to a movable cerebral hemisphere gives an explanation of how trauma in causing movement of the hemisphere might tear across these short veins and so give rise to subdural bleeding. Keegan (42) reports a case in which intermittant bleeding from an opening in the superior longitudinal sinus was actually seen during an operation some two and one half months after the primary or initial injury to the head had occurred.

Kaump (40) in a quite recent article has described the histology of the dura mater as a three layer organ, previously described by Hannah (28), and maintains that the bleeding in chronic subdural hematoma takes place within the vascular middle layer, pushing ahead of it the inner layer. The inner layer as he described it is a loose fibrous connective tissue structure with scanty blood supply and covered with a thin flattened layer of fibroblastic cells. He believes that this inner layer is the limiting membrane of the intradural hematoma until rupture occurs and the blood, or blood from other points of bleeding, comes to lie extradurally. He maintains that the false membrane is formed as the result of the internal dural bleeding and the picture obtained on the surgeon's table is due to the rupture of the inner dural layer or to secondary

hemorrhage into the false membrane. It is his contention that the organization of the clot has its origin in the thickened edematous fibroblastic layer lining the inner portion of the dura. The primary hemorrhage within the dura may continue to bleed as more and more vessels are ruptured as the inner layer is forced away from the vascular middle layer.

One of the outstanding features of the chronic subdural hematoma is the presence of late symptoms. Symptoms which may occur days, weeks, months, or even years after the initial injury have been reported. Hulke (33) reports one case in which there was a ten day interval. Keegan (42) reports a case which did not come to surgery until two and one half months after injury. Lyerly (47) reports a case in which there was an interval of nine months with very few symptoms. Thubert (Putnam 55), as was given in the review of literature, reported a case in which there were but few symptoms until nine years after the first causitive factor. This chronicity, so-called because the symptoms manifest themselves some time after the trauma, has been the problem of a good many writers and the question involved in a great deal of research. The interval between the injury and the final accumulation of symptoms which lead to a diagnosis has come to be known as the latent period.

(Gardner 16). It is this period of latency with which this paper will next deal, and it is hoped that we may arrive at some real conclusions concerning its presence. An understanding of it should lead one into a realization of what one might expect in the way of surgical pathology. Why symptoms of a rather severe type should come on so late after the trauma is the question which this paper shall now make an attempt to answer.

There are several statements made which concern this problem and which are quite dissimilar. However, it is well to understand all sides of a question before making any real decision. Dandy (11) in his article in the Dean Lewis Practice of Surgery asks his reader,

> "How can we explain the fatal course and progressive course when there is a sharply defined and rigid wall surrounding the hematoma which makes it impossible for the original boundary to change?"

His answer is one of cerebral edema which results from the effects of the great mass acting over a length of time as an irritating foreign body. Dandy then would have one believe that the latent interval is due primarily to a large blood clot acting in an irritating manner to the brain causing an edematous condition of the nervous tissue thereby increasing the intracranial

pressure and giving rise to the symptoms as seen and recognized by the clinician. Dandy's idea would seem fit, then, the rather easily understood statement in the introduction wherein we spoke of chronic subdural hematoma as "the formation of an irritating hematoma which over a length of time might be expected to react in some way to its environment". However, the cases which have come to surgery do not seem to bear this out. White (65) states that although the brain is usually much compressed it is not congested and appears perfectly normal. Putnam (55), Munro (51), and others have called attention to edema of the brain following release of tension with the removal or evacuation of the clot but not before.

Kaplan (38) believes that the latent period in the great number of cases can best be explained by slow and intermittent venous bleeding into the subdural space. Gardner (16) says that although the above is the most obvious explanation why is it that hemorrhage acts this way in no other part of the body. Another explanation which Gardner gives is the presence of repeated hemorrhages from the granulation tissue which constitutes the outer wall, either from the capillary source or from the large mesothelial-lined blood spaces. Brodie (4) also believes or did in 1929 that there was a post trau-

matic slowly progressive venous effusion into the subdural space. Until 1932 the theory of increased intracranial pressure over a rather indefinite length of time was explained almost solely upon this progressive venous bleeding theory, and was accepted by the greatest number of authorities.

It was in the year 1932 after some very fine work that Gardner (16) proposed the new explanation for the latent period. He believed that the encapsulated clot, which is probably present in an early subdural hematoma underwent a somewhat gradual and partial liquefaction. This partial decomposition of the clot increased the protein molecular quality of the blood thus increasing the oncotic and osmotic pressure. He, therefore, maintained that the inner lining false membrane acted as a semi-permeable membrane with the liquefied clot on one side and the spinal fluid in the other. Since the protein content on the side of the clot was greatest, there was a passage of fluid into the area of the hematoma, increasing its size, and since there was no escape the hematomatous area began to compress the brain and increase the intracranial pressure. Since liquefaction of the clot took some time the increased pressure would not be noted for a rather long duration of time and this Gardner gave as the explanation of the latent interval.

After Gardner had considered the possibilities of such a theory he set about to prove it. Feeling it was rather difficult to inject the subdural space of men he used dogs in his work. In a series of eight dogs he placed in the subdural space collodion sacs of known weight which contained blood from the femoral vein. Gardner felt that since collodion was a dialyzing membrane there would be a passage of the cerebrospinal fluid into the sac. Since the protein content of the sac would not pass out as it is not dialyzable he felt this would show how increased intracranial pressure might arise. After watching the dogs from three to eight days, during which time they gained thirty nine to one hundred and three percent of their weight and no symptoms of pressure had arisen at all, he came to the conclusion that it is difficult, if not impossible, to reproduce in dogs the clinical picture of subdural hematoma. There was still, however, the possibility of proving that the membrane was a permeable one. Thus he took a portion of an inner cyst wall removed from a patient at operation twenty and one half months after trauma and proceeded to use it in proving that this inner wall did act as a selectively permeable membrane which increased the size of the hematoma as its protein molecular content became greater than that

of the surrounding cerebrospinal fluid. Gardner placed seventeen cubic centimeters of the fluid contents of the hemorrhagic cyst and dialyzed it against fifty-two cubic centimeters of the patient's spinal fluid using the cyst wall removed at operation as a dialyzing membrane. When the hemorrhagic fluid was examined at the end of sixteen hours it had increased twenty-nine percent of its volume. The total protein of the surrounding spinal fluid as estimated by the Kjeldahl method was the same after the sixteen hours as it had been before. It may be understood then that the membrane was not permeable to the protein molecule and since the molecular content of the hemorrhagic cyst is greater than is the protein or molecular content of the spinal fluid, one would expect the fluid to pass into the cyst, increasing it in size. Thus under the pathological conditions as seen in the patient by the clinician, the latent interval, after which signs and symptoms of increased intracranial pressure become evident, comes into its own as an understandable sequence of events due to, not continued bleeding in the main but due to the pull of osmotic or oncotic pressure on the fluid outside the cyst as the clot liquefies and increases its molecular content.

Zollinger (69) reporting on protein determinations

which he made on whole blood and the fluid of subdural hematomas agrees absolutely with the conclusions of Gardner. He makes five conclusions which though they have been stated before it would be well to emphasize here. First, the walls of the subdural hematoma act as a semi-permeable membrane. Second, the blood within a subdural hematoma breaks down slowly over a period of months. Third, disintegration of blood produces a great rise in its effective osmotic pressure and the blood within the subdural hematoma is progressively diluted. Fourth, the resultant augmentation in the size of the lesion causes a rise in the intracranial pressure and the progression of clinical symptoms. Fifth, the late onset of symptoms is therefore directly dependent upon the slow disintegration of the erythrocytes encapsulated within the membrane of the hematoma.

A discussion of the pathological picture which is met with in the various stages of the chronic subdural hematoma as seen by the surgeon and the pathologists should prove of some interest, and it is our purpose to here give these findings. An attempt will also be made to a lesser or greater degree to explain why such a picture does make its appearance. The great majority of workers up until the time of Hannah (28) in 1936 were very much in agreement as to the pathology present.

Hannah, however, has brought forth a new theory which he attempts to prove on the basis of three cases and furthermore he is definitely upheld in his conclusions by Kaump (40) two years later who made a study of some thirty cases. The process which they describe and which they maintain is the true one will be more fully discussed but it is our feeling that other work done previously can not be overlooked. It is this earlier work on the pathology which is to be discussed.

Munro and Merrit (50) made a rather exhaustive study of the surgical pathology of chronic subdural hematoma and have based their findings on a careful analysis of one hundred and five cases which they have examined. Their descriptions of the process as it occurs at varying periods of time throughout the possible existence of a hematoma are most interesting as well as scientific. Their first case was examined two hours after injury and their last two years after injury. It is rather difficult perhaps to understand what part surgical pathology of a two hour period could play in the history of a chronic subdural hematoma as it could not yet be classified as chronic. Yet, if we are to believe Von Storch (64), it most certainly does have a place. Quoting from Von Storch:

> "It is now generally accepted that the pathological picture of chronic subdural hematoma

> is but a late varient of the acute stage or (phase) and that at the late stage the hematoma may be liquid or solid or both, depending upon the osmotic inter-relationships that have been formed".

Munro and Merrit are in complete accord with Von Storch as they show in their study, as well as in the statement they make that the conception of the chronic subdural hematoma as an entity should be abandoned, and the lesion recognized as only the late stages of a previously undiagnosed acute process.

With this as an introduction to these two workers paper, the pathology as they found it, together with that found by other men, will be discussed giving a better understanding, at least for the present it is hoped, of just what does take place after the initial hemorrhage.

These authors divide their hematomata into three groups. The first group is the solid hematoma in which blood is deposited within the subdural space and the organization begins at the periphery and extends into the center. If this organization is rapid and efficient and the clot is not too thick it may remain in just this state for several years. It rarely, if ever, becomes calcified. If the organization is less rapid and less efficient the center remains partially liquefied and a local necrotic process sets in. None of the solid

clot will absorb the cerebrospinal fluid, and since only the center is of a fluid nature this type of hematoma is non-expansible unless there are repeated hemorrhages into the same site. The second type is the mixed type in which there are varying amounts of gross blood or clots and cerebrospinal fluid. The breakdown of the clots into protein molecules has been previously stated and this type of hematoma will increase by osmotic pressure drawing into it the surrounding cerebrospinal fluid of the subarachnoid space. The third type is the fluid hematoma which appears for the greater part as cerebrospinal fluid, the blood having become completely dissolved.

This dissolution of the blood as described first by Gardner forms for the above two workers another problem and they have gone much farther than did Gardner as they postulated the breakdown of the protein into amino acids thus increasing the osmostic pressure to a much greater extent by definitely increasing the molecular content. In their study they showed a very definite increase in the protein of the hematoma for the first few days, which they attributed to the continued bleeding. After the first few days they found a very definite decrease in the amount of protein present but a correspondingly great increase in the amino acid content. Their protein determinations were made by the

methods of Denis and Ayer as described in the Archives of Neurology and Psychiatry 26:1038 November 1931. Thus we have here a much more impressive series of events than those shown by Gardner. It is well known that the smaller the molecule the greater will be the number and the much greater the increase in osmotic pressure activity. With the breakdown of the protein into amino acid molecules we have present a very definite factor in the production of the late symptoms since Gardner has already shown that the membrane separating the two fluids is a semi-permeable one, which by definition is one through which only water can pass. With the realization of just what process is present to account for the symptoms of increased intracranial pressure we may now continue with a discussion of the formation of the permeable membrane which will more or less complete the picture.

The following pathological description is the one as given by Munro and Merrit to explain the process as it occurs in the solid hematoma. The introduction of other worker's findings will also be included but when so done it will be specified as such.

The first cases examined as to early findings consisted of a group of six which came to operation within twenty-four hours after injury. Some fibrin,

as would be expected, was collected around the edge of the clot and already fibroblasts from the connective tissue of the dura were beginning to migrate around it. Here we are seeing the initial effort on the part of the dura to remove or at least render as unirritating as possible the clotted or clotting blood. Dandy (11) has said that the blood in subdural hematomata has lost a good bit of its power to clot because the fibrin has been used to form the membranes. Other writers do not corroborate this and do not mention a decrease in the clotting power. After the twenty-four hour period until the fourth day it has been found that beneath the dura there is a fibroblastic layer which has formed a definite membrane. Gross (23) states that there is no absorption in the subdural space and for this reason the clot is rapidly encysted as the fibroblasts of the dura attempt to wall off this mass from the surrounding normal tissue in order that it may not interfer with the normal physiology. Between the fifth and the eighth days as found in a series of seven cases there is an increase in the thickness of the layer of fibroblasts and by the eighth day a very definite neomembrane has surrounded the clot and there is extension of fibroblasts into the clot. By the eleventh day the blood is almost completely liquefied and

and is broken up into islands by numerous strands of frbroblasts extending between the inner and outer membranes. In twelve cases studied, which came to operation thirteen to seventeen days after trauma, almost all of the blood had been absorbed or at least had disappeared and between the eighteenth and twenty-sixth days the outer formed membrane was as thick as the dura and the inner was about one half the thickness of the dura. There were numerous phagocytic cells around the dura. After six months and until one year after trauma in a series of some fifty cases there was found a thick fibrous neomembrane from which all blood was gone except for a few isolated pigment containing phagocytes. In three cases seen after one year the membrane beneath the dura was histologically almost indistinguishable from it. There was much hyalinized connective tissue and very few fibroblastic nuclei. The differentiation from the dura was possible because all of the fibers of the neomembrane were parallel to one another while those of the dura are seen to run in every direction.

From this picture it is possible to conceive of a clot formation which in time might become completely absorbed in the process of its organization leaving no trace of its presence except for a thickened dura which

would be found only at autopsy. As Munro and Merrit state this should not be called an entity in itself but should be recognized as one of the late stages of an acute process the other late stages being the mixed and fluid hematomata in which are found the marked late symptoms of chronic subdural hematoma.

Rather recently or at least more recently than the above work, there has been some work reported which would lead one to think possibly that the explanation of chronic subdural hematoma was not a settled one and that even the vessels which have been thought to be the source of bleeding may not be at all. Before, however, taking up these cases a review of a case which was rather thoroughly studied in 1927 is to be considered. It is because of certain statements made in the study of the microscopic studies which seem to conform to this later theory that this is being done.

Lord (45) reports a case of apparantly spontaneous subdural hematoma. No history of any trauma whatsoever could be elicited and for this reason it was called spontaneous although some men believe that trauma must have been present sometime (Trotter 63, Griswold 22). The clinical picture presented the possibility of a subdural hematoma and upon exploration this was shown to be the case. The following pathological report was

given. There was a distinct membrane which separated the clot from the pia-arachnoid, and a rather less distinct membrane separating the clot from the dura although the latter was much more vascular than the former. The microscopic picture showed the choroid plexus to have many thrombosed small arteries. The membrane on the arachnoid side was composed of fairly old well-formed fibroblasts between which were large numbers of giant capillaries lined with endothelium. The separation of the membrane from the clot was very clear and distinct. There was no apparant organization spreading from the membrane into the clot. This rather sharp demarcation of clot from membrane with the lack of organization from membrane to clot is the interesting point which it would be well to dwell on in the discussion of the following cases.

It was Hannah (28) in 1936 who upon the basis of a careful study of three cases took it upon himself to change somewhat the prevailing idea concerning chronic subdural hematoma.

Hannah describes three very definite and distinct fibrous connective tissue layers of the dura. The outer layer lines the skull and is composed of very dense fibrous tissue. The middle layer is a looser more areolayer type of connective tissue which is the vascular

layer, composed not only of capillary net works but also of numerous endothelial-lined spaces which may appear either empty or filled with blood cells (Kaump 40). The third layer is separable from the remainder of the dura and is lined on its inner surface with endothelium. It is Hannah's contention that the initial hemorrhage in so-called subdural hematoma occurs in the vascular layer of the dura and pushes ahead or separates the inner layer from the rest of the dura forming a sort of bleb or blister on the inner surface of the dura. Hannah found upon injection into this space that strong pressure failed to rupture this inner layer and produced only these blebs, spoken of above, until some other route of escape developed. As the hemorrhage, which he believes is petechial, pushes ahead the inner membrane, there is a tendency to rupture more capillaries thus increasing the size of the intradural hemorrhage. Hannah states that if the initial hemorrhage were subdural there would be a tendency for the blood to gravitate to lower levels instead of almost always remaining more or less in one position. The greatest majority occur, he says, superiorly and he would expect them to migrate and not be localized. However, such are not the findings of the surgeon, for chronic subdural hematomata are always well delimited

lesions.

The neomembrane which can be proved histologically to be present and which for so long has been believed to be a reaction to the clot and to grow around it is not so explained by Hannah. His explanation is that the neomembrane is the result of or the reaction to the petechial intra-dural hemorrhage and occurs on the free edge of the inner layer, which layer separates it from the clot proper. With the resultant break down of the thin inner layer of the dura or the presence of secondary hemorrhage into a false membrane which has already been formed and which offers osmotic relationships between the blood encysted and the spinal fluid, comes the picture of subdural hematoma as it is seen at autopsy or on the operating table.

Hannah's work is based, as has been stated, upon three cases which he studied. The first two are of an intradural nature, the second showing the formation of a false membrane which is entirely separated from the intradural collection of blood by the third layer of the dura. The third case Hannah believes is the result of a secondary hemorrhage into the false membrane and shows a rather typical picture as it has been previously described. The lack of the fibroblastic extension into the clot and the compressed loose network

of the neomembrane offer proof to Hannah that the hemorrhage he has here found is secondary to the formation of the membrane. In view of these findings it is suggested by him that the best name for the condition is that of "hematoma durae matris" which was originally employed by Virchow.

Kaump (40) agrees in every detail with Hannah and shows in his thirty cases pathological pictures which do coincide with the above theory insofar as Kaump is concerned.

Thus it would seem that in this portion of the paper in which we have attempted to deal with etiology we have very nearly gone in a complete circle. Virchow believed that the membrane was primary and the hemorrhage secondary. Other investigators after him set about proving that the hemorrhage was the primary pathology and the membrane formed as a direct result of the subdural hemorrhage, surrounding the hemorrhage and organizing the clot. Now there is published this latest report of Hannah's and Haump's in which they believe if we understand them correctly that the initial hemorrhage is intradural and although the false or neomembrane is the result of this intradural hemorrhage it is in no way connected with the hemorrhage as it is entirely extradural. Thus not until the neomembrane is completely

formed is it in any way connected with the blood and then only in the event that the inner dural layer ruptures or there is a secondary hemorrage into an already formed pathological tissue layer, the neomembrane. This last would explain the failure of the hemorrhage to migrate or gravitate which so much bothered Hannah. It might also explain the presence of the solid hematoma of Munro in that there had never been anything but intradural hemorrhage for they did not recognize the third layer of the dura. It would not, however, explain the carefully worked out pathology of Munro and Merrit (51) on their own cases. It would appear that more histological work need be done and that more of the early specimens are needed in order to further explain exactly what does take place. The explanation of the latent interval in which there is the greatest clinical interest seems rather well established at this time. The etiology is primarily of academic interest since neither diagnosis nor treatment are directly dependent too much upon the initial pathology. The clinician is far more interested in curing his patient than he is in learning of the why and how.

DIAGNOSIS

An accurate and absolute diagnosis of such a condition as chronic subdural hematoma offers to the neurologist not only many difficulties but also a great many pitfalls in the processes which must be undergone before such a conclusion can be reached. Although for some time now it has been recognized as an entity which must be given adequate consideration in the differential diagnosis of cerebral lesions there are still many questions raised before calling any given cerebral pathology, which makes itself known by certain signs and symptoms, a chronic subdural hematoma. Frequently these signs and symptoms are of such a bizarre nature that it is often wondered whether they are real or functional (Wilkins 67). The neurological signs may vary from hour to hour in a single case thus sometimes rendering impossible any fixed idea concerning a certain set of symptoms which may seem marked now but a short time later will have disappeared (Wilkins 67). Trotter (63) says the patient is very apt to pass from consciousness into unconsciousness and vica versa in a short space of time. Kaplan (38) states that the lethargic state alternating from drowsiness to coma changes within twenty-four hours to periods of alertness and

and surprising response. Although such suggestions do aid very much in making a diagnosis, if they are present, they may be absent and in themselves can not of course be recognized as diagnostic. It is only after a most careful study of many cases that with a grouping together of the findings may one attempt to actually determine certain critera by which one may arrive at a diagnosis of chronic subdural hematoma. Despite the fact that this condition occurs more often than does the much publicised middle meningeal hemorrhage it is frequently unrecognized (Kaplan 38, Wilkins 67).

Although it is not our purpose to herein merely give a group of case reports, we do realize they may be of value and we shall use them to illustrate when it is felt necessary. We should prefer to present certain diagnostic features as given by workers in this field summing up later those points which the greatest number of men feel the most important in their diagnosis of this lesion. Such a problem will be approached from several angles. It is our purpose to take up the discussion from the standpoint of the story as given by the patient or his family, the objective findings of the neurologist, possible laboratory reports which may be of diagnostic value, and the findings of the radiologist.

Such a method of procedure will in some instances entail a certain amount of repetition which may even prove beneficial if only for the purpose of emphasis.

To make a diagnosis of chronic subdural hematoma and prove its presence at surgery without any history of trauma need not necessarily lead one to look for other initial factors concerned in its production. As was stated in the division on etiology some authorities believe that the failure to get such a history merely indicates that the trauma was of so slight a nature that it could not be remembered (Griswold 22, Coblentz 7). The great majority of case reports which one reads in the literature do give a rather definite history of some type of injury be it ever so slight. Jelsma (36), in a study of forty-two cases which he collected from the literature and tabulated gives a very definite history of trauma in eighty-eight percent and a greater percentage of ninety-eight which may well have been traumatic. Thus there was left only two percent with absolute absence of traumatic background insofar as the patient or the family could recall.

According to a great number of men the presence of headache is of great importance as one of the first complaints made by the patient (Furlow 14). Coleman (8) believes it to be the outstanding symptom and says that

it may be unilateral but is more often general and of a persistent throbbing character. Love (46) stresses the presence of headache soon after trauma followed by a period of absence after which time the headaches reappeared and were of a much more severe nature. Jelsma (36) in his series reports an incidence of headache in seventy-nine percent and believes it is of great diagnostic value. Kunkel (43) in a series of forty-eight cases reports the presence of headache in ninety three and six tenths percent. Oekon (54) in his report of a case in which the main symptomatology was that of an acute psychosis without very dominant neurologic manifestations also stresses the presence of frequent and quite severe headaches.

In some instances the symptoms which the family relates to the physician may so far mislead him as to have the patient placed in an asylum because of his psychotic tendencies. Furlow's (15) case report of a man sixty-three years old gives the history of a disease which the neighbors and the family for several months considered on a psychotic basis because of the patient's changes in personality. The symptoms and signs of abnormal sexual practices and slovenliness as portrayed in this patient preceeded by several months the symptoms of increased intracranial pressure by means of which

the physician was enabled to arrive at a differential diagnosis. However, after questioning carefully a very definite history of traum was elicited which had occurred six months previously and which had preceeded all of the abnormal practices and mental aberrations of the patient.

Vomiting, nausea, and vertigo may also play a part in the story as it is given. Kunkel (43) reports sixty-one and nine tenths percent with vomiting and thirty-four percent with vertigo, while Jelsma's (36) cases show only an incidence of twenty-nine percent with vomiting and eleven percent exhibiting vertigo. Kaplan (37, 39), Furlow (14), Craig (9), and others report the presence of vomiting in cases which is usually of a projectile nature. Hall (26) makes the statement that the earlier in life the subdural hematoma developes the more frequent and more severe does the raised intracranial pressure tend to become. He also states that in later years the lessened amount of the increase in the intracranial pressure accounts for the rather vague clinical picture seen. Such a varied history as given by the family and patient, as seen above, may in some cases lead one to a consideration of chronic subdural hematoma which would be confirmed on further examination, but in other cases may merely increase the perlexity

of the attending physician as he attempts to understand a rather bizarre group of complaints.

A careful examination of the patient on the part of the neurologist is the next step in such a procedure if it has been recognized as a neurologic problem. But here there may be signs of such a misleading nature that the pathology will be localized by the examiner on one side of the cerebrum when actually it is on the opposite. Such a discrepancy will be taken into account when the motor signs are discussed.

The finding of eye signs is rather common and constitutes one more important step toward the proper end. Dilatation of the puil may be of some significance. Kaplan (38) believes that unilateral dilatation of the pupils gives a lead as to the presence of central pathology. Wilkins (66) and Daniels (12) also both feel that this is a significant finding since it localizes the pathology on the side of the finding. A much more prevalent eye finding is the presence of choked disc. King (41) in an article on Chronic traumatic subdural hematoma as a cause of choked disc reports in a series of two hundred and twenty-four cases which were reported in the literature an incidence of choked disc in approximately forty percent. That is the largest number of cases reviewed in the literature with any specific

finding in mind as concerns this particular lesion. Jelsma (36) with his series of forty-two reports the presence of choked disc in forty percent while Kunkel finds papillaedema present in fifty-three and two tenths percent in the forty-eight cases which he studied. Coleman (8) reports the presence of choked disc in thirty-three and one third percent after a study of some twenty-four cases. He also points out that a transient diplopia is a symptom which is of great value if present but it makes its appearance in a rather small percentage of cases. The weakness of the extra-ocular muscles according to Daniels (12) is also among the more common findings.

The neurologic findings in a chronic subdural hematoma may be very few and it is rather difficult to localize from them, the lesion. Coblentz (7) believes it is almost impossible to localize the pathology by means of neurologic signs alone. He also says that though pathologic reflexes such as the Babinski and the Hofman are common, they are of no localizing value. Gross (24) states that the pyramidal tract signs are notoriously misleading and will often cause one to localize the pathology on the opposite side of the signs when it really lies on the same side as the findings. Pressure against the opposite pyramidal tract in the region of

the foramen magnum gives this misleading information and must be constantly kept in mind. General weakness, hemiparesis of a spastic nature, increase of deep reflexes and other signs found in upper motor neuron lesions are the ones usually found but as has been said before may lead one to erroneous localization. Few sensory changes over the body have been recorded in the literature and one may theorize from this that the majority of subdural hematomata are anterior or rostral to the somesthetic area or lie laterally over the temporal or silent area of the cortex. Abbott (1) believes the superior temporal region is the region in which to look for these lesions. Keegan (42) uses small frontal or lateral parietal trephine openings for exploration which seems to indicate that the pathology is not usually found over the sensory cortex. The sensory tracts unlike the motor or pyramidal tracts do not lie close to the external portion of the brain stem and thus pressure will not effect them in the same manner as it does the motor tracts. Mental symptoms are such as to make the patient forgetful, disoriented, excitable, and nervous and may be either discovered by the examiner or related by the family (Lyerly 47).

The laboratory is of little value in making a diagnosis in lesions of this type. Love (46) reports the

finding of xanthochromic spinal fluid with a lumber puncture. The laboratory in this case reported the presence of eighty millegrams percent of protein. Kaplan (39) also reports the presence of a xanthochromic spinal fluid but does say that it may often be clear. Martin (48) reports no abnormality of spinal fluid or of spinal fluid pressure as taken by lumbar puncture. Jelsma (36) finds the presence of a xanthochromic fluid in only thirteen percent of the cases he studied and does not consider it of much value as a diagnostic measure.

A rather interesting comment has been made by Rand (56) in his article in which he reviews seven cases. Rand makes the statement that in all of his cases of chronic subdural hematoma there was elicited a decidedly high pitched percussion note over the calvarium. He also states that the note does not appear to be higher on the side of the hematoma, but apparently is rather uniform in its tone over the entire calvarium. Whether such a finding is of importance or not can not be proved on the basis of seven cases but does provide some thought for those who are going to be in a position to see some in the future.

The roentgenogram has proved an invaluable aid in the final step of diagnostic procedure. The use of

anteroposterior and lateral plates of the head, the ventriculograms, and encephalograms have in many instances made possible an accurate diagnosis of the subdural hematoma. Just what findings one might expect with an interpretation of these is the information which is now desired and it is our purpose to present these as they have been given by the radiologist and the neurologist. That there may be some disagreement on any one procedure is certainly to be expected for no one type will yield the exact results in the hands of all investigators when the pathology may vary somewhat.

Flat plates of the head may show some contour changes such as alteration in the shape of the calvarium which may assist in the diagnosis. Hardman (29) gives the following findings in cases of chronic subdural hematoma. He states that there is a hydrocephalic type, in which the skull is much enlarged, which is seen mostly in infants. There may be localized diminution in the size of the skull as well as localized enlargements both of which will appear immediately over the pathology. Fleming (13) believes that the pineal shift seen in this condition is of diagnostic value and stresses the point as does Cabot (5) in a case report. When these pictures are seen they do aid a great deal but so very often one may be unable to detect any alteration in the

shape of the calvarium or to find any shift of the pineal body.

Lindemulder (44) believes that the use of ventriculograms failed to show anything because of increased intracranial pressure with collapse of the ventricle thus allowing no air to pass into the ventricle. He does believe that definite findings are seen with encephalography. Abbott (2) believes there is value in encephalography in that a filling defect in the air in the subarachnoid space may be seen in some cases. Gross (24) and Carmichael (60) both found ventricular shifts away from the area of pathology. Gross (24) states the presence of a depression and a loss of rounded contour of the body of the ventricle on the side of the lesion in a case which he has reported.

Halt (27) has made a rather thorough study of the encephalographic diagnosis and reports early in his article that findings in cases of unilateral subdural hematomata have been reported by several authors. The finding of the wall of the subdural cyst outlined by air is considered by Halt to be pathognomonic of chronic subdural hematoma. When there are small ventricles with a filling of the sulci and the presence of large collections of air over both hemispheres he considers the presence of a bilateral subdural hematoma is rather well

established. If there is a large subdural collection of air over both hemispheres but the ventricles are not seen Halt believes that no reliable encephalographic interpretation can be made. Von Storch (64) believes that encephalography is rarely diagnostic and then only in cases of large chronic clots. He also believes it is unwise to lay too much stress upon the presence of unencysted subdural air as an indication of the presence of subdural hematoma. Von Storch reports four findings in cases which he has described. First, a slight but definite depression of the roof of one lateral ventricle with or without a shift of ventricular septum; second, small multiple or large single cystic areas of subdural hemispheric air associated with a normal or moderately enlarged ventricle and variable amounts of subarachnoid hemispheric air; third, no depression or distortion of ventricle but absence of subarachnoid hemispheric air on one side with contralateral ventricular enlargement; and fourth, unilateral subdural hemispheric air with contralateral ventricular enlargement are the variable findings as reported by Von Storch who believes we may use the encephalogram as an aid but not as an end.

There remains but one more diagnostic procedure which we must discuss. This is one which is absolute.

It consists of a diagnostic trephine with exploration of the dura. Coblentz (7) believes that bilateral trephine openings should be the method of choice since so often localization is impossible or the presence of a bilateral hematoma may make itself known when it was quite unexpected. Keegan (42) makes an exploratory through small frontal or lateral parietal trephines. The presence of a tense dura beneath which is seen a bluish green color is an absolute diagnosis of chronic subdural hematoma. The dura may be incised and there is then no further doubt.

A case of chronic subdural hematoma as may be seen from the above does not lend itself too easily to diagnosis by any set group of signs or symptoms or any cardinal signs and symptoms of unvariability. In the history it is believed that the finding of an initial trauma, with late symptoms of increased intracranial pressure as evidenced by headaches, nausea and vomiting, vertigo, and certain localizing signs such as weakness of one or more extremities and ataxia are the findings which may most likely lead one to a diagnosis. The findings of eye signs such as choked disc, nystagmus, and pupillary dilatation are of value. Pyramidal tract signs, mental aberrations, changes of personality, and psychotic tendencies should all be considered as possible

results of a subdural hemorrhage. The roentgenogram may be of value especially if the presence of a filling defect in the subarachnoid air can be demonstrated. The presence of pineal body shift and of changes in the contour of the calvarium must be considered as suggestive. It is, however, the contention of most writers that an absolute diagnosis of chronic subdural hematoma can be made only by means of the trephine and exploration.

TREATMENT

With a progressively clearer idea of the etiology and diagnosis of any given disease entity, therapeusis becomes of vitally more importance than it is when one is merely treating a patient empirically with no real understanding of the underlying pathological process which is the cause of the patients complaints. Thus it is with the real knowledge of the pathology of chronic subdural hematoma and a clearer analysis of the signs and symptoms of this lesion within our grasp that we may arrive at a definite form of treatment. The treatment of patients who are suffering from the specific lesion which is here under discussion lies essentially in the hands of the neurosurgeon. It is well known that patients with the subdural hematomata have a protracted illness which would frequently end fatally if unrecognized and when treated successfully has an excellent prognosis (Gurgjian 25, Furlow 15, Nash 52). The following is a discussion of the method of treatment as practiced by various men in the field of neurosurgery. All are agreed that the treatment is a surgical problem. The points of disagreement lie mainly in the type of procedure one should follow in draining or removing the clot. There are two main

points of view; one, the use of trephine openings only, for the drainage, and the other, the use of the osteophlastic flap.

The majority of men feel that trephine drainage should be attempted and osteoplastic flaps made when deemed necessary. Keegan (42) believes simple evacuation by sucking through trephine openings and irrigation with normal saline with rubber tissue drainage for forty-eight hours should be preliminary procedure unless a dense organization of the clot is found. He feels that a bone flap is indicated to remove an organized thrombus or to find the source of recurrent hemorrhage if present. The bone flap, however, is not needed for removal of the ordinary thin protective membrane which is usually found surrounding the hematoma. This will disappear in a few months. Harrax (30) on one occasion reflected a bone flap three months after the contents had been sucked out and he found that both the inner and the outer membranes had disappeared. McKenzie (49) feels that the filling of the cavity with saline and the establishment of drainage with a good sized tube not only allows the saline to escape slowly with the obliteration of the cavity but permits complete expansion of the brain. He also states that a large osteoplastic flap may be very dan-

gerous if a bilateral hematoma is present so he favors initial trephining with drainage whenever possible. Fleming (13) suggests that four bilateral trephines be done in such a way that they may be incorporated into an osteoplastic flap if it is thought necessary to have one, and that through and through drainage should be carried out. Woltman (68) reports a case in which two hundred and forty cubic centimeters of dark brown fluid was aspirated from trephine openings and that the patient recovered completely from all of his symptoms except for a slight peroneal weakness which Woltman believed would gradually disappear.

Grant's (21) statement that in a bilateral clot simple drainage is sufficient with through and through drainage agrees with Fleming and McKenzie. Grant also states that if the lesion is unilateral safety and satisfaction have compelled him to perform an osteoplastic flap operation. He has found that if the dura is cut all around and turned over and resutured all of the oozing from its surface will promptly cease. Jaeger (35) has encountered difficulty in pulling off of the capsule in that small vessels are torn, and he believes that preliminary to the craniotomy it is advisable to ligate the middle meningeal artery through a subtemporal decompression opening well down toward the foramen

spinosum. He also believes it is best to leave a subtemporal decompression as the post operative cerebral edema is apt to be considerable. Grant (20) in an early report states that a decompression is a fine way in which to combat brain edema. Most of the recent authors are not in accord with the decompression and feel it is quite unnecessary.

McKenzie (49) found that sometimes the brain would not expand to fill the cavity, a fact due to the adherence of part of the cyst wall to the arachnoid acting as a tight band compressing the cortex. He states that the operator has only to reach through the trephine, sieze and tear this membrane from the cortex and thus allow the brain to pulsate outward and assume its normal position. The advantage of the osteoplastic flap as room for a decompression is one not to be overlooked if the operator feels the necessity of one.

Putnam (55), Munro (51) and others have called attention to the edema of the brain due to the release of tension following evacuation of the clot as a cause of mortality. They believe that post-operative edema should be treated by the injection of fifty percent glucose intravenously given one hundred cubic centimeters at a time. It may be given every two hours as deemed

necessary. As a last resort two hundred cubic centimeters of a two percent solution of magnesium sulfate may be given. This is, however, dangerous and heroic treatment. Munro states and others agree with him that with the patient already in a partial state of shock reoperation is absolutely contra-indicated.

Chronic subdural hematoma then may be treated by simple drainage through trephine openings and the use of oeteoplastic flaps when the operator feels they are needed as in the case of a clot which has rather definitely organized or the presence of thick membranes which must be removed. The use of rubber tissue drainage and the presence of normal saline in the cavity for forty-eight hours are fine postoperative procedures. In the face of postoperative edema the cerebral dehydrating agents such as hypertonic solutions of glucose may be used using a solution of magnesium sulfate only as a terminal act. As has been stated before reoperation for postoperative edema must never be considered.

CONCLUSIONS

1. Chronic subdural hematoma is a clinical and pathological entity which occurs either as a late phase of a previously undiagnosed acute subdural hemorrhage or as a late phase of petechial intradural hemorrhage.

2. The lesion consists pathologically of a neomembrane on the inner surface of the dura attached at its borders to the dura and filled either with a clot in varying stages of organization and liquefaction, or with a fluid mixture of blood and cerebro-spinal fluid.

3. The fluid hematomata increase in size by an increase in their osmotic tension as the clot liquefies and the protein breaks down into amino acid molecules. The neomembrane acting as a semi-permeable membrane allows for the passage of cerebro-spinal fluid into the hematoma within which is the greater molecular content.

4. Any differences between traumatic and spontaneous or the so-called pachymeningitic hematomata are probably those of degree rather than those of type. The majority of "spontaneous" lesions undoubtedly have their origin in a forgotten trauma.

5. A diagnosis of chronic subdural hematoma is not absolute until an exploratory trephine has been done. Subjective symptoms of increased intracranial pressure, neurological findings, and alternating states of alertness with drowsiness do aid a great deal. Very important is the history of an initial trauma. Encephalography may or may not be of value and only with the bilateral trephine is the diagnosis unquestionable.

6. The treatment consists of trephine drainage whenever possible. The osteoplastic flap is used only when deemed necessary, as in the case of an organized thrombus or for the removal of thick newly-formed membranes. The use of saline drainage with rubber tissue for forty-eight hours is also of value. Hypertonic solutions are used to combat post-operative edema of the nervous tissue.

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