KERNIG'S SIGN: ITS PRESENCE AND SIGNIFICANCE IN GENERAL PARESIS AND ARTERIO- CAPILLARY FIBROSIS¹

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Every physical sign in disease, even if not always pathognomonic, is often of value in aiding us in our conclusions in the difficult and at times obscure cases, therefore all of them deserve our attention, and many times our consideration, both in their presence and absence, as helping to make up or modify the symptom complex of disease. As it has always been the clinicians' purpose to know every possible condition in which previous described signs or symptoms might be met with, that he might avail himself of their aid by inclusion or exclusion in his daily attempts at diagnosis of disease in its many and varied forms, I desire to call your attention to the possibility of the further application of Kernig's phenomenon, and at the same time to add additional evidence of its being almost, if not fully pathognomonic of inflammatory changes in the pia mater as first described by Kernig in his original and subsequent description of the sign.

In 1884, Kernig (1) published in the *Berliner klinische Wochenschrift*, a phenomenon which he thought was found only in affections of the pia mater, in inflammation of that membrane, and applied by him in his observations in the epidemic forms of cerebrospinal meningitis, and Herrick (2) says that until Netter, fourteen years later again described the presence of the phenomenon, little or no recognition was given to this most valuable sign. Bull (3), about one year later, in the *Berliner klinische Wochenschrift* stated that it was his belief that it was not pathognomonic of meningitis or even pial trouble, but merely indicated an increase in intra-cranial pressure, and he cited three

¹ Read before the Philadelphia Neurological Society, January 28, 1910.

cases in which he found the flexor contracture sign as proof of these statements as follows: One (1) tuberculous meningitis without autopsy; one of solitary tubercle of cerebellum, but "in the pia over it, a few miliary tubercles" with tuberculosis in the lung, lymph glands, kidney and intestine; one (1) otitis media with caries of the petrous portion of the temporal bone and thrombosis of the transverse sinus and common jugular veins, in the basal pia; especially over the frontal lobe there were ecchymoses, and there were a few teaspoonfuls of serum in the posterior fossa.

These cases, Herrick says, go further to prove Kernig's statement that his sign is found with affection of the pia mater than Bull's that it is not an indication of meningitis, or even of involvement of the pia.

Friis, later in his article also differed with Bull that the phenomenon was due to intra-cranial pressure, and as proof quoted a case of hydrocephalus in which he failed to find Kernig's phenomenon. Later, Netter again writing, stated his belief that its presence permitted one to assert quite positively the existence of a meningitis and from its presence in convalescence, Kernig, Hensch and Netter thought it might be of service in enabling one to make a retrospective diagnosis.

Then follow numerous other writers confirming the value of this sign in the epidemic and tuberculous meningitis, and later Sailer (4) reported three cases in which the sign was present unilaterally in hemiplegia and pneumococcemia, and at the same time Shields (5) reported his observations in one hundred cases, other than meningitis, in which he also found the sign; in three cases the phenomenon being unilateral, and in two cases bilateral; it was present in two uremias, two hemiplegias. Clark's (6) report is included with these; three cases of meningitis in which the phenomenon failed to manifest itself throughout the course of the disease. Later, Wilson (7) in an article upon the negative value of Kernig's sign in 120 non-meningitic cases, quotes 26.8 per cent. of the series, in which the phenomenon could be demonstrated, as against Miller's 28.6 per cent. of similar cases, indicating that there need be no serious brain or cord involvement to produce the phenomenon, and to this I desire to add my observations of the phenomenon in general paresis and in arterio-capillary fibrosis.

My attention was called to the presence of Kernig's phenomenon in general paresis in attempting to reach a conclusion as to the presence of a super-added infectious meningitis in a case of general paresis, and reflecting that as we have a leptomeningitis in paresis, we should also have this phenomenon present. Sometime during the course of the disease and examination of the cases, I confirmed this deduction, and confirmed the observations of Kernig, Netter, Friis and Sailer in other conditions in which the phenomenon was reported, that its persistence varies when frequent observations are made in individual cases; the degree of contracture also varies at times. And here I might further add that the phenomenon may be used as an indicator of the progress of the disease, as it appears with more regularity and also a greater degree of contracture as the disease progresses, and surely remains more constant in the later stages than in the earlier.

With a view of attempting to satisfy myself as to the conditions that might account for the presence of so large a percentage as recorded by Miller and Wilson in their reports of supposed normal individuals who presented this valuable phenomenon, I decided to examine normal cases, normal in the sense that they presented no apparent clinical evidence of meningeal involvement, and in so doing my attention was early directed to the presence of the sign in senility as compared to earlier life, and with further examination and observation I found that in no case where I could exclude fibrosis by palpation of radials, palpation and observation of visible or tortuous temporals, could I demonstrate a true Kernig phenomenon.²

Owing to many new problems arising, I can only make my report preliminary to a more careful and detailed study of this valuable phenomenon in arterio-capillary fibrosis. The problem of deciding why certain cases of fibrosis should present the

² One should avoid confusing a *voluntary Kernig sign*, "a coined term for benefit of description" with the true Kernig phenomenon. A voluntary Kernig sign is present in negativism of the insane, and also present in the cases where the patient fails to cooperate with the examiner. Simple methods of excluding this are by the amount of force with which the leg rebounds when pressure upward is suddenly withdrawn, most marked in the voluntary Kernig state, little resistance noted in true Kernig phenomenon; by marked resistance in voluntary sign in attempting to elicit same, and by the presence of the Kernig phenomenon when the thigh is only partially flexed, "not at right angles." Complete extension of leg is readily obtained in this position in true Kernig's state. Kernig's phenomenon persists in voluntary Kernig's state.

phenomenon, others not, confronted me. Was it an evidence, as Bull claimed, of pressure, as would result in contracture as the result of cerebral fibrosis, and would the phenomenon be the key in deciding the amount of cerebral involvement of fibrosis of the vessels with its consequent atrophy? Here I feel that Somer's admirable work in the abstract compilation of 1,180 autopsies at the Norristown (9) Asylum, aids us. 'Somer's work shows a large percentage of meningeal involvement in some form associated with fibrosis, and to me appears to be the cause of the presence of this phenomenon; also to confirm Kernig and his followers as to its presence indicating some inflammatory changes in the pia mater, and still further to enable us to say which of our cases of fibrosis has cerebral involvement, the nature of it, and the degree. Here I might add that there appears to be a greater degree of contracture in some of the more marked cases of sclerosis.

I will make no attempt this time to account for the presence of meningeal changes in certain cases of fibrosis, though to me there remains some facts to be explained: Why should certain cases of fibrosis show meningeal changes? What is the nature of it and the cause? What amount of meningeal change is necessary to produce the phenomenon? Is location of the meningeal change a factor in the production of the sign? Is the sign present preceding the ability to demonstrate fibrosis? What means should we or can we employ to prevent or limit its appearance? What rôle does lues play in these cases? Would the Wassermann reaction in these cases add any light? And one may go on still further, though at this time I do not deem it necessary to do so, as I feel these questions open a field for further investigation that surely is a broad one, the results of which at this time can only be problematical.

A few remarks of my observation as to the recently described phenomenon of Brudzinsky as described in the *Children's Archives of Medicine*, Paris, October, 1909.

Brudzinsky describes a flexure movement (10) in ankle, knee and hip joint, which he calls the contra-lateral sign, produced upon the forcible flexion of the head upon the chest, and states it to be more constant than the Kernig and Babinski phenomena in tuberculous and epidemic forms of meningitis.

In my observations in general paresis and other cases, I

fail to find the phenomenon, though I do find frequently, especially in the early stages of paresis, what apparently is a modified sign, probably due to similar causes that produce Brudzinsky's phenomenon.

I find in forcible flexion of the head upon the chest a contracture of the muscles on the anterior aspect of the thigh, and at times one can see distinct contracture of the sartorius and gracilis muscles; at other times a contracture of all the muscles on the anterior aspect of the thigh including movement of the patella.

I have examined seventy-five cases of general paresis; in these I could demonstrate Kernig's phenomenon in sixty-three cases, but not in twelve cases. The latter were all early cases, examined but once for the phenomenon, and most likely if examined at frequent intervals would show the sign. Many of these cases have been examined frequently and as previously noted the degree of contracture varies at times. I have examined fifty non-meningitic cases, of which fourteen cases presented the sign, all of these presented evidence of fibrosis, the remaining about equal in number with and without fibrosis, all failed to present the phenomenon. Three cases of uremia also examined presented the phenomenon.³

Through the courtesy of Doctors Spiller and Burr, I have examined twenty-three cases of tabes dorsalis in their wards at the Philadelphia Hospital, for Kernig's phenomenon, and in five of these cases the phenomenon could be demonstrated. Though in all five cases there could be no question of the presence of arterio-capillary fibrosis, in fact in two cases it was pronounced. In no case could the contra-lateral sign be demonstrated.

In conclusion, I will say that at some period in the course of general paresis, one will find Kernig's phenomenon present. It is always present in the late stages of the disease. As to its

³ Since presentation of this paper a necropsy has been obtained in one case of sclerosis reported in this series of cases, in which the clinical diagnosis of arterio-capillary fibrosis, in a man sixty-five years of age, was confirmed at autopsy, and in addition an acute hemorrhagic pachymeningitis, that covered almost the entire cerebrum on its superior surface, involving the pia, was found; no involvement of dura was apparent in the case; no other clinical symptom of meningeal involvement other than Kernig's phenomenon presented itself before death. It has also been my privilege to see a case in which Kernig's phenomenon could be demonstrated in a man thirty-three years of age in which there was marked fibrosis of the radials and temporals, latter firm and markedly tortuous, urinary findings of chronic interstitial nephritis, history of lues questionable.

presence in arterio-capillary fibrosis, and its significance, to me it appears to be an evidence of meningeal involvement, the nature and extent of which I am at this time unprepared to say.

I desire to express my thanks to Dr. Spiller and Dr. Burr for the courtesies and privileges extended me in their wards.

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