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Staff Meeting Report

Immunoelectrophoretic Characterization of Diseases Associated with Defects in the Immune Response*†

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A large number of diseases are commonly associated with enhanced susceptibility to infection.[1] For many of these, however, the relationship in terms of pathogenetic mechanisms is not clear, and the infections appear to result from severe debilitation or some other secondary feature of the disease. The present report will consider three diseases of the hematopoietic tissues that have been shown to have various defects in the immune response.

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Agammaglobulinemia is a disease of the hematopoietic tissues that most commonly reveals itself through enhanced susceptibility to bacterial disease. The striking morphologic anomaly is the virtual absence of plasma cells, together with failure to develop secondary lymphoid follicles. Associated hematologic disturbances in these patients have included: agranulocytosis,[2] aregenertive anemia,[3] alymphocytosis,[4] and thymomas.[5] In addition, approximately one third of the patients with agammaglobulinemia suffer from some disease of the connective tissues.[6] Most commonly this has taken the form of rheumatoid arthritis, but multiple instances of dermatomyositis, scleroderma, and diffuse vascular disease have been reported.[6] Since these patients have

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a defect in the capacity to develop circulating antibodies in response to antigenic stimulation, gamma globulin is virtually absent from the serum. They are, however, capable of developing delayed type sensitivity to various purified proteins and a wide variety of fungal, bacterial, and viral products.[7] Such immunological competence has been offered to explain the ability of these patients to acquire resistance to reinfection with certain viral diseases and their ability to tolerate tuberculous infection and even vaccination with BCG.

Two malignant diseases of the hematopoietic tissues—Hodgkin's disease and multiple myeloma—will also be considered. Both of these have been shown to be accompanied by enhanced susceptibility to infection and by certain specific defects in the immune response. It has been known for a number of years that *Hodgkin's disease* is often accompanied by tuberculosis or certain mycotic infections.[8,9] In contrast, patients with Hodgkin's disease have been shown to have a defect in the capacity to develop or manifest delayed type sensitivity.[10,11] The immunological fault, however, is restricted to this facet of the immune response, and these patients are capable of developing classical circulating antibody in response to antigenic stimulation.

In *multiple myeloma,*[12–14] many patients can be shown to have a defect in the capacity to form circulating antibody.[13–17] Thus, the massive amount of globulin formed in this disease represents nonimmune globulin whose production is apparently associated with a failure in the production of antibody globulins.

Materials and Methods

Patient Material

The 13 patients with agammaglobulinemia studied in this investigation have been previously described.[2,18] In each case, one or more immunochemical measurements of gamma globulin have been made, isoagglutinins have been determined, and bone marrow and lymph node biopsies have been studied in detail. In addition, many have had intensive stimulation with a variety of antigens in an attempt to demonstrate some vestige of immunological capacity. The serum specimens examined in this study were obtained at a time when gamma globulin was not being administered. Serum specimens were obtained from 13 patients with Hodgkin's disease, the diagnosis being based on histologic evidence in each case. All patients were ambulatory at the time the specimens were obtained. One or more serum specimens have been obtained from each of 13 patients with multiple myeloma, the diagnosis again being based on his-

tologic evidence. In all but three of these patients urine specimens were also obtained for electrophoretic analysis.

Preparation of Urine Samples

Urine samples of 50 ml. each were concentrated to one milliliter by dialysis against a 30 per cent solution of polyvinylpyrolidone; in some instances, urine specimens were also concentrated against solid carbowax 20-M. The specimens were stored at 4°C. until analyzed.

Electrophoretic Methods

Paper electrophoresis was performed using a Durrum type cell (Spinco) and an E-C power supply. Analyses were made on 0.01 ml. samples applied to Whatman 3MM filter paper strips. The electrophoretic run was performed with a barbiturate buffer of pH 8.6 and T/2 0.075 for 18 hours at 80 volts. The strips were stained with Amido Black 10B (solution of 0.5 g. in a mixture of 200 ml. of methyl alcohol, 200 ml. of distilled water, and 40 ml. of glacial acetic acid). Immunoelectrophoresis was performed essentially as described by Scheidegger,[19] and the nomenclature used for the various bands is designed to follow this scheme. For the immunological development of the electrophoretically separated proteins three different antisera have been routinely used. One of these was a horse antiserum against whole human serum (Lot 491, Pasteur Institute), and the other two were rabbit antisera against a preparation of human euglobulins and against a commercially prepared Cohn fraction II.

Results

Agammaglobulinemia

Several authors have previously reported the deficiency of three separate serum proteins (γ_2, β_{2A}, β_{2M}) in the typical patient with agammaglobulinemia.[20–22] All three proteins are considered to be antibody globulins. The patients with congenital agammaglobulinemia who have been studied by immunoelectrophoresis in this series all have lacked both the β_{2A} and the β_{2M}, while several with the acquired form of the disease have been shown to have small amounts of one or the other of these two proteins. Both groups of patients with agammaglobulinemia have had very low levels of the major component of the gamma globulin. Those patients with less than about 10 mg/100 ml of gamma globulin measured immunochemically have levels too low to be detected by immunoelectrophoresis. Three previously undescribed serum protein components have been found in the sera from many of these patients; in the terminology of free

electrophoresis all three would be considered to be "fast gamma globulins." In order to avoid inconsistencies with the currently accepted nomenclature of Scheidegger[19] we have designated the two fastest ones as beta 2 globulins (β_{2E} and β_{2K}) and the slowest as gamma 1 globulin (γ_{1A}). As shown in Table 1, all the patients with agammaglobulinemia were observed to have the component designated β_{2K}. Approximately half of the patients have had the β_{2E} and half the γ_{1A}. These last two proteins appear randomly distributed among the sera examined.

TABLE 1

OCCURRENCE OF CERTAIN GAMMA$_1$ AND BETA$_2$ GLOBULINS IN PATIENTS WITH AGAMMAGLOBULINEMIA

	Patient Number	γ_{1A}	β_{2K}	β_{2E}	Immunochemical Gamma Globulin (mg/100 ml)
CONGENITAL	1	+	+	+	3.0
	2	O	+	+	10
	3	O	+	O	14
	4	O	+	+	19
	5	+	+	+	9
	6	O	+	O	2.5
	7	+	+	+	15
	8	+	+	+	8
	Totals	4/8	8/8	6/8	
ACQUIRED	9	+	+	+	6
	10	+	+	O	42
	11	O	+	+	74
	12	+	+	+	81
	13	+	+	O	33
	Totals	4/5	5/5	3/5	

Hodgkin's Disease

The three recognized antibody globulins (γ_2, β_{2A}, and β_{2M}) were found in all these patients. In addition, we observed a striking incidence of the three proteins initially found in patients with agammaglobulinemia. Eleven of the 13 patients examined have had the γ_{1A}, 11 have had the β_{2K}, and the component designated β_{2E} has been seen in five of these patients.

A complete analysis of the proteins present in each of these patients is given in Table 2.

TABLE 2

OCCURRENCE OF CERTAIN GAMMA AND BETA GLOBULINS IN PATIENTS WITH HODGKIN'S DISEASE

Case Number	Gamma Globulins		Beta Globulins			
	γ_2	γ_{1A}	β_{2K}	β_{2E}	β_{2A}	β_{2M}
1	+	O	+	+?	+	+
2	+	+	O	O	+	+
3	+	+	+	O	+	+
4	+	+	+	O	+	+
5	+	+	+	O	+	+
6	+	+	+	+	+	+
7	+	+	O	O	+	tr.
8	+	+	+	+	+	+
9	+	+	+	+	+	+
10	+	+	+	+	+	+
11	+	O	+	O	+	+
12	+	+	+	O	+	+
13	+	+	+	O	+	+
Totals	13/13	11/13	11/13	5/13	13/13	13/13

Multiple Myeloma

The patients in this group have shown a large number of deviations from the normal immunoelectrophoretic pattern. Except for one patient, each has had multiple gamma and beta 2 globulins. Each of the patients with a gamma type myeloma peak revealed by paper electrophoresis has shown an anomaly of the main gamma 2 globulin band. Several have exhibited disturbances in the other two antibody globulins. In four patients, the β_{2M} was not demonstrable, and in one the β_{2A} could not be identified. Two other patients had significant reductions in the amount of the β_{2A} present. The most striking finding has been the almost general occurrence of the β_{2E} component. In only one patient was this protein clearly missing.

DISCUSSION

The various disturbances reported here have been defined through comparisons with the sera of normal individuals. The additional components described, however, must derive their

antigenic determinants from the proteins of normal human serum, since the antisera used in this study have all been prepared against normal human serum or its fractions. It seems likely that these proteins are present in minute amounts. Nevertheless, these determinants in the normal individual may be present in different proteins, or the new proteins may represent incomplete synthetic products associated with particular diseases.

The protein component that we have designated gamma 1A is probably the same as that which Heremans[23] identified as the gamma X. He has found this protein in approximately 50 per cent of the sera from individuals with various diseases. In our own studies we have observed a similar incidence for this protein in the sera of patients hospitalized with a variety of apparently unrelated diseases. This frequency is not greatly different from that noted in the three diseases reported here, though the incidence in the patients with Hodgkin's disease may have been slightly higher.

The frequency with which the two slow beta globulins (β_{2K} and β_{2E}) described here can be found in patients having agammaglobulinemia, Hodgkin's disease, or multiple myeloma reveals that they are most unusual serum protein components. Neither of them can be identified in normal individuals, and only very rarely have they been found in patients with other diseases. They probably do not represent proteins specific for the diseases reported, though they may well reflect some common derangement of the immune response.

A far more complex situation exists in the case of patients with multiple myeloma. Here we have found multiple components in the gamma and beta 2 globulin area in addition to those mentioned earlier. This heterogeneity of the myeloma globulins has also been demonstrated by starch gel electrophoresis.[24] Grabar and associates[25] have suggested that some of these proteins may be the same as those found in the urine. In the majority of the paired samples of serum and urine examined in their study, little if any similarity was found between the multiple proteins of serum and urine. The same workers also found the β_{2A} globulin to be most commonly absent, although in the present study it was regularly observed; thus the two groups of patients probably represent different distributions of myeloma types.

The antibody globulins of normal human serum have been shown to cross react in various studies, and they would appear to contain certain common antigenic determinants.[23,26] Immunoelectrophoretic demonstration of further cross reaction between normal constituents and the myeloma proteins has recently been

TABLE 3

OCCURENCE OF CERTAIN SERUM PROTEINS IN PATIENTS WITH MULTIPLE MYELOMA

Myeloma Component Paper Electrophoresis		IMMUNOELECTROPHORETIC COMPONENTS Anomalous Bands		Defined Proteins						
Serum	Urine	γ	β_2	γ_2	γ_{1A}	β_{2K}	β_{2E}	β_{2A}	β_{2M}	Specimen
$GAMMA_2$	No Specimen	3	1	A*	0	+	0	+	+	Serum
	Beta (minor γ_2)	1	0	A	0	0	+	+	+	Serum
		4	0							Urine
	None (Small γ ; mainly Alb;α_1 & β)	1	0	A	0	0	+	+	+	Serum
		4	0							Urine
	Beta (trace γ ; trace α_2)	1	0	A	0	0	+	+↓	0	Serum
		2	0							Urine
	none (trace γ_1)	1	0	A	0	0	+	+	+	Serum
		1	1							Urine
	No Specimen	1	1	A	0	0	+	+	+	Serum
	None (trace γ)	1	0	A	0	0	+	+	+	Serum
		2	0							Urine
	No Specimen	1	0	A	0	0	?	+	+	Serum
$GAMMA_1$	$Beta_1$ (Small γ)	1	0	A	0	0	+	+	0	Serum
		3	0							Urine
$BETA_2$	Beta (trace γ ; alb.)	0	1	+	+	0	+	0	0	Serum
		1	2							Urine
	Gamma	2	1	+	0	0	+	+↓	0	Serum
		1	2							Urine
NORMAL	Gamma	0	0	+	+	0	+	+	+	Serum
		1	2							Urine
	Gamma (Small B_2; Alb.)	2	0	+	+	0	+	+	+	Serum
		1	2							Urine

*A—denotes anomalous configuration unlike normal γ_2 band
+—component present; 0—component absent; +↓—component reduced

reported.[27,28] A number of such instances have been observed in the present study. The relative specificity of these proteins is still undetermined. Of great importance would be the demonstration of similar proteins in patients who did not have multiple myeloma. Among the many samples of serum examined in other investigations, one such specimen was found—this from a patient considered to have Laënnec's cirrhosis. Paper

electrophoresis showed that this patient had a normal total amount of gamma globulin, although prominent peaking occurred in the gamma 2 area. By immunoelectrophoresis this specimen was demonstrated to be essentially the same as the sera of the gamma type myeloma.

Summary

1. The appearance of three unusual gamma and beta 2 globulins has been described in the sera of three groups of patients having certain defects in the immune response. Patients with agammaglobulinemia often show all three components, while in patients with Hodgkin's disease only two of these components (β_{2K} and γ_{1A}) are generally demonstrable. In patients with multiple myeloma there is a very striking incidence of the β_{2E} globulin.

2. Additional protein abnormalities in the sera and urine of patients with multiple myeloma have been described, and their possible relationship to normal serum proteins has been discussed.

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Staff Meeting Report

Sexual Dysfunction Following Abdominoperineal Resection of the Rectum for Cancer: An Anatomic and Physiologic Study*†

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It is a prime dictum of surgery for cancer that a wide margin of normal tissue should be excised *en bloc* with the tumor in addition to known lymphatic pathways of spread of the malignant lesion. Whenever possible, however, vital structures, particularly nerves and major blood vessels, are not traumatized. For example, in surgery for thyroid cancer, the recurrent laryngeal nerves are protected meticulously except when directly involved by the extension of the cancer. Numerous other examples could be cited.

DAVID M. LONG

Unfortunately, the incidence of sexual impotence, presumably secondary to nerve injury, is very high following surgery for carcinoma of the rectum. Although major disturbances of micturition are much less frequent, this latter aspect of genitourologic function should also be considered because of the close etiologic relationship.

The general surgeon or proctologist may regard sexual function as relatively unimportant in view of the gravity of the primary disease process. Nevertheless, the psychological significance of impotence must be considered for the particular patient. As was revealed in the survey by Kinsey and his co-workers,[1] one should not assume that the patient older than 60 years of age is disinterested in his sexual ability. In younger patients of course, the postoperative impotence is

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even more likely to lead to severe psychic trauma and depression.[2] This factor should be evaluated by the surgeon, and the patient should be made fully cognizant of the high probability of this complication. Fortunately, from 73 to 95 per cent of patients with urinary bladder dysfunction recover after the first few postoperative weeks.[3–6] Many patients with cancer of the rectum are in the age group in which they would have occult vesical outlet obstruction, and retention of urine may be precipitated by minor surgical procedures or physical problems; transurethral resection of the obstructing tissue is usually all that is necessary to relieve this complication.[3] Occasionally, vesical dysfunction may be due to nerve damage and hence may persist permanently. Impotence and the inability to ejaculate semen, on the other hand, are almost always permanent. This paper will examine the surgical anatomy and physiology involved in the impotence and sterility which follow abdominoperineal resection of the rectum.

Although numerous papers have been published on micturition difficulties following rectal surgery, very few reports have appeared on the frequency of sexual impotence. Goligher[7] reported that 72 per cent of his patients were capable of erection, and 64 per cent were capable of sexual intercourse following synchronous combined and perineoabdominal resection of the rectum for carcinoma. Sixty-one per cent of these patients were capable of ejaculation. These patients however, were selected by Goligher, so that they included only those men under 60 years of age who were married and leading an active sexual life. Watson and Williams[6] noted that two patients out of 50 had the pelvic nerve syndrome, which includes impotence and neurogenic vesical dysfunction; impotence in patients without the pelvic nerve syndrome was not recorded. In 41 patients undergoing combined abdominoperineal resection of the rectum, Bruch[8] found only five (12 per cent) who were free from disturbance in sexual function. Thirteen patients (32 per cent) had partial disturbance, and 23 (56 per cent) were completely impotent. Jones[4] reported impotence in approximately 95 per cent of patients with abdominoperineal resection of the rectum.

In patients with abdominoperineal resection for ulcerative colitis, Dennis[9] observed no loss of sexual function. The operation was carried out by removal of no more than the rectal wall, thus avoiding injury to the *nervi erigentes*. Stahlgren[10] reported that in five out of 25 men partial but not complete disturbance of sexual function followed abdominoperineal resection for ulcerative colitis.

Surgical Anatomy of the Hypogastric Plexus and the Pelvic Nerves

The hypogastric plexus and the pelvic nerves and their ramifications were dissected in two cadavers and in ten fresh autopsy specimens. In general, the anatomic configurations noted closely conformed to those described in other investigations[11–16] and in standard anatomy textbooks.

Several points relating to the anatomic relationships of these nerves and other pelvis structures should be emphasized: The sympathetic fibers to the hypogastric plexus originate from the lower thoracic and upper lumbar segments of the spinal cord and enter the lumbar sympathetic trunk via the white *rami communicantes.* Branches from the various lumbar ganglia form a network anterior to the aorta. The aortic plexus descends to the pelvis, where it continues as the hypogastric plexus (Fig. 1). As the hypogastric plexus descends over the bifurcation of the aorta, it enters the tissue plane of the middle sacral artery and the median common iliac lymph glands and sacral lymph glands. The hypogastric plexus then divides to form the right and left pelvic plexi. The lymphatic structures mentioned above are intimately related to the hypogastric plexus and are also frequently the sites of metastases from carcinoma of the rectum.[17,18] Therefore, in any adequate radical lymph node dissection, the hypogastric plexus should be identified and should be extirpated.

The two main divisions of the hypogastric plexus traverse inferolaterally on the wall of the pelvis as the pelvic plexi and fuse with the pelvic parasympathetic outflow or pelvic nerves (Fig. 1). This point of fusion of principal nerve plexi is intimately related to the middle hemorrhoidal vessels. The distal divisions of these plexi pass around the middle hemorrhoidal vessels to innervate the rectum, urinary bladder, seminal vesicles, prostate gland, and penis. Surgical exposure in this area is frequently poor, and the middle hemorrhoidal vessels are often cut without being identified or ligated. This combination of poor exposure and intimate relationship of vital structures makes destruction of the pelvic plexus an imminent possibility. Blind attempts to secure hemostasis with clamping or stick tying are likely to produce nerve injury. Fortunately, the pelvic plexi are diffuse structures and do not constitute a solitary nerve trunk. In addition, experimental data[19] indicate that unilateral destruction of the pelvic plexus or pelvic nerve apparently has little demonstrable effect.

The pelvic nerves are branches of S2, 3, and 4 spinal nerves

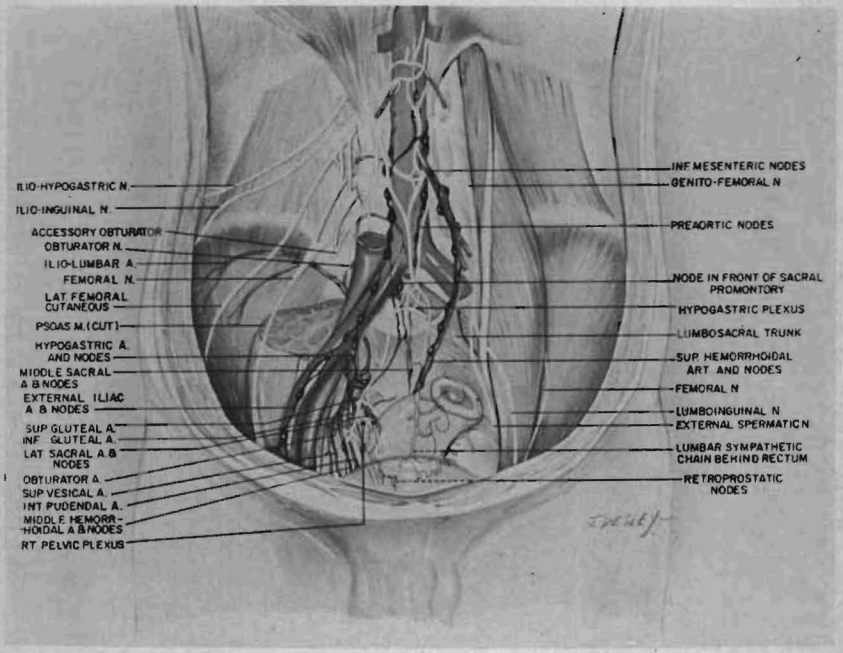

Fig. 1. Detailed drawing of the lower abdominal and pelvic structures. Particular emphasis has been placed on the relationship of autonomic nerves to the lymphatic structures and blood vessels.

and contain the sacral parasympathetic nervous outflow (Fig. 2). These nerves are by far the most important structures in the maintenance of erection and of micturition. As the pelvic nerves pass anterolaterally on the wall of the pelvis, they are covered by a condensation of connective tissue known as Waldeyer's fascia,[6] which varies in density and prominence in different individuals. Usually it can be seen to provide a fascial plane between the pelvic nerves and the presacral plexus of veins posterolaterally and medially and the pararectal tissue anteriorly. Dissection through this plane may result in injury to the pelvic nerves and severe bleeding from the presacral plexus of veins. Nevertheless, in lesions located posteriorly and penetrating deeply, dissection through the plane of Waldeyer's fascia may be necessary.

The nerves of the vesical plexus course anteriorly and inferiorly from the pelvic plexus in contiguity with the inferior vesi-

Fig. 2. Photograph of wax model reconstruction of the sympathetic nerves and parasympathetic nerves innervating the pelvic structures and sexual organs.

cal artery to enter the area of the trigone of the urinary bladder (Fig. 2). The seminal vesicles are innervated by branches of the vesical plexus. The prostatic plexus arises from the pelvic plexus between the vesical and rectal plexi and courses through the prostatic capsule, intermingling with the prostatic plexus of veins. Extensions of the prostatic nerve plexus form the cavernous plexus and supply the nerves of erection to the penis. When veins. Extensions of the prostatic nerve plexus form the cavernous plexus and supply the nerves of erection to the penis. When cutting through the rectovesical septum in dissecting the results in injury to the autonomic nerve fibers. Excision of the nerves serving the function of erection may be more probable in patients with low anterior carcinomatous growths. In addition,

the nerves of erection are apparently particularly vulnerable to injury at this point, as evidenced by the high incidence of impotence following perineal prostatectomy or open perineal biopsy of the prostate.[20,21]

Physiology of Sexual Function

In 1863 Eckhard[22] produced erection of the penis by stimulating the pelvic nerves in the dog. He called these nerves the *nervi erigentes.* The turgidity of the penis produced by stimulating the pelvic nerves has since been demonstrated by others in many species, including the Rhesus monkey[16] (Fig. 3). Henderson and Roepke[23] demonstrated an increase in blood flow through the dorsal vein of the penis by stimulating the nervi erigentes, and by giving intra-arterial injections of acetylcholine and intravenous injections of physostigmine. A rapid rise in venous pressure was also measured in the corpora cavernosa during erection. These investigators concluded that erection is not due to compression of the efferent veins by skeletal muscle.

Semans and Langworthy[24] found stimulation of the pelvic nerve roots did not produce erection when the aorta was constricted. In addition, if erection was elicited by nerve stimulation and the arterial blood flow was then interrupted, the turgidity of the penis subsided. Induced erection was also complete when the bulbocavernosus and ischiocavernosus muscles were transected and dissected free from the *corpora cavernosa.* Although the precise mechanism of erection is not understood, it apparently involves increased blood flow to the penis, as well as decreased egress of venous blood.

Impotence has also been reported following thoracolumbar[25] and lumbar sympathectomy and in the syndrome of sympathetic dysfunction also involving orthostatic hypotension and anhidrosis.[26] Although the physiologic evidence is lacking, White *et al.*[27] postulated that the impotence following sympathectomy is due to the borrowing-lending phenomenon, or shunting of blood flow from the pelvic structures to other denervated areas and viscera. The work of Semans and Langworthy[24] seems to lend some credence to this hypothesis. In addition, one can also cite the impotence found in Leriche's syndrome, or arteriosclerosis obliterans of the aortic bifurcation and common iliac arteries.

Astute observations during the performance of sympathectomies in man were made by Learmonth;[28] he found that stimulating the sympathetic nerves to the pelvis produced contraction of the seminal vesicles with expulsion of the semen from

Functional Neuroanatomy Schema
Parasympathetic Nerves to Pelvic Structures

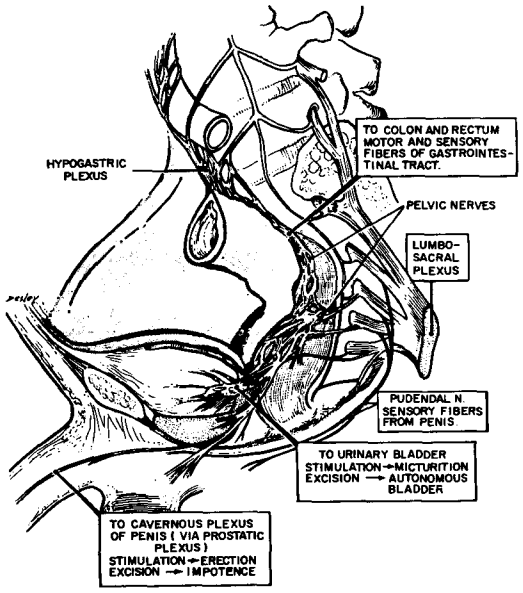

Fig. 3. Schematic presentation of the sacral parasympathetic outflow illustrating the function of the parasympathetic nerves as demonstrated by experiments involving the stimulation and the excision of the pelvic nerves.

Functional Neuroanatomy Schema
Sympathetic Nerves to Pelvic Structures

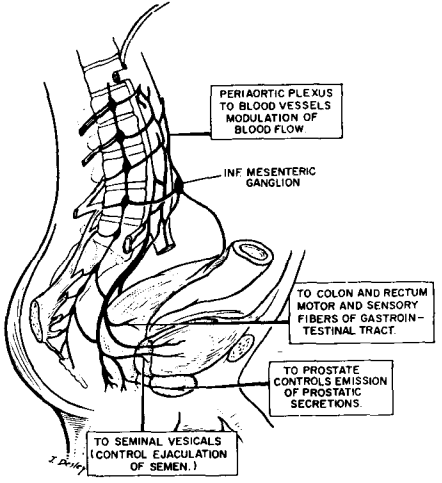

Fig. 4. Schematic presentation of the function of the sympathetic nerves innervating the pelvic viscera and the sexual organs.

Critical Areas for Nerve Injuries

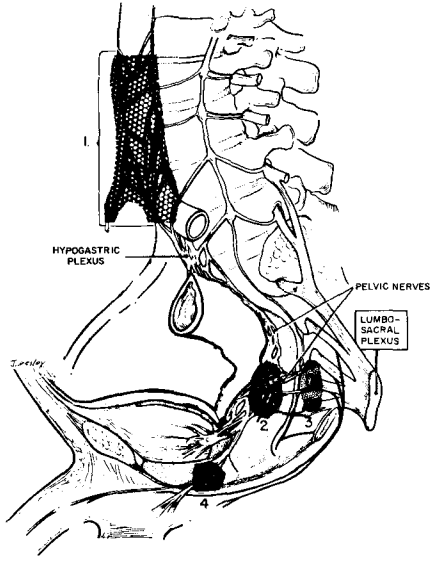

Fig. 5. The four most probable sites of injury to the autonomic nerves modulating sexual function are: (1) the aortic plexus and the hypogastric plexus, (2) the large nerve plexus marking the fusion of the hypogastric plexus and the pelvic nerves, (3) the pelvic nerves near their origin at the lumbosacral plexus, (4) the nervi erigentes as they pass through the prostatic nerve plexus intermingling with the prostatic venous plexus.

the ejaculatory ducts and expulsion of prostatic secretion as a result of contraction of the smooth muscle in the prostate (Fig. 4). He concluded that in man the efferent fibers for ejaculation were contained in the fibers of the first lumbar root. Bacq[29] observed in 1931 that emission of semen, or ejaculation, did not occur following sectioning of the sympathetic nerve pathways in rats. Semans and Langworthy[24] verified the observations that stimulation of the abdominal sympathetic trunk or hypogastric plexus produced ejaculation and that stimulation of the parasympathetic fibers in the sacral roots led to subsidence of erection. Whitelow and Smithwick[25] found permanent loss of ejaculation capacity in six (54 per cent) of 11 patients with bilateral lumbar sympathectomy. Rose[30] reported a 10 per cent incidence of asemenorrhea in 30 cases of lumbar sympathectomy.

Discussion and Summary

Excision of the lower portion of the aortic plexus and the hypogastric plexus is presumably inevitable in the thorough elimination of lymph nodes and lymphatic ducts that are known areas of metastases for carcinoma of the rectum (Area 1, Fig. 5). This conclusion is based on well-documented facts regarding anatomic relationships. Excision of the sympathetic plexi in this area will result in severing the connections of the lower thoracic and upper lumbar sympathetic centers for control of ejaculation and the smooth muscle of the seminal vesicles, vas deferens, and prostatic ducts. The result will be asemenorrhea, or the inability to ejaculate. The patient is not necessarily impotent but is sterile. Impotence, however, has also been documented as a complication of sympathectomy.

The main trunk of the pelvic nerve is likely to be severed deep in the pelvis near the origin of this nerve from the sacral spinal nerves (Area 3, Fig. 5). To avoid this possibility, the tissue plane deep to Waldeyer's fascia should not be entered. Excessive hemorrhage from the presacral plexus of veins can also be prevented by taking this precaution when possible. Bilateral injury to the main pelvic nerves results in the pelvic nerve syndrome, which includes impotence and neurogenic vesical dysfunction. The patient exhibiting the pelvic nerve syndrome is not necessarily sterile and even if impotent may still enjoy the sensual pleasure of ejaculation. Experimental evidence suggests that unilateral injury of the pelvic nerves could be without demonstrable clinical effects.

The pelvic plexus of nerves, which is a fusion of the hypogastric plexus (sympathetic nerves predominantly) and the

pelvic nerves (parasympathetic nerves), is particularly susceptible to injury (Area 2, Fig. 5). Such damage probably occurs at the time of severance or ligation of the middle hemorrhoidal vessels. Injuries at this point can be of a mixed nature. A study of the anatomy in this area suggests, however, that the nerves of erection and ejaculation are more likely to be injured than are those of the vesical plexus to the urinary bladder.

Another vulnerable part of the nervi erigentes is that portion in the prostatic plexus (Area 4, Fig. 5). Injuries to this structure probably occur during the perineal phase of the dissection, especially when the fascia around the prostate is entered. Such trauma would result in impotence just as in perineal biopsy of the prostate. Anatomically, the innervation of the seminal vesicles could still conceivably be intact and the patient impotent but capable of ejaculation.

A number of other factors, along with primary nerve injury, must also be kept in mind when considering the etiology of the impotence secondary to nerve injury. Disturbances of vascular flow resulting from arterial occlusion or alterations in the nervous modulation of the blood vessels can cause failure of erection both experimentally and clinically. Whether or not elderly patients with some arterial insufficiency are more prone to impotence following some types of sympathectomy can only be conjectured. Impotence may also be related to hysteria,[3] although the introduction of this variable is probably not necessary in accounting for the postoperative condition of most of these patients.

A review has been made of 150 men patients undergoing surgery for cancer of the rectum or sigmoid colon. In this group, of the 68 individuals who were under sixty years of age, only two were found to have normal sexual function. When the surgery was performed for carcinoma of the sigmoid colon, the inferior mesenteric artery was ligated near its origin and the periaortic lymphatic vessels and nodes were removed *en bloc* with the sigmoid colon. If the rectum was not removed, inability to ejaculate semen has been the rule. Following combined abdominoperineal resection, impotence and failure of ejaculation have occurred.

Is sexual impotence a necessary complication following surgery for cancer of the rectum and sigmoid colon? Some authors[31–33] have described operations for cancer of the rectum which have been followed by an incidence of sexual dysfunction of between only 10 and 15 per cent. In describing the merits of these operations, these authors have indicated that no compromise has been made in removing lymphatic-bearing tis-

sues and that the procedures are as radical as the conventional Miles type of operation. Now, it is not our intention to espouse the cause of the radical or extended radical operations for cancer of the rectum and sigmoid colon, nor to take issue with those who feel that more conservative surgical procedures are entirely justified. We do want to indicate, however, that surgical procedures designed to remove the greatest part of the lymphatics which drain the rectum and sigmoid colon must include the removal of the pathways of the sympathetic and parasympathetic nerves upon which normal sexual function is dependent. Moreover, we believe that the incidence of sexual dysfunction following operations for cancer of the rectum and sigmoid colon is a reliable index of the amount of lymphatic-bearing tissue which has been removed during the procedure.

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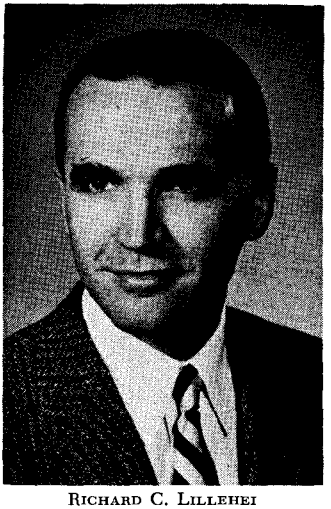
Medical School News

DR. RICHARD C. LILLEHEI NAMED MARKLE SCHOLAR

Dr. Richard C. Lillehei, 32-year-old Fellow in Surgery at the University of Minnesota Medical School and Hospitals, has gained the exemplary honor of being named a Markle Scholar in Medical Science.

His appointment by the John and Mary R. Markle Foundation, New York City, carries a $30,000 grant to the Medical School, which will use it to help support Dr. Lillehei's research program for the next five years.



RICHARD C. LILLEHEI

The honor is given in recognition for outstanding progress and promise in the field of medical research, and enables the scientist to pursue research of his own choice and wherever he wishes.

Dr. Lillehei is a graduate of the University of Minnesota Medical School in 1952, and interned at Minneapolis General Hospital. He was the winner of several merit awards, among them was the Southern Minnesota Medical Association's Medal and Prize in 1951 for maintaining the highest four-year average in Medical School. He was Interfraternity welterweight boxing champion and is a member of Phi Rho Sigma fraternity, Phi Beta Kappa, Iron Wedge, Alpha Omega Alpha, and Grey Friars.

Dr. Lillehei lives in Minneapolis with his wife, the former Elizabeth Jean Larson, and three sons.

Other Minnesotans named Markle Scholars in recent years include Drs. Leonard Peltier, Gilbert Campbell, Lloyd MacLean, Mitchell Spellman, Richard Egdahl, George Moore, Russell Nelson, William Scherer, Robert Ulstrom, and Robert A. Good.

The Medical School was host Feb. 11-13, 1960 to the 21st annual meeting of the Society of University Surgeons, an organization of surgeons affiliated with medical schools throughout the country.

About 300 member and guest surgeons in attendance heard presentations of 45 papers devoted to recent advances in Sugery. The University of Minnesota delegates presented 22 papers.

Dr. Henry T. Randall, New York City, President of the Society, gave an address on "Postgraduate Education in Surgery" at the annual banquet. Dr. Bernard Zimmerman, Professor of Surgery at the University of Minnesota, was chairman of the host committee.

Dr. Robert B. Howard, Dean of Medical Sciences, attended a meeting of Deans of all U.S. medical schools in Chicago on Jan. 9 under auspices of the American Association of Medical Colleges. The one-day conference dealt with proposed Federal legislation affecting medical education and research.

Dr. Howard, and Drs. James Carey, Richard Magraw, Albert Sullivan, and H. Mead Cavert represented the Medical School Feb 6-9 in Chicago at the 56th Annual Congress on Medical Education. At the Congress, Dr. Maurice B. Visscher, Head of the Department of Physiology, presented a paper, "Medicine as a True University Graduate Study—Fact, Fiction and the Future."

LECTURES

Dr. Herbert H. Jasper, Professor of Experimental Neurology at the Montreal Neurological Institute, McGill University, Montreal, Canada, lectured on "Petit Mal Epilepsy" Feb. 18, 1960, when the Medical School presented the J. B. Johnston Lecture.

Dr. Gordon K. Moe delivered the annual George E. Fahr Lecture Feb. 9, 1960, at the Medical School on the topic "The Mechanism of Atrial Fibrillation." Dr. Moe is Professor and Head of the Department of Physiology at State University of New York College of Medicine, Syracuse, New York.

Dr. D. Arn. Van Krevelen, of the University of Leiden, Holland, delivered a special lecture on "Psychopathology of the Only Child" March 2 at the Medical School under auspices of the Department of Psychiatry and Neurology. He is President of the International Association for Child Psychiatry and Allied Professions.

Departmental News

ANATOMY

Dr. Arnold Lazarow, Professor and Head, and Dr. Frederick C. Goetz of the Department of Medicine were in Los Angeles, Calif. Jan. 22-23 to participate in a U.S. Public Health Service workshop for program directors who are recipients of USPHS diabetes training grants.

Dr. Edward Allen Boyden, Professor Emeritus, was one of ten men named recently by Modern Medicine magazine as recipients of its Distinguished Achievement awards. He was cited for his contribution to knowledge of renal, biliary tract, and lung anatomy.

PHYSICAL MEDICINE AND REHABILITATION

Dr. Frederic J. Kottke, Professor and Head, lectured at a graduate teaching seminar on physical medicine and rehabilitation Jan. 28-30 at the University of Florida Medical School, Gainesville, Fla. He attended meetings of the American Registry of Physical Therapists and the American Physical Therapy Association in New York Feb. 5, and attended the meeting of the Medical Directors of Physical Therapy Schools in Chicago Feb. 6.

Dr. Kottke gave two lectures on "Rehabilitation of the Cardiac Patient" Feb. 10 in a continuation course at the University of Oklahoma School of Medicine.

Dr. Glenn Gullickson, Assistant Director of the University of Minnesota Rehabilitation Center, attended a meeting of the Conference of Rehabilitation Centers and Facilities in Evanston, Ill., January 25.

Dr. Richard M. Steidl has been appoined a Medical Fellow Specialist in the Department, and will conduct research on the histopathology of neuromuscular diseases.

UNIVERSITY HOSPITALS

Mr. Ray M. Amberg, Director of University of Minnesota Hospitals, presented the 1960 Samuel W. Melendy Memorial Lecture Feb. 2 at the 21st annual Pharmacy Institute at the University. His topic was "Pharmacy in Hospitals." He is immediate past president of the American Hospital Association.

Also participating in the Institute were Dr. Wesley W. Spink, Professor of Medicine, who discussed "The Clinical Problem of Antibiotic Resistant Organisms," and Dr. Claude R. Hitchcock, Associate Professor of Surgery, who spoke on "Control of Infections in a General Hospital."

PHYSIOLOGY

Dr. Maurice Visscher, Professor and Head of the Department, lectured January 19, 1960 in Davenport, Iowa, before the Iowa Association for the United Nations. His topic was "The Work of the World Health Organization."

Dr. E. B. Brown, Professor, spoke Feb. 4, 1960 in International Falls, Minn. on "The University's Enrollment Paradox." His audience was the Minnesota Alumni Club of International Falls.

SURGERY

Dr. Owen H. Wangensteen, Chairman, was a faculty member at the 13th Annual Symposium presented by the Indiana University School of Medicine in Indianapolis, Ind. March 29-30, 1960. Title of the Symposium was "Malignancy of the Colon."

PSYCHIATRY AND NEUROLOGY

Dr. Bernard C. Glueck, Jr., Professor of Psychiatry, has resigned effective July 15, 1960 to become Director of Psychiatric Research at the Institute of Living, Hartford, Conn. For the past five years he has headed the research section of the Division of Psychiatry, University of Minnesota.

Dr. Frank Kiesler, Assistant Professor of Psychiatry, has accepted a post as Director of the Community Mental Health Clinic, Grand Rapids, Minnesota, effective July 1, 1960. He has been director of the Department's training program since 1958, and formerly was Director of the Out Patient Psychiatric Clinic.

Dr. George Dorsey, Instructor in Psychiatry at Minneapolis General Hospital, resigned his position recently to enter private practice in Minneapolis.

ALUMNI DEATHS

Dr. Richey Laughlin Waugh (Med. '15), retired Medical Director of the U.S. Public Health Service, died Nov. 24, 1959 in Arlington, Mass. He was 71 years old and holder of a Founders Medal of the Association of Military Surgeons of the United States. He was a fellow of the American College of Surgeons and member of the American Academy of Orthopaedic Surgeons and the American Association for the Surgery of Trauma.

Dr. Emanuel S. Lippman (Med. '28), Minneapolis pediatrician, died Feb. 24, 1960, at the age of 54 years. He was Assistant Clinical Professor of Pediatrics, a member of the Minnesota Medical Foundation, and former Chief of Pediatrics at Mount Sinai Hospital in Minneapolis. He served as a medical officer with the U.S. Army in World War II. Among the survivors is a brother, Dr. Hyman S. Lippman (Med. '19) of St. Paul.

Dr. Arthur C. Johnson (Med. '20) died February 3, 1960 in Omaha, Nebraska, where he had practiced for the past 34 years. He was on the staff of Creighton University Medical School and in partnership with a group of private physicians. Following graduation from Medical School he spent five years as first assistant in surgery to Dr. William Mayo at the Mayo Clinic, Rochester, Minn. His wife, two sisters, and two brothers survive, among them Dr. Walter R. Johnson, (Med. '24), of Asheville, N.C.

Dr. Albert M. Snell (Med. '18), died February 6, 1960, at the age of 64 years, in Palo Alto, Calif. He was born in Lake Park, Minn., practiced at the Mankato, Minn. Clinic and Mayo Clinic, Rochester, and in 1950 moved to California to become Clinical Professor of Medicine at the University of California. He had since been Chief of the Division of Medicine at Stanford University. Dr. Snell served as a U.S. Navy Medical officer in the two World Wars, and was a Consultant in Medicine for the Veterans Administration. His wife and five children all reside on the West Coast.

Dr. Chester J. Olson (Med. '25), died October 20, 1959 in St. Mary's Hospital, Rochester, Minn. He was in practice at Belle Plaine, Minn., and a member of the American Academy of General Practice. Dr. Olson died at age 64. He had served as a lieutenant commander in the U.S. Navy during World War II.

Progress Report

Class of 1959 Scholarship Fund

RECENT CONTRIBUTORS

John D. Banovetz

William J. Broussard

Carl G. Evers

Douglass A. Lowe

Richard A. Meland

Richard R. Oslund

Darline D. Smith

Total Contributions to Date	$60.00
Number of Contributors	41
Number of Class Members	113
Percent of Participation	36%
Current Year's Goal	$125.00
*Objective by 1963	$500.00

*To underwrite a "Class of 1959 Scholarship" for a meritorious medical student in September 1963.

Coming Events

University of Minnesota Medical School

Courses in Continuation Medical Education During 1960

April 7–9	Emergency Surgery for General Physicians
April 11–13	Radiology for General Physicians
April 21–23	Otolaryngology for General Physicians
May 2–6	Intermediate Electrocardigoraphy for General Physicians and Specialists
May 9–11	Cardiovascular Diseases for General Physicians and Specialists
May 16–18	Psychiatry for General Physicians
May 23–27	Proctology for General Physicians
June 13–15	Gynecology for Specialists
Continuous in 1960 .	Cancer Detection for General Physicians

Courses are held at the Center for Continuation Study or at the Mayo Memorial Auditorium on the campus of the University of Minnesota. Usual tuition fees are $10 for a one-day course, $40 for a three-day course, and $65 for a one-week course. These are subject to change under certain circumstances.

Register early. For further information write to:

Director
Dept. of Continuation Medical Education
1342 Mayo Memorial — University of Minnesota
Minneapolis 14, Minnesota

A Word About

Memorial Gifts

Many people have adopted the appropriate custom of sending memorial gifts to worthy organizations in time of bereavement or other occasion. Such funds have lent significant strength to the fight against the major diseases known to Americans.

The Minnesota Medical Foundation welcomes your memorial gifts when an appropriate occasion arises. Memorial gifts serve the living and pay thoughtful tribute to the memory of a friend, associate, or relative. The Foundation will acknowledge gifts with suitable cards mailed promptly to both the donor and the family of the deceased. The gift will help finance the Foundation's program of support for the Medical School of the University of Minnesota.

Special memorial funds may be created within the Foundation on request to serve as a permanent repository for continuing contributions.

When making memorial gifts to the Foundation, include the names and addresses of the deceased, next of kin, and the donor.

MINNESOTA MEDICAL FOUNDATION
1342 Mayo Memorial
University of Minnesota
Minneapolis 14, Minnesota