A STUDY OF THE SKELETAL MUSCLES OF SHEEP WITH SPECIAL REFERENCE TO SCRAPIE DISEASE

A Ph.D. THESIS SUBMITTED BY

T. J. HULLAND

VOLUME II

TABLES AND FIGURES

VOLUME II

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CLINICAL ASPECTS OF SCRAPIE

TABLE 1 NATURALLY SCRAPIED CASES

Case Number	Breed	Age	Sex	Clinical Symptoms					
				Observed (days)	Pruritis	Ataxia *	Loss of Body Condition	Other	Predominant Clinical Sign
S-1	Suf.	4	M	27	++	++	+	-	ataxia
S-2	Suf.	3	F	29	+++	+++	++	-	ataxia
S-3	Chev.	3	F	30	++	+	++	-	pruritis
S-4	Suf.	2	F	30	++	++	+	-	ataxia
S-5	Chev.	2	F	24	+++	+	++	-	pruritis
S-6	Chev.	3	F	23	+	+++	+++	hyperaes.**	hyperaes.
S-7	Swale.	2	F	20	+++	+	+++	-	pruritis
S-8	Swale.	2	F	22	+++	+	+	-	pruritis
S-9	Chev.	3	F	28	+++	++	+++	hyperaes.	hyperaes.
S-10	Swale.	2	F	44	++	+	+++	-	pruritis
S-11	Chev.	4	F	35	+++	++	+++	-	pruritis
S-12	Suf.	3	F	0	+	++	+	-	ataxia
S-13	Swale.	2	F	58	+++	+	+++	-	pruritis
S-14	Swale.	3	F	76	++	+	++	-	pruritis
S-15	Suf.	3	F	24	+	+++	+	-	ataxia
S-16	Swale.	2	F	107	++	+	+++	-	pruritis
S-17	Chev.	3	F	98	+++	++	+++	-	pruritis
S-18	Suf.	3	F	55	++	+++	-	-	ataxia
S-19	Chev. X	3	F	30	+	+	+++	hyperaes.	hyperaes.
S-20	Border L.	3	M	24	++	++	+	-	pruritis
S-21	Suf. X	3	F	30	+++	+	+	-	pruritis
S-22	Swale.	3	F	28	++	+	++	hyperaes.	hyperaes.
S-26	Suf.	3	F	65	+++	+++	++	-	ataxia
S-27	Chev.	2	F	5	+	+	++	hyperaes.	hyperaes.
S-28	Chev.	4	M	0	+	++	+	-	ataxia
S-29	Wens.	2	F	45	++	++	++	-	pruritis
S-30	Border L.	5	F	31	++	++	+	-	ataxia
S-31	Suf.	3	M	23	++	+++	++	-	ataxia
S-32	Chev. X	2	F	24	+++	+	+	-	pruritis
S-33	Chev. X	3	F	30	++	++	+	-	pruritis
S-34	Suf.	2	F	20	++	++	++	-	pruritis
S-35	Suf.	2	F	22	++	+++	+	-	ataxia
S-36	Chev. X	2	F	4	++	++	+	hyperaes.	hyperaes.
S-37	Suf.	3	F	10	+	++	+	-	ataxia
S-38	Chev. X	2	F	7	+	++	++	hyperaes.	hyperaes.
S-39	Suf.	3	F	20	+	++	+	-	ataxia
S-40	Chev.	3	F	6	++	++	+	-	pruritis
S-41	Suf.	3	F	10	+++	+++	+	-	ataxia
S-42	Chev.	2	F	14	+++	+	++	-	pruritis
S-43	Chev.	3	F	32	++	+	+	-	pruritis
S-44	Chev.	2	F	17	++	+	++	-	pruritis
S-45	Suf.	3	F	9	+	++	+	-	ataxia
S-46	Chev.	3	F	20	+++	++	+++	-	pruritis
S-47	Chev.	2	M	10	++	+	++	-	pruritis
S-48	Chev.	3	F	18	+++	+	+	-	pruritis
S-49	Suf.	2	F	27	++	+	+	-	pruritis
S-50	Chev.	2	F	11	++	+	++	-	pruritis
S-51	Chev.	3	F	19	+++	++	+	-	pruritis
S-52	Suf.	3	F	0	+	+	-	-	pruritis
S-53	Suf.	2	F	4	+	+	+	rupture	pruritis
S-54	Suf.	2	F	14	++	+	++	-	pruritis
S-55	Suf.	2	F	7	+	+	+	dystocia	pruritis

S-56	Suf.	3	M	45	+++	++	+	-	pruritis
S-57	Dales.	3	F	65	++	+	++	-	pruritis
S-58	Suf.	3	F	36	+	+	+	-	pruritis
S-59	Chev.	3	F	28	++	+	++	-	pruritis
S-60	Suf.	3	M	38	++	++	+	-	pruritis

Suf. - Suffolk

Chev. - Cheviot

Swale. - Swaledale

Border L. - Border Leicester

Wens. - Wenslydale

Dales. - Dalesbred

X indicates a cross-bred

- - none + - slight ++ - moderate +++ - severe

* The term "ataxia" here is used to indicate all locomotor disturbances.

** "hyperaes." - hyperaesthesia and includes all types of altered responses.

CLINICAL ASPECTS OF CASES

TABLE 11 EXPERIMENTALLY SCRAPIED CASES

Case Number	Breed	Age	Sex	Clinical Symptoms					
				Observed (days)	Pruritis	Ataxia *	Loss of Body Condition	Other	Predominant Clinical Sign
E-1	Chev.	1	F	22	++	++	+	-	ataxia
E-2	Chev.	1	C	11	+++	++	+	-	pruritis
E-3	Chev.	1	C	28	++	+	+++	-	pruritis
E-4	Chev.	1	C	130	++	+++	++	-	ataxia
E-5	Chev.	1	C	37	++	+++	+	-	ataxia
E-6	Chev.	1	C	67	++	++	+++	-	ataxia
E-7	Chev.	1	C	20	+++	++	++	-	pruritis
E-8	Chev.	4	C	70	++	+++	++	-	ataxia
E-9	Chev.	1	C	67	++	+	++	-	pruritis
E-10	Chev.	1	C	87	+	++	+	-	ataxia
E-11	Chev.	1	C	48	++	++	+++	-	ataxia
E-12	Chev.	1	C	44	+	++	++	-	ataxia
E-13	Chev.	2	C	59	++	+	++	-	ataxia
E-14	Chev.	2	C	51	+++	+	+	-	pruritis
E-15	Chev.	2	C	43	+++	+	+	-	pruritis
E-16	Chev.	4	C	70	++	+++	+	-	ataxia
E-17	Chev.	2	C	81	++	++	+	-	pruritis
E-18	Chev.	1	C	28	++	+	+	-	pruritis
E-19	Chev.	1	C	49	++	+	+	-	pruritis
E-20	Chev.	1	C	87	++	+++	++	-	pruritis
E-21	Chev.	1	C	35	+++	++	++	-	pruritis
E-22	Chev.	1	F	65	++	+	++	-	pruritis
E-23	Chev.	1	C	83	++	++	+++	-	pruritis
[illegible]	[illegible]	1	C	[illegible]	++	++	+++	[illegible]	[illegible]
E-25	Chev.	7	F	120	++	+	+++	-	pruritis
E-26	Chev.	7	F	130	++	+	++	-	pruritis
E-27	Chev.	7	F	153	++	+++	+++	-	ataxia
E-28	Chev.	2	C	72	++	+	++	-	pruritis
E-29	Chev.	2	C	120	++	++	++	-	pruritis
E-30	Chev.	2	C	93	++	+	+	-	pruritis
E-31	Chev.	2	C	88	+	+	+	-	pruritis
E-32	Chev.	2	C	80	++	+	++	-	pruritis
E-33	Chev.	7	F	120	++	+	+++	-	pruritis
E-34	Chev.	1	C	64	++	++	+	-	pruritis

Chev. - Cheviot

C - castrate

* - The term "ataxia" here is used to indicate all locomotor disturbances.

- - none + - slight ++ - moderate +++ - severe

CLINICAL ASPECTS OF CASES

TABLE 111 CONTROL GROUP

Case Number	Breed	Age	Sex	Loss of Body Condition	Clinical Symptoms	Post-mortem Diagnosis
C-1	B-F.	1	C	++	-	urethral calculi
C-2	Chev.	2	F	++	scouring	Johne's disease
C-3	Suf.	1	F	-	-	normal
C-4	Suf.	1	F	-	-	normal
C-5	Suf.	1	F	-	-	normal
C-6	Chev.	1	F	-	-	normal
C-7	Chev.	1	F	-	-	normal
C-8	Chev.	1	F	++	dyspnoea	pneumonia
C-9	Chev.	1	F	++	dyspnoea	pneumonia
C-10	Chev.	1	F	++	scouring	Johne's disease
C-11	Chev.	2	F	+	-	pneumonia
C-12	Chev.	2	F	-	-	normal
C-13	Chev.	2	F	+	-	normal
C-14	Chev.	3	F	-	-	normal
C-15	Chev.	3	F	-	-	normal
C-16	Chev.	3	F	+++	pining	purulent peritonitis
C-17	Chev.	3	F	-	-	normal
C-18	Chev.	3	F	+	-	normal
C-19	Chev.	3	F	+	-	normal
C-20	Chev.	2	F	-	-	normal
C-21	Chev.	2	F	+	-	normal
C-22	Chev.	3	F	++	scouring	Johne's disease
C-23	B-F.	4	F	++	pining	tick pyemia
C-25	Chev.	3	F	+++	scouring	Johne's disease
C-26	Chev.	2	F	-	-	normal
C-27	Chev.	2	F	-	-	normal
C-28	Chev.	3	F	++	ataxia	louping ill
C-29	B-F.	3	M	++	prostrate	brain abscess
C-30	Chev.	1	F	+	-	normal
C-31	Chev.	1	F	+	-	normal
C-32	Chev.	7	F	-	-	normal
C-33	Chev.	2	F	+	hyperaesthesia	muscle fibrosis
C-34	Chev.	4	F	+++	scouring	Johne's disease
C-35	Chev.	7	F	-	-	normal
C-36	B-F.	5	F	+	-	normal
C-37	Chev.	1	F	++	lameness	osteodystrophy
C-38	Chev.	3	F	++	-	Johne's disease
C-39	Suf.	2	M	+	lameness	foot rot
C-40	Suf.	3	F	+++	lameness	foot rot
C-41	Chev.	2	F	++	scouring	Johne's disease
C-42	Chev.	1	F	+	-	normal
C-43	Chev.	1	F	+	-	normal
C-44	B-F.	1	F	+++	prostrate	osteodystrophy
C-45	Suf.	2	F	+	dyspnoea	pneumonia
C-46	Suf.	2	F	+	rupture	muscular dystrophy
C-47	Suf.	4	F	-	dystocia	muscular dystrophy
C-48	Suf.	1	F	+	dystocia	muscular dystrophy
C-49	Chev.	6	F	++	rupture	muscular dystrophy
C-50	Chev.	6	F	++	-	muscular dystrophy

C-39	Suf.	2	M	+	lameness	foot rot
C-40	Suf.	3	F	+++	lameness	foot rot
C-41	Chev.	2	F	++	scouring	Johne's disease
C-42	Chev.	1	F	+	-	normal
C-43	Chev.	1	F	+	-	normal
C-44	B-F.	1	F	+++	prostrate	osteodystrophy
C-45	Suf.	2	F	+	dyspnoea	pneumonia
C-46	Suf.	2	F	+	rupture	muscular dystrophy
C-47	Suf.	4	F	-	dystocia	muscular dystrophy
C-48	Suf.	1	F	+	dystocia	muscular dystrophy
C-49	Chev.	6	F	++	rupture	muscular dystrophy
C-50	Chev.	6	F	++	-	muscular dystrophy
C-51	B-F.	3	F	-	-	normal
C-52	Chev.	6	F	+++	pining	neoplasm
C-53	B-F.	2	F	+	-	normal
C-54	B-F.	2	F	+	-	normal
C-55	Chev.	6	F	+	-	normal
C-56	Chev.	3	F	++	-	muscular dystrophy
C-57	Suf.	2	F	-	-	normal
C-58	Suf.	6	F	-	-	normal
C-59	Chev.	2	F	+	-	normal
C-60	Suf.	5	F	+	dyspnoea	pneumonia
C-61	Chev.	4	F	++	dyspnoea	pneumonia
C-62	Chev.	7	F	+	-	pneumonia
C-63	Chev.	3	F	+	-	hypomagnesaemia
C-64	Chev.	6	F	+	-	pneumonia
C-65	Chev.	6	F	+	dyspnoea	pneumonia
C-66	Chev.	6	F	-	-	pneumonia
C-67	Chev.	1	C	+	prostrate	brain abscess
C-68	Chev.	3	F	++	prolapse	uterine fistula
C-69	B-F.	3	F	++	-	septicemia
C-70	Chev.	1	C	+	-	normal
C-71	Chev.	1	C	+	-	normal
C-72	Chev.	3	F	+++	pining	muscular dystrophy
C-73	B-F.	4	F	+++	-	muscular dystrophy
C-74	B-F.	2	F	+++	-	muscular dystrophy
C-75	Border L.	3	M	+	paraplegia	spinal abscess
C-76	Chev.	7	F	-	rupture	muscular dystrophy

B-F. - Black Face

Chev. - Cheviot

Suf. - Suffolk

Border L. - Border Leicester

C - castrate

- - none + - slight ++ - moderate +++ - severe

MACROSCOPIC AND MICROSCOPIC FINDINGS

TABLE 1V NATURALLY SCRAPIED SHEEP

Macroscopic			Microscopic							
				Number of Muscles Showing ____						
Case No.	Carcass Emaciation	Lesions Visible in Muscles	No. of Muscles Sampled	Type 1	Type 11	Type 111	Type 1V	Type V	Myositis	Infarction
S-1	+	0	0	-	-	-	-	-	-	-
S-2	+	0	0	-	-	-	-	-	-	-
S-3	++	0	20	8	-	-	-	-	-	-
S-4	+	0	20	4	-	-	-	-	-	-
S-5	++	0	20	7	-	-	-	-	-	-
S-6	+++	0	20	-	-	-	-	-	-	-
S-7	++	0	20	8	-	-	-	-	-	-
S-8	+	0	20	8	-	-	-	-	-	-
S-9	+++	0	20	2	-	-	-	-	-	-
S-10	+++	0	20	1	-	-	-	-	-	-
S-11	+++	0	20	2	-	-	-	-	-	-
S-12	-	0	20	5	1	-	-	-	-	-
S-13	+++	0	20	4	-	-	-	-	-	-
S-14	++	0	50	33	-	3	-	-	-	-
S-15	-	0	44	21	-	-	-	-	-	-
S-16	+++	0	20	4	-	-	-	-	-	-
S-17	+++	0	42	4	-	-	-	-	-	-
S-18	-	0	18	5	-	-	-	-	-	-
S-19	+++	10	16	9	-	-	10	-	-	-
S-20	+	0	6	2	-	-	-	-	-	-
S-21	+	0	10	4	-	-	-	-	-	-
S-22	+	0	8	7	-	-	-	-	-	-
S-23	+	0	8	3	-	-	-	-	-	-
S-24	++	0	8	3	-	-	-	-	-	-
S-25	++	0	6	4	-	-	-	-	-	-
S-26	++	0	4	1	-	-	-	-	-	-
S-27	+	6	16	5	-	-	6	-	-	-
S-28	+	0	0	-	-	-	-	-	-	-
S-29	++	0	3	1	-	-	-	-	-	-
S-30	+	0	5	3	-	-	-	-	-	-
S-31	+	0	5	2	-	-	-	-	-	-
S-32	+	0	5	1	-	-	-	-	-	-
S-33	+	0	5	2	-	-	-	-	-	-
S-34	++	0	4	3	-	-	-	-	-	-
S-35	+	0	0	-	-	-	-	-	-	-
S-36	+	0	4	2	-	-	-	-	-	-
S-37	+	0	2	-	-	-	-	-	-	-
S-38	+	0	2	1	-	-	-	-	-	-
S-39	+	0	0	-	-	-	-	-	-	-
S-40	+	0	0	-	-	-	-	-	-	-
S-41	+	0	0	-	-	-	-	-	-	-
S-42	++	0	0	-	-	-	-	-	-	-
S-43	+	0	0	-	-	-	-	-	-	-
S-44	++	0	0	-	-	-	-	-	-	-
S-45	+	0	0	-	-	-	-	-	-	-
S-46	+++	0	0	-	-	-	-	-	-	-
S-47	+	0	0	-	-	-	-	-	-	-
S-48	+	0	0	-	-	-	-	-	-	-
S-49	+	0	0	-	-	-	-	-	-	-
S-50	++	0	0	-	-	-	-	-	-	-
S-51	+	0	0	-	-	-	-	-	-	-
S-52	-	1	5	3	-	-	-	-	1	-
S-53	-	5	10	2	-	-	5	-	-	-
S-54	+	0	0	-	-	-	-	-	-	-
S-55	+	3	5	-	-	-	3	-	-	-
S-56	+	0	3	2	-	-	-	-	-	-
S-57	+	1	1	-	-	-	-	-	1	-
S-58	+	2	2	-	-	-	-	2	-	-
S-59	++	0	0	-	-	-	-	-	-	-
S-60	+	2	2	-	-	-	-	2	-	-

- or 0 - none or no + - slight ++ - moderate +++ - severe

MACROSCOPIC AND MICROSCOPIC FINDINGS

TABLE V EXPERIMENTALLY SCRAPIED SHEEP

Macroscopic			Microscopic							
				Number of Muscles Showing ___						
Case No.	Carcass Emaciation	Lesions Visible in _ Muscles	No. of Muscles Sampled	Type I	Type II	Type III	Type IV	Type V	Myositis	Infarction
E-1	+	0	0	-	-	-	-	-	-	-
E-2	+	0	0	-	-	-	-	-	-	-
E-3	+++	0	20	1	-	-	-	-	-	-
E-4	++	0	18	18	-	-	-	-	-	-
E-5	+	0	20	7	-	-	-	-	-	-
E-6	++	0	22	12	1	-	-	-	-	-
E-7	++	0	20	10	2	-	-	-	-	-
E-8	-	1	20	5	-	-	-	-	1	-
E-9	++	0	20	8	-	-	-	-	-	-
E-10	++	0	20	3	-	1	-	-	-	-
E-11	++	0	20	7	-	-	-	-	-	-
E-12	++	0	20	5	-	-	-	-	-	-
E-13	++	0	20	2	-	-	-	-	-	-
E-14	+	0	16	3	-	-	-	-	-	-
E-15	+	0	16	4	-	-	-	-	-	-
E-16	+	0	16	5	-	-	-	-	-	-
E-17	+	0	20	9	-	-	-	-	-	-
E-18	+	0	7	3	-	-	-	-	-	-
E-19	+	0	7	5	-	-	-	-	-	-
E-20	++	0	7	4	-	-	-	-	-	-
E-21	++	0	10	6	-	-	-	-	-	-
E-22	++	0	6	2	-	-	-	-	-	-
E-23	+++	0	6	4	-	-	-	-	-	-
E-24	+++	0	5	1	-	-	-	-	-	-
E-25	++	5	11	2	-	-	5	-	-	-
E-26	++	0	3	2	-	-	-	-	-	-
E-27	++	1	5	4	-	1	-	-	-	-
E-28	++	0	0	-	-	-	-	-	-	-
E-29	++	0	0	-	-	-	-	-	-	-
E-30	+	0	0	-	-	-	-	-	-	-
E-31	+	0	0	-	-	-	-	-	-	-
E-32	++	0	0	-	-	-	-	-	-	-
E-33	+++	0	5	3	-	-	-	-	-	-
E-34	+	0	0	-	-	-	-	-	-	-

- or 0 - none or no + - slight ++ - moderate +++ - severe

MACROSCOPIC AND MICROSCOPIC FINDINGS

TABLE V1 CONTROL SHEEP

Macroscopic			Microscopic							
				Number of Muscles Showing ___						
Case No.	Carcass Emaciation	Lesions Visible in ___ Muscles	No. of Muscles Sampled	Type 1	Type 11	Type 111	Type 1V	Type V	Myositis	Infarction
C-1	++	0	6	3	-	-	-	-	-	-
C-2	++	0	4	3	-	-	-	-	-	-
C-3	-	0	0	-	-	-	-	-	-	-
C-4	-	0	0	-	-	-	-	-	-	-
C-5	-	0	0	-	-	-	-	-	-	-
C-6	-	0	0	-	-	-	-	-	-	-
C-7	-	0	0	-	-	-	-	-	-	-
C-8	++	0	7	2	-	-	-	-	-	-
C-9	++	0	4	2	-	-	-	-	-	-
C-10	+	0	4	2	-	-	-	-	-	-
C-11	-	0	20	8	-	-	-	-	-	-
C-12	-	0	6	1	-	-	-	-	-	-
C-13	+	0	20	14	-	-	-	-	-	-
C-14	-	0	6	2	-	-	-	-	-	-
C-15	-	0	24	23	-	-	-	-	-	-
C-16	+++	0	14	4	-	-	-	-	-	-
C-17	-	0	24	8	-	-	-	-	-	-
C-18	+	0	22	14	-	-	-	-	-	-
C-19	+	0	20	3	-	-	-	-	-	-
C-20	-	0	20	15	-	-	-	-	-	-
C-21	+	0	20	17	-	-	-	-	-	-
C-22	++	0	15	12	-	-	-	-	-	-
C-23	++	0	20	17	-	-	-	-	-	-
C-24	+++	0	20	11	-	-	-	-	-	-
C-25	+++	0	20	8	-	-	-	-	-	-
C-26	-	0	0	-	-	-	-	-	-	-
C-27	-	0	0	-	-	-	-	-	-	-
C-28	++	0	15	6	-	-	-	-	-	-
C-29	+	0	0	-	-	-	-	-	-	-
C-30	+	0	3	-	-	-	-	-	-	-
C-31	+	0	3	-	-	-	-	-	-	-
C-32	-	0	5	4	-	-	-	-	-	-
C-33	+	3	10	2	-	2	-	-	1	-
C-34	++	0	5	2	-	-	-	-	-	-
C-35	-	0	4	-	-	-	-	-	-	-
C-36	+	0	0	-	-	-	-	-	-	-
C-37	+	0	0	-	-	-	-	-	-	-
C-38	+	0	2	-	-	-	-	-	-	-
C-39	+	0	2	1	-	-	-	-	-	-
C-40	+++	0	5	2	-	-	-	-	-	-
C-41	+	0	5	1	-	-	-	-	-	-
C-42	+	0	5	-	-	-	-	-	-	-
C-43	+	0	5	-	-	-	-	-	-	-
C-44	+++	0	3	1	-	-	-	-	-	-
C-45	+	3	3	-	-	-	3	-	-	-
C-46	+	12	14	-	-	-	12	-	-	-

C-26	-	0	0	-	-	-	-	-	-	-
C-27	-	0	0	-	-	-	-	-	-	-
C-28	++	0	15	6	-	-	-	-	-	-
C-29	+	0	0	-	-	-	-	-	-	-
C-30	+	0	3	-	-	-	-	-	-	-
C-31	+	0	3	-	-	-	-	-	-	-
C-32	-	0	5	4	-	-	-	-	-	-
C-33	+	3	10	2	-	2	-	-	1	-
C-34	++	0	5	2	-	-	-	-	-	-
C-35	-	0	4	-	-	-	-	-	-	-
C-36	+	0	0	-	-	-	-	-	-	-
C-37	+	0	0	-	-	-	-	-	-	-
C-38	+	0	2	-	-	-	-	-	-	-
C-39	+	0	2	1	-	-	-	-	-	-
C-40	+++	0	5	2	-	-	-	-	-	-
C-41	+	0	5	1	-	-	-	-	-	-
C-42	+	0	5	-	-	-	-	-	-	-
C-43	+	0	5	-	-	-	-	-	-	-
C-44	+++	0	3	1	-	-	-	-	-	-
C-45	+	3	3	-	-	-	3	-	-	-
C-46	+	12	14	-	-	-	12	-	-	-
C-47	-	2	2	-	-	-	2	-	-	-
C-48	+	3	3	1	-	-	2	-	-	-
C-49	++	4	4	-	-	-	4	-	-	-
C-50	++	3	7	-	-	-	3	-	-	-
C-51	-	1	1	-	-	-	-	-	1	-
C-52	+++	4	6	-	-	-	4	-	-	-
C-53	+	0	0	-	-	-	-	-	-	-
C-54	+	0	0	-	-	-	-	-	-	-
C-55	+	0	0	-	-	-	-	-	-	-
C-56	++	3	3	-	-	-	3	-	-	-
C-57	-	0	0	-	-	-	-	-	-	-
C-58	-	0	0	-	-	-	-	-	-	-
C-59	+	0	1	1	-	-	-	-	-	-
C-60	+	8	8	-	-	-	8	-	-	-
C-61	++	16	16	-	-	-	16	-	-	-
C-62	+	0	5	1	-	-	-	-	-	-
C-63	+	0	0	-	-	-	-	-	-	-
C-64	+	0	0	-	-	-	-	-	-	-
C-65	+	0	0	-	-	-	-	-	-	-
C-66	-	0	2	-	-	-	-	-	-	-
C-67	+	4	4	-	-	-	3	-	-	1
C-68	++	3	3	-	-	-	2	-	-	1
C-69	+	0	0	-	-	-	-	-	-	-
C-70	+	0	0	-	-	-	-	-	-	-
C-71	+	0	0	-	-	-	-	-	-	-
C-72	++	8	8	-	-	-	8	-	-	-
C-73	+++	3	4	-	-	-	3	-	-	-
C-74	++	7	7	-	-	-	7	-	-	-
C-75	-	13	14	-	-	-	-	-	-	13
C-76	-	8	8	-	-	-	3	-	5	-

- or 0 - none or no + - slight ++ - moderate +++ - severe

TABLE V11

THE INCIDENCE OF TYPE 1 LESIONS CORRELATED WITH AGE

Age	No. of Animals	No. of Muscles	No. of Muscles Showing Type 1	%
1	25	368	106	28.8
2	33	372	142	38.2
3	28	356	189	53.0
4	8	103	43	41.7
5	1	5	3	60.0
6	3	8	3	47.7
7	7	32	17	53.1

Average age of scrapied sheep 2.4 years. 35.6% of muscle samples contained type 1 changes.

Average age of non-scrapied sheep 2.9 years. 45.5% of muscle samples contained type 1 changes.

TABLE VIII

COMPARISON OF MUSCLE CHANGES IN

SCRAPIED AND NON-SCRAPIED SHEEP

Group	Totals		Type of Microscopic Change Shown													
			Type 1		Type 11		Type 111		Type 1V		Type V		Myositis		Infarction	
	No. of Muscles Sampled	No. of Cases	No. of Muscles	No. of Cases	No. of Muscles	No. of Cases	No. of Muscles	No. of Cases	No. of Muscles	No. of Cases	No. of Muscles	No. of Cases	No. of Muscles	No. of Cases	No. of Muscles	No. of Cases
Natural Scrapie	535	60	177	35	1	1	3	1	24	4	4	2	2	2	0	0
Experimental Scrapie	362	34	135	26	3	2	2	2	5	1	0	0	1	1	0	0
Control	491	76	173	30	0	0	2	1	83	16	0	0	7	3	15	3

TABLE 1X

A COMPARISON OF NEUROMUSCULAR SPINDLES FROM NORMAL AND SCRAPIED SHEEP AND FROM MUSCLES SHOWING TYPE 1V CHANGE

Group	Diameter of Spindles (in Microns)		Number of Fibres Per Spindle		Diameter of Fibres (Microns)	
	Range	Average	Range	Average	Range	Average
Normal	60 - 190	100	4 - 9	6.4	4 - 25	13.0
Scrapied	40 - 300	100	3 - 10	6.2	3 - 26	11.5
Type 1V	50 - 220	101	1 - 9	4.6	4 - 30	11.8

TABLE X

THE INCIDENCE OF INTIMAL SCLEROSIS IN SCRAPIED AND NON-SCRAPIED SHEEP

Group	Spleen		Kidney		Liver		Heart		Pancreas	
	* No. +	** %	No. +	%	No. +	%	No. +	%	No. +	%
Natural Scrapie (20 Animals)	20	100	18	90	16	80	10	50	9	45
Experimental Scrapie (15 Animals)	12	80	5	33	4	26	5	33	4	26
Non-scrapie (15 Animals)	15	100	11	73	8	53	8	53	6	40

* No. + - Number of animals rated as showing intimal sclerosis.

** % - Percentage of animals rated as showing intimal sclerosis.

TABLE XI

THE INCIDENCE OF INTIMAL SCLEROSIS WITH AGE

Group	Spleen		Kidney		Liver		Heart		Pancreas	
	* No. +	** %	No. +	%	No. +	%	No. +	%	No. +	%
Foetuses (15 Animals)	1	7	0	0	1	7	0	0	0	0
0 - 1½ Yrs. (15 Animals)	13	87	7	47	5	33	8	53	4	27
1½ - 3 Yrs. (13 Animals)	12	92	11	84	11	84	7	54	7	54
Over 3 Yrs. (18 Animals)	18	100	17	94	13	72	14	78	10	56

* No. + - Number of animals rated as showing intimal sclerosis.

** % - Percentage of animals rated as showing intimal sclerosis.

TABLE XII

TYPE AND RANGE OF DETECTABLE CHANGES IN PERIPHERAL MOTOR NERVES FOLLOWING DENERVATION

Change Observed	Days Post-operative					
	3	6	9	20	30	40
Axon Fragmentation	+	+	+	+	-	-
Copper Sulphide Deposits	+	+	+	+	+	+
Neutral Fat in Myelin Sheaths	-	+	+	+	+	+
Interstitial Fat Deposition	-	-	-	-	+	+

Fig. 1 Camera lucida drawing of muscle fibre cross sections. Trapezius muscle from a slightly emaciated normal control animal showing a lack of fibre uniformity. Case C-13.

Fig. 2 Fibre frequency curves of the trapezius muscles of a normal sheep (C-17) and two emaciated sheep (C-16, S-9) from which the diameter of six hundred fibres was plotted against frequency.

Fig. 1 X 170

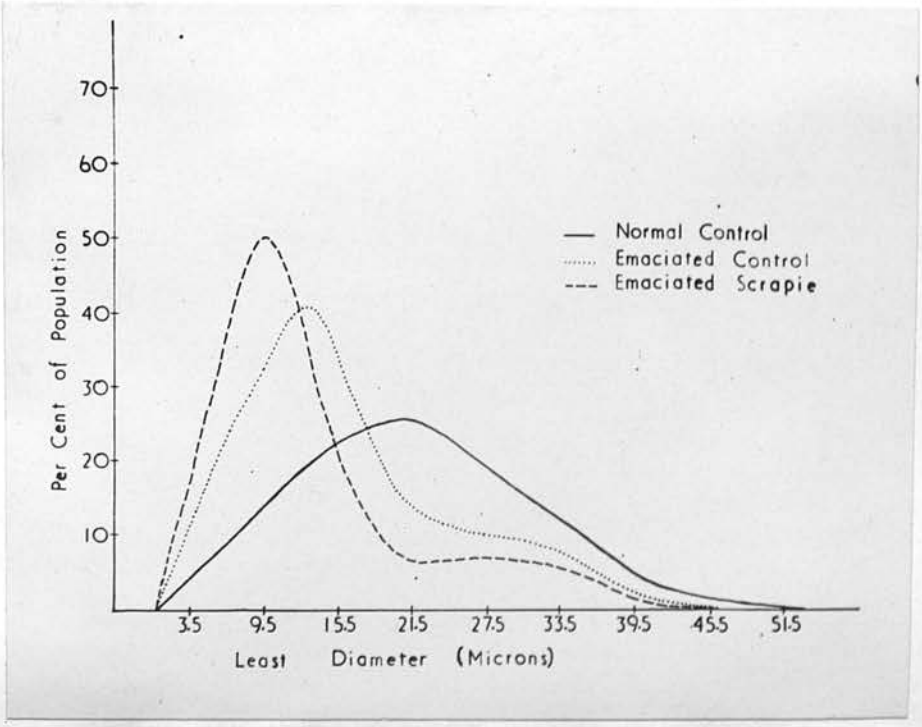

Fig. 2

Fig. 3 Camera lucida drawings of (A) longissimus dorsi, (B) trapezius and (C) supraspinatus muscles of a normal control animal of the Cheviot breed. Case C-14.

Fig. 4 Camera lucida drawings of the same three muscles, in the same order as in Fig. 3, from an emaciated control Cheviot. Case C-16.

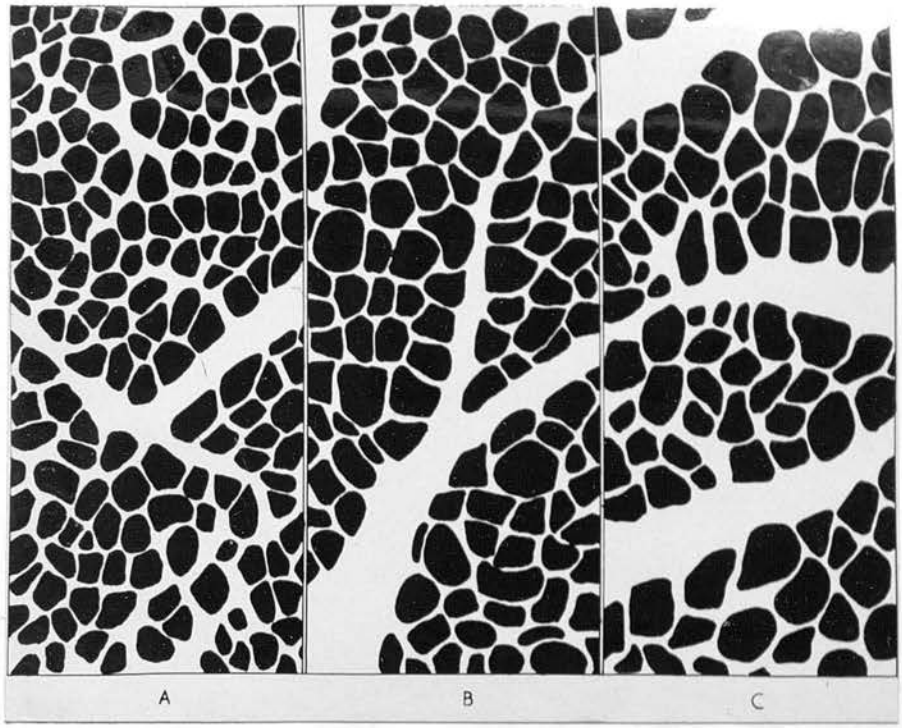

Fig. 3 X 170

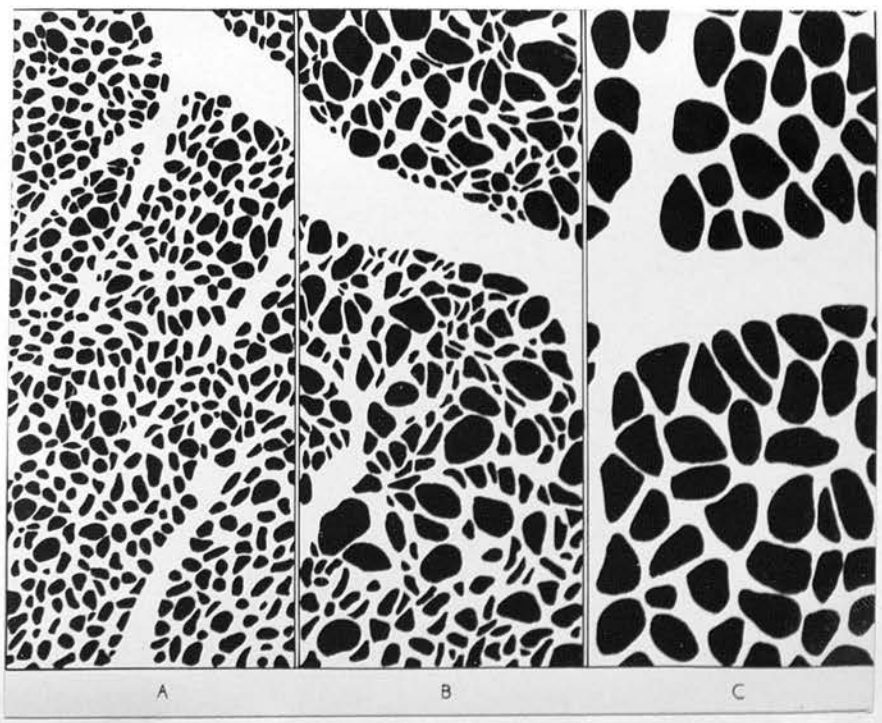

Fig. 4 X 170

Fig. 5 Camera lucida drawing of fibre cross sections from a superficial pectoral muscle of a scrapied Suffolk sheep showing a prominent population of large fibres even though there is little general reduction in fibre diameters. Case S-18.

Fig. 6 Camera lucida drawing of a serratus muscle from an emaciated Swaledale sheep showing general reduction of fibre diameter and a prominent population of large fibres. Compare with Fig. 5. Case S-13.

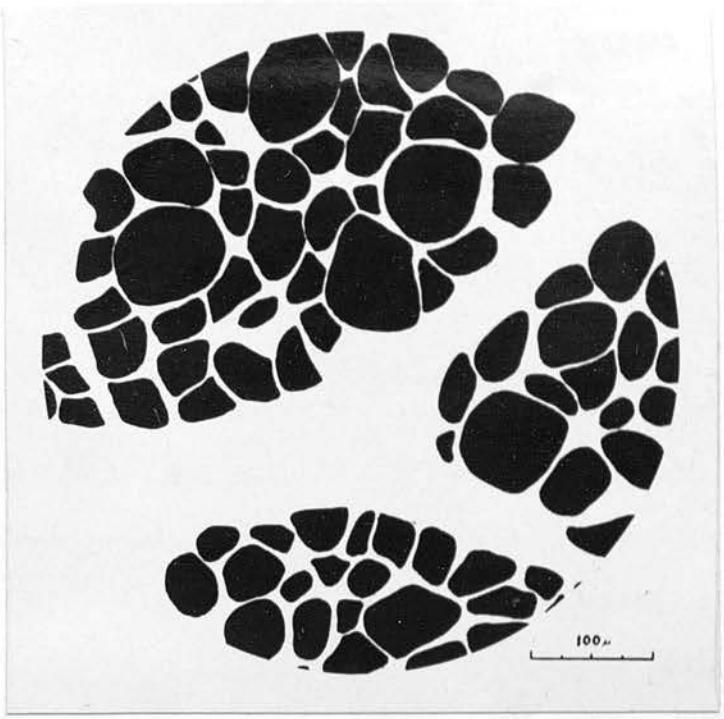

Fig. 5 X 170

Fig. 6 X 170

Fig. 7 Camera lucida drawing of fibres from two muscles from a single animal showing a disparity of fibre sizes. Left, semitendinosis muscle, right, biceps femoris muscle. Case S-5.

Fig. 8 Camera lucida drawings of two regions of the same trapezius muscle showing a disparity of fibre diameters. Case E-9.

Fig. 7 X 170

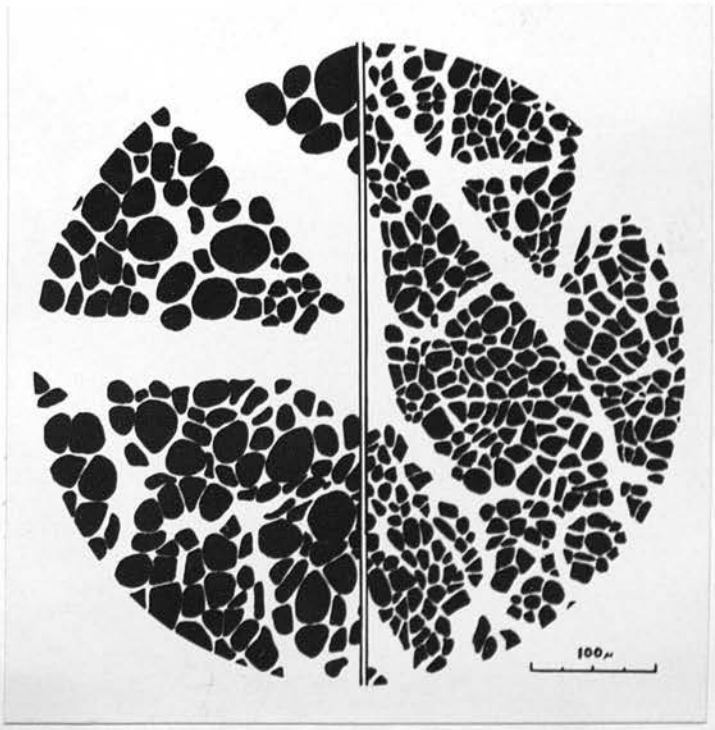

Fig. 8 X 170

Fig. 9 Camera lucida drawing of two areas of a single gracilis muscle demonstrating differences in fibre size. Case E-14.

Fig. 10 Camera lucida drawing of a single microscopic field of an atrophic trapezius muscle. Despite the general reduction in fibre diameter, the large fibres at the left are as large as or larger than any in a comparable normal muscle. Case S-17.

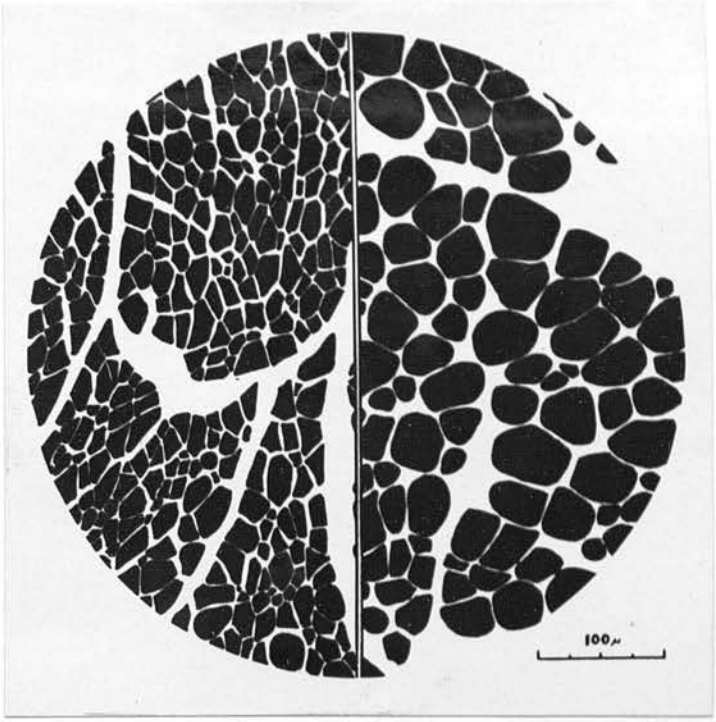

Fig. 9 X 170

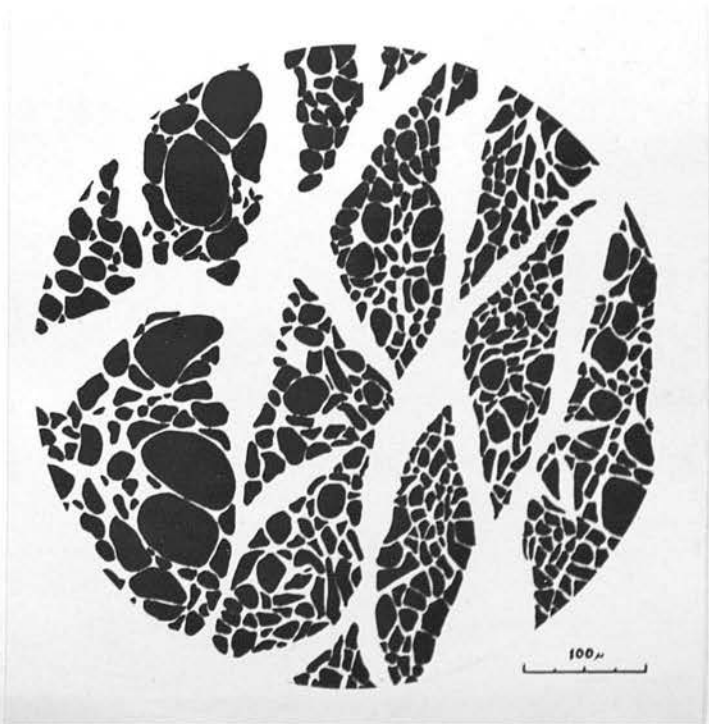

Fig. 10 X 170

Fig. 11 Camera lucida drawing of a single field of an atrophic trapezius muscle showing a difference in fibre diameters across a major perimyseal band but not between primary bundles on the same side of the band. Case E-9.

Fig. 12 Mixed large fibres and small angular fibres observed in advanced emaciation. The shape of some of the small fibres appears to depend on the pressure exerted by adjacent large fibres. Note the absence of cellular degenerative changes even in the smallest fibres. Case S-17, H&E.

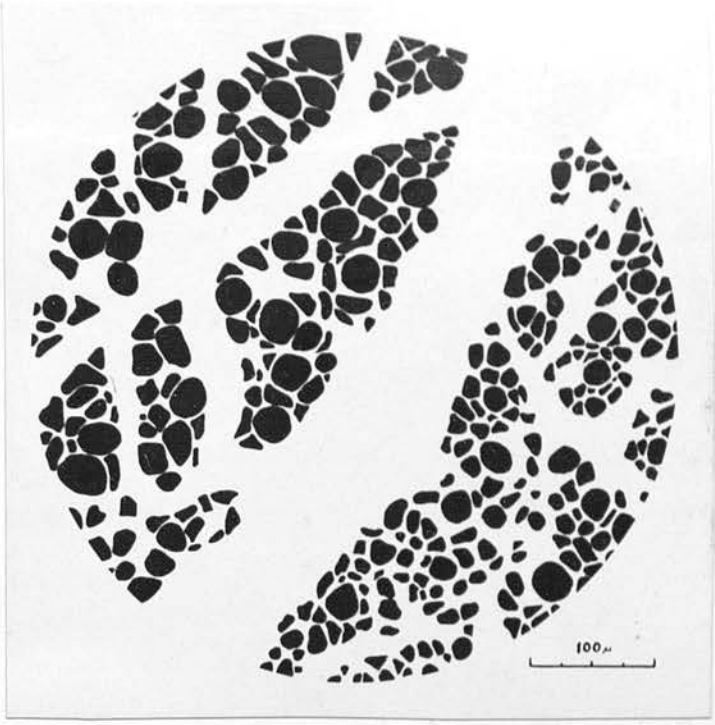

Fig. 11 X 170

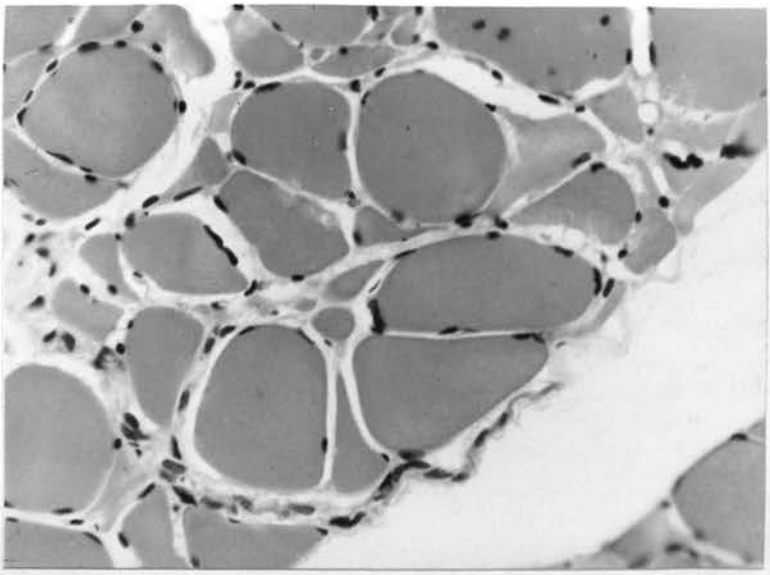

Fig. 12 X 485

Fig. 13 Type 1 reaction surrounding a fibre containing a small sarcocyst. A second sarcocyst to the right is not surrounded by cells. Case E-4, H&E.

Fig. 14 Type 1 change showing proliferating muscle nuclei replacing muscle substance. Note the central nuclei in adjacent, apparently normal muscle fibres. Case C-11, H&E.

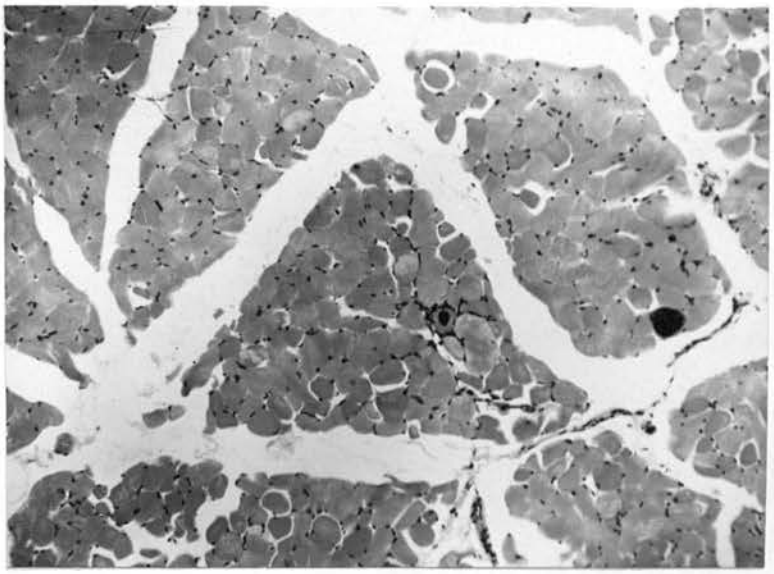

Fig. 13 X 190

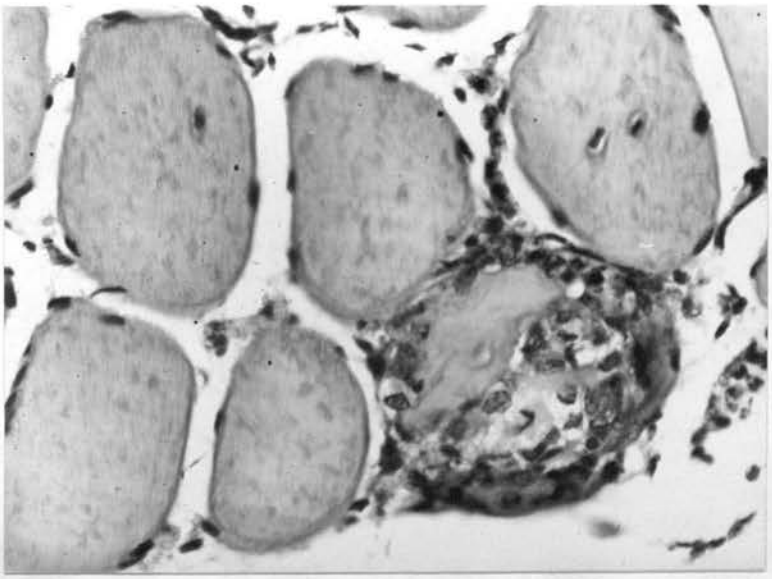

Fig. 14 X 485

Fig. 15 Type 11 change showing a diffuse increase in muscle nuclei. For comparison of nuclear density, see Fig. 13. Case E-7, H&E.

Fig. 16 Type 11 change in the same general area as Fig. 15. At least three fibres are ringed in muscle nuclei. Case E-7, H&E.

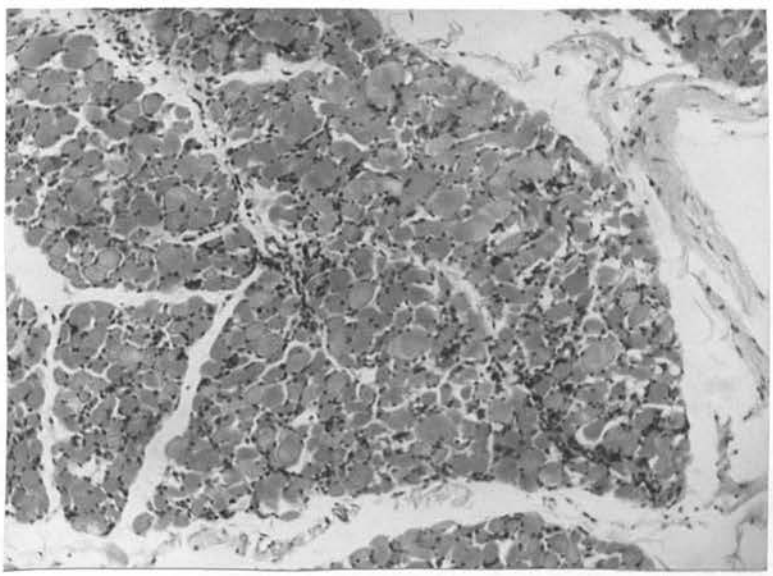

Fig. 15 X 190

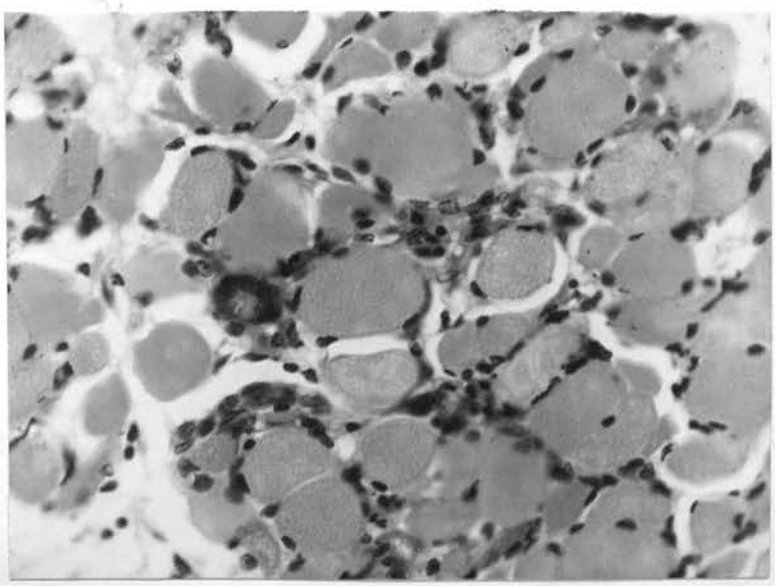

Fig. 16 X 485

Fig. 17 Type III fibrotic lesion showing occasional degenerate muscle fibres and heavy connective tissue bands, particularly around the intramuscular nerve in the upper left of the figure. Case S-14, H&E.

Fig. 18 Type III lesion showing an increase in perimyseal and endomyseal connective tissue. A small muscle fibre (arrow) appears to have escaped from the general fibre organization and has grown into the perimyseum. Case C-33, kiton fast red - light green.

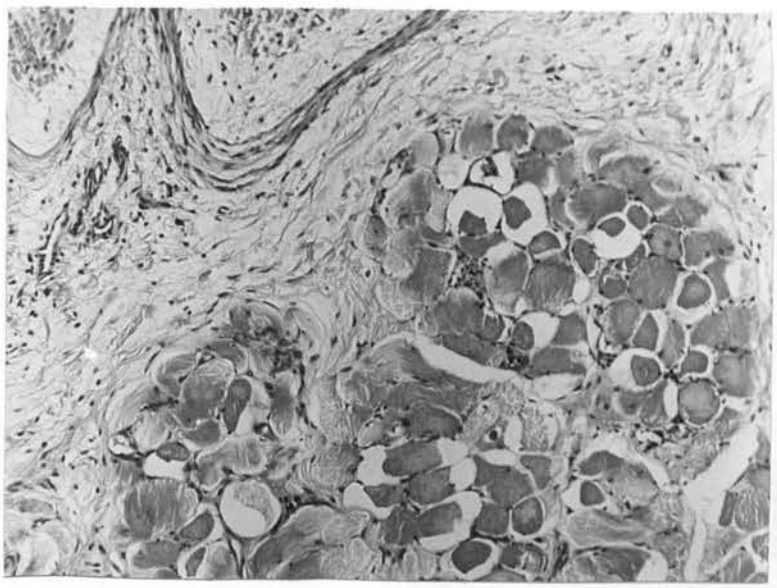

Fig. 17 X 190

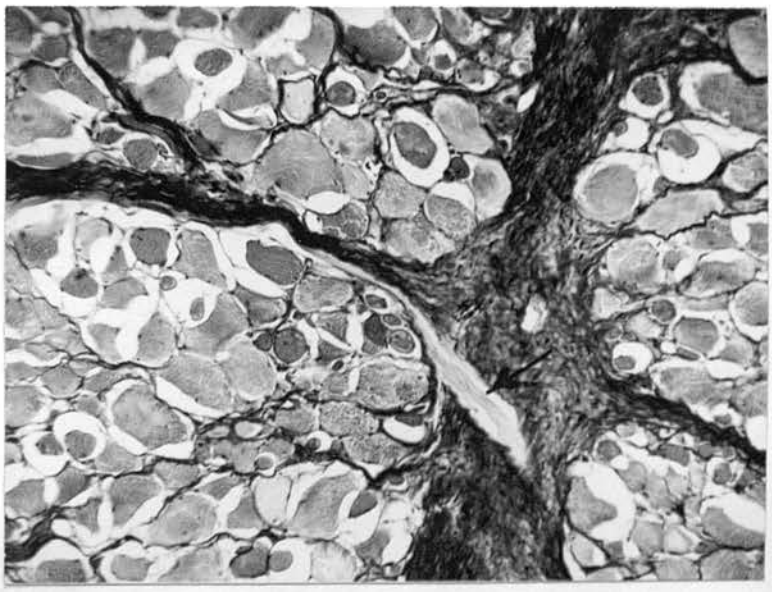

Fig. 18 X 190

Fig. 19 Acute type IV degeneration. The two primary bundles at lower right contain many apparently normal fibres, while the fibres in the three bundles above are vacuolated and those to the left of the figure are sparse, fragmented and swollen. Red blood cells lie between the primary bundles. Case C-61, P.T.A.H.

Fig. 20 Longitudinal section from an area comparable to that in Fig. 19. Note the contracted fragments and the vacuolation in a few fibres. Case C-52, H&E.

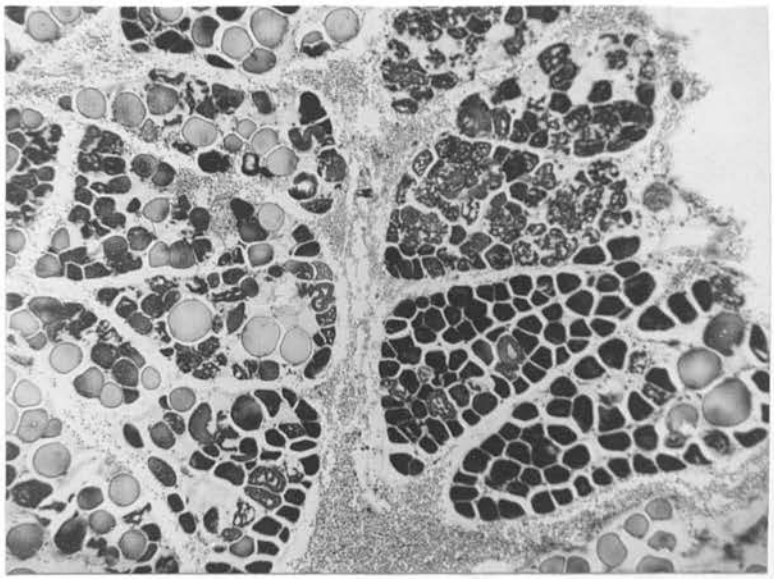

Fig. 19 X 190

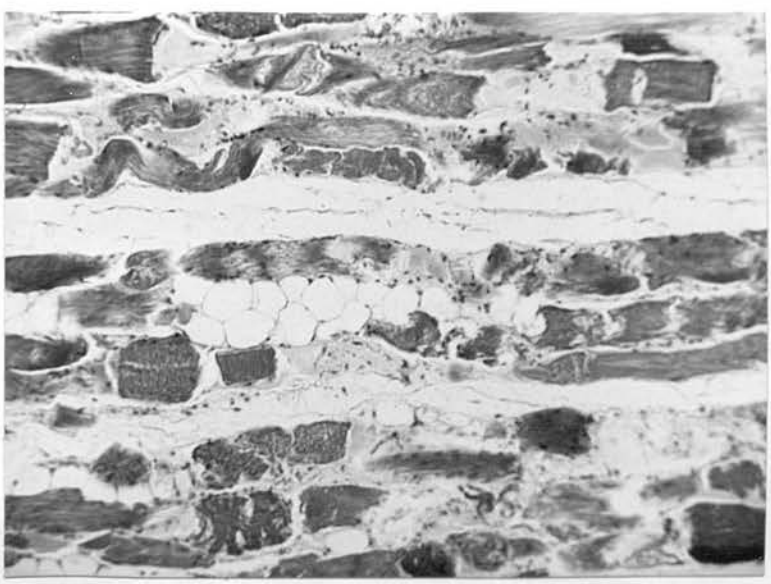

Fig. 20 X 190

Fig. 21 Type 1V degeneration. Fragmented fibres with little or no reaction surrounding them lie between thin, twisted fibres. Some central nuclei are visible. Case C-52, H&E.

Fig. 22 Type 1V degeneration showing a vacuolated muscle fragment above and many small fibres below, some of which have rows of central nuclei. Case C-46, H&E.

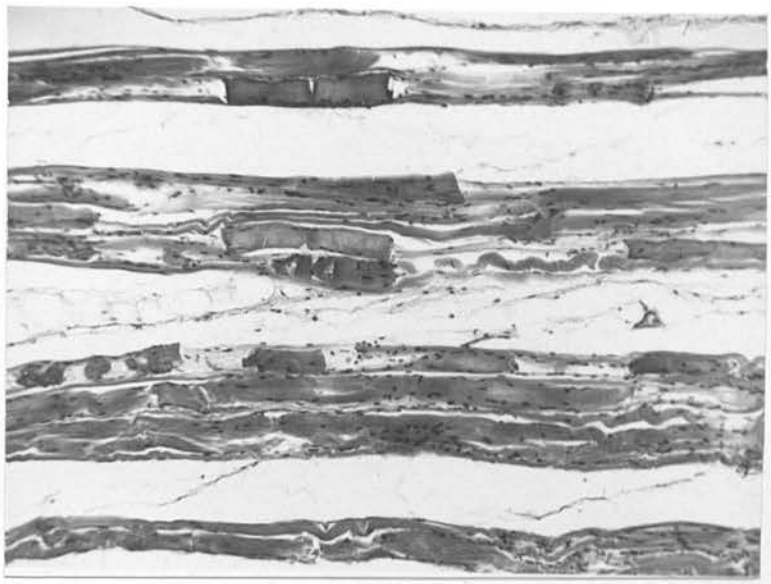

Fig. 21 X 190

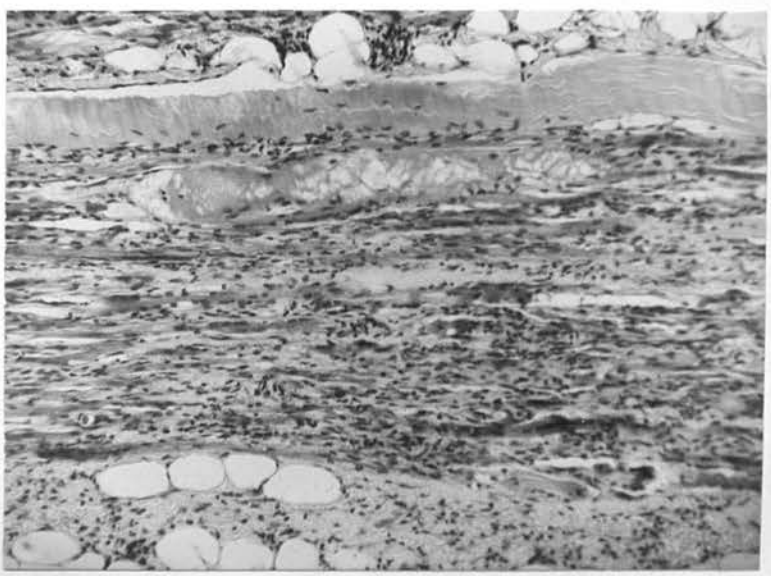

Fig. 22 X 190

Fig. 23 Type 1V degeneration showing (a) sarcolemmal nuclei proliferating around selectively degenerate muscle fibres and (b) calcification in the same area. Case S-19, (a) H&E (b) von Kossa.

Fig. 24 Type 1V degeneration. Note the granular remnants of two fibres and the presence of multinucleated cells. Case S-19, H&E.

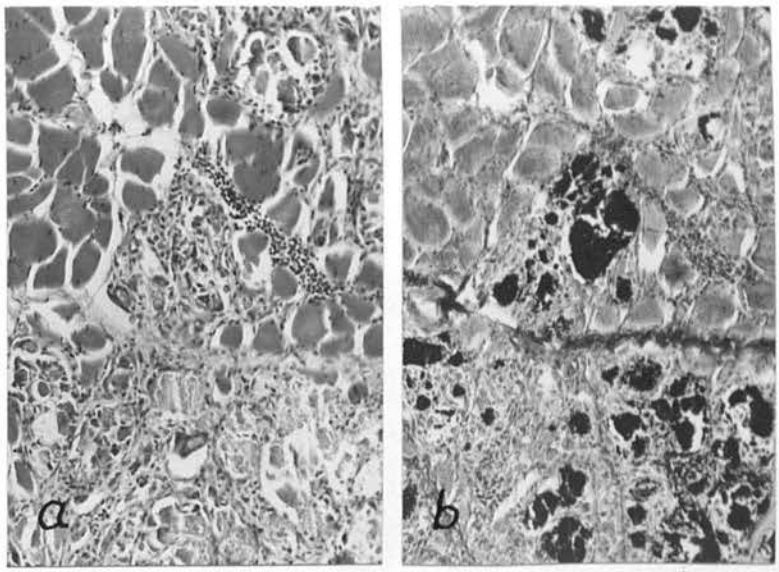

Fig. 23 X 190

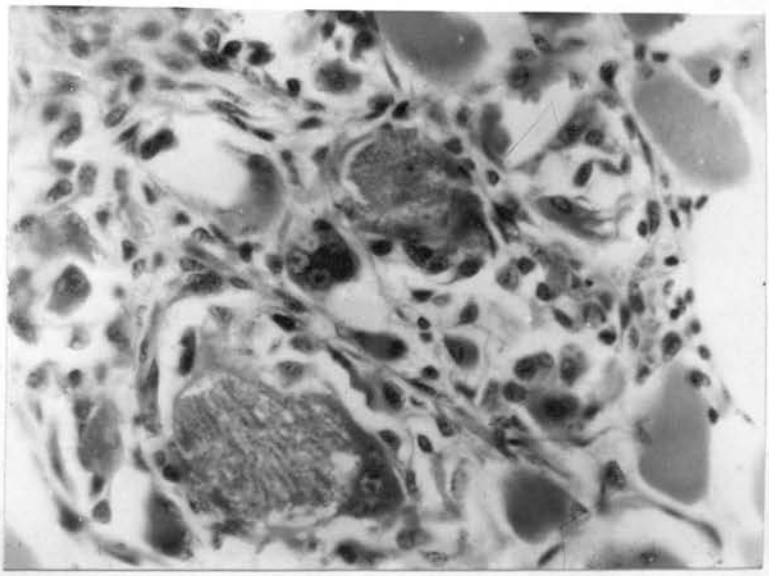

Fig. 24 X 485

Fig. 25 Type 1V degeneration with granular, degenerate fibres in the upper part of the figure and many apparently normal fibres in the lower part. Lamb muscle, H&E.

Fig. 26 Type 1V degeneration. Many small fibres and a few large, granular and normal fibres are embedded in connective tissue. Fat cells are visible as clear vacuoles. Case C-50, H&E.

Fig. 25 X 190

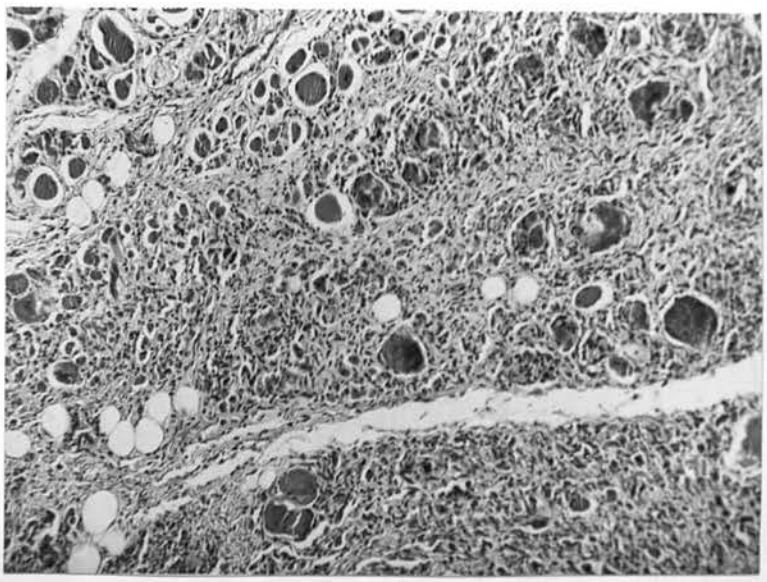

Fig. 26 X 190

Fig. 27 Type 1V degeneration. One of many foci of round cells visible in a muscle section composed uniformly of very small fibres. Primary bundle divisions have been obscured. Case C-46, H&E.

Fig. 28 Type 1V degeneration showing fibres of extremely variable size embedded in dense connective tissue. Primary bundles are difficult to define. Case C-76, H&E.

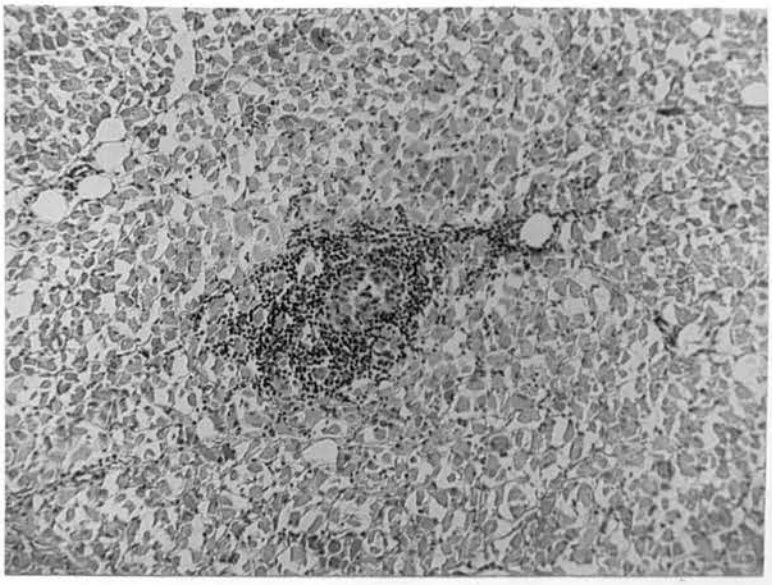

Fig. 27 X 190

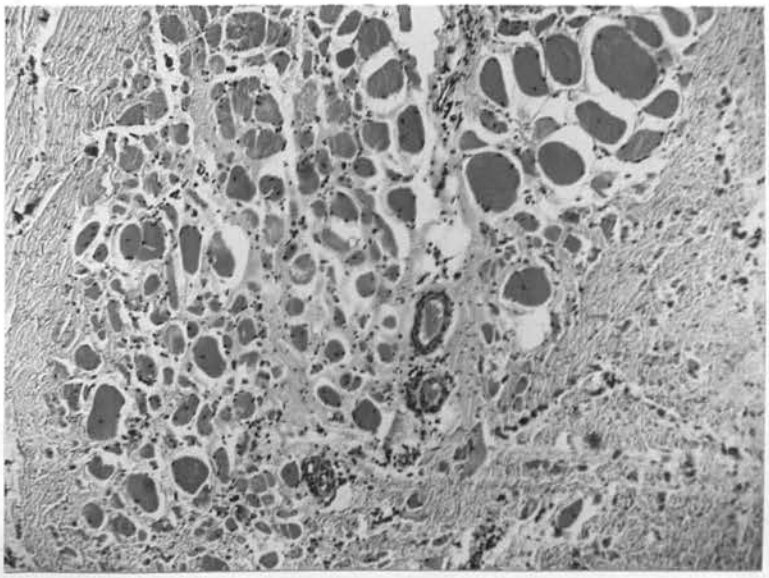

Fig. 28 X 190

Fig. 29 Type 1V change with a diffuse increase in muscle nuclei and rows of fat cells apparently replacing fibres which have degenerated. Case C-48, H&E.

Fig. 30 Type 1V degeneration with foci of necrotic fibres. The two islands of large fibres on the left appear to lack normally distributed nuclei while those on the right do contain marginal nuclei. Case S-55, H&E.

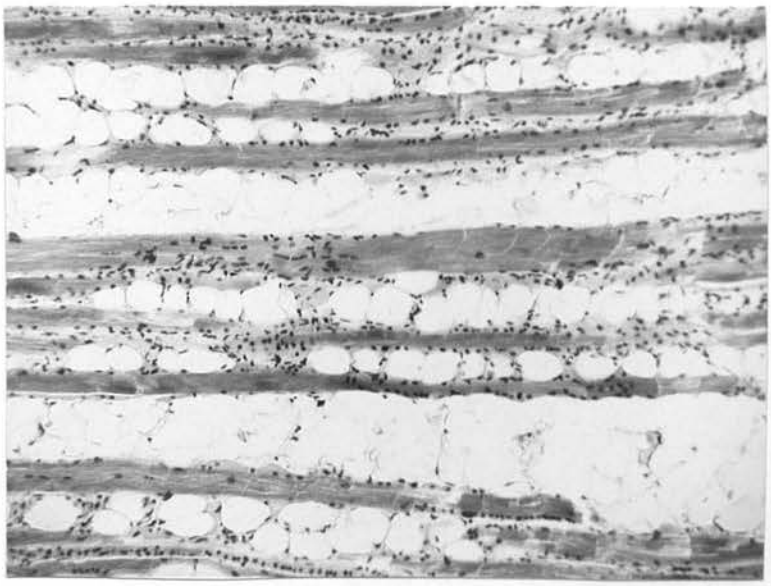

Fig. 29 X 190

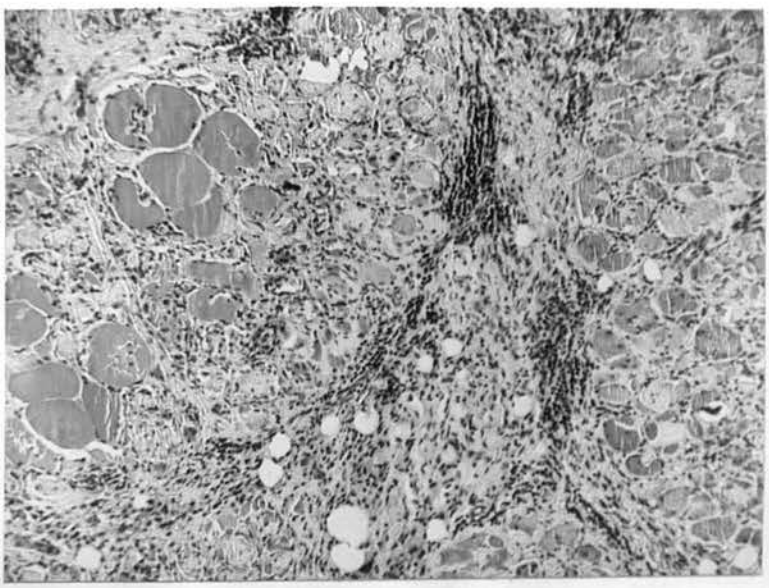

Fig. 30 X 190

Fig. 31 Type V muscular lesion illustrating mixed hypertrophy and atrophy. Several fibres contain central nuclei and fat deposits distort the muscle architecture. Case S-58, H&E.

Fig. 32 Type V lesion showing an atrophic group of muscle fibres and a mass of connective tissue (upper) in which are rows of nuclei suggestive of muscle fibre remnants. The surrounding fat is extensive. Case S-58, H&E.

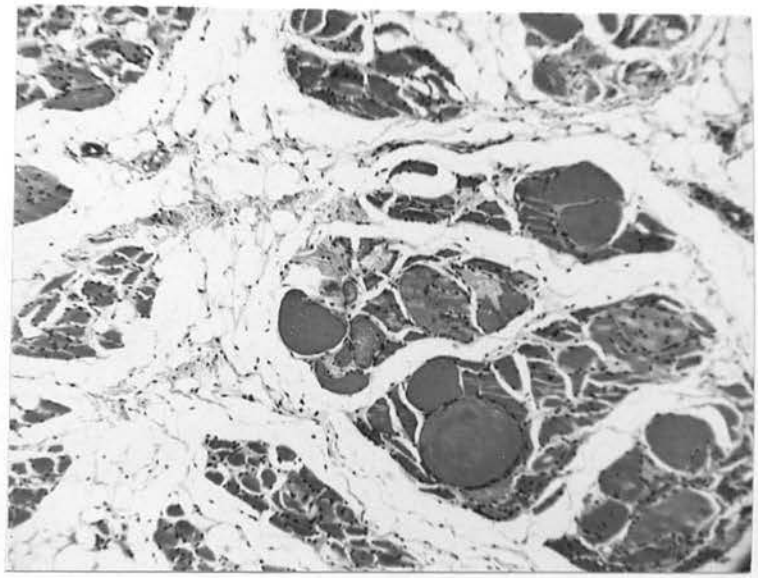

Fig. 31 X 190

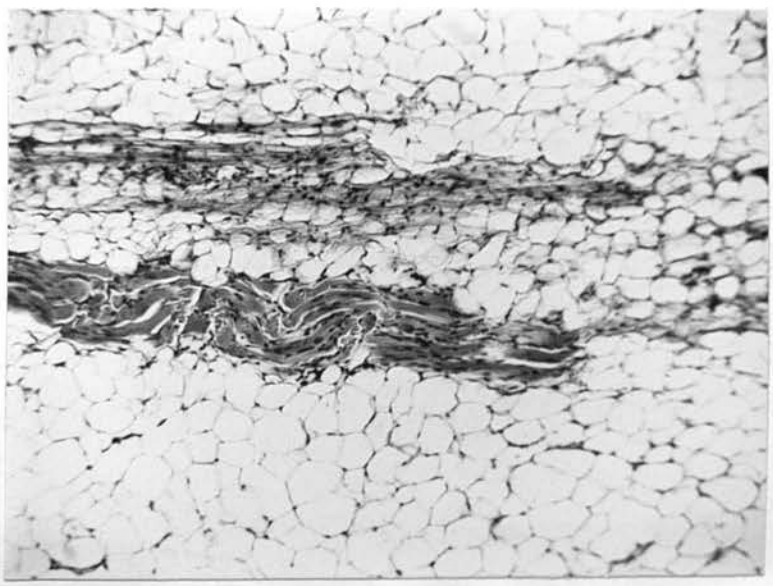

Fig. 32 X 190

Fig. 33 Type V lesion. (a) Trapezius muscle illustrating a few very large fibres, some of which have central nuclei. The large irregular fibre at lower center is incompletely divided by septae and a group of small fibres above are embedded in heavy connective tissue. (b) Comparable normal muscle. Cases S-60 and S-18, (a) and (b) van Gieson.

Fig. 34 Type V degeneration. Longitudinal section of a large fibre similar to those in Fig. 33. The central clefts appear to begin and end at any point along the fibre. Case S-60, kiton fast red - light green.

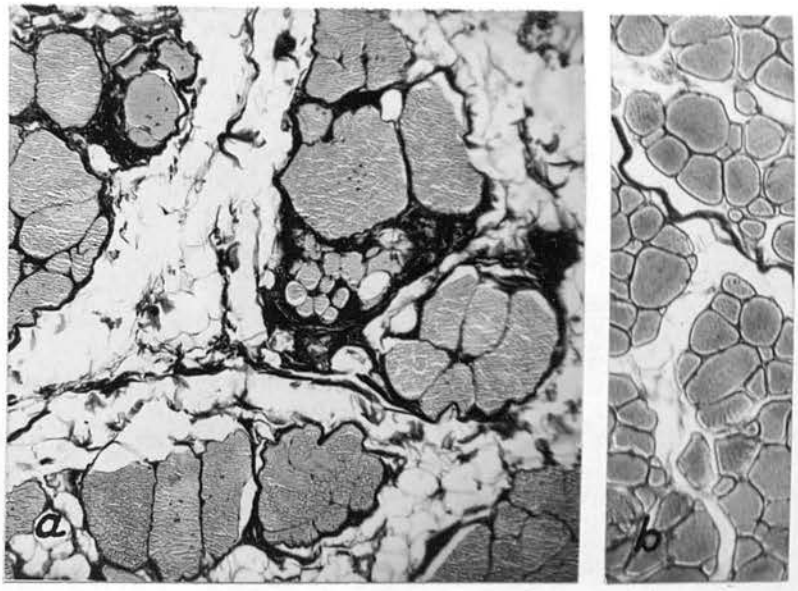

Fig. 33 both X 190

Fig. 34 X 190

Fig. 35 Parasitic myositis. An anuclear mass of debris can be observed in the lower left hand corner. An eccentric zone of granulation tissue (pale area) is surrounded by a very cellular zone. Separated muscle fibres appear at the right. Case C-51, H&E.

Fig. 36 Mycotic myositis associated with a "ray" colony of bacteria. Multinucleated cells can be seen surrounding the central focus. Muscle fibres are separated by infiltrating cells. Case C-76, H&E.

Fig. 35 X 50

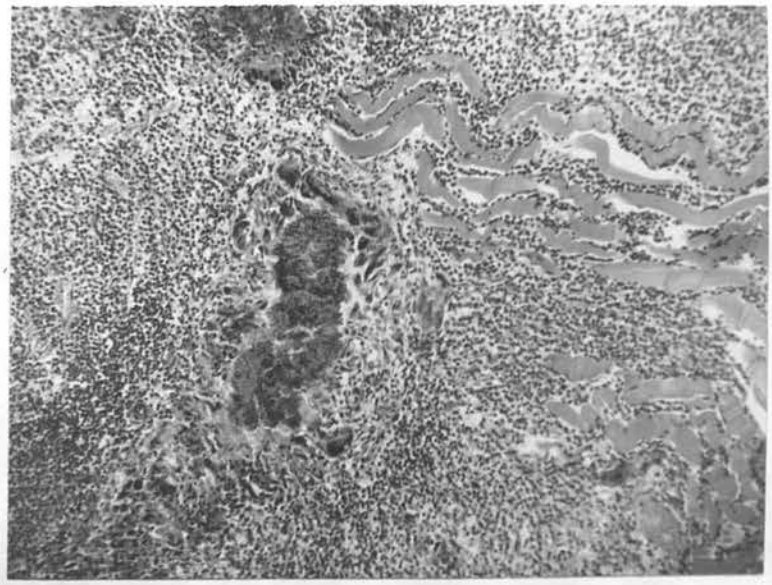

Fig. 36 X 190

Fig. 37 Infarction. A zone of necrotic, anuclear muscle fibres separates a mass of crystals (upper right) and viable but small muscle fibres (lower left). Case C-67, H&E.

Fig. 38 Infarcted muscle at upper right is undergoing organization by round cells and small, dark muscle fibres. A zone of atrophic fibres is just visible at the lower left. Case C-75, H&E.

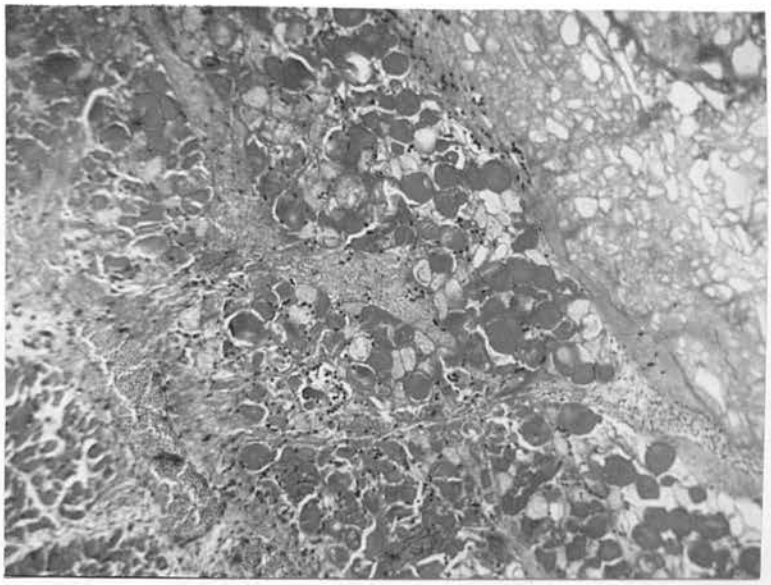

Fig. 37 X 190

Fig. 38 X 190

Fig. 39 The cross striations on the broad central fibre appear to lack the Qh and Z lines in the middle of alternate dark and light bands. In the other three fibres the Qh lines (dividing the dark bands) can be seen and the Z lines are visible at the bottom left of the figure (arrow). Case C-74, kiton fast red - light green.

Fig. 40 (a) The myofibrils of the central fibre are completely interrupted to the right and the sarcolemmal sheath is indented. At the left, Qh and Z lines as well as the Q and J bands are clearly visible. Between these extremes are several areas which have lost one or both of the lines. (b) Discoid degeneration in which the bands and lines are clearly visible throughout the fragmented area. (a) Lamb muscle (b) Case C-74, kiton fast red - light green.

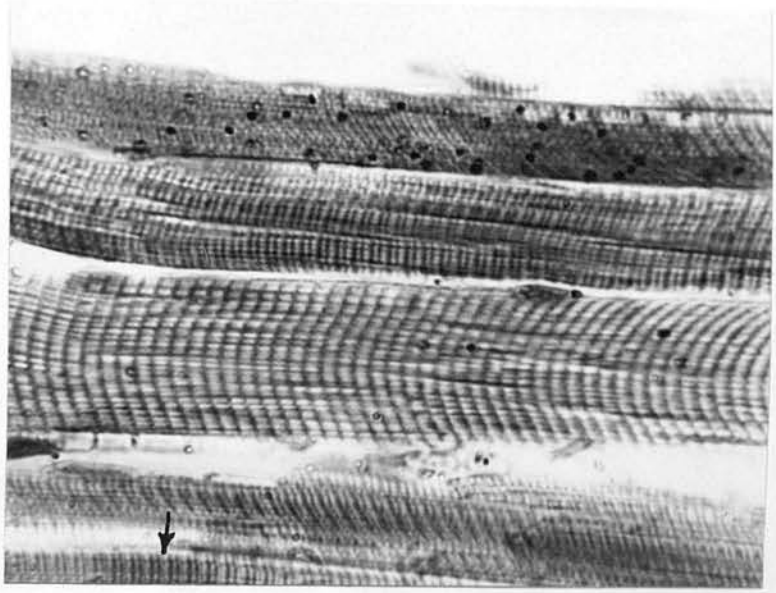

Fig. 39 X 1200

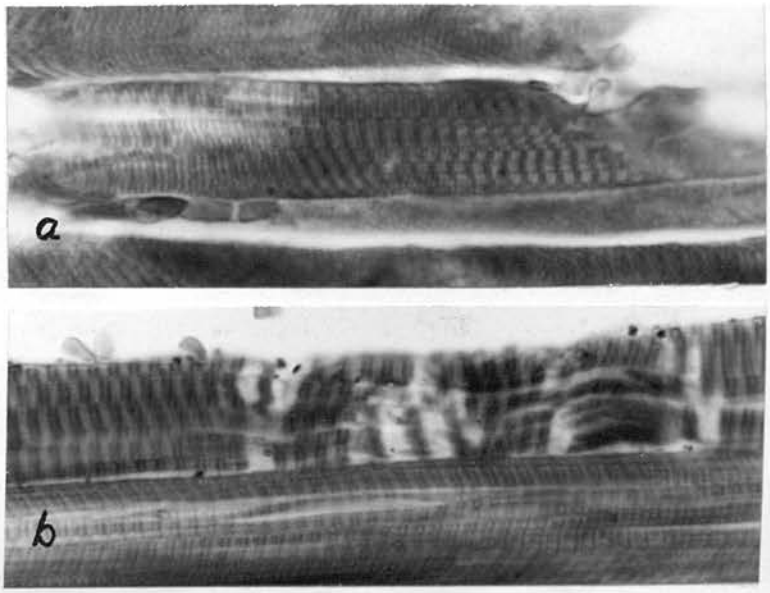

Fig. 40 X 1200

Fig. 41 A very large sarcocyst distends a muscle fibre to many times normal size. The septae in the capsule of Miescher's tube are clearly visible. Case S-32, H&E.

Fig. 42 A sarcocyst in a muscle fibre is surrounded by nuclei, a few of which can be seen penetrating the muscle fibre. Rainey's corpuscles are not clearly visible. Case C-20, H&E.

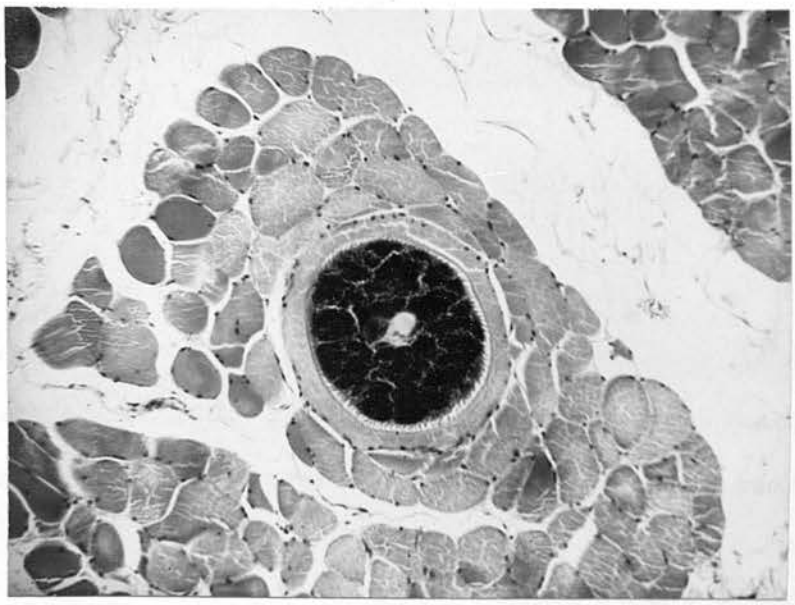

Fig. 41 X 190

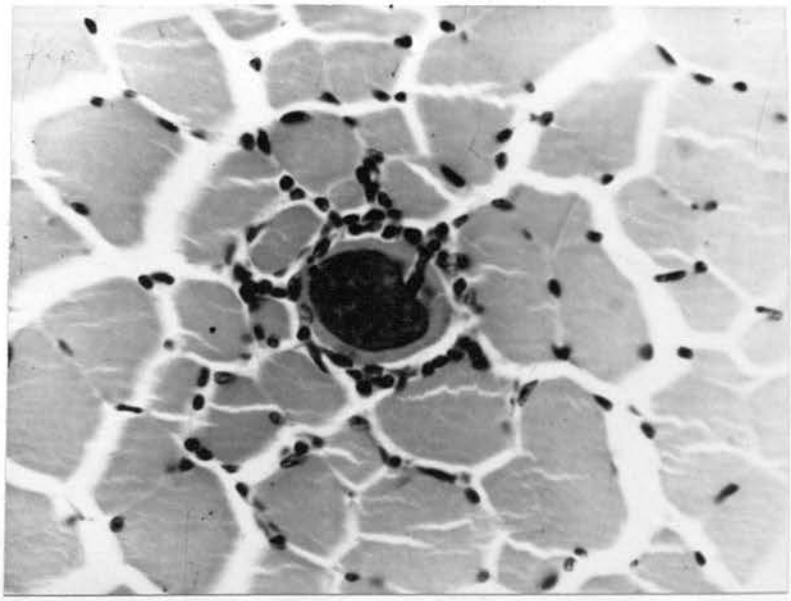

Fig. 42 X 485

Fig. 43 Striated muscle-fibre annulets from a semitendinosus muscle lesion. (a) Cross sectional appearance of a complex ring. The small groups of vertical myofibrils (arrows) have been separated from the main vertical fibre by the annulet. (b) Longitudinal section of a simple ring which appears to be constricting the main fibre. Case C-33, (a) kiton fast red - light green, (b) Holmes' silver.

Fig. 44 Longitudinal section of two striated muscle-fibre annulets surrounding a single fibre in a semitendinosus muscle. The two groups of encircling myofibrils appear to be quite distinctly separated by an expanded portion of the vertical fibre. Case C-33, Holmes' silver.

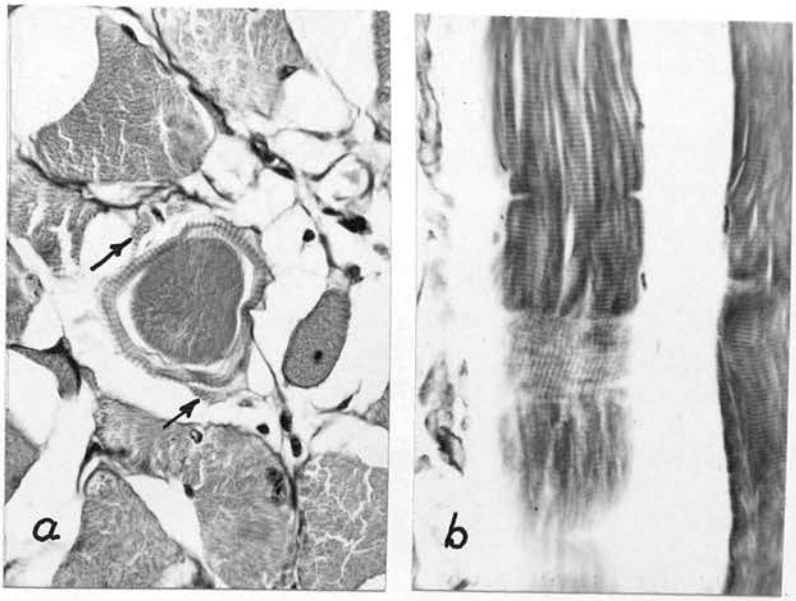

Fig. 43 X 485

Fig. 44 X 485

Fig. 45 This simple annulet is completely sub-sarcolemmal. The nucleus in the ring is apparently oriented parallel to the encircling fibrils. Case C-33, kiton fast red - light green.

Fig. 46 Sarcoplasmic masses in two muscle fibres in which the red myofibrils have been reduced in number and / or volume - and pressed to one side. The light green masses are apparently sarcoplasm. Case S-14, kiton fast red - light green.

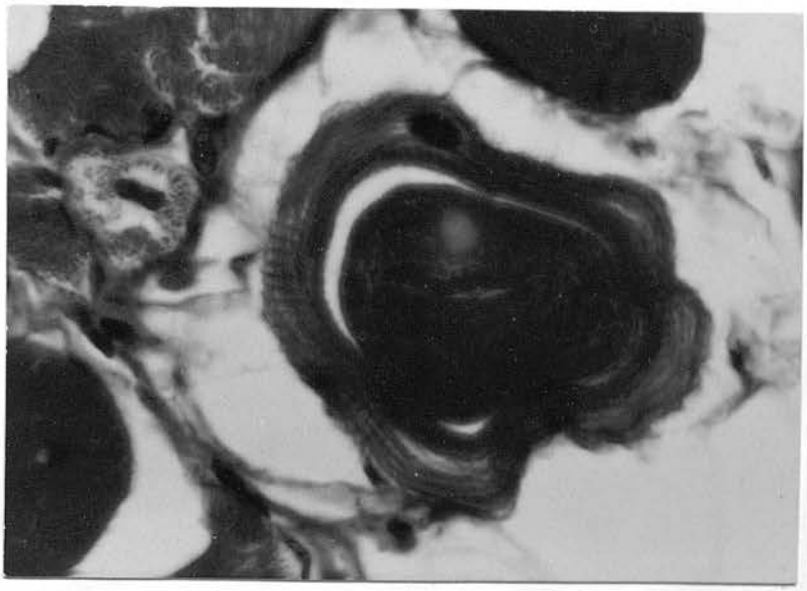

Fig. 45 X 1000

Fig. 46 X 900

Fig. 47 Neuromuscular spindle in which the intrafusal fibres are fragmented, as are the extrafusal fibres. Case C-61, P.T.A.H.

Fig. 48 Intrafusal muscle fibres. The fibre to the left has fragmented and formed a contracted fragment and an empty sarcolemmal sheath (above). The spindle capsule is to the right. Case E-25, H&E.

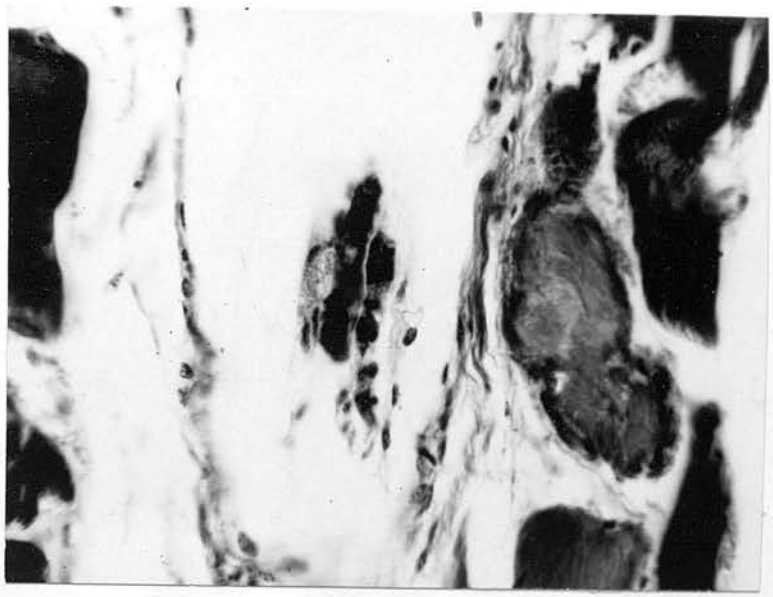

Fig. 47 X 485

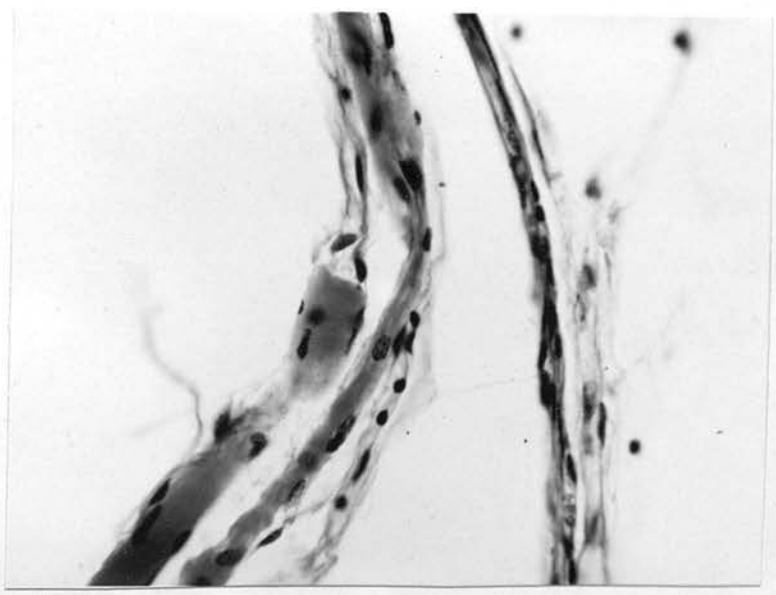

Fig. 48 X 1200

Fig. 49 Complex neuromuscular spindle containing seven groups of intrafusal fibres. One pair of spindles appears to be incompletely divided. Case C-72, H&E.

Fig. 50 Fibrotic spindle. Connective tissue partially fills the intrafusal space and forms a heavy sheath around extrafusal muscle fibres and primary bundles. Case S-14, kiton fast red - light green.

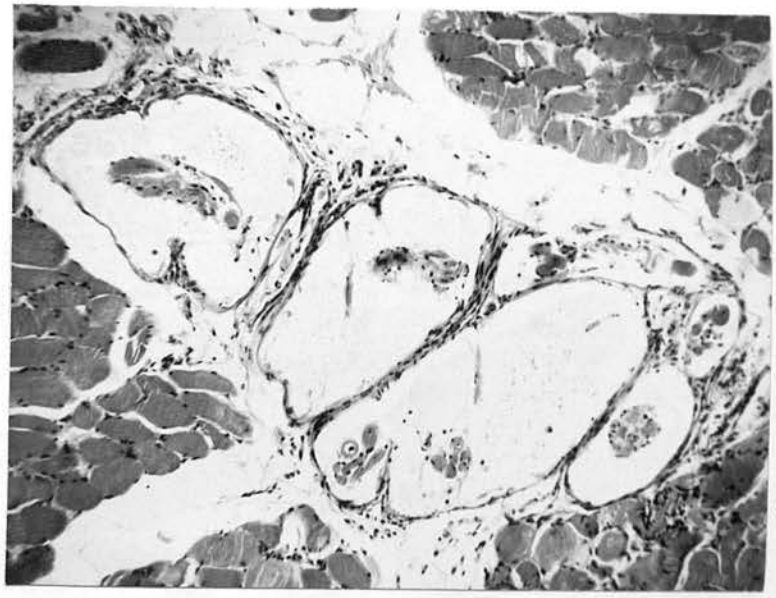

Fig. 49 X 190

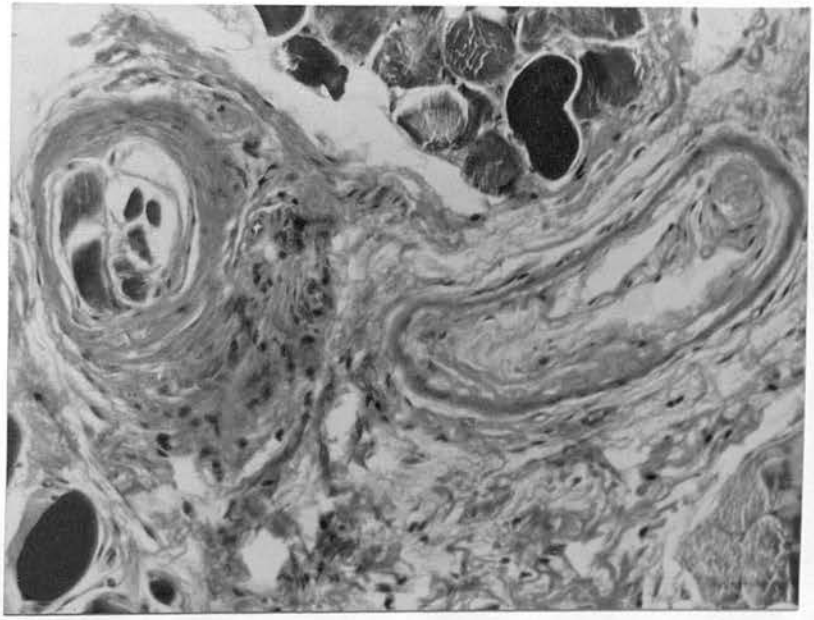

Fig. 50 X 225

Fig. 51. Normal motor end plates in sheep muscle demonstrated by the gold chloride - paraffin section method. Note the pleomorphism of end plates and the varicosity of terminal axons. Gracilis muscle, case C-34.

Fig. 52 Normal motor end plates, gold chloride - paraffin section method, to show muscle fibre striations and sole plate nuclei (grey). Case C-34.

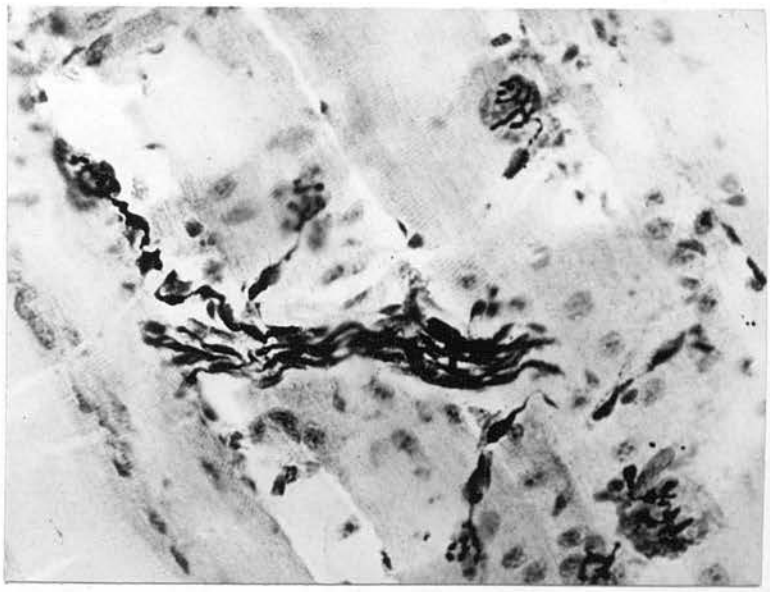

Fig. 51 X 485

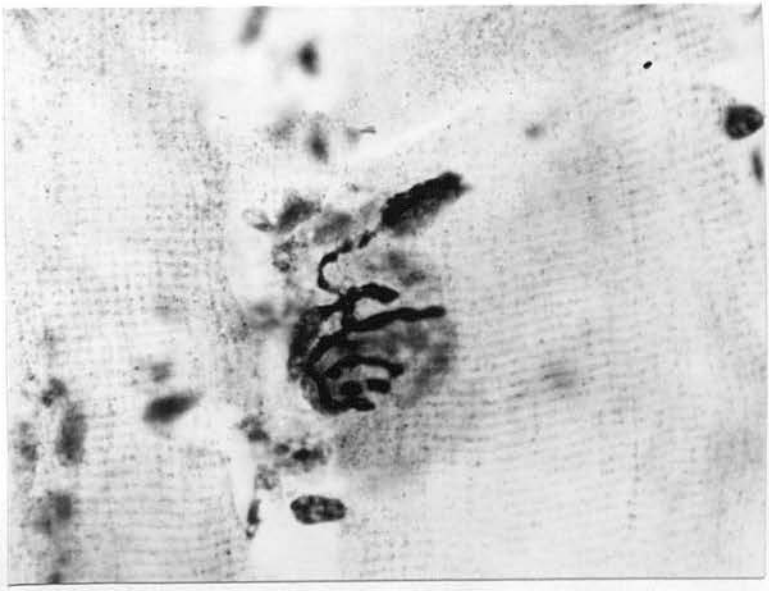

Fig. 52 X 1200

Fig. 53 Motor end plates in an atrophic trapezius muscle showing variation in the size of adjacent plates. Gold chloride - frozen section method. Case C-40.

Fig. 54 Two normal motor end plates one of which is enclosed in a distinct Doyère's hillock in profile. Gold chloride - frozen section method. Semimembranosus, case C-35.

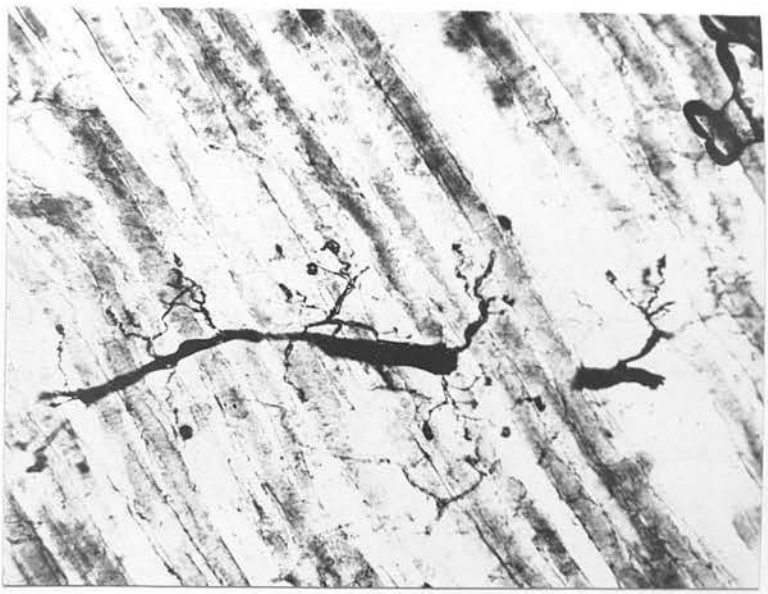

Fig. 53 X 190

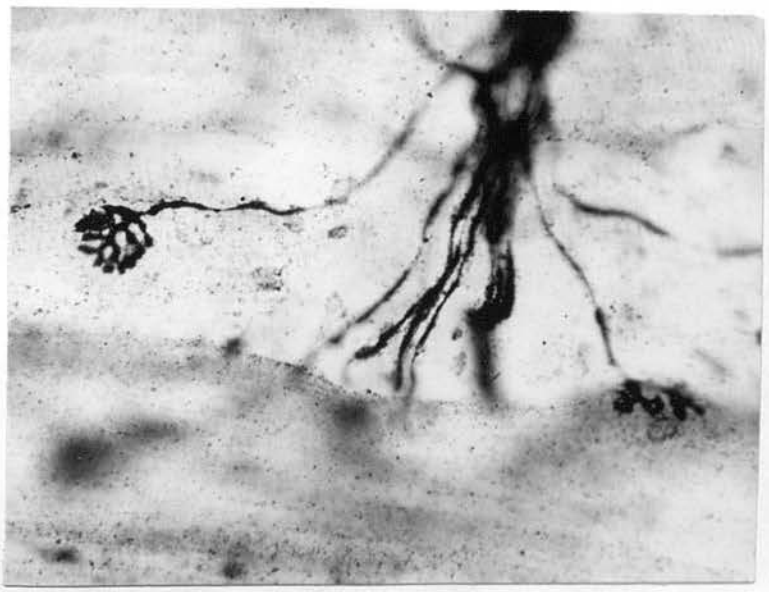

Fig. 54 X 485

Fig. 55 (a) Normal motor end plates demonstrated by the cholinesterase - silver method. Latissimus dorsi muscle. (b) Cross section of the same muscle, H&E. Case C-43.

Fig. 56 (a) Motor end plates from an emaciated control animal demonstrating a reduced end plate size. Cholinesterase - silver method. (b) Cross section of the same muscle (latissimus dorsi) stained by H&E. Note the relatively uniform reduction in end plate and fibre diameter. Case C-44.

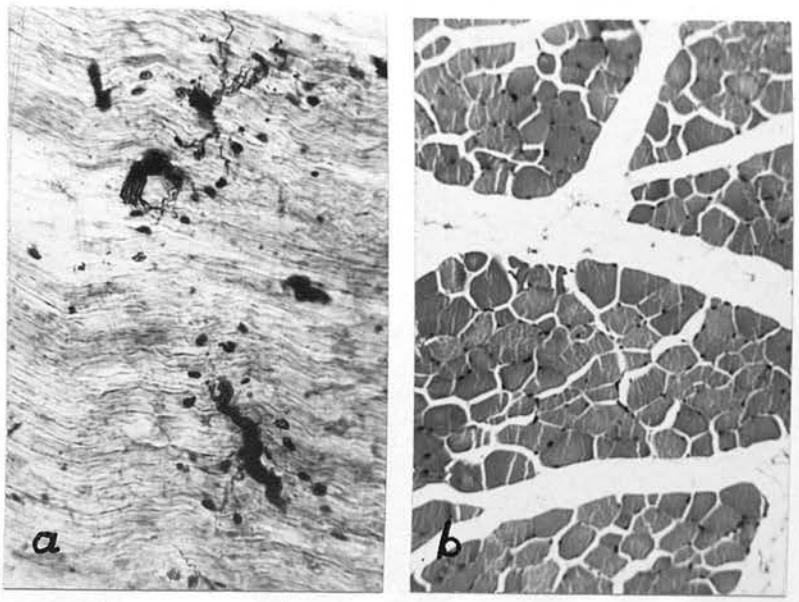

Fig. 55 X 190

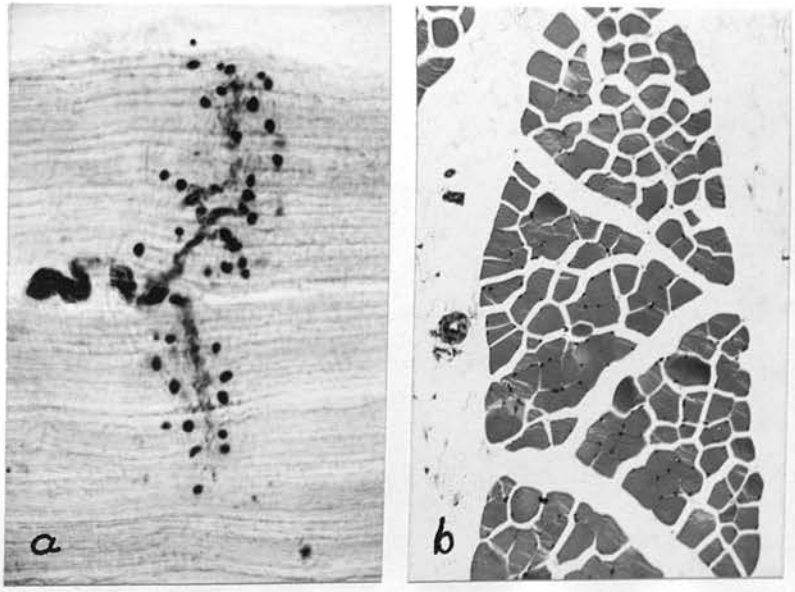

Fig. 56 X 190

Fig. 57 (a) Motor end plates in an atrophic trapezius muscle. Cholinesterase - silver preparation. (b) Cross section of the same muscle, H&E. Note the variation in fibre and end plate size.

Fig. 58 Motor endings in an atrophic supraspinatus muscle. Cholinesterase - silver preparation. The axons are very crenated in some areas. Case S-57.

Fig. 57 X 190

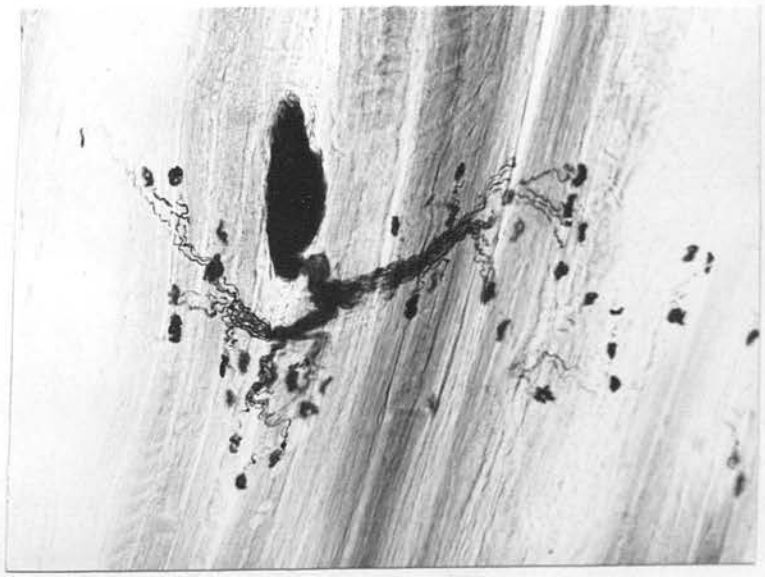

Fig. 58 X 225

Fig. 59 Motor nerve end plate in a fibrotic type lll muscle lesion demonstrated by Holmes' silver method. The teliodendria appear to have reunited in the coarse, circular end plate visible (arrow). Compare to Fig. 65. Case C-33.

Fig. 60 Sub-terminal branching in the same muscle as Fig. 59. The axon at "a" goes to a motor end plate at "b" and a sub-terminal branch "c" is given off. A second fine, sub-terminal branch is given off at "d". Case C-33, Holmes' silver method.

Fig. 59 X 485

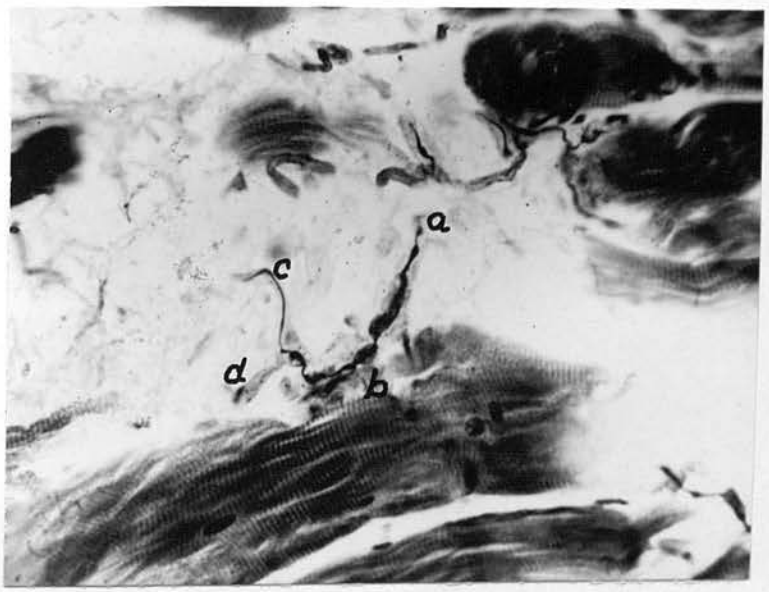

Fig. 60 X 485

Fig. 61 Motor innervation in a type IV muscle lesion. The small bundle of axons is fragmented and disconnected end plates appear above and to the right. Cholinesterase - silver preparation. Case S-53.

Fig. 62 Higher power of the box in Fig. 61. The end plates lack definite form and the axons appear as argentophilic beads and fragments.

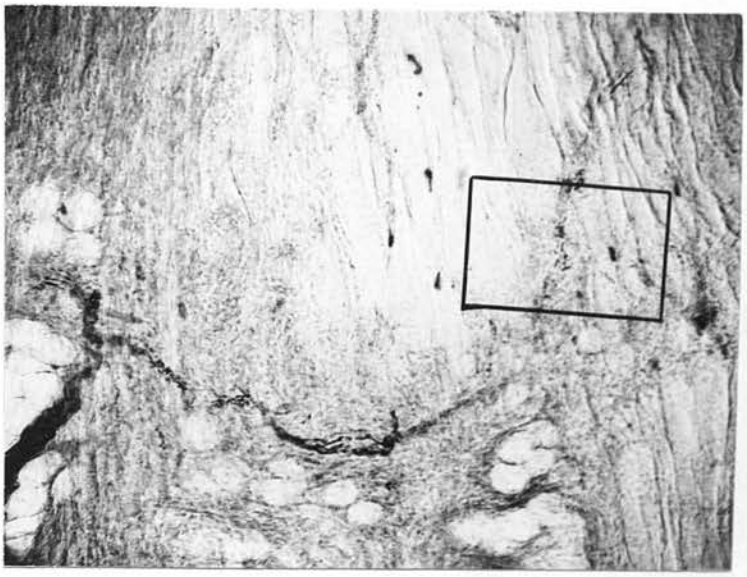

Fig. 61 X 190

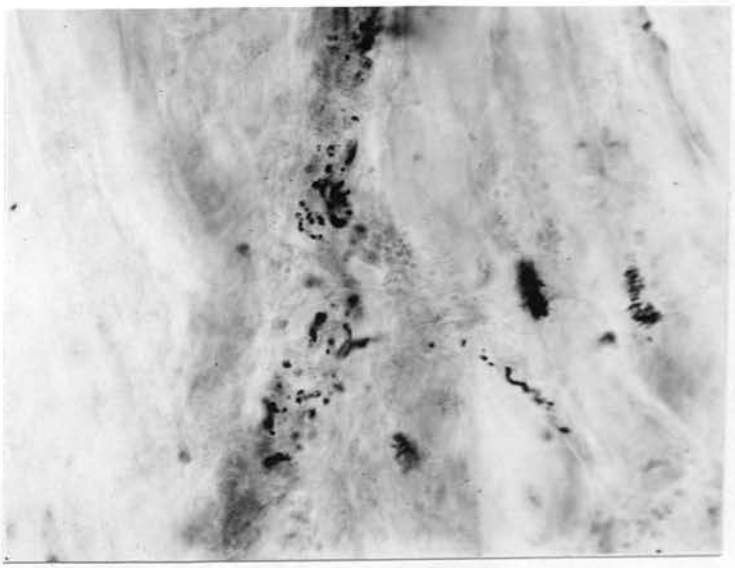

Fig. 62 X 485

Fig. 63 Motor innervation in a type 1V muscle lesion. The axons to the left are starting to break up and the end plates at right lack definite form. Cholinesterase - silver method. Case S-53.

Fig. 64 (a) Motor innervation in a type 1V lesion. Two normal end plates can be seen (above and below) while the third lacks definite form. The axons appear intact. Cholinesterase - silver method. (b) Cross section of the same muscle in which two fibres are undergoing degeneration. H&E.

Fig. 63 X 485

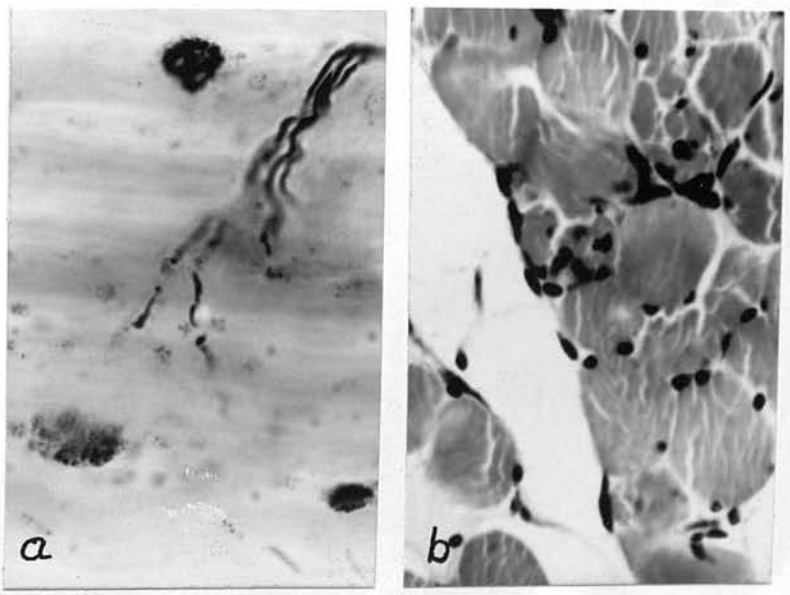

Fig. 64 X 485

Fig. 65 Muscle innervation in a type IV lesion. Three normal end plates lie to the right of the tangled mass of axons. The teliodendria appear as a single black line amid the sole-plate nuclei. Case S-27, Gross-Bielschowsky (bulk) method.

Fig. 66 From the same general region as Fig. 65. A normal axon connects to an end plate which appears contracted (compare to Fig. 65). The muscle fibre innervated appears to be undergoing granular degeneration. Case S-27, Gross-Bielschowsky (bulk) method.

Fig. 65 X 485

Fig. 66 X 485

Fig. 67 Three striated muscle-fibre annulets (left, right and center) from a rectus oculi muscle of a two year old animal of the normal control group. Case C-21, kiton fast red - light green.

Fig. 68 Three striated muscle-fibre annulets, one of which is very complex, from a three year old ewe's rectus oculi muscle. Case C-40, kiton fast red - light green.

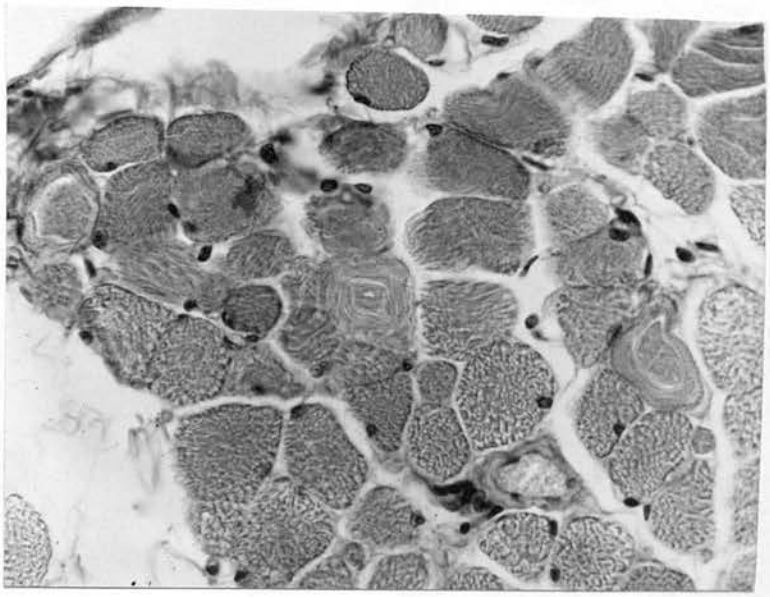

Fig. 67 X 485

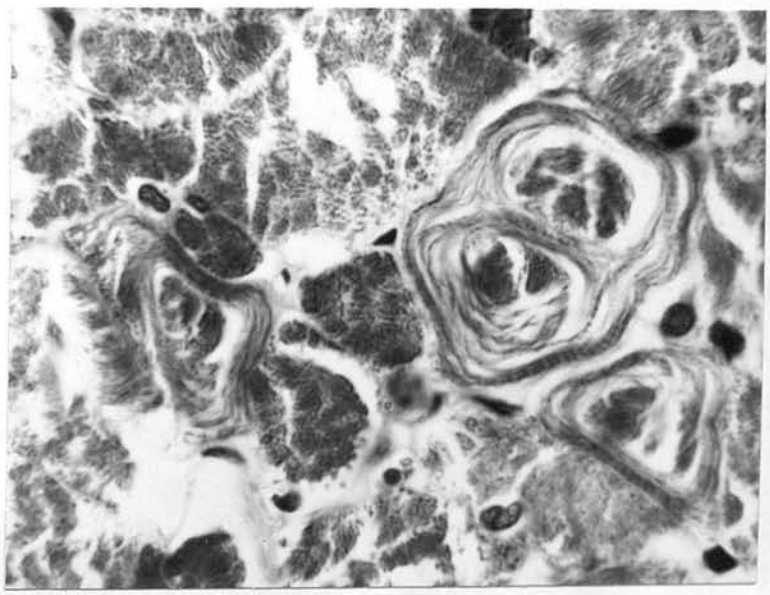

Fig. 68 X 1200

Fig. 69 Extrinsic ocular muscle showing vacuolation of muscle fibres. The two vacuoles in one fibre in the center of the figure appear to be well formed entities. Case C-40, kiton fast red - light green.

Fig. 70 Extrinsic ocular muscle showing vacuolation of an intrafusal muscle fibre of a neuromuscular spindle. Case C-19, kiton fast red - light green.

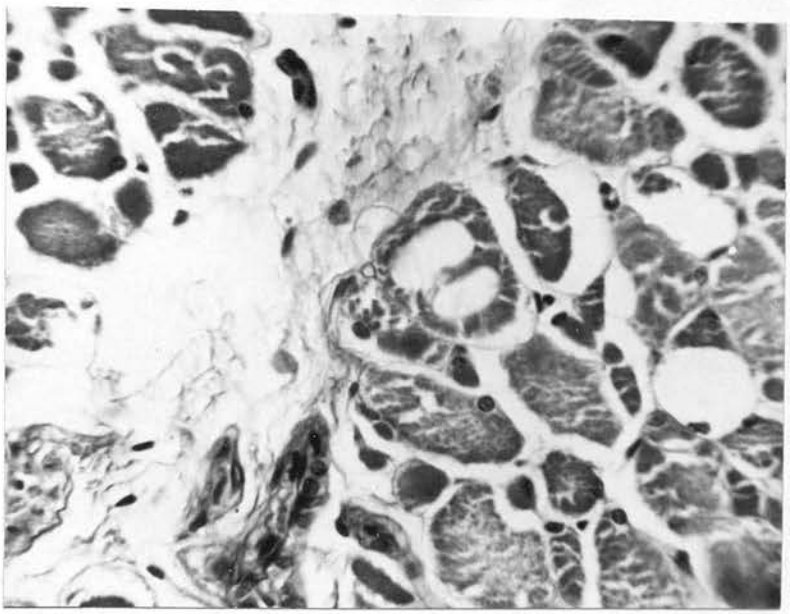

Fig. 69 X 485

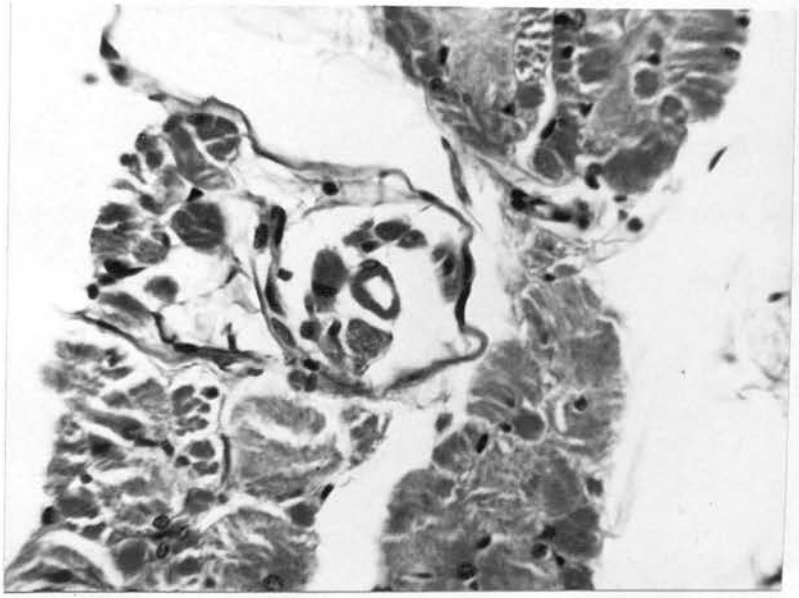

Fig. 70 X 485

Fig. 71 Diagramatic reconstruction from serial sections of a short length of a splenic artery from a six year old ewe. The confluent plaques are shown as irregular shading on the inner surface, black on the cut surface.

Fig. 72 As Fig. 71, from a one year old ewe. Note that one branch artery is free from plaque and that one plaque has no direct connection with a branch artery.

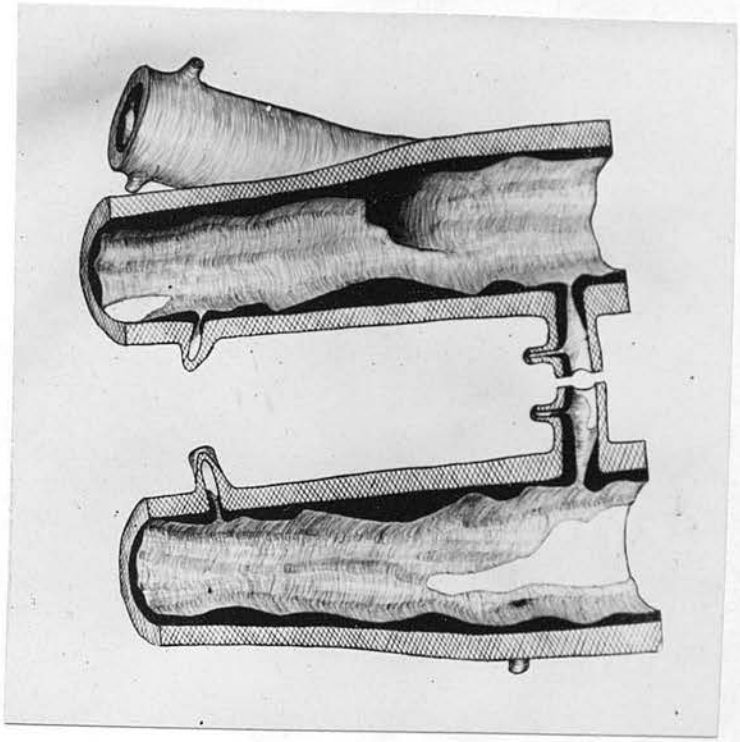

Fig. 71

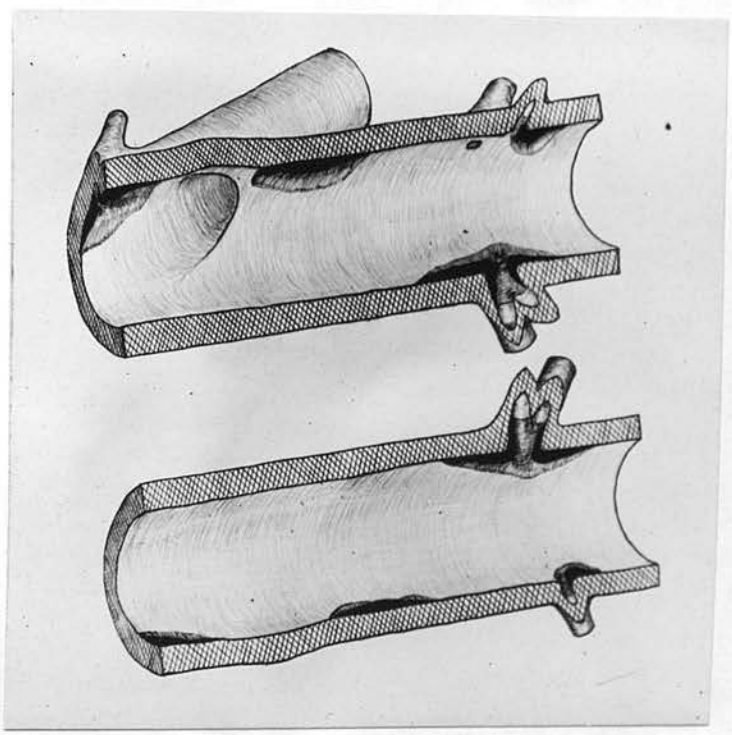

Fig. 72

Fig. 73 A small hepatic artery showing moderate occlusion. Red blood cells are prominent (black) in the media and mark the vasa. Case C-11, aldehyde fuchsin - Lendrum's stain.

Fig. 74 From the same section as Fig. 73 showing relatively greater occlusion and a large number of red blood cells in the adventitia, presumably by-passing the obstructed area.

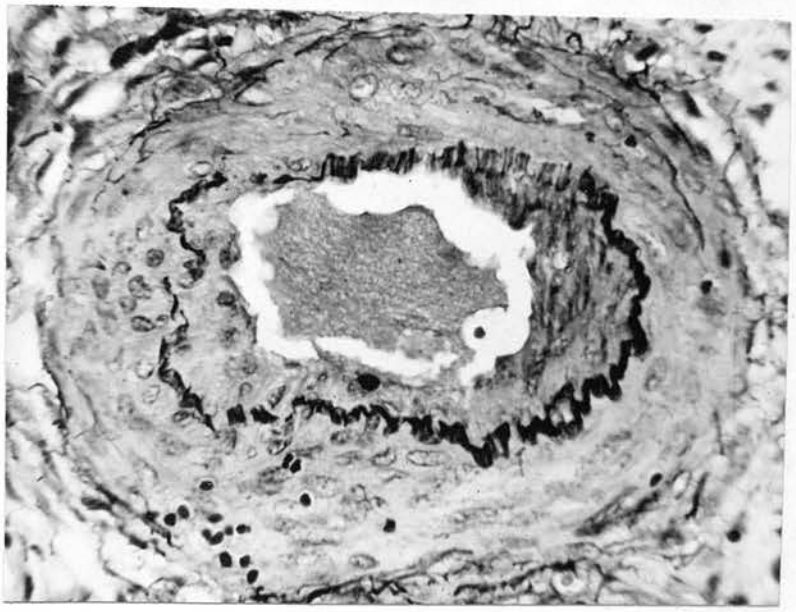

Fig. 73 X 485

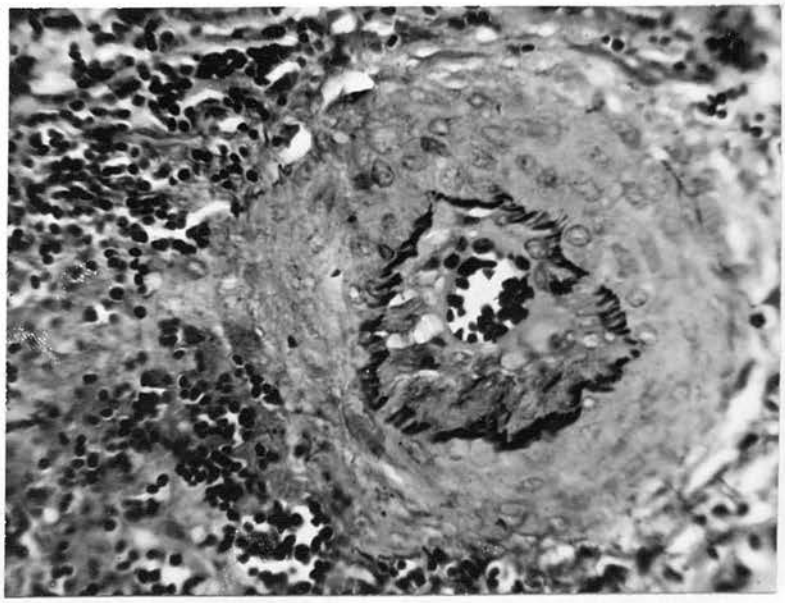

Fig. 74 X 485

Fig. 75 From the same section as Fig. 73 and 74 showing what appears to be the residual elastic membrane of an occluded artery and a mass of red blood cells occupying a new channel, the wall of which contains no elastic membrane.

Fig. 76 Splenic artery from a two year old ewe showing prominent and numerous endothelial cells. Red blood cells and debris are lying on the surface. Case C-11, aldehyde fuchsin - Lendrum's stain.

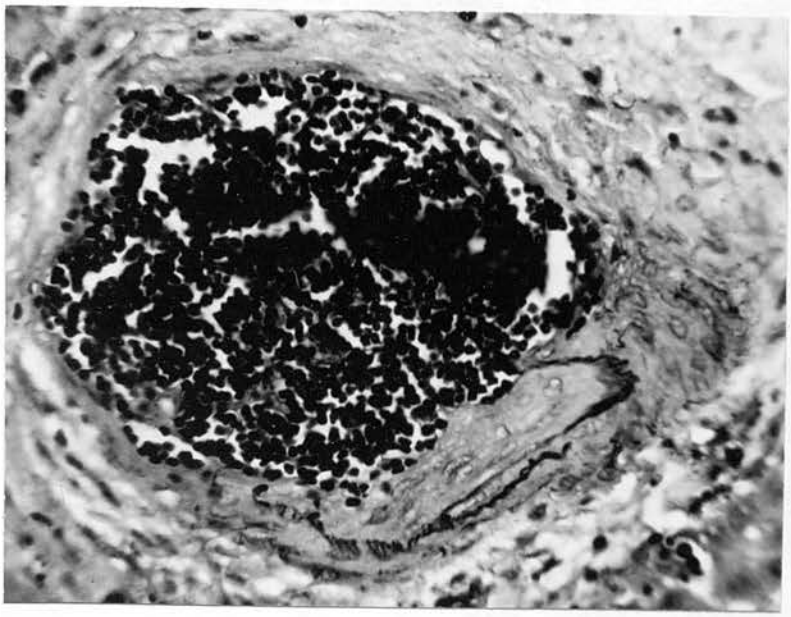

Fig. 75 X 485

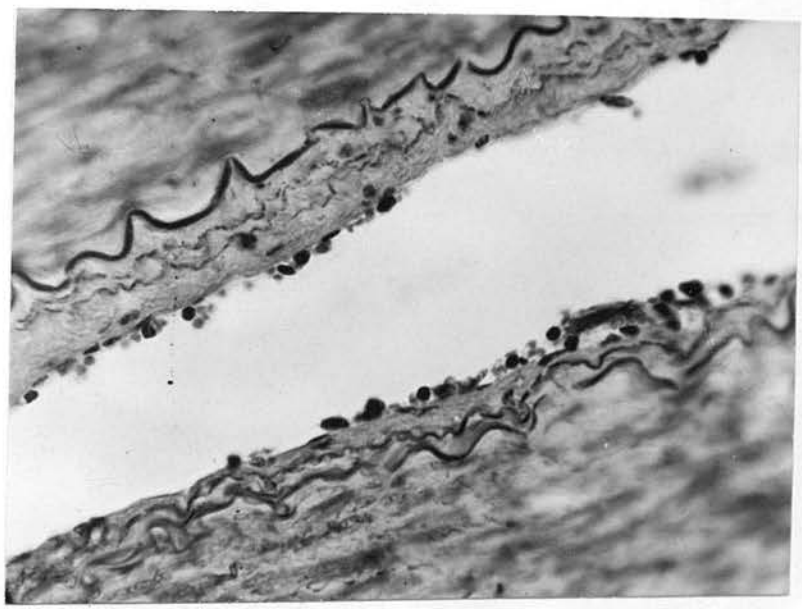

Fig. 76 X 485

Fig. 77 Splenic artery from a six year old ewe showing confluent plaques which are poor in elastic fibrils. The pattern of the medial muscle fibres is distorted adjacent to breaks in the inner elastic membrane and the red-staining muscle fibres can be seen penetrating. Case C-49, aldehyde fuchsin - Lendrum's stain.

Fig. 78 Splenic artery from a three year old ewe showing a laminated plaque, the outer layer of which is rich in elastic fibrils and smooth muscle fibres; the inner layer contains neither. Case C-4, aldehyde fuchsin - Lendrum's stain.

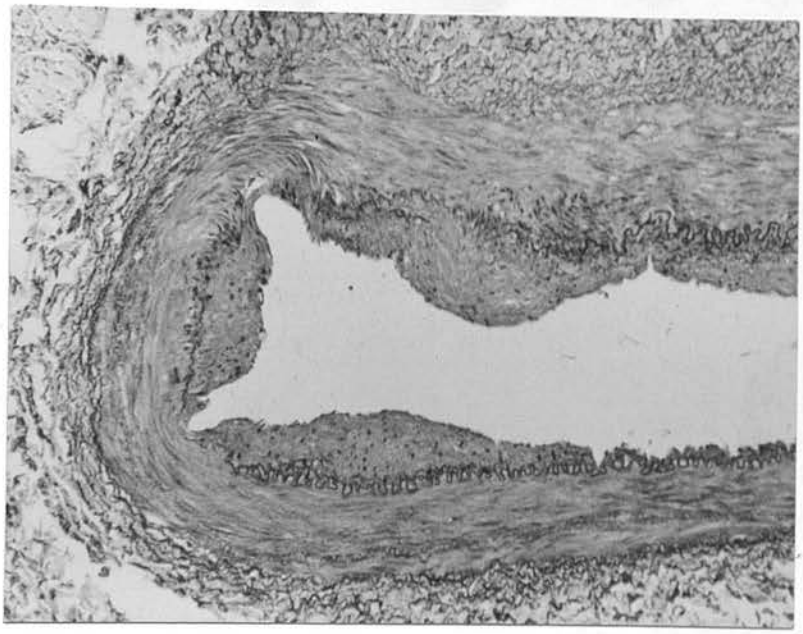

Fig. 77 X 140

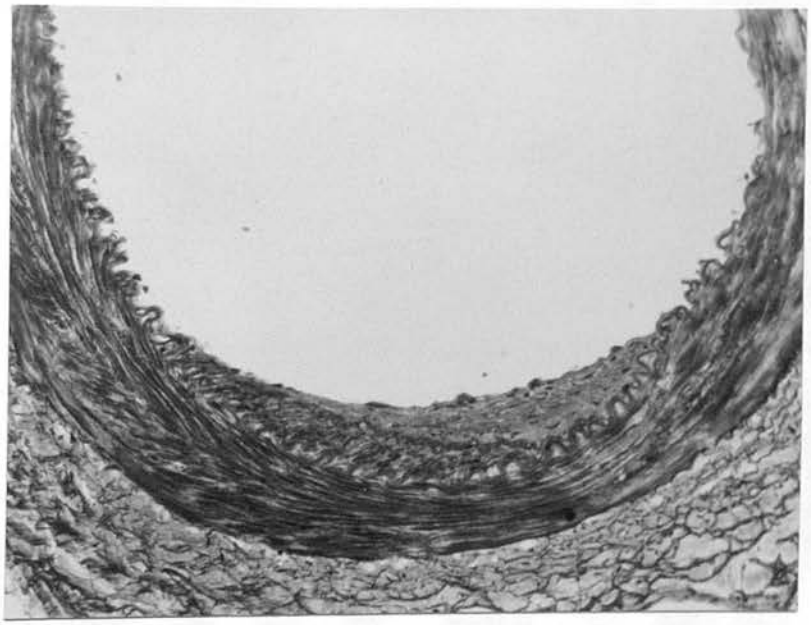

Fig. 78 X 225

Fig. 79 Intramuscular artery from a four year old wether showing medial necrosis and two foci of calcification. Case E-23, von Kossa - van Gieson.

Fig. 80 This plaque on the aortic intima contains fine droplets of sudanophilic material rather diffusely scattered through a thickened area. Case C-64, Sudan IV - haematoxylin.

Fig. 79 X 100

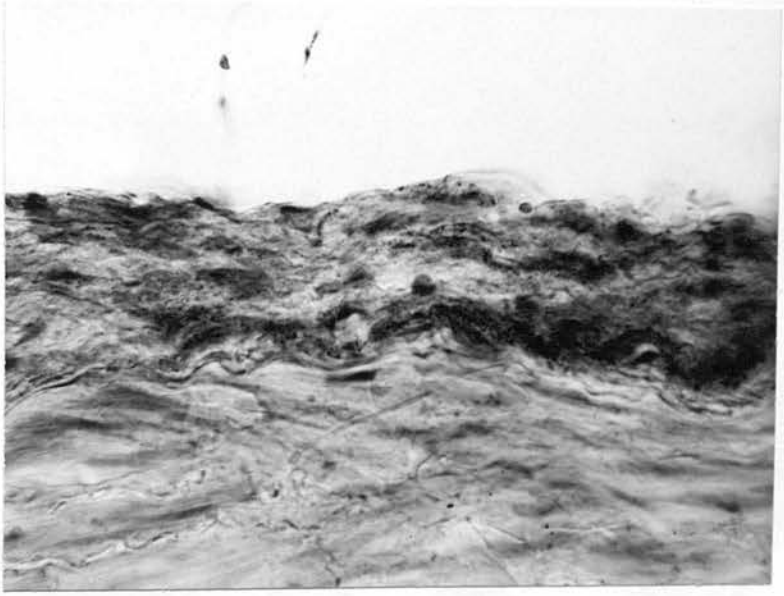

Fig. 80 X 485

Fig. 81 This intramuscular artery from a two year old ewe contains a large organized thrombus. From a gold chloride preparation (toluidine blue). Case C-33.

Fig. 82 The graph illustrates the relative incidence of intimal plaques in arteries of various organs as they were associated with age. The size of the plaques is not taken into consideration here.

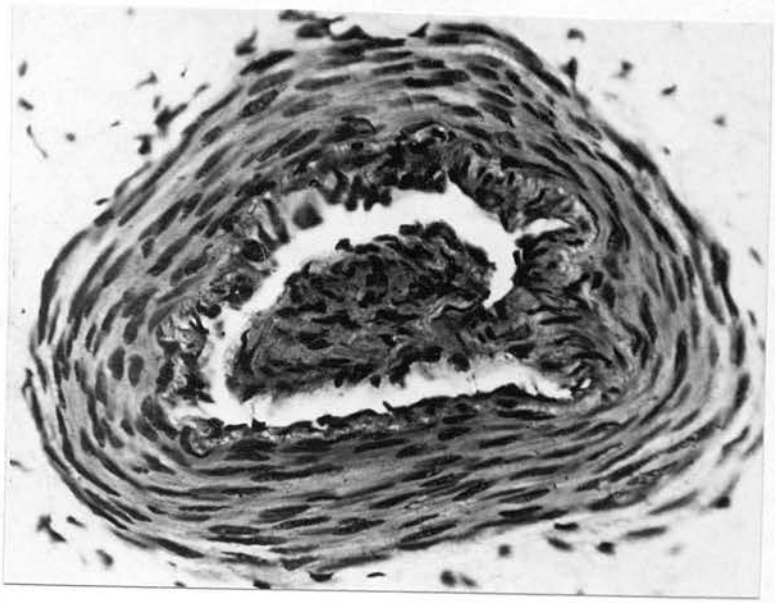

Fig. 81 X 485

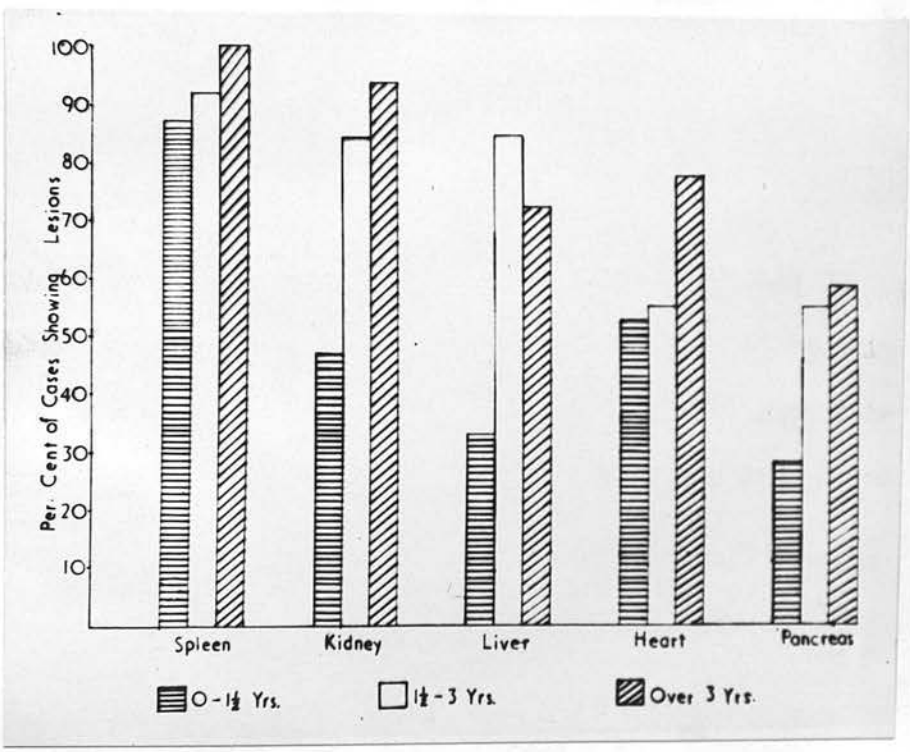

Fig. 82

Fig. 83 Motor nerves in normal guinea pig thigh muscle. The end plates are distributed in clusters, and even in normal muscle show pleomorphism. Cholinesterase - silver preparation.

Fig. 84 Terminal axon and end plate structures in a normal guinea pig thigh muscle. Note the bifurcated terminal axon attached to the elongated end plate in the lower portion of the figure. Cholinesterase - silver preparation.

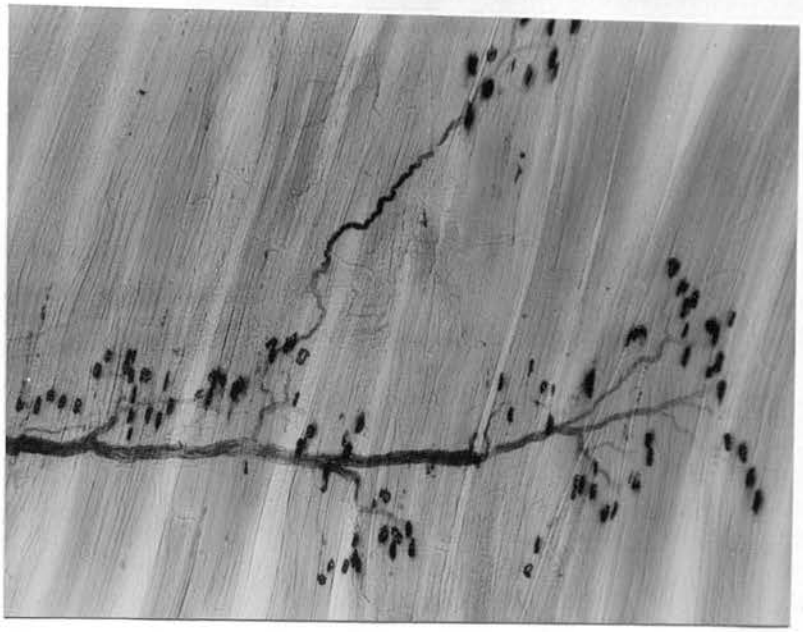

Fig. 83 X 50

Fig. 84 X 225

Fig. 85 Motor endings three days after sciatic neurectomy in the guinea pig. The end plates appear normal but the fine terminal axons (arrows) show evidence of fragmentation. Cholinesterase - silver preparation.

Fig. 86 Motor endings twenty days after denervation. The end plates appear more numerous per unit area and somewhat smaller than normal. The large motor trunk (arrow) is barely discernable but sensory nerves accompanying blood vessels are normal. Cholinesterase - silver method.

Fig. 85 X 190

Fig. 86 X 190

Fig. 87 Nerve bundles twenty days after neurectomy. The myelin sheaths are marked by rows of red fat droplets. Sudan IV - haematoxylin.

Fig. 88 Motor end plates forty days after denervation. The density of the plates is striking (compare to Fig. 85) and no axons are visible. Interstitial fat is evident (above) between fibres. Cholinesterase - silver preparation.

Fig. 87 X 225

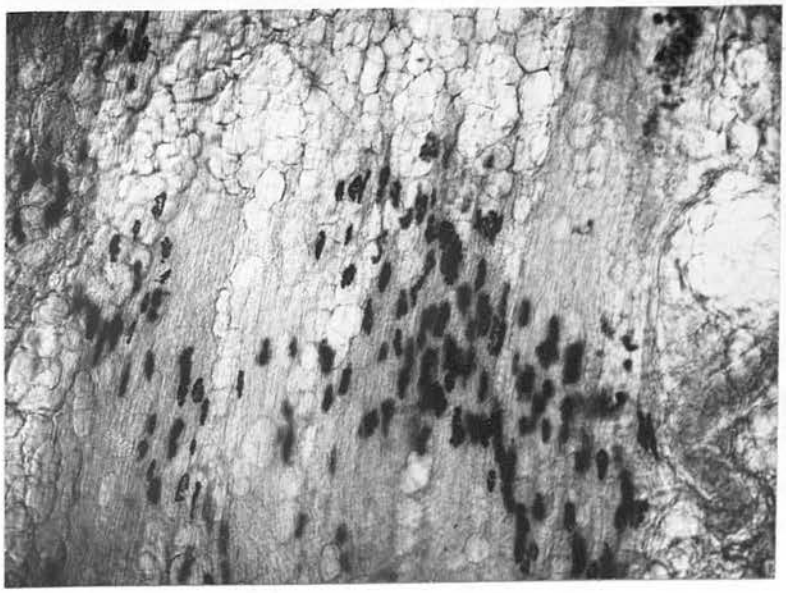

Fig. 88 X 190

Fig. 89 Cross section of guinea pig thigh muscle forty days after denervation. The large fibres represent an area which has had an intact motor innervation and the small one a denerved area. Van Gieson.

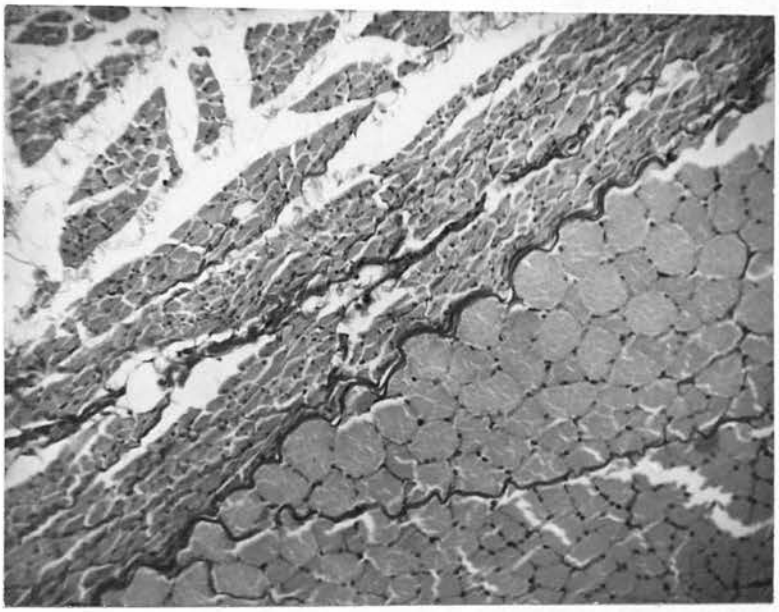

Fig. 89 X 190