

**Staff Meeting Bulletin  
Hospitals of the . . .  
University of Minnesota**

**Giant Cell  
Tumors of Bone**

STAFF MEETING BULLETIN  
HOSPITALS OF THE . . .  
UNIVERSITY OF MINNESOTA

Volume VIII

Thursday, March 11, 1937

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during the school year, October to May, inclusive.

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William A. O'Brien, M.D.

I. LAST WEEK

Date: March 4, 1937.  
Place: Nurses' Hall  
 Recreation Room  
Time: 12:15 to 1:15 P.M.  
Program: Abstract: Roentgenologic  
 Diagnosis of Spinal Cord  
 Lesions.  
 Analysis of University of  
 Minnesota Hospitals Cases.

Case Reports

Movie: The Seeing Eye

Present: 134

Discussion: H. O. Peterson  
 W. T. Peyton  
 J. C. McKinley  
 L. G. Rigler

II. MOVIE

Title: Heart of the Sierras

Released by: R.K.O.

III. ABSTRACTGIANT CELL TUMORS OF BONE

R. W. Koucky

History

1860 - Nelaton: accredited with the  
 first modern description.

1876 - Virchow: contributed to the  
 subject but labeled the  
 tumor as malignant--  
 "myelogene schalige Riesen-  
 zellensarkome."

1891 - Recklinghausen: described  
 generalized osteitis fibro-  
 sa cystica. Association of  
 this with giant cell tumor,  
 however, was not made at  
 this time.

1910 to 1925: Numerous contributors;  
 Silver, Bloodgood, Elmslie,  
 Ewing, Konjetzny, Lewis, etc.:  
 established giant cell tumor  
 as a clinical entity and as  
 a benign tumor.

1927 - Kolodny: Report of the Bone  
 Tumor Registry studies.

1929 - Geschickter and Copeland:  
 400 cases studied, with  
 theories of genesis. Giant  
 cell tumor and bone cysts  
 placed in same genetic group.

1930 - Same authors: Review of 400  
 cases with emphasis on  
 benign nature.

1930 to 1936: Numerous studies on  
 hyperparathyroidism. Proof  
 developed that this condition  
 produces tumor-like areas  
 similar to giant cell tumors.

Questions today

Are giant cell tumors a separate  
 entity?

Are they neoplasms or a manifestation  
 of a systemic change?

Pathology and X-ray

So much of the pathology of all bone  
 lesions has been developed through x-ray  
 studies that descriptions of the tissue  
 changes must include the roentgenologi-  
 cal appearance.

The tumors are made up of a fibrous  
 tissue stroma within which are embedded  
 giant cells. The stroma varies con-  
 siderably in appearance. It may be  
 dense, like scar or keloid tissue, or

it may be cellular with numerous nuclei of plump dimensions such as are present in rapidly growing tissue. Stimulation by infection, hemorrhage, fracture or radiation commonly increases this cellular or sarcomatous appearance. Degeneration with the formation of cysts is common.

The giant cells are large, frequently containing 50 to 100 nuclei. In contrast to the phagocytic or foreign body type of giant cell, these have the nuclei arranged loosely in the center rather than ring-like about the periphery. The cells are numerous within the tumor, 20 to 30 per low power field. However, they are most abundant in tumors with a cellular stroma and they may be much reduced in numbers in the fibrous types.

The tumors vary greatly in size; according to the size of the bone involved and also according to the rapidity of development and their duration.

The location, in long bones, is invariably in the epiphysis. In the small bones, the relation to the ossification centers is lost because of the small size of the entire bone. In the skull, the tumors are limited to those areas preformed in cartilage. This relationship to secondary ossification centers within bones arising in cartilage is striking (constant?).

The earliest tumors studied apparently begin in the cancellous bone just under the cortex on one side of the epiphysis. As growth continues, the cancellous portions are destroyed first and bulging of the cortex appears only after the central part has been extensively filled.

The expansion of the bone is not a simple distention but active absorption of bone on the inner surface with deposition under the periosteum. When the rate of destruction is greater than that of construction, perforation through the cortex takes place. While perforation generally indicates rapidity of growth, it is not an indication of malignancy.

The tumor, in its growth, tends to be multilobular. Remnants of bone remain between these lobulations and give to the

x-ray or dried specimen a polycystic or foam-like appearance. Remnants of bone persist within the masses of tumor and these are visualized as coarse and usually irregular striations in the x-ray. Especially in fibrous types, some of these bone spicules are regenerated bone. This is probably a healing reaction rather than tumorous bone formation such as is present in the sarcomata.

So-called "hybrid" types of giant cell tumors are not uncommon. Exaggerated cystic changes with maturation of the fibroblastic stroma and atrophy of the giant cells may take place in varying degrees. It is said that bone cysts characterized by a prominent cystic space, dense fibrous walls containing few or no giant cells represent the extreme (healed) form of this same process. On the other hand, especially under the stimulation of infection, trauma, fracture or similar agents, the fibrous stroma may manifest active growth in the same manner as in organizing scars or granulations. Microscopic examination during such stages may give rise to the erroneous diagnosis of malignancy.

Osteogenic sarcomata may contain giant cells. The fundamental cell of bone sarcomata has the potentiality of developing any of the cells associated with bone and mere presence of giant cells within such tumors does not classify them with the true giant cell tumors. Errors in pathological diagnosis arise in such cases and such errors have caused confusion regarding the nature of giant cell tumors.

From the study of these hybrid types, 5 groupings can be made:

1. Bone cysts (said to be healed giant cell tumors).
2. Spindle cell or fibrous variants (said to be healing stages)
3. Typical giant cell tumors.
4. Cellular variants. (The term "malignant variant" is often used but since the increased

growth is a response to external stimuli (?) and has only the appearance of malignancy it seems that such a term is confusing).

5. Osteogenic sarcoma containing giant cells. (Not related to any of the above).

### Metastasis

Metastasis from giant cell tumors are reported.

Local recurrence is relatively common; 31 cases in 222 tumors. The opinions regarding the factors governing recurrence will be touched on under "treatment." As in other tumors, such local recurrences, however, do not indicate that the tumor is malignant.

Study of the so-called metastasis from giant cell tumors (8 instances in "well over 500 cases") has lead to certain definite conclusions. "First, in no cases has a nodule of typical giant cell tumor ever been found in the lung, for whenever these metastatic nodules have been examined, they have shown the histology of osteogenic sarcoma. Second, in no case has the association of an originally benign and typical giant cell tumor in the bone with a secondary metastatic osteogenic sarcoma in the lung been proved." (Geschickter and Copeland, 1930). In a considerable number of these so-called metastasizing giant cell tumors, careful study of the primary tumor has shown it to be an osteogenic sarcoma containing giant cells. In regard to the remainder, "the only plausible deduction is that in a few isolated instances an apparently benign lesion of bone, when subjected to unsuccessful treatment and to trauma, may by its failure to heal, provide a fertile site for the subsequent development of osteogenic sarcoma." (Geschickter and Copeland, 1930). Writers subsequent to this time generally hold to these same views.

### Development of Giant Cell Tumors

Geschickter and Copeland (1929 and 1930) believed that giant cell tumors were

neoplastic perversions of normal bone formation initiated by trauma. In the simplest terms, the tumor represented an overgrowth of osteoclasts producing local destruction of bone. Under the influence of good blood supply and the restraining effect of thick bone, both of which conditions are present in the shaft of the bone, these tumors tend to heal with the production of bone cysts. In epiphysis where the blood supply is not as great (less anastomotic) and where the thin bony structures offer no restraint, the process once initiated does not tend to heal but remains in its original giant cell form. These authors even at this time (1930) observed atypical multiple tumors and stated "some complicating factor is usually present" and in foot note (page 224) refer to a reported case of hyperparathyroidism.

### Hyperparathyroidism

Since 1930, as knowledge of parathyroid overactivity became general, there was a rapid accumulation of information regarding the pathology of the bone lesions in this illness.

The multiple bone cysts and solid masses within bone defects found in hyperparathyroidism were shown to have a histology identical to the cysts and giant cell tumors previously described as separate disease entities. (See previous staff meeting bulletin).

The natural reaction was to conclude that all giant cell tumors and bone cysts are a manifestation of or result of parathyroid overactivity. Such a tendency is well shown in the following table compiled from Morse (1933).

<u>Disease of Bone Rarefaction</u>	<u>Explanation</u>
Osteogenesis Imperfecta Fragilitas ossium	Mesoblastic defects; Hereditary and familial.
Rickets Osteomalacia	Defect of lime absorption or fixation. Insufficient supply or unusual demand for calcium; vitamin deficiency.
Bone fragilities in other (not para- thyroid) endocrine disturbances.	Hyperthyroidism, diabetes, basophil adenoma of pituitary, adrenal tumors.
Renal rickets	Nephritis with high phosphorus levels and compensatory hyperparathyroid activity.
Osteitis fibrosa Osteitis deformans (Paget's disease) Leontiasis ossium Giant cell tumors Ankylosing polyarthritis (Oppel) Multiple myeloma	"At the present time, we feel are caused by either a primary parathy- roidism or at least an overactivity of the parathyroid gland brought on by some special cause."
Miscellaneous bone defects	Schuller-Christian's, Gaucher's, Niemann- Pick's, Hodgkin's disease, carcinoma- tosis, etc.

The reaction to such a sweeping deduction has already appeared. Pierce-- "I do not feel that the evidence is sufficient at the present time to place giant cell tumor in the group of primary parathyroid osteomalacias"; Ballin-- "There are some giant cell tumors that do not belong to the parathyroid group." The outstanding features indicating that a distinction is still warranted are:

<u>Hyperparathyroidism</u>	<u>Giant Cell Tumor</u>
1. Diffuse skeletal disease.	1. Local destruction.
2. Multiple tumors.	2. Isolated, single tumors.
3. Present in shaft.	3. Limited to epiphyses.
4. Severe disturbances of blood calcium and phosphorus.	4. Blood calcium and phosphorus normal.
5. Cured (?) by parathyroid surgery.	5. Cured by local therapy.

Nevertheless, much good has been accomplished in that atypical giant cell tumors, especially about the jaws, are now being investigated as possible parathyroid disturbances.

#### Clinical features

Age:  
Years (2 series combined)

Clinical featuresAge: Years (2 series combined)

	<u>Cases</u>	
Under 10	16	)
10 - 20	80	)-- 83%
20 - 30	103	)
30 - 40	54	)
40 - 50	36	
50 - 60	11	
Over 60	5	

These figures are approximate because they are taken from graphs. Simmon's series of 116 cases show 84% under 40 years of age.

Sex:

Kolodny - female to male ratio, 6 to 5.

Simmons - 54.5% female, 45.5% male.

Bone involved:

<u>Simmons:</u>	<u>Cases</u>	
Femur	36	)
Tibia	24	) - (54%
Radius	12	)
Jaws	10	
Humerus	8	
Ulna	5	
Vertebra	4	
Fibula	3	
Ribs	2	
Others	12	

<u>Kolodny</u>	<u>%</u>
Lower extremity	56
Upper extremity	23
Axial and girdle bones	21

Combined series of Geschickter and Copeland and Christensen. (Note that tumors of the skull are not included).

Lower femur	123
Upper tibia	92
Lower radius	62
Upper humerus	27
Lower ulna	22
Upper femur	16
Upper fibula	15
Lower tibia	14
Rib	9
Scapula	8
Upper ulna	7
Lower fibula	4
Upper radius	3

Trauma

Trauma as an etiological factor is considered to be important. Geschickter and Copeland believe that this apparently is the exciting factor. Its presence was noted as follows:

Simmons	64.3%
Kolodny	"very frequently"
Geschickter and Copeland	42.0% - the "initial event" producing the tumor.
Murphy	"undoubtedly the first step."

Duration of Symptoms

Geschickter and Copeland - 226 cases - average duration prior to observation - 14 months.

Symptoms

Pain, tumor, fracture is the sequence of symptoms almost uniformly given by various authors.

The pain is not severe. No special features concerning it are noted. The development of swelling varies with the speed with which the tumor grows. Usually it develops slowly. In 14% none of these symptoms had been observed and fracture was the first sign. A considerable number of tumors are discovered in roentgenological studies for other purposes.

Constitutional symptoms are absent.



Radiation preferably by deep therapy rather than by radium implantation is today considered as the first line of treatment. Only when this is not available or has failed are surgical methods to be carried out. It is emphasized that the first reaction to the treatment is one of increased growth and tenderness (see pathology). After 3 to 4 months, the tumor begins to recede.

### Results

Simmons outlines the results in his series as follows:

	<u>Cures %</u>
Amputation	100
Resection or excision	100
Curettage	63
Curettage and radiation	72
Radiation alone	75
Coley's toxin (7 cases)	42

Geschickter and Copeland's series:

	<u>Cases</u>	<u>Recurrences</u>
Amputation	30	0
Resection and excision	34	2 (both excisions)
Curettement	105	31 (29.5%)
Radiation	5	No estimate of results.

### Recurrences

In Geschickter and Copeland's group, there were 31 recurrences in 222 cases. All of these followed curettage, totaling 29.5% of this group. Microscopic study of these recurrent cases showed no histological characters to account for the recurrence. "The cause is to be ascribed either to poor selection in choosing the type of treatment for the individual case or to an incomplete operation." However, the average age of patients with recurrences was definitely higher than that for the general group - 42% over 30 years of age. Of especial interest is that while the upper end of the tibia produced 8% recurrences, and the lower femur 39%, the lower end of the radius showed 50% recurrences. "Obviously cur-

ettement in a patient over 35 years of age with a giant cell tumor in the lower end of the radius - invites recurrences."

Sixteen of these 31 cases of recurrence were cured by second or third curettage.

Infection is a serious complication. In 105 cases, there were 7 instances which resulted in 3 amputations for the infection. The influence of this factor in inducing malignancy has been mentioned above.

### Summary

1. From 1860 to 1930, giant cell tumors were identified as a distinct disease entity. Since 1930 with the advent of information regarding hyperparathyroidism, the status of giant cell tumor has become very much confused.

2. The pathology of the isolated giant cell tumors apparently differs in no way from that found in the multiple tumor like growths occupying the rarefied areas in the bone in hyperparathyroidism.

3. Certain authors have made the statement that giant cell tumors merely represent another manifestation of parathyroid disease.

4. This idea, however, is not accepted by and in all possibility the isolated epiphyseal giant cell tumor will remain as distinct tumor entity.

5. Considerable evidence is present to show that giant cell tumors vary greatly in their rate of growth and that some of the tumors recede. Theoretically when the tumor is healed, cysts result and on the other hand under stimulation by infection or trauma or inadequate treatment, the rate of growth may be increased. During the period of increased growth, the tumor may have the histological appearance like that of a malignant tumor of bone.

6. Local recurrences after removal are not uncommon but such recurrences do not

indicate a malignant nature.

7. In rare instances, metastases with giant cell tumors have been observed. However, studies of these cases show that the metastatic nodules have the histology of osteogenic sarcoma.

8. Two explanations are available: (1) that the original tumor was actually an osteogenic sarcoma in which an erroneous diagnosis of giant cell tumor was made (this is most likely); (2) that a previously benign giant cell tumor subjected to stimulation by trauma or inadequate treatment subsequently becomes the site of a true osteogenic sarcoma.

9. About 85% of the tumors develop before the age of 40, and there is a slightly greater incidence in women. The lower end of the femur, the upper end of the tibia, the lower end of the radius, the upper end of the humerus are the areas most frequently involved. Tumors in the jaw are not uncommon.

10. Trauma is considered to be an important etiological factor. The earliest lesions which have been observed have been present at the edge of the bone in the epiphysis in such a position that trauma could easily have been the exciting factor in their development.

11. In two hundred and twenty-six cases the average duration prior to observation was 14 months. Pain, tumor and fracture in that order are the most common symptoms. In 14% fracture was the first sign observed. Constitutional symptoms are usually absent.

12. The diagnosis in some cases is not easy. The x-ray picture may be confusing. The differentiation between giant cell tumor and osteogenic sarcoma may be difficult. The histological criteria are not infallible. Trauma, infection and radiation may stimulate the tumor to the extent that a diagnosis of malignancy may be made. On the other hand, true osteogenic sarcoma may be considered to be giant cell tumor because of the presence of giant cells.

13. In the older series, treatment by amputation was quite common. While the

method is not so commonly used today, there are still occasions when it is the treatment of choice.

14. Resection is the favorite method in areas where the loss of the bone is of no significance or where the loss of the bone may be repaired by plastic procedures such as bone graft. Curettage when it is completely done is a reliable method of treatment but large tumors, especially where the cortex has been extensively destroyed, are not suitable for this method; resection is preferable. Radiation, preferably by deep therapy, is apparently the method of choice today. About 75% of the cases are cured by this method; the remainder must be treated by surgical methods.

15. Amputation and resection give practically 100% cures; curettage alone about 63%; radiation alone, 75%; curettage and radiation, about an equal percentage.

16. In one series of cases, 29% recurred after curettement. The recurrence is said to be due to incomplete removal.

17. Older individuals apparently have a higher rate of recurrence and the recurrence rate varies with the site; 8% in the tibia; 39% in the femur; 50% in radius.

18. Infection is a serious complication, and is an important consideration in the choice of treatment, for example, radium implantation versus deep x-ray therapy.

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#### IV. CASE REPORT

, age 27.  
Admitted 3-8-36;  
Discharged 3-25-36.  
Readmitted 2-25-37;  
still in hospital.

#### Onset of pain

1934 - Observed occasional drawing pains in region of left knee.

#### Swelling

1935 - Observed swelling of left knee; this gradually increased; pain

became more marked.

10- -36 - Consulted a physician. The appearance of the knee at this time suggested arthritis. Hot packs and rest were advocated. Condition grew worse.

#### Out-patient Department

12-19-35 - Admitted to Dispensary. Left knee swollen, apparently warmer than the right; skin reddened; complete flexion not possible because of pain. Clinical impression: arthritis.

12-19-35 - X-ray: Extensive tumor in distal end of left femur extending into epiphysis with marked trabeculation through it; has expanded the cortex and penetrated to some extent into the soft tissue. Conclusion: Probable extensive giant cell tumor at distal end of femur with possible beginning malignant degeneration. X-ray of chest: negative.

#### Deep X-ray Treatment

1-4-36 - 120% S.E.D. given to medial and lateral fields of lower end of left thigh in 6 treatments over 12 days.

3-2-36 - X-ray: Tumor has increased its destruction and has extended since the last examination, suggesting progression; no evidence of healing. X-ray of lungs: negative.

#### Admitted to Hospital

3-8-36 - Physical examination - essentially as above. Other laboratory work and remainder of physical examination, nonessential.

#### Operation

3-13-36 - There has been no response to the x-ray treatment. Operation: The distal end of the femur was removed; this incision extended 4 or 5 cm. above the upper limits of the tumor. The bone was dissected out from its muscle and ligamentous attachments. The crest of the tibia was then removed and used as a graft fitted between the head of the tibia and the lower end of the femur.

Pathological examination

Gross: Tumor of femur. Specimen consists of lower 18 cm. of femur, shelled out from adjacent soft tissue. Tumor is 12 x 7.5 x 8 cm., occupying the distal end of the segment. The tumor extends to the cartilage but shows no perforation through it. At the upper end, the tumor is sharply demarcated through the adjacent medulla. On one side, the tumor has penetrated through the cortex of the bone over a distance of about 6 cm. and has projected outward for 1.5 cm. On the opposite side, the periosteum is lifted away from the rest of the adjacent cortex and no bone is palpable in this area but the tumor itself is not projecting to any appreciable extent. On cross section, the tumor is composed of solid portions and large multilocular cyst-like spaces. The largest of these cysts are about 3 cm. in diameter. Some of the cysts are filled with blood and others are filled with clear fluid. The tumor itself is of uniform grayish-white appearance, and coarse spicules of bone can be palpated within the tumor substance.

Microscopic: Sections from various parts of the tumor show approximately the same structure. There persist small spicules of bone which are being absorbed and none of which show any evidence of new growth. The bulk of the tumor is a uniform fibrous tissue stroma. The nuclei vary considerably in appearance; some are very definitely atrophic and have the appearance of nuclei within heavy scar tissue; in other parts, the nuclei are plump and suggest recent or active division. Isolated within this fibrous tissue stroma are very occasional giant cells; the giant cells are small, average about 10 nuclei within their center.

Conclusion: Giant cell tumor. This tumor is atypical in that it has such a large amount of fibrous tissue stroma which in some areas appears to be actively growing and because of the poverty of giant cells. However, this appearance is probably due to the radiation which the tumor has received. The fibroblastic proliferation probably represents a healing or repair process

within the mass.

Discharged

3-25-36 - Patient discharged, in a cast; advised to return for observation.

Readmitted

6-15-36 - Returned for check-up. X-ray showed that the bone graft had increased its diameter but is still too small to allow any weight bearing. Discharged on 6-18-36.

Readmitted

2-25-37 - Patient has gained weight. There had been no disturbing symptoms. In September, part of the cast had been removed to allow massage and manipulation of the foot, intended to stimulate the formation of callus. An x-ray taken during this interval showed no change in the condition of the bone.

Physical examination on this admission was negative throughout.

X-ray Examination of the femur showed considerable atrophy of the tibia and fibula. The bone graft appeared to be firmly united in its upper end, but there was no definite evidence of union in the lower end. There was very slight angulation of the alignment of the graft with little proliferation of bone tissue from the distal end of the femur.

Opinion

3-4-37 - As soon as the skin which has been under plaster for a long period of time is in good condition, exploration of the operative site is to be carried out and either another bone graft laid side by side with the old one or else removal of the cartilage from the tibia and the placing of bone chips about the insertion of the graft into the tibia will be carried out.

## V. GOSSIP

The Minnesota State Department of Health, organized in 1872, had for its first year's work an appropriation of \$500.00; Massachusetts was first (1879) to be formed; California second (1870), and Minnesota third. Dr. Albert J. Chesley, our present health officer, was a medical student at the University of Minnesota at the outbreak of the Spanish-American War in which he enlisted as a private. Dean Harold S. Diehl's interest in public health work dates back to the World War, when as a student he went to Poland with the Red Cross with Dr. Chesley.....Thomas J. Kinsella, formerly located at the Glen Lake Sanatorium in a full-time capacity, is now in private practice, limiting his work to thoracic and abdominal surgery and the surgery of the tuberculous.....In a letter of appreciation from one of our former patients, she deeply regrets her inability to thank each one of her physicians in person. For 3 days about a month before she became sick, she had a vision that something was going to happen to her but it would be all right. She feels that we are part of the prophecy.....Another one of our patients, long a sufferer from bowlegs, thinks she now has the cure as one of her legs is straight. What the great mystery is, is a secret, and it is for sale.....The 7th Annual Institute for Public Health Nursing sponsored by the Minnesota State Department of Health will be held March 22 to 24, 1937, in the Medical Science Building. Among others, Dr. Haven Emerson, Institute of Public Health, Columbia University, will speak. During the first part of the meeting, there will be a summary of this year's discussion groups on "Mental Hygiene." Anyone interested is cordially invited to attend.....Dr. Lewis G. Jacobs, formerly a member of our staff in the department of irradiation therapy, is now at the University of Wisconsin in the roentgenologic department. Dr. Jacobs came to Minnesota from California, and left many friends who predict much success for him.....The Rudolph Kouckys and the Karl Andersons are anxiously awaiting the spin of the wheel of fortune--Will it be a boy or will it be a girl???......The Women's Field Army of the American Society for the Control of Cancer in cooperation with the Minnesota State Medical Associa-

tion is putting on a drive for members and funds the week preceding Easter. It is a dollar to join to support an educational campaign against cancer in Minnesota. In the present drive, emphasis is being placed on four points: (1) See your physician when you have a sore which does not heal; (2) a lump which appears or an old lump which starts to grow; (3) any discharge of blood or other material from the interior of the body; (4) any change in digestive or elimination habit. All the other warnings are now listed under the head of precancerous changes which include moles. The publicity in national magazines increases. An especially good article is to be found in the current issue of the American Legion publication by Dr. Greenough of Harvard. ....There will be a silver tea by the auxiliary of the hospital on Sunday afternoon, March 14th, 5 to 6 P.M., in the lounge of the Nurses' Hall. Everyone is cordially invited to balance a cup and drop a coin.....The Minnesota State Hospital Association is sponsoring the short course for small hospital administrators at the Center for Continuation Study on March 18, 19 and 20th, 1937. Professor Raymond Michael Amberg is very busy organizing his faculty. Nearly 60 reservations have been made by Minnesota hospital executives.....There will be the usual spring recess of the Staff Meetings on Thursday, March 25th. They will be resumed with the opening of school in the spring quarter the following week.....More people were killed and injured in accidents in 1936 than during any previous year. For the first time in 8 years, the injuries received at home were responsible for more deaths than injuries received on the highways. In 1935, traffic caused 37,000 deaths; occupations, 16,500; home, 31,500; miscellaneous, 18,000; total - 100,000. In 1936, traffic caused 38,500 deaths; occupations, 18,000; home 39,000; miscellaneous, 19,000; total - 111,000. In addition to the deaths in 1936 there were 400,000 permanently disabled and 10,300,000 temporarily disabled. Safety authorities point out that we must not concentrate too much on the drunken drivers as the careless driver is responsible for more deaths.....

Adios.