

1947

Male climacteric

Robert Ellsworth Fitch
University of Nebraska Medical Center

This manuscript is historical in nature and may not reflect current medical research and practice. Search [PubMed](#) for current research.

Follow this and additional works at: <https://digitalcommons.unmc.edu/mdtheses>

Recommended Citation

Fitch, Robert Ellsworth, "Male climacteric" (1947). *MD Theses*. 1453.
<https://digitalcommons.unmc.edu/mdtheses/1453>

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.

THE MALE CLIMACTERIC

Robert E Fitch

SENIOR THESIS

PRESENTED

TO

COLLEGE OF MEDICINE

UNIVERSITY OF NEBRASKA

OMAHA

1947

INTRODUCTION

This paper is to be written on a very controversial question and a definitely undecided question. The discussion presented here, obviously, will not be the final answer but will attempt to present the work and theories of various investigators and this writer's conclusions drawn from those ideas.

This paper will discuss the male climacteric from the following points of view;

1. Is there a male climacteric?
2. If there is a male climacteric, what causes it?
3. What are the signs and symptoms of the climacteric in the male?
4. Diagnosis of the condition.
5. Treatment of the male climacteric.

IS THERE A MALE CLIMACTERIC

The greatest controversy about the subject of the male climacteric is simple: Is there such an entity? Numerous investigators and people who have studied carefully the formers' work all have presented their conclusions. There are so many claims and counter claims, each with considerable definity, that it is difficult to present a well organized discussion. Consequently this writer has chosen to present the belief of each man and the arguments in favor of each belief and to follow this with my own conclusions after reviewing the literature.

It also was difficult to decide whether to discuss separately or jointly the two questions:

- (1) Is there a male climacteric?
- (2) If there is a male climacteric, what is it and what causes it?

Because of a fear that the point of each discussion might become confused or lost in a joint discussion, it was decided to discuss them separately. There will of necessity be some unavoidable overlapping and repetition.

Since Werner has done the most extensive work on this syndrome and is the most outspoken man in contending

that there definitely is such an entity, it is fitting that his theories and arguments be given first. Since 1939 Werner has written several articles concerning his observations of a group of climacteric patients which has grown to a total of 273 at present. His first paper (23)(24) in 1939 covered his investigation of 37 patients. At that time he wrote that in spite of the fact that the male climacteric had been a mirth producing diagnosis for years he felt it reasonable to believe that many if not all men pass through a period like the female menopause, lacking only the outward sign of the cessation of menstruation; that they had the same decline of their sex gland function which resulted in an endocrine imbalance which in turn caused an upset autonomic system just as occurred in the female of the species. On the other hand he admits that the change occurs later in man, between 48 and 52 years of age, and usually causes less disturbance. Because of this and the fact that there is a disbelief and ignorance of the existence of a climacteric in man, it is often misdiagnosed, overlooked or ignored. Just as all women don't lose their libido and potency at menopause, neither do all men, but this doesn't necessarily militate against the evidence that many men in their 6th decade begin a decline of potency and show clinical signs which often

are relieved by treatment with male sex hormone.

In 1943 Werner's (25) investigations led him to state that existence of the male climacteric is well established, and that the syndrome was the same as in the female menopause except for the cessation of menstruation. He explains the severity of symptoms in both sexes varies according to their constitutional, function, and mental makeup.

In his article in 1945 Werner (27) laments that too much importance is placed in arguments against the existence of a male climacteric upon the fact that man has no definite outward sign of the decline of sex function. Man has no outward sign of a sex function such as menstruation, and yet no one denies his part in the act of reproduction. Therefore it is not reasonable to conclude that just because he does not have a corresponding outward manifestation that he does not have a decline of sex function and can not have a climacteric.

In his later article in 1946 Werner (28) repeats that the male climacteric is an established fact. He feels that it would be found that just as many men experience an endocrineautonomic imbalance and climacteric

symptoms following a decline of sex function if the syndrome were recognized and the information were available. He feels that the syndrome is often overlooked because of three reasons.

- (1) Lack of an outward sign.
- (2) Symptoms often milder.
- (3) Syndrome not even considered due to ignorance or disbelief.

Douglas (7) feels much the same as Werner, concludes that it is accepted, and feels the only confusion is over the age of onset. Even though he states it is an accepted entity, he feels the syndrome is more common than usually thought and is overlooked often because of mildness of symptoms and especially because of a lack of an outward sign like the end of the menstruation in female.

Ayres (2) states many authorities either deny there is a male climacteric or avoid discussion yet insists there are profound physical and mental changes at the time of decline of sex function which most certainly occurs. Ayres apparently feels that every man passes through a climacteric but agrees that it is overlooked more often than diagnosed. He rationalizes that it is not surprising that since man's part of reproduction is comparatively small that his change in life would come

later and with milder manifestations. This writer believes Ayres is getting close to the truth with these statements and his contention that in a few aggravated cases men show all symptoms of the female climacteric replacing those peculiar to the female with urinary and prostatic symptoms.

Eidelsberg (10) observes that following bilateral removal of testes the pituitary gonadotrophic hormone increases in blood and urine and the pituitary enlarges; the same occurs at menopause in the female. He concludes therefore that similar findings in men over 50 years of age indicate without any question that man has a definite decline of sex function after a certain age.

Thus far in this discussion there have been presented the ideas of men who are pretty much convinced that the male climacteric is a definite entity. There are several investigators who keep near the middle of the road in the controversy and fail to say yes or no definitely.

Thompson (22) who has done a great deal of investigation in the realm of male sex hormone says in some men the production of the male sex hormone falls off as they grow older. This decrease may be great enough to

produce definite symptoms similar to those seen in women at menopause. However Thompson is clear in his contention there is no clear cut epoch in man as in female, and that the male climacteric is not common. He further emphasized that every old man who is tired is not suffering from the climacteric, and that the doctor must make a careful and fastidious search for some other basis. This writer would hasten to remind Dr. Thompson that this last statement could apply just as well to women. It must be kept in mind that the physical and mental symptoms of the female menopause (which few, if any, would deny) only persist for a varied but limited period, and few doctors try to blame the menopause for troubles seen in women after this period.

Another writer who tries to give the impression that there is no climacteric in the male is Boswell (5), but it seems to me that he turns right around and admits that it does occur. He starts by saying there is no significant dependable change in man as there is in woman; then continues with the statement there is a gradual unimportant decline in sex function starting at age 48 approximately; and finishes by admitting that sometimes there occurs a typical climacteric with symptoms very similar to those seen in female plus changes in and

symptoms from the prostate gland and seminal vesicles. It would seem to me that Boswell's stand would need further evaluation in regard to his use of the words 'significant' and 'unimportant'. To some men the gradual decline of sex function might be quite significant and significant enough to cause functional or nervous symptoms.

Heller and Myer (13) present an organic basis for justifying the claim that the male climacteric is a true clinical entity. In studying 23 patients with symptoms and signs similar to those seen in the female, they found an increase of gonadotrophic hormone in urine and blood just as in bilateral castration and primary gonadal degeneration. Histological study of 8 of the patients all showed testicular atrophy and degeneration. These men made fairly certain these patients were pure climacterics for they showed relief of symptoms only on androgen therapy and return of symptoms on withdrawal of the androgens. However these same investigators after studying and investigating gonadotrophic excretion, histology of testicular tissue, sex history, and physical findings in elderly men concluded both germinal and hormonal function of the testes is preserved well into senility in the average man. Their final conclusion is that the male climacteric

is a clinical entity and can occur as early as the third decade, but further that the male climacteric is an infrequent and pathological accompaniment of the aging process and consists of testicular failure which can easily be confused with psychoneurotic impotency.

There are some writers who feel strongly that there definitely is no such clinical entity as male climacteric. There was a very good unsigned editorial (9) in the Journal of the American Medical Association which expresses such an opinion after evaluating the work of certain endocrinologists. The writer of the article arrived at his conclusion from the following information: Text books say female mammals below the primates do not pass through a menopause since they do not menstruate. In female primates the primary phenomena at menopause is ovarian failure; the ovary becomes atrophic and secretes only a reduced amount of estrogens; there is a compensatory increase in secretion of gonatrophic hormone as manifested by an increased titer in the urine. In women vascular and psychoneurological complications are common, also headaches, giddiness, and rheumatic pains. If man passes through a similar stage he should show:

1. Testicular insufficiency and decreased secretion of androgens.

2. Pronounced compensatory increase of gonadotrophic hormone.
3. Vasmotor and similar phenomena seen in women at menopause.

The writer states there is no real evidence of this occurring abruptly in man and cites the work of several men in support of this conclusion.

Dingemans, Borchart, and Laquer (6) have shown there is a definite decrease of androgens in the urine of older men. The average excretion per liter of urine in men ages 20-34 years was 40-50 international units, * and in men 57-64 years of age the average is 19 international units. Fraser, Forbes, Albright, Sulkowitch and Reifenstein (11) found 1.8-4.8 mgm. per liter of urine from men 71-75 years of age and an average of 13.8 mgm. in younger men. However neither of these groups could demonstrate any abrupt drop in androgens secreted. Nor was either able to demonstrate any sudden increase of gonadotrophic hormone, and both suggested that this may be due to the fact that there is less pituitary secretion in

* NOTE: An international unit of androgen: That amount of the hormone which when injected daily for 5 days yields an average of 5 mm. increase in length and height of combs of at least 5 brown leghorn capons (10).

older men. If such were the case the decrease of testicular function would be secondary. Also this would nullify much of the criteria many investigators are using in deciding about the existence of a male climacteric, i.e., the decrease of androgens and the increase of gonadotrophic hormone in the urine.

The editorial being discussed says that vascular and nervous phenomena as seen in the female menopause are comparatively rare in male of older age but may be seen in cases of primary testicular insufficiency (which most writers contend is the cause of the climacteric) accompanied by increased secretion of gonadotrophic hormone even surpassing that seen in female menopause. This change in hormone secretion does not occur if the testicular insufficiency is secondary to pituitary insufficiency (which no one contends is the basis of the climacteric). According to the work done by Heller and Serveringhaus (18) this primary testicular failure very seldom occurs spontaneously, but a few cases have been reported. Hess, Kunstadter and Saphir (15) have shown the climacteric state possibly present in bilateral cryptorchism with a resultant decrease in testicular secretion and supranormal amounts of gonadotrophic hormone in the urine. They have reported a similar syndromes following

orchitis, traumatic castration and surgical castration (hernia, tumor, hydrocoele). It is obvious in such cases that there is a testicular failure, and it would seem to me if one could show this decrease of androgens and increase in gonadotropins in older men that one would have to conclude there is a decrease of sex function and, so, a climacteric.

The editorial in question concludes from the above material that in the normal male a sudden spontaneous onset of a primary testicular insufficiency is seen very infrequently, and there is no real evidence of a regular sudden decrease of androgens with a compensatory increase of gonadotrophic hormone in the urine of every male as in female. The writer of this editorial should keep in mind that most men who think there is a male climacteric admit that the decrease in sex function in male is more gradual than in female but that nevertheless the decline is sufficient to cause psychic and endocrine autonomic upsets which result in signs and symptoms of the climacteric.

However other men also feel much the same as the article discussed above. Bauer (3)(4) contends the male climacteric

can truly exist only if there is a cessation of testicular function at a definite period in life, and that there is no real evidence of this, only of a slackening of function with age. He feels only occasional mental depression coming with the end of sexual potency is not a justification for existence of an entity called a male climacteric. He further feels that since the syndrome is seen in some men under 30 years of age and only rarely in the older group that the syndrome is misnamed and should be called "testicular failure".

SUMMARY OF ARGUMENTSPRO AND CONPRO

1. Presentation by several investigators of series of patients presenting symptoms the exact counterpart of those seen in the female menopause.
2. Symptoms in male are later in life, milder, and more gradual and therefore are often missed or ignored.
3. Too much emphasis on man's lack of an outward sign of the decline of sex function.
4. Report by some investigators that in older men there are less androgens and more gonadotrophins in the urine just as following bilateral orchidectomy in younger men.

CON

1. Most men show no symptoms similar to those of female climacteric.
2. Even in men where climacteric symptoms are seen, they are much milder than in women with few exceptions.
3. Men lack an outward manifestation of a cessation of sex function similar to cessation of menstruation.
4. Many men show no decrease of libido and potency until very advanced ages. Numerous times active spermatozoa are seen in prostration secretion stripped from men 70 and even 80 years of age.

PRO cont'd.

5. Less disturbance in males because sex life usually not as great a part of their lives as in females.

6. Histological studies of testes of men showing climacteric symptom reveal testicular atrophy.

7. Response of climacteric syndrome to testosterone medication.

CON cont'd.

5. Some say there are no differences between level of gonadotrophic hormones in urine of older and younger men.

6. Decline of sex function in male is no physiological instance in man's life but merely part of the aging process.

7. Mammals below primates do not pass through climacteric since they don't menstruate.

8. No one has been able to demonstrate any abrupt decline of sex function in male except in pathological or surgical instances.

CON cont'd.

9. Orchiectomy for carcinoma of prostate in older men is effective in a high percentage of cases. Would indicate some hormonal activity of testes late in life.

SUMMARY:

Among the men who have become genuinely interested in this subject and have done some investigation, the majority, led by Werner who seems to have done the most exhaustive work in attempting to find the answer, seem to have become convinced in varying degrees of the existence of a clinical entity called the male climacteric. They feel that most men go through the same decline of sex function with a decrease of androgens secreted and a compensatory increase of gonadotrophic hormone; these phenomena result in an endocrineautonomic upset which together with accompanying decline of libido and potency can cause profound physical and mental symptoms similar to those seen in the female at menopause, even though in most cases the symptoms are too mild to gain attention or cause concern enough for the man to go to a doctor. These men contend that this change of life in man comes on more gradually, at a later age, and with less disturbance in the average case than in woman. Because of these differences and the fact that man lacks an outward sign similar to the cessation of menstruation, Werner and the others who agree with him feel that the male climacteric is often misdiagnosed, overlooked, or ignored because of ignorance or disbelief.

There are many doctors who never have considered that such a syndrome even exists and therefore never look for it as a basis for signs and symptoms in a patient. These doctors usually are informed inadequately if asked what they think about the male climacteric.

On the other side of the controversy are the investigators who have concluded that the male climacteric is a medical entity only in infrequent and pathological instances. These have reached this decision from the facts that there is no clear cut significant epoch in male as in female, that there is no outward sign such as the cessation of menstruation, that there is no demonstrable abrupt decrease in androgen secretion and no increase of gonadotrophic hormone in the urine as in the female menopause, and that the symptoms of the female menopause are seen less frequently and with less severity in male.

It would seem to this writer that the arguments on both sides of the controversy are right as far as they go. Those who are on the negative side are emphatic in saying the male climacteric as an exact counterpart of the female climacteric does not exist except in comparatively rare cases. These same men would not deny that man certainly does reach a stage in his life when his sex function declines, and this might manifest itself in ways similar to

the ways of the female menopause. Moreover it seems to me that is exactly what those on the positive side are saying, i.e., that every man experiences a gradual sexual decline which may cause symptoms like those in the female and infrequent cases as severely as in the female. In my opinion this last statement is approximately the situation that exists. It would seem that male reaches a point, approximately between ages 48 - 52 years, when the activity of the testes begins to decrease slowly. In a very few men, either because of an extreme sexual drive or psychological imbalance, this gradual decline will cause symptoms similar to those of the female menopause. However there are several reasons why it seems apparent that this sex decline in male is very slow, and that some function of testes may remain even into very advanced age. Facts which would indicate this last conclusion are the active sperm seen in prostatic strippings of men in their 70's and 80's, semination by men of similar ages, and the effectiveness of bilateral orchidectomy for prostatic carcinoma in older men.

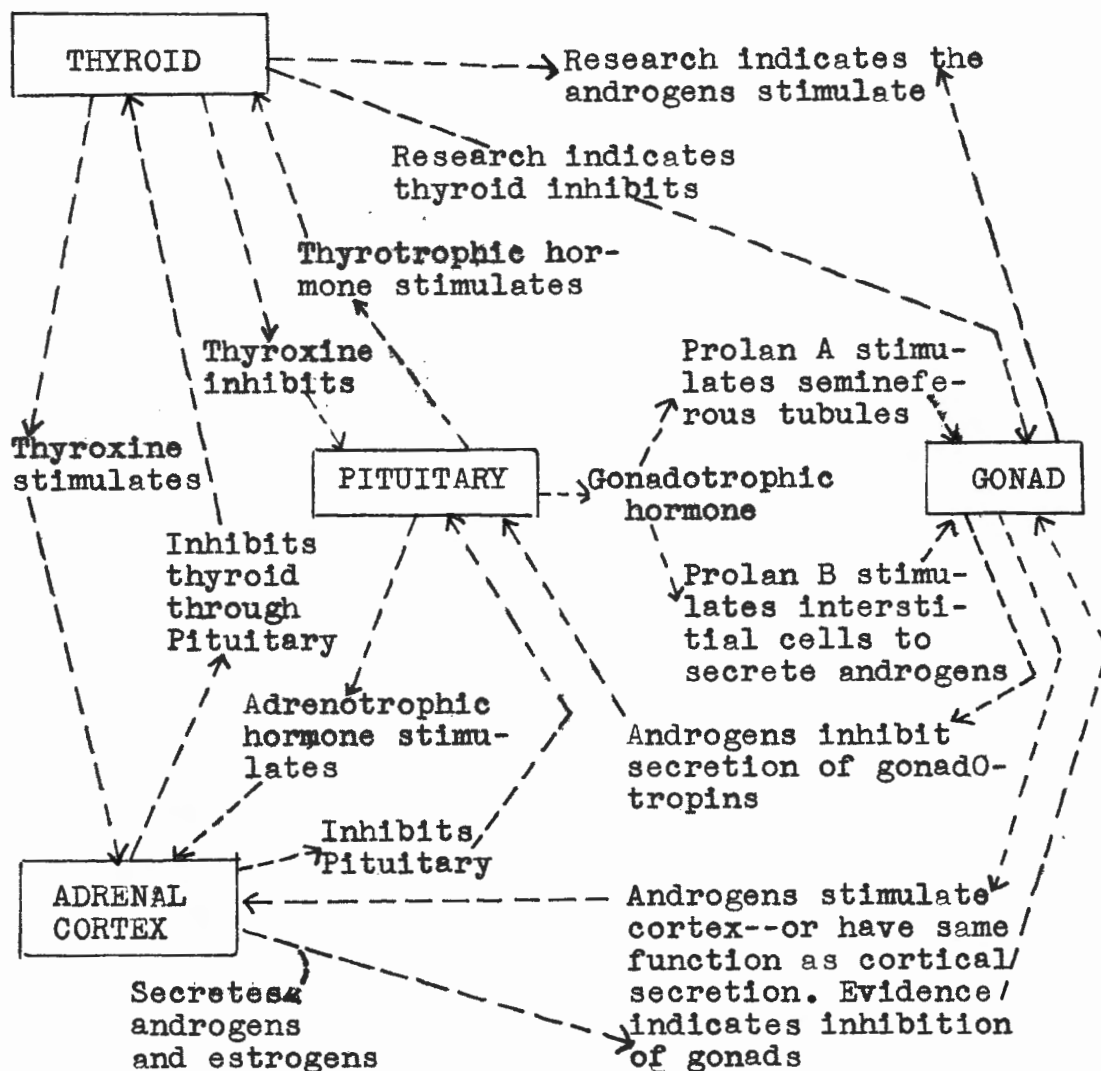
One might ask why the climacteric usually causes less disturbance and less severe symptoms in the male. This probably is explained by two things; first, because the

decline of sex function is more gradual in man, and second, to most women the bearing of children and the raising of a family is often by far the most important part of their lives, while man in addition to this part of his life usually has a business and career which is also a great, if not the greater, part of his life; consequently the decline of sex function does not cause as often such a psychic upset as seen in more females.

In both sexes the severity of symptoms at the time of sex function decline depends on the individual's constitutional and mental makeup. The man or woman who has been able to find complete satisfaction of his or her sexual desire throughout most of life may suddenly find his or her libido and potency on the decline, and this may cause severe repercussions physically and mentally. On the other hand the individual such as the bachelor or 'old maid' who earlier in life had had to divert his or her sex drive into other activities and thus accomplish his or her adjustment earlier in life will pass through the climacteric changes with little or no trouble.

WHAT IS THE MALE CLIMACTERIC

The following diagram is presented to help visualize the changes possibly involved in the male climacteric. The diagram attempts to show the principle relationships of endocrine glands in normal man. All the relationships are not known for certain but those below represent the consensus.



As expressed by Dr. Ayres of Memphis (6) the main purpose in life is to reproduce and perpetuate the race--this function and the living of a happy and comfortable life depends on an endocrine balance. All stages of life depend on this balance; growth, vigorous adulthood, age of involution, and old age. The pituitary is the center of this balance; it produces the gonadotrophic hormone among its other important functions. This hormone stimulates secretion of the gonadal hormone which in turn inhibits the anterior pituitary secretion of the gonadotrophic hormone. This balance continues until the response of the testes to the pituitary secretion decreases. Then the gonadal secretion decreases, and since this means less inhibition of gonadotrophic hormone formation in the pituitary the amount of gonadotropins in the body increases. Thus this failing activity of the testes causes an endocrine imbalance.

It is this endocrine imbalance that Werner (25) thinks is the basic phenomena underlying the male climacteric. He believes that the disturbed and over active pituitary in turn results in autonomic nervous system upset which causes the vasomotor symptoms described as part of male climacteric. He further claims that the vasomotor symptoms together with the symptoms of hypogonad function, chiefly loss of potency, cause a psychic upset

which manifests itself in the various nervous symptoms of the syndrome. (27) He believes this because the symptoms are very similar to those of syndromes wherein endocrine imbalance is an accepted fact, eunuchism and bilateral cryptorchism for instance. He theorizes that although the decreased androgen secretion and increased gonadotrophic secretion are proven facts, the endocrine autonomic upset may be even more extensive with the thyroid and adrenals secondarily involved since their function is under pituitary control. (28)

Further discussion of these claims of Werner may help the reader decide in his own mind what the male climacteric is, if it is. The best way to discuss these points is to review an article by Bauer (4). In discussing Heller and Myer's work about gonadotrophic hormone decrease with testosterone medication (see section on treatment), Bauer insists that many questions are unanswered. These questions are:

1. Why, if, as Heller and Myer claim, the increase in gonadotrophic hormone is due to the failure of failing testes to utilize it, does the gonadotrophic hormone decrease when parenteral testosterone is administered? - This is because the testosterone, a substitute for testicular secretion, inhibits

the pituitary secretion just like the latter secretion does in the normal man. (25)

2. Why do some patients with clinical symptoms but no testicular degeneration as in male climacteric and thus classed as psychotic show not even psychological response to testosterone? - Howard and Vest (16) and Ketchom (18) have both demonstrated that a man lacking male sex hormone are super-sensitive to testosterone and respond to dosages that have no effect on men not lacking the hormone.

3. Why are there no transitional or combination of hypo function and psychoneurotics symptoms? - Werner claims to have proven that in any male climacteric wherethe symptoms are severe enough for the man to seek treatment the symptoms are a combination of vasomotor symptoms from hypo-function and nervous signs from psychic upset. (27)

4. Why one never encounters spontaneous hot flashes in the male? - Several authorities have reported frequent instances of such symptoms. These include Werner (25), Boswell (5), and Douglas (7). Werner does admit that the hot flashes and other vasomotor symptoms in the male climacteric are less severe because of a more gradual development of hypo function than in female.

This writer personally thinks most of Bauer's points are poorly taken, but answering them gives a good opportunity to further explain Werner's opinions and theories.

There are some investigators who do not agree with the foregoing conclusions. The work of Sevringhaus and Heller (14) showed the levels of the gonadotrophic hormone in the urines of older and younger men show no consistent differences, and consequently they claim to have proven there is no scientifically consistent increase of urinary gonadotrophic hormone in the male climacteric. They report that the amount of this hormone in the urine of male at any age, young or old, never reaches anywhere near the levels seen in the average female at menopause. These workers are not disclaiming the existence of a male climacteric, but only that there is no correlation between the level of gonadotrophic hormone in urine and the psychic and vasomotor symptoms of the climacteric. This lack of correlation is further borne out by the fact that these men found some older men with involutional psychoses whose urines showed gonadotrophic titers at least approaching those in menopausal women and also some men of similar ages with the same symptoms but having low gonadotrophic titers. - There is no assurance that these workers were not failing to differentiate

between patients suffering from psychic symptoms from testicular hypofunction and patients with true psychotic depressions.

Heller and Sevringhaus also have shown that gonadotrophic titers in the urine of castrate males approximate the titers in urine of castrate and elderly females. They conclude that this shows there is a possibility of testes continuing to play at least a reduced role in gonadal - pituitary relationship in later life in male and thus use up some of gonadotrophic hormone secreted. This is supported by the observation that secondary sex characteristics in males rarely fade as in female after menopause. These facts also agree with the conclusion that the male climacteric is more gradual and less severe than the female menopause.

With some investigators saying there is an increase of gonadotrophic hormone secretion in males after a certain age and others saying there is not such an increase, it is difficult to decide which is the exact case. It is difficult to discount the contentions of Werner who has done such extensive work on the subject. Moreover there are several men who have done considerable work on the treatment of the syndrome who are certain the levels of

this hormone is raised before testosterone medication and decreased there after.

There are a few writers who have completely different ideas on the cause of the male climacteric. However all these men have done little or no real research on the subject and are merely presenting their theories which have no other substantiation. Abarbanel (1), after careful observation of 2 patients feels that the climacteric is not necessarily the result of testicular failure per se but may be the result of a disturbance in heat regulation due to a drop in the level of certain steroids within the endocrine system of the body.

Douglas (7) evidently believes that the underlying cause of the climacteric is the result of gonadal function decline which initiates an endocrine upset. However Douglas does not agree with Werner and the others that this causes an autonomic nervous system upset. He contends that the endocrine upset is the result of androgens becoming insufficient to counteract the estrogens secreted within the male system. He further theorizes that this endocrine imbalance results in an auto-intoxication which manifests itself in the symptoms and signs of the climacteric.

LaMar (20) also disregards the endocrine-autonomic imbalance theory and claims that the male climacteric is merely part of the aging process, and that the symptoms are merely manifestations of the undoing of all products of the gonadal secretions due to a gradual decrease of testes function. As described in the section on symptoms, LaMar mentions many symptoms and signs not mentioned by other investigators.

SUMMARY:

It is in this aspect of the subject that there are so many divergent claims and theories that it is very difficult to decide what the climacteric actually is or what cause it. However it seems there is considerable, in fact almost complete, agreement that underlying the syndrome is a gradual decrease of testicular function. Whether or not there is a compensatory increase of gonadotrophic hormone is undecided although it would seem logical since the androgen inhibition of the pituitary is decreased. There is a possibility that the capacity of pituitary to produce gonadotrophic hormone is also decreasing at the same time. At any rate it seems safe to assume that with the hypo function of the testes the endocrine system of the body is upset. Since the autonomic system depends on the integrity of the hormonal system, the vasomotor symptoms attributed to the male climacteric undoubtedly are the result of the endocrine upset.

The same hypofunction of the testes and decline of androgen secretion can explain from a purely physiological basis the decrease of potency.

Most authorities also agree that the vasomotor symptoms and decrease of potency result in psychic changes that

cause the nervous manifestations of the climacteric. This writer found it interesting that in discussing this subject with a member of the local staff the opinion which follows was forth coming. The opinion was that the change of life in both sexes is primarily a psychic matter with endocrine changes playing a very minor role in any manifestations seen.

Why symptoms of the change of life in man are less severe and less frequently observed, and why symptoms vary within the male sex are explained in the first section of this paper.

SYMPTOMS OF MALE CLIMACTERIC

Although there is the endless and as yet unanswered controversy as to whether there is a male climacteric, and, if there is what it is exactly, there is a fairly complete agreement among those investigators who believe there is a climacteric in male as to what the signs and symptoms of the syndrome are. Many of the investigators enumerate many of the same symptoms; however some list certain manifestations not mentioned by others.

Perhaps the most exhasutive investigation of this syndrome has been done by Dr. Werner (25) (27) of the University of St. Louis and his compilation of signs and symptoms of the male climacteric is the longest and most complete. He states subjective symptoms are functional since they are due to neuro-encorine imbalance. These symptoms are divided into three groups, nervous, circulatory, and general. These symptoms are:

A. Nervous

1. Nervousness and tension:

This tension usually consists of an inward tremulousness which is seen most frequently during the night or in the morning upon arising. Any ordinary excitement or fatigue is exaggerated.

2. Irritability and excitability:

In addition to any excitement being exaggerated the man suffering from the climacteric gets irritated, upset, and excited about things which ordinarily he would not.

3. Sleep poorly at night:

This often results in more sleeping in the day time, and this must not be confused with somnolence.

4. Paraesthesia:

May consist of numbness, tingling or even itching and prickling on any area of the body.

5. Formication:

This consists of a feeling of little bugs crawling all over the body.

6. Headaches:

These are not migrainous but may be dull ache recurring irregularly or continuously. The location varies, but the most diagnostic locations are vertex and cervico-occipital. These headaches are often accompanied by mental haziness.

7. Decrease of memory and power of concentration:

The loss of memory is especially concerned with recent events. These two symptoms, of course, are important features of senile dementia or cerebral arteriosclerosis, but they occur, at an unusually early age in the climacteric and are dramatically relieved by testosterone which certainly does not occur in the other two conditions.

8. Depression and mild melancholia:

This is expressed in an obvious loss of interest in work, home, family problems, hobbies, and social activities. Patients also may cry or show other signs of emotional instability for no reason at all; they know something is wrong and become introverts; they are ill at ease, apprehensive, and unnecessarily worried; they lose all self confidence and develop a feeling of hopeless futility and show an uncommon desire to be alone; all this may often go to the extent of verging on a real psychotic depression to the point of self

accusation and, even in extremely rare cases, self destruction. This mental condition is often referred to as "climacteric psychoses".

b. Circulatory

1. Hot flushes:

Due to dilatation of superficial capillaries; often accompanied by profuse perspiration and occasionally by vertigo. The flushes chiefly involve the upper face, neck, and body. They usually are short but may last for an hour, and patients often feel like they are smothering. In some patients the flushes may alternate with chills.

2. Tachycardia, palpitation and dyspnea:

In case of such symptoms must be certain there are no other causes. These symptoms may even awake the patient during the night.

3. Vertigo with change of position:

This often is associated with tinnitus and scotomata; again must rule out cardiovascular disease as possible cause.

c. General

1. Lassitude and fatiguability:

Patient is either constantly tired or arise tired in the morning.

2. Widespread vague pains:

3. Decrease or loss of potency;

4. Libido effects are different:

The libido operates through the conscious mind and depends on the patient's mental reaction rather than endocrine action. Consequently, though low libido often is present, libido may be high even though potency is low or absent, and this can be quite confusing to the general practitioner.

5. Constipation:

Werner feels that constipation seemingly is more coincidental than part of the syndrome of endocrine imbalance; is more from improper diets and habits and gastrointestinal disease.

6. Gastric distention and eructation:

Usually secondary to nervousness and constipation.

7. Low basal metabolic rate:

In most patients the B.M.R.'s are in the normal range. Some may be slightly on the negative side, but none are on the positive side. However the low B.M.R.'s are not from hypothyroid for there usually are no other clinical signs of hypothyroid, and the B.M.R.'s are not raised by thyroid medication.

A table showing the frequency with which Werner (5) found various symptoms in patients are as follows:

	<u>Per cent</u>
1. Nervousness, subjective.....	100.0
2. Potency decrease or loss.....	94.9
3. Depression.....	89.4
4. Decreased memory and concentration.....	86.5
5. Fatigability and lassitude.....	75.7
6. Loss of interest and self confidence.....	70.0
7. Sleep disturbed.....	64.9
8. Irritability.....	59.4
9. Excitability.....	51.3
10. Ill at ease.....	51.3
11. Cervico-occipital aching.....	51.3
12. Numbness and tingling.....	51.3

13. Vertigo.....	48.6
14. Hot flushes.....	46.0
15. Headache.....	46.0
16. Constipation.....	43.2
17. Tachycardia, palpitation, dyspnea.....	40.5
18. Crying.....	37.8
19. Sweating.....	35.0
20. Scotomata.....	35.0
21. Itching.....	29.7
22. Unsociability.....	27.0
23. Desire to avoid crowds.....	27.0
24. Vague pains.....	24.3
25. Cold hands and feet.....	21.6
26. Thoughts of self destruction.....	19.0
27. Psychoses.....	13.5
28. Formication.....	13.5
29. Tinnitus.....	10.8
30. Self accusation.....	5.4
31. Attempted suicides.....	5.4

As shown above the main symptoms and those seen most frequently by werner in his series of 254 patients are:

- a. Nervousness and tension
- b. Loss or decrease of potency

- c. Depression and mild melancholia
- d. Decrease memory & power of concentration
- e. Lassitude and fatigability

It is seldom or never that all symptoms listed appear in each patient but several almost always will be found and quite often all five in the shorter list above.

Obesity, was strikingly infrequent in Werner's patients. This might seem to intimate this syndrome is seen more often in more active men and in those who have found necessary a minimum of glandular readjustment during life.

Werner's patients ranged in age from 41-64 years, and the average age was 53 years.

There are several other men who include other signs and symptoms in the climacteric syndrome or present a somewhat different point of view of some aspects of the picture as presented by Werner. Abarbanel (1) found a generalized pruritus in several of his climacteric patients but finally concluded that it usually was not manifested until the 6th decade. He also found a frequent incidence of regression of testes and insisted that such was not to be considered any more pathological than the ovary

ceasing function; he further states that in either case the climacteric is physiological and not pathological. This same investigator is alone in his contention that the loss of libido and impotency are not part of the climacteric in either sex but merely are coincidental.

Boswell (5) and Douglas (7) both describe various urinary symptoms which would seem to be a part of the climacteric syndrome in male. Boswell lists vague pains over the bladder, loss of force of urinary flow and a partial incontinence. Douglas explains this incontinence by a decreased function of the bladder neck. He says he usually finds no residual urine or overdistention in climacteric patients to explain the pains over the bladder region. These urinary symptoms could be explained at least in part by the prostate changes described by these same men. Boswell merely says there is a change of shape, size, consistency and function of the prostate. Douglas describes these changes more fully. He says in the male climacteric the prostate is enlarged and boggy, and the seminal vesicles are enlarged and lacking in tone due to decreased secretion of androgens; on carefully stripping these structures all show signs of inflammation, debris, and pus cells. Several men with prostates in this condition were treated in many different ways but improved

only after the accepted climacteric treatment, i.e., testosterone.

Dunn (8) says the symptoms of impotency and decreased libido may precipitate profound psychic upset and an occasional individual conceives himself as utterly useless and hopeless and burdensome even to the point of self destruction. This point of view by Dunn would seem to support the conclusion that all symptoms are not the result of neuro-endocrine imbalance directly.

LaMar (20) who contends the climacteric is more gradual includes several other signs and symptoms which would seem to be more a part of the general aging process; however the climacteric is considered by some as merely a part of that aging. These manifestations presented by LaMar are relieved by testosterone, he claims, and therefore certainly are worthy of mention here. They are an atrophy of the testes and penis, thinning of hair and a change in its color; bones and muscles losing some of their hardness and strength; the larynx, enlarged by testosterone, now relaxes and atrophies, and the voice gets its typical crackling of senility; fat deposits on abdomen, face, shoulder and buttocks decrease.

It must be admitted that all manifestations mentioned as part of the male climacteric could be caused by other things including psychoneuroses, central nervous system lesions, cardio-vascular disease, hypo- or hyperthyroidism, gastro-intestinal disease, and urinary or prostatic conditions. However, it should be safe to assume that each investigator before presenting any symptom or sign as a part of the syndrome under discussion had carefully ruled out any possible other cause, and had seen said sign or symptom relieved by climacteric treatment.

Viewing all the manifestations presented by various investigators as a whole it would seem that the syndrome claimed to represent the male climacteric is a near counterpart of the female climacteric in its psychic, circulatory, and general aspects. The only differences seem to be the lack of such a definite outward sign as the cessation of menstruation and the presence of symptoms and signs from structures peculiar to the male, the testes, prostate, seminal vesicles, and the penis.

DIAGNOSIS OF MALE CLIMACTERIC

The diagnosis of the male climacteric often is missed because of ignorance, disbelief, or more often because at the age it usually occurs the patient has other pathology which disguised it. The syndrome is usually diagnosed from symptoms alone (13), symptoms appearing at the appropriate age with sudden onset with no previous history of psychic trauma.

The finding of low titer of androgens in the urine may be a great aid to diagnosis (5). The method of measuring androgens in the urine by injecting into leghorn capons is set forth earlier in this article. The androgens are secretions of the testes, and therefore the amount secreted in the urine is a fairly good measure of testicular hormone function. It is very important to make sure that there are present no other causes for testicular failure such as eunuchodism, testicular hypoplasia, testicular atrophy following disease, infantilism, or crypt orchism. A somewhat similar aid to diagnosis is the measuring of gonadotrophic hormone excretion in the urine and finding an increase. However it must be kept in mind that whereas testicular failure or inadequate testicular stimulation are the only instances with decreased androgen secretion there are several other causes for increased gonadotropins, including adenoma of the anterior pituitary (Cushings'

disease). However, it is very indicative if one finds both an increase of the gonadotropins and a decrease of androgens in the same patient.

Some advocate the use of a therapeutic test with androgen medication, but this can be dangerous for it may relieve some of the symptoms without uncovering the underlying causes (2). However, if, after a careful history taking and examination, the patient shows improvement on androgen therapy, the clinician can be fairly certain of his diagnosis.

The important aspect in the diagnosis of this syndrome is a careful and painstaking elimination of all other possible causes of the symptoms. It should be remembered that a man's responsibilities, stresses, worries, and activities are often at their peak at 45 - 55 years of age and may cause nervousness which easily could be confused with climacteric syndrome. Since quite often the patient has a low B.M.R., diagnosis of hypothyroidism often is made erroneously, but the patient will be given no relief from thyroid medication (25). Some of the organic pathology that must be ruled out absolutely are:

(5)

1. Gastric neurosis - could cause the nervousness,

tension, irritability, and gastro-intestinal symptoms and many of the other symptoms.

2. Early nephritis - could explain the poor sleep, headaches, lassitude and fatigue, pains, and the decreased sexual activity.
3. Cardio-vascular disease - pathology of this type must be excluded very carefully for it could include arterio-sclerosis and hypertension and could cause almost all the symptoms listed under the male climacteric.

It must be remembered that any fairly serious organic disease could cause enough psychic upset in some individuals to initiate all the nervous symptoms mentioned, including the decline of libido.

Heller and Myder (1) discuss at great length the confusion that may arise from a psychoneurotic impotency and explain that just a little careful investigation will clear up such a case. There would be no decrease of androgens or increase of gonadotropins in the urine; even more conclusive would be the failure of the patient to respond under androgen therapy.

SUMMARY:

The diagnosis of the male climacteric is made almost exclusively from the symptoms alone and the really important aspect of such a diagnosis is the careful elimination of any other possible causes of the symptoms.

TREATMENT

In the treatment of the male climacteric there is almost no controversy. Except for a few slight differences of opinion concerning the exact dosage of drug to be used there is complete accord among all the men who have worked on the problem. They agree on what drug to use, how to use it, the results to expect from it, and what the limitations, contra-indications, and abuses of the drug are.

DRUG USED

The specific drug to be used in treating the male climacteric is an androgen according to Thompson (22), who has done more work than any one with the subject of the male sex hormone. Testosterone in active chemical combination is the male sex hormone. It was isolated by Laqueur, Dingemause, and Freud in 1935 and synthesized by Ruzecka and Butenandt a short time afterwards. Testosterone used as its propionate salt has since then become the only medication for the male climacteric. Earlier methods of treatment included attempts to stimulate with gonadotrophic hormone but this was very unsatisfactory for two reasons: (1) it is fairly well accepted that testicular failure rather than inadequate testicular stimulation is the underlying cause of the climacteric; and the increase of gonadotrophic hormone in the system was emphasizing the endocrine imbalance causing a great

many of the symptoms. Other improved but still inadequate methods were (21).

1. Dessicated testes tissue;
2. Hombreal, an urinary androgen extract.

With appearance of testosterone upon the scene the other methods of treatment almost completely disappeared. Testosterone as a substitution for the secretion of the failing testes suppresses the anterior pituitary, thus decreasing the excess secretion of gonadotrophic hormone and restoring the endocrine balance. In addition to its inhibitory action testosterone by direct stimulation causes in most cases an increase of libido and potency (2). Testosterone thus relieves both the vasomotor symptoms from endocrine-autonomic upset and psychic symptoms from loss of libido and potency.

According to Werner (25) 90% of the climacteric patients are given nearly complete relief of all symptoms and increased feeling of well being. There is improved physical and mental effort, relief of depression and irritability, disappearance of tension, often a beneficial weight gain in thin patients, improved facial color and appearance, relief of vasomotor symptoms, increased potency, and increased libido possibly due in some part to the relief of

mental symptoms. Werner cautions that occasionally a patient is relieved of symptoms but gets no increase of potency; in such cases greater dosages with androgens can not be certain to increase potency, for the impotency in such cases can be the result of senility setting in, thus reiterating the fact that Werner believes the climacteric in man is a definite physiological phenomena and not the result of getting old. Lake (10) also sounds the same warning concerning the possible failure of potency to respond to endocrine therapy. He says a better remedy for failing potency in a man is "a long second honeymoon with an affectionate understanding and sexually well trained wife".

One slight dissenter to testosterone therapy as the final answer could be McCullagh (21) who states that testosterone is the only androgen thus far isolated from the testes, and it is not proven that testosterone is the only androgen secreted by testes, or that it will completely replace the endocrine action of that gland. However the dramatic response to testosterone therapy in climacteric patients has been demonstrated repeatedly and will be shown in case studies presented later in this paper. All writers make a point to remind readers that treatment with androgens is a substitution therapy, and while it gives symptomatic

relief it does not correct the underlying changes. The patient should be told that, although as time goes on smaller doses may suffice, the medications must continue as long as physiological drives demand relief from symptoms.

Some people have suggested that the relief of symptoms from testosterone medication is purely psychic and more from suggestion and assurance of doctor rather than any pharmacuetical action of the drug. This usually can be shown to be erroneous by merely withdrawing the drug and observing the return of symptoms (13). Douglas (7) proved even a little more conclusively the results of testosterone treatment were not entirely psychic. Following a later relapse in a case treated earlier with testosterone, there was no response to injections of alcbhol, water and mineral oil; in another case good response was obtained in a patient who did not know what he was being treated for or what drug was being used.

METHODS OF ADMINISTRATION:

Almost complete agreement exists about the fact testosterone given intramuscularly in oil and as propionate salt is the effective method of administration. The reason it is given as the salt is that as such it is

absorbed more slowly and necessitates fewer injections; the same is the reason for injecting the drug in sesame oil (5). However, there are other methods of administration and forms of drug to use. Experiments by Greene, Burrill; Oppenheimer, and Nelson at Northwestern University (12) have shown the potency of androgens varies with vehicle used and with route of administration. They used testosterone, testosterone propionate, and methyl testosterone, and gave them to rats by the following routes;

1. Subcutaneously in ointment,
2. Percutaneously in ointment,
3. Subcutaneously in alcohol,
4. Subcutaneously in pellet form.

Their observations showed the following results:

1. Each drug used was most effective when used in pellet form;
2. Each was more effective in alcohol than in ointment;
3. There was no difference in effectiveness of different drugs when all administered by the same routes.

Heller and Myer (13) sum up very well what the majority of investigators feel about the giving of oral methyl

testosterone. On the whole the use of the drug in this way has been disappointing. It is true that a relief of symptoms can be obtained this way, but to do so effectively necessitates such large doses that it can be quite expensive and often causes severe nausea and vomiting. Thompson (22) dismisses the subject by saying it is less effective. On the other hand Lake (19) advises the general practitioner who may not be too well trained in endocrinology to treat patients with oral extracts of whole testes. He recommends this because in his practice he has been able to give these extracts over long lengths of time with good results and without patients complaining about endless injections. He comments that patients can be told to regard the medication as a specialized addition to their diet much like the taking of vitamins, etc. This oral medication can be made more effective by supplementing it with occasional (weekly or monthly) injections of testosterone propionate in oil.

Boswell (5) mentions that testosterone can be mixed with wool fat and applied locally. Both he and Dunn (8) feel pretty certain that the drug is readily absorbed from such a surface, and that symptoms are relieved by such medication. The disadvantage of this route of medication is again that it takes an expensively large

dose to be effective and necessitates bandaging of the area.

The only method of giving the drug that may eventually rival or supplant the intramuscular is the subcutaneous implantation of testosterone pellets. Heller and Myer (1) recommend use of 4-8 testosterone pellets of 75 mgm. each placed subcutaneously in the thigh every 6 months, if patient needs a greater dosage than can be given conveniently intramuscularly, and Thompson (15) admits that such medication may have a great future in the field. The great advantage of this method of medication is that it is absorbed even more slowly than the intramuscular oil injection and thus facilitates even less frequent administration. In 1939 Howard and Vest (17) presented a new injection instrument for placing such pellets subcutaneous without making an incision which till then had been the draw-back of the method. All three groups of investigators mentioned in this paragraph give positive assurances that there are no undesirable therapeutic effects and a minimum of foreign body reaction about the site. At present there are not enough reports on the use of these pellets to give the final answer. Only the future holds the answer to this.

DOSAGE:

Although there are a few men who advocate greater or smaller doses the majority of men recommend the use of 50 to 100 mgm. a week in divided doses until symptoms are brought under control; certain small adjustments are usually made for the weight of the patient and the severity of the patients.

Thompson (22) recommends 25 mgm. of testosterone propionate in oil injected by hypodermic intramuscularly three times a week.

Howard and Vest (16) when using the intramuscular route almost exclusively employ 25 mgm. testosterone propionate in sesame oil injected intramuscularly twice a week. They claim less satisfaction and no better results with larger dose. These men have been unable to demonstrate any increased tolerance to the drug when given up to 30 months. For the benefit of certain critics who have noted that the same dosages cause no physiological changes in normal men, Howard and Vest explain they have proven a definite super sensitivity to androgens in men who are lacking in the hormone. Ketcham (18) confirms this contention.

Heller and Myer (13) think the best dose is the same as that recommended by Howard and Vest. However the former have some further recommendations. Although some other men think it is not a good idea, Heller & Myer suggest a therapeutic test before embarking on a long range androgen course. They say to administer 25 mgm. of testosterone propionate intramuscularly 5 times weekly for 2 weeks and observe the results. If there is no relief of symptoms, the patient either is not suffering from the climacteric or needs such high doses it would be financially unpractical to continue. If there is a response to the high dose of the therapeutic test, the doctor can find the minimal dose for control by trial and error; usually end up using 25 mgm. three times weekly just as Thompson.

Werner recommends a little higher dosage, using 10 - 25 mgm. every other day, and states that response, if the diagnosis is correct, will be visible after three or four doses, and complete relief is usually effected in two weeks; after this a smaller dose for maintenance can be determined.

Dunn (22) and Boswell (5) also favors a slightly higher dose, ranging from 30 -150 mgm. weekly with a total medi-

cation of from 300 to 400 mgm. Dunn presents as a typical case a patient to whom he gave 25 mgm. three times weekly for four weeks followed by 10-25 mgm. weekly for three weeks. Dunn also seems to have a very logical idea in giving the patient some sedation for the first week or so on androgen therapy, since it usually takes that long before there is an response to the drug.

Douglas stands on the other side of the picture and advocates 10 mgm. intramuscularly twice weekly and not in any case to exceed 50 mgm. weekly. He claims there usually is a response at 30 mgm. and one of his patients showed response at 10 mgm. He further claims that recovery is fairly complete after 100 mgm.

CONTRA-INDICATIONS AND ABUSES OF TESTOSTERONE:

Heller and Myer (13) caution doctors about the use of testosterone in the following conditions;

a. Carcinoma (especially of prostate):

Androgens stimulates growth of malignancies.

b. Edema:

Testosterone produces sodium and would increase the edema.

c. Normal testicular function:

Androgens would inhibit this function.

Dunn (8) warns that excessive stimulation of libido and increase of potency may not be desirable in male climacteric in any cases with any suggestion of hypertension or angina.

Thompson (22) laments that testosterone too often is prescribed carelessly and abusively. The most common abuses of the use of testosterone mentioned by him are:

a. May cause sterility:

The secretion of any internal secreting gland will inhibit the action of that gland. If the dosages discussed above are adhered to, however, there is little danger to any activity remaining in the failing testes.

b. May accelerate growth of prostatic carcinoma.

c. Is dangerous in old men with coronary sclerosis or hypertension:

May stimulate patient to enough increased activity to increase risk of coronary thrombosis, cardiac decompensation, or cerebral hemorrhage.

d. May cause acne vulgaris.

3. May cause hypermetabolism.

Thompson admits there is not much danger of these accidents at dosages not greater than 75 mgm. weekly but should be kept in mind when considering possible higher doses.

There is one possible drawback to testosterone treatment that was not mentioned in any of the articles. That is the cost of even the average course of medication. The average cost, at least locally, is \$10.00 - \$11.00 for 3 ampules of 25 mgm. each - it is possible to obtain the same amount of drug wholesale for about 60% of the above price. It is possible that this might limit the use of testosterone to the man with a fairly good income.

Dr. Payson Adams of Omaha, Nebraska, reminds the writer of another contraindication to the use of testosterone. Testicular secretion is known to stimulate the growth of the prostate. Therefore, the use of testosterone would be ill-advised in any patient with prostatic hypertrophy.

SUMMARY:

It is fairly well agreed that the treatment of the male climacteric is the weekly intramuscular injection of 50-100 mgm. of testosterone propionate in oil in divided doses. It may be well to use sedation during the first week of treatment. The use of subcutaneous testosterone pellets may warrant greater use in the future. Practitioners should be cautioned about using testosterone in patients with any malignancies, any angina, or hypertension, an any edema.

There are many case studies that could be included in this article for the sake of illustration, but one will illustrate just as well.

CASE STUDY:

HISTORY.-

J.K. was a married man aged 49. For the past three years he had noticed a decrease in libido and potency. He was subjectively very nervous, irritable and excitable. He was depressed and cried whenever anyone tried to talk to him. He had hot flushes frequently. There was a decrease in memory and inability for mental concentration; he was decidedly ill at ease, and he had a fear of impending danger and worried very much. There was a loss of interest in everything and a loss of self confidence. He disliked company and crowds and wanted to be alone. There was a feeling of frustration, and on several occasions he thought of committing suicide. He had vertigo with change of position and scotomata appearing as black threads before his eyes. There was occipito-cervical and retro-orbital aching. He was always fatigued and would rise unrested in the morning. He had tachycardia, palpitation, and dyspnea on light effort, and he would be awakened during the

night by tachycardia and palpitation. There was numbness and tingling of the fingers and his hands and fingers felt swollen. He also had generalized itching at times. He slept poorly and awoke early in the morning. There had been a lumbosacral backache at times during the last four years.

PHYSICAL.-

Height was 71 inches, and weight in the nude was 151 pounds. The patient had been overweight as a child. The heart was normal; the pulse rate was 85 while he was lying down, and the blood pressure was 135 systolic and 80 diastolic. The genitals were normal, and the prostate was only slightly increased in size and consistency. The physical otherwise was normal.

LABORATORY.-

Hemoglobin, red and white cells and the differential count were all within normal limits. Urinalysis was negative. The nonprotein nitrogen in the blood was 35 mgm. and the fasting value of the blood sugar was 85 mgm. per hundred cubic centimeters. The Kahn test was negative, the cholesterol level of the blood was 160 mgm. per hundred cubic centimeters, and the B.M.R. was \nearrow 10 per cent.

TREATMENT.-

The patient was given intramuscular injections of 25 mgm. testosterone propionate every other day, plus a mild sedative four times a day. At the present time he has been treated for two months and has improved. He will need more treatment.

This case history was one presented by Werner (28) in his latest article.

SUMMARY

There are already many repetitions within the article. Consequently there is no necessity to try to summarize all theories, arguments, claims and counter claims already mentioned. This summary contains only the conclusions of this writer after evaluating all literature reviewed.

It seems apparent to me that men with few exceptions do not pass through a period in life that is the exact counterpart of the menopause of women. However it would seem that most men pass through a more gradual decline of testicular function usually between the ages of 48 to 52 years. In only a few men does this cause signs and symptoms of enough significance to concern the man or his doctor. In those individuals who develop clinical manifestations, the latter are of a result of a two way mechanism; with the decline of testicular function there is a decrease secretion of androgens and probably a compensatory increase of gonadotrophic hormone from the pituitary resulting in an endocrine - autonomic upset which causes a vasomotor disturbance; the decline in the function of the sex glands causes a decrease of libido and potency which together with vasomotor symptom can cause a psychic upset which results in the depressive nervous symptoms of the syndrome.

Symptoms of the male climacteric can be divided into circulatory (hot flashes, profuse perspiration, tachycardia, palpitation, dyspnea, etc.) nervous (nervousness, irritability, paraesthesia, melancholia, etc.) and general (lassituded, vague pains, decrease potency, urinary and prostatic signs, etc.). The five most frequent symptoms observed are nervousness, and tension, loss and decrease of potency, depression and mild melancholia, decrease of powers of memory and concentration, and lassitude and fatigability.

The diagnosis of the syndrome is made chiefly from signs and symptoms and is sometimes aided by a study of the levels of gonadotrophic and androgen hormones in the urine. The important feature of such a diagnosis is the careful elimination of other possible causes of the symptoms.

There is almost unanimous agreement that the treatment of the male climacteric once diagnosed is intramuscular injections of testosterone propionate in oil in dosages totaling 50-100 mgm. weekly. Dramatic results in relief of symptoms from this therapy are the rule. Oral and topical use of the same synthetic androgens can bring results but are less effective for they both require ex-

pensively large doses. In the future subcutaneous implantation of testosterone pellets may replace the intramuscular therapy. Testosterone medication is only a replacement therapy and may have to be continued for long periods.

As can be said of many subjects in medicine, there is much to be learned about the various aspects of the male climacteric.

BIBLIOGRAPHY

1. Abarbanel, A.R.: The Male Climacteric: J.A.M.A.: (Feb. 17, 1945)
2. Ayres, J.C.: Change of Life - Male and Female: Memphis Med. J.: p. 157, (Aug. 1942)
3. Bauer, Julius: Constitution and Disease: Applied Constitutional Pathology: (New York, Grune & Stratton): p. 83, (1942)
4. Bauer, Julius: The Male Climacteric: J.A.M.A., (Dec. 2, 1944)
5. Boswell, Clarence H.: The male Climacteric: Illinois Med. J.: p. 280, (Oct. 1942)
6. Dingemanse, Eliz.: Borchart, Helene: and Laquer, E.: Capon Growing Substances in Human Urine of Male and Female of Various Ages: Biochem. J.: 31: p. 500, (April 1937)
7. Douglas, Robert J.: The Male Climacteric: Its Diagnosis and Treatment: J. Urol.: 45: 404-410, (1940)
8. Dunn, Charles W.: Male Hormone Therapy at Male Climacteric and Gonadal insufficiency State: Delaware State Med. J.: 11: 76-84, (May 1939)
9. Editorial: Climacteric in Aging Men: J.A.M.A.: p. 458, (Feb. 7, 1942)
10. Eidelsberg, Jos: The Male Sex Hormone: M.Clin. of No. Amer.: 22: 1537-1544, (Sept. 1938)
11. Fraser, R.W.; Forbes, Anne P.; Albright, Fuller; Sulkowitch, Hirsch; and Reifenstein, E.C., Jr.: Colorimetric Assay of 17-Ketosteroids in Urine: A Survey of the Use of this Test in Endocrine Investigation, Diagnosis and Therapy: J. Clin. Endocrinology: 1: 234, (March 1941)
12. Greene, R.R.; Burrill, M.W.; Oppenheimer, E.; Nelson, D.; (Northwestern University - Physiology and Pharmacology): Conditions Modifying Effectiveness of Testosterone: J. Of Urol.; p. 735, (May 1942)

13. Heller, Carl G. and Myer, G.B.: The Male Climacteric: J.A.M.A., (Oct. 21, 1944)
14. Heller, E.J. and Sevringhaus, C.G.: Gonadotrophic Hormone: Endocrinology: 29 #1, (July, 1941)
15. Hess, J.H.; Kunstadter, R.H.; and Saphir, Wm.: Urinary Excretion of Gonadotrophic Hormone in Cryptorchism: J.A.M.A.: 108: p. 352, (Jan. 30, 1937)
16. Howard, John E. and Vest, S.A., Jr: Clinical Experiments with Male Sex Hormone: Amer. J. Med. Sc.: 198: 823-237, (1939)
17. Howard, J.E. and Vest, S.A., Jr.: A Method of Implantation of Crystalline Testosterone: J.A.M.A.: 113: 1869-1872, (Nov. 18, 1939)
18. Ketcham, W. Merritt: The Male Sex Hormone: J. Missouri State M. A.: 36: 427-430, (Nov. 1939)
19. Lake, George B.: Endocrines and the General Practitioner: Illinois Med. J.: 77: 162-165, (Feb. 1940)
20. LaMar, Carlos P.: Clin. Endocrinology of the Male With Special Reference To the Male Climacteric: J. Florida Med. Ass., (Feb. 1940)
21. McCullagh, E. Perry: Treatment of Testicular Deficiency with Testosterone Propionate: J.A.M.A.: 112 #1, (March 18, 1939)
22. Thompson, W.O.: Uses and Abuses of Male Sex Hormone: J.A.M.A.: 132 #4: p. 185, (Sept. 28, 1946)
23. Werner, A.A.: Male Climacteric: J.A.M.A.: 112: p. 1441, (April 15, 1939)
24. Werner, A.A.: The Male Climacteric: Merck Report: (Oct. 1939)
25. Werner, A.A.: Male Climacteric: Additional Observation of 37 Patients: J. Urol.: 49: 872-882, (June, 1943)
26. Werner, August A.: Male Climacteric: J. Missouri State Med. Assn.: (Sept, 1943)

27. Werner, A.A.: Male Climacteric: Observation of 54 Patients: J.A.M.A.: (March, 24, 1945)
28. Werner, A.A.: The Male Climacteric: Report of 273 Patients: 132 #4: p. 188, (Sept. 28, 1946).