

1987

Lanier Brugh, Inc v. Bernice Steward : Brief of Appellant

Utah Court of Appeals

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IN THE COURT OF APPEALS OF THE STATE OF UTAH

LANIER BRUGH, INC., AND/OR	:	
WORKERS' COMPENSATION FUND	:	Court of Appeals
OF UTAH,	:	#870418CA
	:	
Defendants/Appellants,	:	Industrial Commission
	:	#86000198
vs.	:	
	:	Administrative Law Judge:
BERNICE STEWARD, Widow of	:	Judge Richard G. Sumsion
DALE STEWARD, Deceased, and	:	
INDUSTRIAL COMMISSION OF UTAH,	:	Argument Priority #6
	:	
Applicant/Respondents.	:	APPEAL

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TABLE OF CONTENTS

	<u>Page</u>
STATEMENT OF ISSUE PRESENTED FOR REVIEW	1
DETERMINATIVE STATUTES	2
STATEMENT OF THE CASE	2
A. NATURE OF THE CASE, COURSE OF PROCEEDINGS, AND DISPOSITION BY THE INDUSTRIAL COMMISSION	2
B. STATEMENT OF FACTS	3
SUMMARY OF ARGUMENT	4
ARGUMENT	5
 POINT I: IN ACCORDANCE WITH THE "ALLEN TEST," BECAUSE THE DECEDENT HAD A PRE-EXISTING CONDITION WHICH CONTRIBUTED TO HIS INJURY, AND HE DID NOT PUT FORTH UNUSUAL OR EXTRAORDINARY EXERTION, DECEDENT'S EMPLOYMENT WAS NOT THE LEGAL CAUSE OF HIS DEATH, AND THEREFORE THE ACCIDENT IS NOT COMPENSABLE	
	5
 POINT II: THE INDUSTRIAL COMMISSION ERRED IN CONCLUDING THAT THERE WAS SUFFICIENT EVIDENCE TO SUPPORT A FINDING THAT THE STRESS OR EXERTION REQUIRED BY DECEDENT'S JOB WAS THE MEDICAL CAUSE OF HIS DEATH.	
 ACCORDINGLY, BECAUSE THERE IS NO MEDICALLY DEMONSTABLE CAUSAL LINK BETWEEN THE WORK- RELATED EXERTIONS AND THE DEATH, THE ACCIDENT IS NOT COMPENSABLE	
	11
 POINT III: THE MEDICAL CAUSATION ISSUE SHOULD PROPERLY BE REFERRED TO A MEDICAL PANEL	
	22
 POINT IV: THE INDUSTRIAL COMMISSION FAILED IN ITS STATUTORY DUTY TO . . . "REVIEW THE ENTIRE RECORD . . . " AS IS REQUIRED BY §35-1-82.54 (EFFECTIVE THROUGH DECEMBER 31, 1987)	
	24
CONCLUSION	24

APPENDICES	27
APPENDIX I:	
UTAH CODE ANNOTATED	
\$35-1-45 COMPENSATION FOR INDUSTRIAL ACCIDENTS TO BE PAID	28
\$35-1-77 MEDICAL PANEL-DISCRETIONARY AUTHORITY OF COMMISSION TO REFER CASE - FINDINGS AND REPORTS - OBJECTIONS TO REPORT HEARING EXPENSES	29
\$35-1-82.54 REVIEW OF CASES AND ORDERS BY COMMISSION-PROCEDURES-EFFECT OF AWARD	30
\$35-1-83 REVIEW BY COURT OF APPEALS	31
APPENDIX II:	
FINDINGS OF FACT, CONCLUSIONS OF LAW AND ORDER MAY 28, 1987	32
APPENDIX III:	
ORDER DENYING MOTION FOR REVIEW AUGUST 28, 1987	41
APPENDIX IV:	
WORKERS' COMPENSATION RULES AND REGULATIONS GUIDELINES FOR UTILIZATION OF MEDICAL PANEL	47
APPENDIX V:	
1986 PHYSICIANS DESK REFERENCE PRODUCT INFORMATION	50
APPENDIX VI:	
EVALUATION BY DR. JOSEPH PERRY JULY 29, 1986	56

TABLE OF AUTHORITIES

	<u>Page</u>
<u>Allen v. Industrial Commission</u> , 729 P2d 15 (Utah 1986)5, 6, 8 9, 11, 12 24
<u>Bryant v. Masters Machine Co.</u> , 44 A.2d 329, 337 (Me. 1982)	6
<u>Champion Home Builders v. Industrial Commission of Utah</u> , 703 P2d 306, 308 (Utah 1985)	22, 23
<u>Holloway v. Industrial Commission</u> , 729 P2d 31, 32 (Utah 1986)	9, 11
<u>Hone v. J.F. Shea Company</u> , 728 P.2d 1008 (Utah, 1986)	11, 23
<u>Kaiser Steel Corporation v. Monfredi</u> , 631 P.2d 888 (Utah 1981)	11
<u>Lancaster v. Gilbert Development</u> , 736 P2d 237 (Utah 1987)	15, 16
<u>Ocean Accident & Guarantee Corporation v. Industrial Commission</u> , 245 P. 343, 345-346 (1926)	19
<u>Prestige Homes, Inc. v. Legouffe</u> , 658 P.2d 850 (Colo. 1983)	20, 21
<u>Price River Coal v. Industrial Commission</u> , 731 P.2d 1080 (Utah, 1981)	11
<u>Ross v. Gardner</u> , 365 F.2d 554 (6th Cir. 1966)	21
<u>Sayers v. Gardner</u> , 380 F.2d 940 (6th Cir. 1967)	21
<u>Spencer v. Industrial Commission</u> , 20 P2d 618 (Utah 1933).	18
<u>Tintic Standard Mining Company v. Industrial Commission</u> , 110 P2d 367, 369 (Utah 1941)	20

OTHER AUTHORITIES CITED

	<u>Page</u>
Utah Code Annotated, Section 35-1-45	2, 4, 5 6, 7
Utah Code Annotated, Section 35-1-77	2, 23
Utah Code Annotated, Section 35-1-82.54	2, 24
Utah Code Annotated, Section 35-1-83	2
Workers' Compensation Rules & Regulations, Rule 1.2.33	2, 22

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STATEMENT OF ISSUE PRESENTED FOR REVIEW

1. Was the Industrial Commission correct in concluding that the decedent's death was the result of an injury by accident arising out of or during the course of his employment; or was decedent's death the result of a non-compensable heart attack that occurred as the result of an underlying heart condition and not because of his employment?

More specifically, the issue may be stated as follows: Was there substantial competent evidence in the record to warrant a finding of both legal causation and medical causation linking decedent's death with activities of his employment?

DETERMINATIVE STATUTES

Section 35-1-45 COMPENSATION FOR INDUSTRIAL ACCIDENTS TO BE PAID

Section 35-1-77 MEDICAL PANEL-DISCRETIONARY AUTHORITY OF COMMISSION TO REFER CASE - FINDINGS AND REPORTS - OBJECTIONS TO REPORT HEARING EXPENSES

Section 35-1-82.54 REVIEW OF CASES AND ORDERS BY COMMISSION-PROCEDURES - EFFECT OF AWARD

Section 35-1-83 REVIEW BY COURT OF APPEALS
WORKERS' COMPENSATION RULES & REGULATIONS, RULE 1.2.33

(See Appendix I for Text of Statutes)

STATEMENT OF THE CASE

A. Nature of the Case, Course of Proceedings, and Disposition by the Industrial Commission.

This case arises under the Utah Workers' Compensation Act, and the Utah Court of Appeals has jurisdiction over this matter pursuant to §78-2a-3, Utah Code Annotated (1953, as amended). The applicant, Bernice Steward, widow of Dale W. Steward, filed a Claim for Dependent's Benefits and/or Burial Benefits on February 17, 1986. (R 2). An evidentiary hearing was held on November 5, 1986 (R 19). On May 28, 1987 an Order was entered by Administrative Law Judge Richard G. Sumsion entitling Bernice Steward to dependent's benefits. (R 268) Said Order was based upon a finding by the Administrative Law Judge that the applicant had met her burden of proof in establishing both legal and medical causation and that the decedent's death was precipitated by the conditions of his employment.

On June 12, 1987 appellants - Workers' Compensation Fund of Utah moved for review of the order. (R 276) The Industrial Commission denied the Motion for Review, and adopted the Findings of Fact and Conclusions of Law of the Administrative Law Judge. (R 286) (See orders attached as Appendices II and III).

B. STATEMENT OF FACTS

1. On November 11, 1985 Dale W. Steward, the decedent, was employed as a truck driver for Lanier Brugh Corporation. Lanier Brugh is a contract carrier with the United States Postal Service for delivery of mail between various locations. (R 30-32)

2. Mr. Steward's job entailed driving a truck between Salt Lake City and Denver. On November 11, 1985 Steward departed from Salt Lake City at approximately 5:30 A.M. (R 27, 31)

3. Mr. Steward drove through a snow storm on the way to Denver, and arrived at approximately 5:30 P.M. on November 11, 1985. (R 44, 49)

4. Mr. Steward commenced his return trip to Salt Lake City at approximately 11:15 P.M. on November 11, 1985. (R 86)

5. Approximately fifteen to thirty minutes after Mr. Steward had departed Denver for Salt Lake City, his truck was observed moving over two lanes of traffic to his left. The truck then hit the median and lodged on top of it. (R 104) The accident report indicates that Mr. Steward died of a heart attack prior to the truck coming to rest on the median. (R 99)

6. Mr. Steward's body was transported to Humana Hospital where it was determined that the cause of death was "apparent acute myocardial infarction." (R 3)

7. At the time of the accident the road was dry and there were no adverse weather conditions. (R 99-100)

8. At the time of the accident, Mr. Steward had been taking

dexedrine or dextroamphetamine because he suffered from narcolepsy. (R 38, 46, 111-112)

9. At the date of his death, decedent was 56 years old. (R 55)

10. Mr. Steward had a 36 year smoking history in which he would smoke approximately one and one half packs of cigarettes a day. (R 55)

11. Mr. Steward was diagnosed as having emphysema. (R 55, 125)

12. Mr. Steward was, at the time of his death, approximately five feet nine inches tall, and about 200 pounds.

SUMMARY OF ARGUMENT

Section 35-1-45, Utah Code Annotated (1953, as amended) provides for an award of benefits to the dependents of an employee who is killed "by accident arising out of or in the course of his employment" In order for such an accident to be compensable, the claimant must show that the decedent's employment was both the legal cause and the medical cause of his death.

Because decedent had several risk factors for heart disease (a pre-existing condition), to show legal causation the claimant must prove that decedent put forth unusual or extraordinary exertion in carrying out his employment duties. Inasmuch as decedent did not put forth any such unusual exertion, decedent's employment did not contribute anything substantial to increase the risk he already faced in non-employment life. Accordingly,

decedent's employment was not the legal cause of his death, and the claim is not compensable.

The Industrial Commission also erred in concluding that decedent's death was medically caused by his employment. There is no competent evidence in the record to support a finding of a medically demonstrable causal link between any work related exertion and decedent's death. Therefore, the Commission's findings regarding this issue are arbitrary and capricious, and subject to reversal.

Further, in determining that medical causation existed, the Administrative Law Judge improperly took judicial notice of, and relied upon medical literature which was not introduced into evidence at the hearing thereby precluding defendants from providing countering evidence and authority. The administrative law judge also failed to refer the case to a medical panel contrary to the Commission's own rules. (See Workers' Compensation Rules and Regulations attached as Appendix IV.) The Commission failed to review all of the evidence and record as the transcript of the hearing was not prepared until after the Petition for Writ of Review was served.

ARGUMENT

POINT I

IN ACCORDANCE WITH THE "ALLEN TEST," BECAUSE THE DECEDENT HAD A PRE-EXISTING CONDITION WHICH CONTRIBUTED TO HIS INJURY, AND HE DID NOT PUT FORTH UNUSUAL OR EXTRAORDINARY EXERTION, DECEDENT'S EMPLOYMENT WAS NOT THE LEGAL CAUSE OF HIS DEATH, AND THEREFORE THE ACCIDENT IS NOT COMPENSABLE.

Section 35-1-45, Utah Code Annotated (1953, as amended) provides for an award of benefits to the dependents of an employee who is killed "by [an] accident arising out of or in the course of his employment. . . ." While the Workers' Compensation Act does not define this phrase, the recent case of Allen v. Industrial Commission, 729 P2d 15 (Utah 1986) sets forth the analysis to be followed in accident cases involving internal failures. In Allen, the sole issue on appeal was whether the claimant who had suffered pre-existing back problems and was injured as the result of exertion usual and typical for his job, was injured "by accident arising out of or in the course of employment." In addressing this issue, the Utah Supreme Court set forth as follows:

This statute creates two prerequisites for a finding of a compensable injury. First, the injury must be "by accident." Second, the language "arising out of or in the course of employment" requires that there be a causal connection between the injury and the employment.

* * *

The language "arising out of or in the course of his employment" found in U.C.A., 1953, Section 35-1-45 (Supp. 1986), was apparently intended to ensure that compensation is only awarded where there is a sufficient causal connection between the disability and the working conditions. The causation requirement makes it necessary to distinguish those injuries which (a) coincidentally occur at work because a preexisting condition results in symptoms which appear during work hours without any enhancement from the work place, and (b) those injuries which occur because some condition or exertion required by the employment increases the risk of injury which the worker normally faces in his everyday

life. See Bryant v. Masters Machine Co., 44 A.2d 329, 337 (Me. 1982). Only the latter type of injury is compensable under U.C.A., 1953, Section 35-1-45. There is no fixed formula by which the causation issue may be resolved, and the issue must be determined on the facts of each case.

Professor Larson has suggested a two-part causation test which is consistent with the purpose of our workers' compensation laws and helpful in determining causation. We therefore adopt that test. Larson suggests that compensable injuries can best be identified by first considering the legal cause of the injury and then its medical cause. Larson, *supra* Section 38.83 (a), at 7-273. "Under the legal test, the law must define what kind of exertion satisfies the test of 'arising out of the employment'...[then] the doctors must say whether the exertion (having been held legally sufficient to support compensation) in fact caused this [injury]." Larson, *supra* Section 38.83 (a), at -276 to -277.

1. Legal Cause - Whether an injury arose out of or in the course of employment is difficult to determine where the employee brings to the work place a personal element of risk such as preexisting condition.

* * *

To meet the legal causation requirement, a claimant with a preexisting condition must show that the employment contributed something substantial to increase the risk he already faced in everyday life because of his condition. This additional element of risk in the work place is usually supplied by an exertion greater than that undertaken in normal, everyday life. This extra exertion serves to offset the preexisting condition of the employee as a likely cause of the injury thereby eliminating claims for impairments resulting from a personal risk rather than exertions at work. Larson, supra, Section 38.83 (b), at 2-278. Larson summarized how the legal cause rule would work in practice as follows:

If there is some personal causal contribution in the form of a [preexisting condition], the employment contribution must take the form of an exertion greater than that of nonemployment life....

If there is no personal causal contribution, that is, if there is no prior weakness or disease, any exertion connected with the employment and causally connected with the [injury] as a matter of medical fact is adequate to satisfy the legal test of causation.

Id. Thus, where the claimant suffers from a preexisting condition which contributes to the injury, an unusual or extraordinary exertion is required to prove legal causation. Where there is no preexisting condition, a usual or ordinary exertion is sufficient.

729 P2d at 18, 24-26

Regarding the legal causation requirement, it is appellant's contention that the Administrative Law Judge misapplied the Allen legal causation standard. In his Findings of Fact, Conclusions of Law, and Order, the Administrative Law Judge used the lower standard of the Allen test which assumes that usual or ordinary exertion is sufficient to show legal causation when there is no pre-existing condition.

The Judge stated, "In Mr. Steward's case, there is no evidence of any previously diagnosed heart condition." (R. 270)

Appellant's take exception to the utilization of this lower legal causation standard, and contend that inasmuch as the decedent had several risk factors for heart disease, the higher standard of causation as outlined in Allen was the appropriate test to use. The risk factors included a 36 year smoking habit in which decedent smoked one and one-half packs of cigarettes a day,

high levels of amphetamine use prescribed for narcolepsy, emphysema, and obesity. (R.55, 94, 125) In addition, decedent at the time of his death, was 56 years of age. Further, prior to his death, the decedent had demonstrated other suspicious symptoms of heart problems. In fact, Dr. M. Peter Heilbrun indicated on November 18, 1985 that, "In retrospect, it makes me wonder if some of [Dale Steward's] recent neck and shoulder pain was possibly myocardial in origin." (R. 135)

It should also be noted that the pre-existing condition of which Allen speaks need not be patent nor "previously diagnosed." See Justice Zimmerman's concurring opinion in Holloway v. Industrial Commission, 729 P2d 31, 32 (Utah 1986) wherein it was stated:

With respect to the focus of this case on remand-whether Holloway had a preexisting condition-I would observe that the preexisting condition of which Allen speaks need not be patent; in fact, it need not have been known or knowable to anyone before the injury. The sole question is whether the worker came to the work place with a condition that increased his risk of injury. If he did and that condition contributed to the injury, then Allen's higher standard of legal causation comes into play so as to place that worker on the same footing as one who did not come to work with a preexisting condition.

It is appellants contention that the decedent came to the workplace with pre-existing conditions (heart disease and narcolepsy treated by amphetamines),. Therefore, before legal causation can be established there must be a finding that the employment activities of decedent involved exertion in excess of the normally expected level of nonemployment activity. Decedent

was diagnosed as having narcolepsy and he had a predisposition to fatal arrhythmia due to his amphetamine consumption to treat his narcolepsy. (R 111 and 288 paragraphs 4 & 5) That is, decedent at the time of his death, was taking dexedrine and ritalin for treatment of his narcolepsy. (R 130, 132, 133). The use of these prescribed drugs makes one more susceptible to suffering a fatal arrhythmia. (See Physician's Desk Reference attached as Appendix V). Accordingly, because decedent was predisposed to suffering a fatal arrhythmia, he had a pre-existing condition. The higher standard requiring unusual exertion should have been utilized.

In the Order Denying Motion for Review, the Industrial Commission states that even if the unusual exertion standard was applied, the facts relate exertion and/or stress that goes beyond what the average person encounters in everyday nonemployment life; specifically, that decedent had to drive on snow covered roads, he had little rest in between the trip to Denver and the return trip to Salt Lake City, and that decedent had been taking amphetamines. (R. 287). Appellants refute this contention and maintain that the facts do not support a finding that decedent put forth an exertion greater than that of nonemployment life.

In this case the decedent had not undergone any unusual or extraordinary exertion in the time frame immediately preceding his fatal heart attack. At the time of his heart attack, the decedent was just outside of Denver, Colorado. (R 86). The road conditions were clear and dry. (R 99-100. See also photographs of accident scene at R 101). Further, decedent had been driving only

15 to 30 minutes on his return trip to Salt Lake City after a five hour and forty-five minute layover in Denver when he suffered the heart attack. (R 44, 49, 86)

Decedent's employment did not contribute anything substantial to increase the risk he already faced in everyday life. Rather, the heart attack coincidentally occurred during work hours without any enhancement from the work place or conditions surrounding his work place. Accordingly, because decedent's heart attack was not legally caused by his employment, the "accident" herein did not "arise out of or in the course of employment," and is therefore not compensable.

Moreover, because the Commission's findings are not supported by the facts in the record, the finding is arbitrary and capricious and subject to reversal. Kaiser Steel Corporation v. Monfredi, 631 P.2d 888 (Utah 1981). For other recent Utah cases which are consistent with Allen, and supportive of appellants position, see the following: Holloway v. Industrial Commission, 729 P.2d 31 (Utah 1986); Price River Coal v. Industrial Commission, 731 P.2d 1080 (Utah, 1981); Hone v. J.F. Shea Company, 728 P.2d 1008 (Utah, 1986).

POINT II

THE INDUSTRIAL COMMISSION ERRED IN CONCLUDING THAT THERE WAS SUFFICIENT EVIDENCE TO SUPPORT A FINDING THAT THE STRESS OR EXERTION REQUIRED BY DECEDENT'S JOB WAS THE MEDICAL CAUSE OF HIS DEATH.

ACCORDINGLY, BECAUSE THERE IS NO MEDICALLY DEMONSTRABLE CAUSAL LINK BETWEEN THE WORK-RELATED EXERTIONS AND THE DEATH, THE ACCIDENT IS NOT COMPENSABLE.

The Industrial Commission concluded that there was a medically demonstrable causal link between decedent's work-related exertions and his death. Inasmuch as this conclusion is not supported by substantial medical evidence, appellant asserts that the finding of medical causation is arbitrary and capricious.

As with the issue of legal causation, Allen is an appropriate place to begin a discussion concerning medical causation. The Utah Supreme Court set forth the following:

[10] 2. Medical Cause-The second part of Larson's dual-causation test requires that the claimant prove the disability is medically the result of an exertion or injury that occurred during a work-related activity. The purpose of the medical cause test is to ensure that there is a medically demonstrable causal link between the work related exertions and the unexpected injuries that resulted from those strains. The medical causal requirement will prevent an employer from becoming a general insurer of his employees and discourage fraudulent claims. With the issue being one primarily of causation, the importance of the medical panel becomes manifest. It is through the expertise of the medical panel that the Commission should be able to make the determination of whether the injury sustained by a claimant is causally connected or contributed to by the claimant's employment.

Schmidt, 617 P.2d at 697 (Wilkins, J., concurring). Under the medical cause test, the claimant must show by evidence, opinion, or otherwise that the stress, strain, or exertion required by his or her occupation led to the resulting injury or disability. In the event the claimant cannot show a medical causal connection, compensation should be denied. (Emphasis added)

729 P.2d at 27.

Thus, in accordance with Allen, in order for an accident to be compensable, it must be determined that the injury was medically caused by the stress, strain, or exertion required by ones employment.

In the proceedings below, the Administrative Law Judge held- and the Industrial Commission affirmed-that medical causation was established because the extra stress the decedent faced during the 24 hours preceding his death most likely led to the arrhythmia which resulted in the fatal heart attack.

The extra stress factors cited by the Administrative Law Judge included:

1. Anxiety caused by the late arrival of decedent's truck thereby delaying his departure from Salt Lake City.
2. Fatigue from having to drive on slippery roads and during a snow storm, both to and from Denver.
3. Fatigue from inadequate rest prior to his departure from Denver.
4. The use of amphetamines, probably in greater amounts than usual because of the lack of adequate rest. (R. 270)

It should be noted at this juncture that two of the above referenced stress factors are not supported by the factual record in this case. Specifically, there is no competent evidence in the record that the decedent was driving on a slippery road "both to and from Denver." Road conditions at the time of the decedent's return trip from Denver were, as noted earlier, clear and dry. Also, the decedent's use of amphetamines was not due to "lack of

adequate rest." Rather, the decedent had a long-standing condition of narcolepsy which was treated through prescription use of high doses of amphetamines. The Judge's statement that the use of amphetamines was increased due to the decedent's level of fatigue as a result of his truck driving just prior to the time of his heart attack was not argued at the hearing and assumes facts not supported by the record. It is appellants' belief that the record is void of any substantial evidence which would support a finding of medical causation.

In concluding that the medical causation standard was met, the Administrative Law Judge quoted and adopted the entire report of Dr. Perry. (R 270-272) Interestingly, Dr. Perry indicated that the decedent's heart attack was idiopathic. (R 111-112). However, the Administrative Law Judge found that the four factors outlined above precipitated the decedent's fatal heart attack.

Appellant's aver that the Administrative Law Judge's opinion that the noted factors were the medical cause of the decedent's heart attack oversteps the bounds of a fact finder and a judge charged to rule in matters of law. A lay person is not qualified to propound medical opinion. In this matter, the Administrative Law Judge was not weighing and choosing between competing medical opinions in the record. The judge, though stating he relied on the report of Dr. Perry, in reality disregarded the key points.

I think trauma can be excluded with a
reasonable degree of confidence in this
case . . .

* * * *

In terms of medical probability it is most likely that he experienced a fatal cardiac arrhythmia while driving, lost consciousness a few seconds later thus losing control of the vehicle and having the accident as reported. It is possible that his dextroamphetamine was related to his death because it may worsen arrhythmias in (sic) susceptible individuals.

An autopsy would have been supporting this diagnosis, but likely would not have confirmed it with an absolute degree of certainty. I suspect it would have shown amphetamines present and coronary artery disease present. At this point in time (sic) exhuming the body would not shed any light on the presence of amphetamine. It would, however, document the presence or absence of coronary artery disease. In the absence of drugs it is extremely unusual for cardiac arrest to occur in a person with normal coronary arteries. . . .

* * * *

In summary in terms of reasonable medical probability, the patient suffered a fatal arrhythmia while driving and the accident was simply the result of his death and subsequent loss of control of the vehicle. While other possibilities exist, they are far less likely. To (sic) exume and perform a post-mortum examination of the body would later those probabilities to an extent, but it is highly unlikely it would provide definite answer.

(R 110 and 111 entire report attached hereto as Appendix V hereto.)

Instead of following Dr. Perry's considered opinion, the Judge rendered his own medical opinion and this is reversible error.

The medical causation standard was further discussed in the context of a heart attack case in Lancaster v. Gilbert Development, 736 P2d 237 (Utah 1987). There, the Utah Supreme Court quoted Dr. J. Joseph Perry, the same cardiologist involved in the present case. The Supreme Court noted:

Dr. Perry, the chairman of the medical panel and a cardiologist, testified that it was "likely" that the conditions under which Mr. Lancaster was working aggravated his pre-existing heart condition. However, Dr. Perry also was less than certain about the causal connection between the work conditions and the myocardial infarction. In his report to the Administrative Law Judge, Dr. Perry identified and ranked the role of various risk factors, including those associated with work, in precipitating the claimant's myocardial infarction. When asked to quantify the contribution of pre-existing risk factors and work factors to the claimant's myocardial infarction, Dr. Perry assigned a value of 90 percent to pre-existing conditions and 10 percent to work conditions. Dr. Perry explained, however, that his assessment of the factors was "a fairly random guess."

736 P2d at 240.

Dr. Perry undertook the same type of analysis in this case as he did in Lancaster. He briefly discussed several possible, but not entirely probable, medical causes of decedent's heart attack. Then, Dr. Perry concluded that the heart attack was idiopathic in origin and not in all probability, caused by the alleged industrial events.

In Lancaster, the Utah Supreme Court further stated:

Thus, although there may have been some connection between the heart attack and the cold weather and high altitude [the industrial factors], the evidence of any such connection is inconclusive. Not one of the doctors was willing to state with medical certainty that the claimant's injury was caused by work-related factors. Thus, there is competent and comprehensive medical evidence in the record upon which the Administrative Law Judge could rely in concluding that medical causation was lacking. Although the medical evidence was conflicting, it is the responsibility of the

Administrative Law Judge to resolve factual conflicts.

736 P2d at 240-241.

In the case at bar, there is no competent evidence which shows a medical causal link between the alleged industrial events and the decedent's heart attack. The only evidence that the claimant could provide was the ex post facto statement of Dr. Heilbrun that because the decedent,

[H]ad no prior history of cardiac disease,
...his death should be considered to be an
industrial-related cardiac event. (R. 245)

It is noteworthy that this letter of Dr. Heilbrun appears to be a statement prepared at the widow's request and suggestion. In this light, Dr. Heilbrun wrote yet another letter to the claimant on the same date, July 25, 1986, and stated,

I am enclosing the following letter. Please read through it and advise me if you feel it satisfactorily explains my thoughts on the industrially related nature of Dales heart attack. (R 244)

Further, Dr. Heilbrun's July 25, 1986 causation letter appears to contradict his prior statement of November 18, 1985 wherein he postulated that the decedent's neck and shoulder pain just prior to his death was "possibly myocardial in origin."

(R. 135) The foundation for Dr. Heilbrun's view of causation is lacking because the preexisting narcolepsy treatment with amphetamines makes one more susceptible to the arrhythmias that took Mr. Steward's life. (See Physicians Desk Reference at Appendix V.)

Moreover, to substantiate his conclusion, at least in part, the Administrative Law Judge discussed certain copyrighted medical literature which was not fully quoted. (R 272) Apparently, none of the physicians in this case used or relied upon this literature. However, the Administrative Law Judge consulted the same for his own reference during his decision-making process. Appellants contend that such literature cannot be used in ruling on this matter. Appellants contention is supported by the following line of case law.

Spencer v. Industrial Commission, 20 P2d 618 (Utah 1933) contains language which is supportive of appellant's point of view.

We recognize that the Industrial Commission is not a court and is not bound by the usual common law or statutory rules of evidence or by the technical or formal rules of procedure...Yet, it is fundamental that in investigations such as the Industrial Commission is authorized to make, any party to a cause or proceeding is entitled to be advised of and afforded an opportunity to meet such evidence as the commission may consider and rely on in the making of it's findings and decision. Unless such evidence is brought into the case, and in some lawful manner made a part of the record, it cannot be regarded as competent evidence, and must be excluded in determining the sufficiency of the evidence to support the findings of the Industrial Commission.

20 P2d at 620

In the case at bar, certain copyrighted medical literature which was not presented at the hearing was considered and relied upon in determining that medical causation was established. In

accordance with Spencer, this medical literature is not competent evidence and should not have been considered.

The Workers' Compensation Fund was entitled to be advised of and afforded an opportunity to meet and refute any evidence which the Administrative Law Judge considered and relied upon in making his decision.

If appellant had been afforded such an opportunity, it would have introduced evidence substantiating its contention that because decedent was taking certain prescription drugs for his narcoleptic condition, he was predisposed to suffering a fatal arrhythmia and the employment was not the cause of decedents death. (See Physicians Desk Reference attached as Appendix IV). Because the Fund was not afforded this fundamental right, reversible error was committed.

See also Ocean Accident & Guarantee Corporation v. Industrial Commission, 245 P. 343, 345-346 (1926) wherein the Court stated as follows:

The Legislature, has, however, by express provision (Comp. Laws Utah 1917, Section 3149) stated that the Industrial Commission is not bound by the usual common or statutory rules of evidence or by any technical or formal rules of procedure. The commission is given the power to adopt its own rules of procedure. Rules promulgated by the commission must not, of course, deprive the parties of their constitutional right of having their day in court and of having the cause determined after an impartial hearing.

* * *

Doubtless the members of the Industrial Commission or other fact finding body not limited by the ordinary rules relating to the

introduction of testimony are at times justified in adopting "short cuts" to get at the ultimate facts in a matter under consideration. But in adopting those short cuts or new rules for determining facts care should be exercised not to deprive any party of every fair means of eliciting the facts to be finally determined or not to unnecessarily limit cross-examination of witnesses before it.

It is appellants' contention that in the case at bar, the Administrative Law Judge improperly took a "short cut" by considering evidence which appellant had no opportunity to refute. This "short cut" had the net effect of depriving appellant of its constitutional right of having its day in court and of having the cause determined after an impartial hearing.

See also Tintic Standard Mining Company v. Industrial Commission, 110 P2d 367, 369 (Utah 1941) wherein the Court set forth:

[T]he Commission should not receive evidence on disputed matters where a hearing is held after the hearing is closed, since the party adversely affected would have no opportunity to meet such evidence...

Another case which is highly supportive of the Fund's position is Prestige Homes, Inc. v. Legouffe, 658 P.2d 850 (Colo. 1983). Legouffe was concerned with a workmen's compensation claim which was filed by an employee who was seeking recovery for permanent disability suffered as the result of a heart attack allegedly precipitated by an industrial accident. The expert testimony at the hearings on the issue of causation was in conflict. The Industrial Commission denied claimant any benefits, affirming a finding that claimant had not established a causal

connection between the accident and the injury. Claimant appealed, and the Colorado Court of Appeals reversed the Commission's final order. In reversing the order, the Court of Appeals took judicial notice of certain scientific propositions found in medical treatises and concluded that one of the expert's testimony was based on an erroneous assumption of scientific fact and therefore could not serve as a basis for concluding that no causal connection had been shown.

The Colorado Supreme Court granted certiorari and addressed the issue of judicial notice. The Court concluded that the Court of Appeals' judgment should be reversed because of its erroneous application of the judicial notice rule.

The Court of Appeals relied on medical treatises not offered or admitted into evidence, and not cited by either Dr. Lissauer or Dr. Mutz, for its finding that an electric shock caused by contact with a 220 volt power line can cause serious injury without leaving a visible burn mark. The Court in effect assumed the role of an expert medical witness by discrediting the opinion of Dr. Mutz based on independent research and interpretation of medical texts which properly should be interpreted only by experts in the appropriate field. See Sayers v. Gardner, 380 F.2d 940 (6th Cir. 1967); Ross v. Gardner, 365 F.2d 554 (6th Cir. 1966). To accept the court's substitution of its own fact findings for those of the referee in this instance would expand the judicial notice rule far beyond its intended scope.

658 P.2d at 853-854

The rule concerning judicial notice, as set forth in Legouffe, is applicable to the case at hand. By relying upon certain medical literature which was not introduced as evidence at

the hearings, the Administrative Law Judge usurped the role of an expert medical witness by undertaking independent research and interpreting a medical text which he was not properly qualified to interpret. Accordingly, because it was improper for the Administrative Law Judge to take such judicial notice, and because the notice improperly taken was used to substantiate a finding of medical causation, such a finding of medical causation cannot stand.

POINT III

THE MEDICAL CAUSATION ISSUE SHOULD PROPERLY BE REFERRED TO A MEDICAL PANEL.

As noted in POINT II above, appellants contend that there is insufficient competent evidence in the record to support a finding of medical causation and therefore a reversal is warranted. In the alternative, appellants seek a referral of the medical causation issue to a medical panel. To do this, the Administrative Law Judge could pose the four risk factors noted by him, and ask an appropriate specialist(s) to discuss those factors in terms of reasonable medical probability. By doing this, the Commission would be provided with a very specific medical opinion as to medical causation. The surmise and supposition of lay people relative to this medical issue would, therefore, not be necessary. Further, the Commission's own rule requires that in these circumstances a referral to a medical panel is the mandated course. (Workers' Compensation Rules & Regulations, Rule 1.2.33; see Appendix IV).

The following line of authority is supportive of submitting this issue to a medical panel.

In Champion Home Builders v. Industrial Commission of Utah, 703 P2d 306, 308 (Utah 1985), the Supreme Court, in discussing the role of a medical panel, set forth:

Finally, Champion Home Builders argues that the Administrative Law Judge abused his discretion by not referring this case to a medical panel. Utah Code Ann. 1953, Section 35-1-77 (Supp. 1983), provides that where the employer or insurance carrier denies liability, "the commission may refer the medical aspects of the case to a medical panel appointed by the Commission..." (Emphasis added). In some cases, such as where the evidence of causal connection between the work-related event and the injury is uncertain or highly technical, failure to refer the case to a medical panel may be an abuse of discretion.

Another recent case which cites Champion Home Builders with approval is Hone v. J.F. Shea Company, 728 P2d 1008 (Utah 1986) wherein the Utah Supreme Court stated:

Although referral to the medical panel is not required by statute, we believe in this case that the findings of that panel would aid the Administrative Law Judge.

728 P2d at 1012

While it is true that Dr. Perry evaluated this case at the request of the Workers' Compensation Fund of Utah, and set forth various possible causative factors regarding decedent's death, the medical aspects of this case were not referred to a medical panel appointed by the commission. Because the evidence of casual connection between the work-related activities and decedents's

death is uncertain and highly technical, failure to refer the case to a medical panel was an abuse of discretion.

POINT IV

THE INDUSTRIAL COMMISSION FAILED IN ITS STATUTORY DUTY TO . . . "REVIEW THE ENTIRE RECORD . . . " AS IS REQUIRED BY §35-1-82.54 (EFFECTIVE THROUGH DECEMBER 31, 1987).

§35-1-82.54 (Effective Through December 31, 1987), requires the Commission to review the entire record when a matter is submitted to them pursuant to a motion for review.

The commission, upon referral of a case to it by an administrative law judge, or upon a motion being filed with it to review its own order, or an administrative law judge's supplemental order, shall review the entire record made in said case, and, in its discretion, may hold further hearings and receive further evidence, and make findings of fact and enter its award thereon. The award of the commission shall be final unless set aside by the Supreme Court as hereinafter provided. (Emphasis added.)

The Commission did not review a transcript of the hearing as it was not transcribed until October 20, 1987, (R 98), nearly a month after the Petition for Writ of Review was filed by appellants. (R 325-328). Likewise, there is not any indication in the record that the Commission reviewed the entire record in some other manner. A review of the entire record would have revealed the idiopathic nonindustrial nature of the unfortunate death of Mr. Steward.

CONCLUSION

Appellants seek a reversal of the Industrial Commission's opinion in this case because the Allen standards of legal and

medical causation were not met by the claimant herein. More specifically, inasmuch as the decedent had a preexisting heart condition complicated by his amphetamine intake for narcolpesy and he did not put forth any unusual or extraordinary exertion in the course of carrying out his job tasks, decedent's employment was not the legal cause of his death. Further, because there was insufficient competent evidence in the record indicating that the stress or exertion required by decedent's job was the medical cause of his death, the finding of medical causation was arbitrary and capricious, and must be overturned.

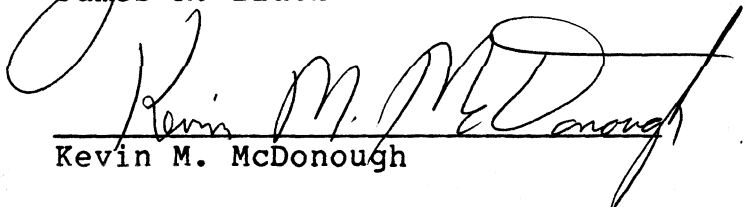
Additionally, the Commission committed reversible error in failing to refer the case to a medical panel for evalulation and also in failing to review the entire record as statutorily mandated in the administrative appellate process.

Accordingly, because the Allen standards of legal and medical causation were not met, the accident is not compensable.

DATED this 24 day of December, 1987.

BLACK & MOORE


James R. Black


Kevin M. McDonough

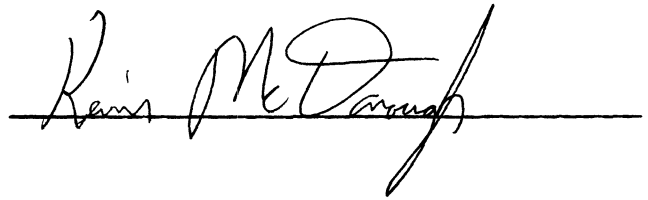
CERTIFICATE OF MAILING

I hereby certify that four (4) true and correct copies of the Brief of Appellant was mailed, postage fully prepaid, on the 24 day of December, 1987, to the following:

Thomas R. King
2121 South State, Suite 205
Salt Lake City, UT 84115
Attorney for Bernice Steward

Barbara Elicerio
Industrial Commission of Utah
160 East 300 South
P.O. Box 45580
Salt Lake City, UT 84145-0580

Attorney General's Office
236 State Capitol Bldg.
Salt Lake City, UT 84114

A handwritten signature in cursive script, appearing to read "Kevin M. Drough", is written over a horizontal line.

APPENDIX I

UTAH CODE STATUTES

35-1-45. COMPENSATION FOR INDUSTRIAL ACCIDENTS TO BE PAID.
35-1-77 MEDICAL PANEL-DISCRETIONARY AUTHORITY OF COMMISSION
TO REFER CASE - FINDINGS AND REPORTS - OBJECTIONS TO REPORT
HEARING EXPENSES.

35-1-82.54 REVIEW OF CASES AND ORDERS BY COMMISSION-
PROCEDURES - EFFECT OF AWARD

35-1-83 REVIEW BY COURT OF APPEALS

35-1-45. Compensation for industrial accidents to be paid.

Every employee mentioned in Section 35-1-43 who is injured, and the dependents of every such employee who is killed, by accident arising out of or in the course of his employment, wherever such injury occurred, if the accident was not purposely self-inflicted, shall be paid compensation for loss sustained on account of the injury or death, and such amount for medical, nurse, and hospital services and medicines, and, in case of death, such amount of funeral expenses, as provided in this chapter. The responsibility for compensation and payment of medical, nursing, and hospital services and medicines, and funeral expenses provided under this chapter shall be on the employer and its insurance carrier and not on the employee.

35-1-77. Medical panel — Discretionary authority of commission to refer case — Findings and reports — Objections to report — Hearing — Expenses.

Upon the filing of a claim for compensation for injury by accident, or for death, arising out of or in the course of employment, and where the employer or insurance carrier denies liability, the commission may refer the medical aspects of the case to a medical panel appointed by the commission and having the qualifications generally applicable to the medical panel set forth in section 35-2-56. The medical panel shall then make such study, take such X-rays

and perform such tests, including post-mortem examinations where authorized by the commission, as it may determine and thereafter make a report in writing to the commission in a form prescribed by the commission, and also make such additional findings as the commission may require. The commission shall promptly distribute full copies of the report of the panel to the applicant, the employer and the insurance carrier by registered mail with return receipt requested. Within fifteen days after such report is deposited in the United States post office, the applicant, the employer or the insurance carrier may file with the commission objections in writing thereto. If no objections are so filed within such period, the report shall be deemed admitted in evidence and the commission may base its finding and decision on the report of the panel, but shall not be bound by such report if there is other substantial conflicting evidence in the case which supports a contrary finding by the commission. If objections to such report are filed the commission may set the case for hearing to determine the facts and issues involved, and at such hearing any party so desiring may request the commission to have the chairman of the medical panel present at the hearing for examination and cross-examination. For good cause shown the commission may order other members of the panel, with or without the chairman, to be present at the hearing for examination and cross-examination. Upon such hearing the written report of the panel may be received as an exhibit but shall not be considered as evidence in the case except as far as it is sustained by the testimony admitted. The expenses of such study and report by the medical panel and of their appearance before the commission shall be paid out of the fund provided for by section 35-1-68.

**35-1-82.54. Review of cases and orders by commission —
Procedure — Effect of award [Effective until
January 1, 1988].**

The commission, upon referral of a case to it by an administrative law judge, or upon a motion being filed with it to review its own order, or an administrative law judge's supplemental order, shall review the entire record made in said case, and, in its discretion, may hold further hearings and receive further evidence, and make findings of fact and enter its award thereon. The award of the commission shall be final unless set aside by the Supreme Court as hereinafter provided.

35-1-83. Review by Court of Appeals [Effective until January 1, 1988].

Within 30 days after the commission has given notice of its award, provided a motion was previously filed in accordance with this act for review of the order or supplemental order upon which the award was based, any affected party, including the Division of Finance, may file an action in the Court of Appeals for review and determination of the lawfulness of the award.

APPENDIX II

FINDINGS OF FACT CONCLUSIONS OF LAW AND ORDER
May 28, 1987

THE INDUSTRIAL COMMISSION OF UTAH

Case No. 86000198

BERNICE STEWARD, Widow of	*	
DALE W. STEWARD, Deceased,	*	
	*	
Applicant,	*	FINDINGS OF FACT
	*	
vs.	*	CONCLUSIONS OF LAW
	*	
LANIER-BRUGH and/or	*	AND ORDER
WORKERS COMPENSATION FUND	*	
OF UTAH,	*	
	*	
Defendants.	*	
	*	
* * * * *		

HEARING: Hearing Room 334, Industrial Commission of Utah, 160 East 300 South, Salt Lake City, Utah, on November 5, 1986, at 1:00 o'clock p.m. Said hearing was pursuant to Order and Notice of the Commission.

BEFORE: Richard G. Sumsion, Administrative Law Judge.

APPEARANCES: Applicant was present and represented by Thomas R. King, Attorney at Law.

Defendants were represented by Shaun Howell, Attorney at Law.

The sole issue to be resolved by the Industrial Commission in this case is whether the decedent's death was the result of an injury by accident during the course of his employment, or whether his death was the result of a non-compensable heart attack that occurred as the result of an underlying heart condition and not because of his employment.

FINDINGS OF FACT:

The decedent's employer, Lanier-Brugh, is a contract carrier with the U. S. Postal Service for delivery of mail between various locations. The decedent's job involved driving a truck between Salt Lake City and Denver. He customarily departed Salt Lake City around 11:00 p.m. and usually arrived in Denver, Colorado, around 9:00 a.m. the following morning. He would then rest in preparation for a return trip leaving Denver around 11:00 p.m. and arriving back in Salt Lake City, the next day.

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER
PAGE TWO

On the evening of November 10, 1985, the decedent was notified that his truck would be late arriving from Nevada, because of bad weather. He was advised to be on standby and to call back periodically. Mrs. Steward testified that the anxiety caused by the truck arriving late, combined with bad weather, caused the applicant to have a very restless night. Finally, the decedent was notified the truck had arrived at 5:00 a.m. and would be ready for departure about 5:30 a.m..

Upon his arrival in Denver, Mr. Steward called his wife as she had requested. This was around 6:20 p.m.. Mrs. Steward testified her husband said the roads were bad and he was very tired but was, nevertheless, scheduled to make the return trip leaving around 11:00 p.m..

Apparently, the decedent started on the return trip to Salt Lake City around 11:00 p.m. or shortly thereafter. Enroute on the interstate highway, some fifteen to thirty minutes after his departure, the decedent's truck was observed moving from the right lane, crossing over two lanes to the left, then coming to rest on top of the cement median wall dividing the highway. The truck did not overturn and there is no indication that it came to an abrupt stop. An eye witness, traveling behind the decedent's truck at the time, stopped and tried unsuccessfully to obtain assistance. Finally, another motorist arrived. They found Mr. Steward on the passenger side of the cab with his arms hanging above his head, making it impossible for them to administer CPR. He had no pulse. The Highway Patrol was notified at 11:39 p.m. and arrived at the scene five minutes later. CPR was administered enroute to the hospital. So far as can be determined from the records, there was no indication of life, but efforts were made to revive him.

As stated above, the key issue in this case, like most internal failure cases, is whether the injury "arose out of or in the course of employment." Compensability of the applicant's claim depends on evidence showing both legal and medical causation. One-half of the test of legal causation set down in the recent case of Allen v. Industrial Commission, 46 Utah Advance Report 3 (1986), is clearly met inasmuch as the decedent's death was obviously unexpected. The test is different, however, if there is some personal causal contribution in the form of a pre-existing condition. If this is the case, the employment contribution must take the form of an exertion or stress greater than that of non-employment life. As suggested in the case cited in Footnote 8 of the Allen decision:

"The result would depend on whether there was a personal causal element in the form of a previously weakened heart. If there was not, compensation would be awarded, since the employment contributed something and the employee's personal life nothing to the cause of the collapse. If there was a previously weakened heart, compensation would be denied in spite of the medical causal contribution, because legally the personal causal contribution was substantial, while the employment added nothing to the usual wear and tear of life."

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER
PAGE THREE

There may be some question as to the definition of what is meant by a "previously weakened heart." In Mr. Steward's case, there is no evidence of any previously diagnosed heart condition. This is true of many heart attack victims. It is arguable, however, that despite the absence of any symptoms or diagnosis of a previously weakened heart, the decedent may have had coronary artery disease because it is well known that heart attacks almost never occur absent an underlying disease. Cardiac arrhythmias are sometimes an exception to this general rule.

Assuming that legal causation can be **established**, there is still a need to prove the decedent's death was medically the result of an exertion or stress that occurred as a result of his employment. As noted in the Allen case, "the purpose of the medical cause test is to insure that there is a medically demonstrable causal link between the work-related exertions and the unexpected injuries that resulted from those strains".

The Administrative Law Judge finds the decedent was subjected to the following stress factors: (1) anxiety caused by the late arrival of his truck delaying his departure from Salt Lake City, (2) fatigue from having to drive on slippery roads and during a snow storm, both to and from Denver, (3) fatigue from inadequate rest prior to his departure from Denver, and (4) the use of amphetamines, probably in greater amounts than usual because of the lack of adequate rest.

The Administrative Law Judge believes the various possible causative factors are succinctly addressed in the report of Dr. J. Joseph Perry, a cardiologist, who evaluated the case at the request of the Workers Compensation Fund of Utah. To properly understand this assessment of medical causation, the Administrative Law Judge believes it is necessary to quote rather extensively from Dr. Perry's letter of July 29, 1986. He states:

"It seems clear that Mr. Steward was not conscious when his rig swerved to the left and ran off coming to rest on top of the median wall without apparently overturning or coming to an abrupt stop as it would with a collision. Additionally on arrival in the Emergency Department there was no gross evidence of physical injury or trauma according to the emergency physician. Additionally, the photographs sent to me demonstrate very little trauma to the rig he was driving. Thus, I think trauma can be excluded with a reasonable degree of confidence in this case.

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER
PAGE FOUR

Complicating the issue is the mention of narcolepsy in the medical record. The patient had given a history of sudden sleep attacks while driving and had been treated with amphetamines by his physician for several years prior to his demise. I find no studies in the record to document narcolepsy, thus this diagnosis is not secure in this individual.

Of the two scenarios which may have occurred, that is falling asleep then suffering a cardiac arrest sometime after contacting the median wall, or having the cardiac arrest while driving, only the latter seems to have firm support. Had he suffered narcolepsy while driving, he would have awakened when he left the road (I speak from experience) and there seemed to be no event which would have been of sufficient severity to cause his death. It is remotely possible that the shock and fear of waking up in the middle to a serious accident would have been sufficient to engender the fatal cardiac arrhythmia, but this does not seem very probable.

In terms of medical probability, it is most likely that he experienced a fatal cardiac arrhythmia while driving, lost consciousness a few seconds later thus losing control of the vehicle and having the accident as reported. It is possible that his dextroamphetamine was related to his death because it may worsen arrhythmias in susceptible individuals. (Emphasis added).

An autopsy would have been supporting this diagnosis, but likely would not have confirmed it with an absolute degree of certainty. I suspect it would have shown amphetamine present and coronary artery disease present. At this point in time exhuming the body would not shed any light on the presence of amphetamine. It would, however, document the presence or absence of coronary artery disease. In the absence of drugs it is extremely unusual for cardiac arrest to occur in a person with normal coronary arteries. If the absence of coronary artery disease could be documented, then the scenario of striking the median wall, waking up and then suffering a fatal arrhythmia would become somewhat more plausible. Whether or not that has any legal significance is of course not within my area of expertise.

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER
PAGE FIVE

In summary in terms of reasonable medical probability, the patient suffered a fatal arrhythmia while driving and the accident was simply the result of his death and subsequent loss of control of the vehicle. While other possibilities exist, they are far less likely. To exhume and perform a post-mortem examination of the body would alter those probabilities to an extent, but it is highly unlikely it would provide definitive answer."

For the most part, the Administrative Law Judge adopts the foregoing opinions of Dr. Perry as his own Findings of Fact. However, the Administrative Law Judge finds that Dr. Perry's opinions do not appear to be based upon all of the facts as reflected by the record and for this reason the Administrative Law Judge differs from Dr. Perry with respect to the ultimate question of medical causation. Considering all of the evidence, the Administrative Law Judge finds the applicant has met her burden of proof in establishing both legal and medical causation and that the decedent's death was precipitated by the conditions of his employment.

The possibility exists that despite the absence of any great amount of trauma, the decedent may have sustained some significant trauma and it may have contributed to his death. It is more probable, however, that he was already dead, or at least unconscious, when he struck the median wall because all of the attention of the witnesses and the hospital staff were focused on what appeared to be a heart attack. There was little or no attention paid to the possibility of traumatic injuries resulting from a collision.

The Administrative Law Judge has not made any extensive research of medical literature relative to the causes of cardiac arrhythmias. He has reviewed, however, a commentary on cardiac arrhythmias which strongly suggests, the emotional stress and fatigue under which the decedent was driving may well have precipitated his fatal cardiac arrhythmia and the Administrative Law Judge so finds. The information relied upon is not quoted herein because it is copywrited material. (See Sagall, E. L. and Reed, B. C.: The Heart and the Law, The McMillan Co., New York, 1968, page 536.)

The decedent's average weekly wage at the time of his death is alleged to have been \$424.00 per week. This qualifies for the maximum death benefit of \$275.00 per week.

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER
PAGE SIX

CONCLUSIONS OF LAW:

The totality of the evidence showing the decedent's use of amphetamines, having a restless night before his departure from Salt Lake City, driving in bad weather enroute to Denver, arriving fatigued and apparently somewhat distressed over the prospects of having to make the return trip with only a few hours rest, and having embarked upon the return trip in a snowstorm, demonstrates a body of evidence that preponderates in favor of the applicant in showing the precipitating cause of the decedent's fatal cardiac arrhythmia was due to the conditions of his employment and is therefore compensable under the Workers Compensation Act.

ORDER:

IT IS THEREFORE ORDERED that the defendants, Lanier-Brugh and/or Workers Compensation Fund of Utah, pay applicant compensation at the rate of \$275.00 per week at intervals of not more than every four weeks for a period of 312 weeks for a total of \$85,800.00. Accrued amounts, less attorney's fees plus interest at the rate of 8% per annum from the date each payment would have otherwise been due and payable, shall be paid in a lump sum.

IT IS FURTHER ORDERED that the defendants, Lanier-Brugh and/or Workers Compensation Fund of Utah, pay the medical expenses associated with the decedent's hospitalization following his fatal accident, in addition to the statutory funeral allowance of \$1800.00, plus the additional cost of transporting the decedent's body from Colorado to Utah.

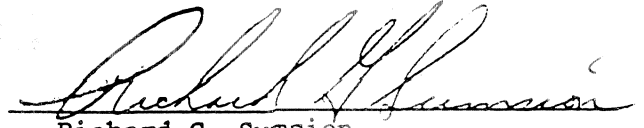
IT IS FURTHER ORDERED that when the above payments end, the decedent's widow is to contact the Industrial Commission regarding possible additional benefits under Section 35-1-68, Utah Code Annotated.

IT IS FURTHER ORDERED that immediate notice is to be given by the decedent's widow to the Industrial Commission and the insurance carrier in the event of her remarriage.

IT IS FURTHER ORDERED that the defendants, Lanier-Brugh and/or Workers Compensation Fund of Utah, pay to Thomas R. King, attorney for the applicant, the sum of \$10,830.00, for services rendered in this proceeding. This fee is in accordance with the Industrial Commission's rule pertaining to contingent fees in contested death cases.

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER
PAGE SEVEN

IT IS FURTHER ORDERED that any Motion for Review of the foregoing shall be filed in writing within fifteen (15) days of the date hereof specifying in detail the particular errors and objections, and, unless so filed, this Order shall be final and not subject to review or appeal.


Richard G. Sumsion
Administrative Law Judge

Passed by the Industrial Commission
of Utah, Salt Lake City, Utah, this

28th day of May, 1987.

ATTEST:

/s/ Linda J. Strasburg

Linda J. Strasburg
Commission Secretary

CERTIFICATE OF MAILING

I certify that on May 28th, 1987, a copy of the attached Findings of Fact, Conclusions of Law and Order, in the case of Bernice Steward, Widow of Dale W. Steward, Deceased, issued May 28th 1987, was mailed to the following persons at the following addresses, postage paid:

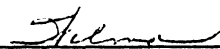
Bernice Steward, 1410 West 600 South, SLC, UT 84104

Thomas R. King, Atty., 2121 South State Street, Suite 205, SLC, UT 84115

Shaun Howell, Atty., Workers Compensation Fund of Utah, 560 South 300 East, SLC, UT 84111

Lanier-Brugh, Inc., 815 N. W. 11th, Portland, OR 97209

THE INDUSTRIAL COMMISSION OF UTAH

By 
Wilma

APPENDIX III

ORDER DENYING MOTION FOR REVIEW
August 28, 1987

009

THE INDUSTRIAL COMMISSION OF UTAH

Case No: 86000198

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased,

Applicant,

vs.

LANIER-BRUGH and/or
WORKERS COMPENSATION FUND OF UTAH,

Defendants.

ORDER DENYING

MOTION FOR REVIEW

* * * * *

On May 28, 1987, an Administrative Law Judge of the Industrial Commission issued Findings of Fact, Conclusions of Law and Order awarding the applicant in the above-captioned case dependent death benefits. The applicant's husband died of a heart attack (acute myocardial infarction) on November 12, 1985 while driving a truck from Denver to Salt Lake City in the course of his employment with the defendant/employer, Lanier-Brugh. The Administrative Law Judge found the facts supported both a finding of legal causation as well as medical causation. Allen vs the Industrial Commission, 46 Utah Advanced Reports 3 (Utah 1986).

With respect to legal causation, the Administrative Law Judge determined there was no previously diagnosed heart condition and so the Administrative Law Judge found only normal or usual exertion was required to establish legal causation. The Administrative Law Judge found that medical causation was established because the extra stress the deceased faced during the 24 hours preceding his death most likely led to the arrhythmia which resulted in the myocardial infarction. The Administrative Law Judge relied on a medical text discussing the causes of arrhythmia to arrive at this finding. Furthermore, the Administrative Law Judge noted that the physician who analyzed the possible causes for the deceased's heart attack found that the amphetamines the deceased took as treatment for narcolepsy could have contributed to the occurrence of the arrhythmia. As both medical and legal causation were established, the Administrative Law Judge awarded benefits to the widow.

On June 12, 1987, counsel for the Workers Compensation Fund filed a Motion for Review. Counsel for the Workers Compensation Fund contests both the finding of legal causation as well as the finding of medical causation. With respect to legal causation, counsel for the Workers Compensation Fund notes the following personal risk factors which counsel finds should have been

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER DENYING MOTION
PAGE TWO

considered by the Administrative Law Judge in determining whether the higher unusual exertion standard applied; obesity, age, moderate to heavy smoking, use of amphetamines as treatment for narcolepsy, emphysema and some record of possible myocardial related symptoms. In the alternative, if the Administrative Law Judge does not find these factors are sufficient to warrant application of the higher legal causation standard, counsel for the Workers Compensation Fund states the Administrative Law Judge should at least present the question of personal risk to a medical panel for its determination. With respect to the issue of medical causation, counsel for the Workers Compensation Fund finds the list of stress factors existing during the 24 hours preceding the heart attack cited by the Administrative Law Judge, as insufficient for purposes of establishing medical causation. Counsel for the Workers Compensation Fund states what medically caused the heart attack needs to be determined by a physician and cannot be decided by the Administrative Law Judge by way of reference to medical literature.

The Commission finds the issues to be decided on review are whether the Administrative Law Judge's conclusions regarding medical and legal causation are supported by the facts in this case. With respect to legal causation, the Commission finds that even if the unusual exertion standard is applied, the facts relate exertion and/or stress that goes beyond what the average person encounters in everyday non-employment life. The Administrative Law Judge found the deceased was driving under unusually stressful circumstances due to the fact his departure was delayed, he had to drive on snow covered roads, he had little or no rest in between the trip to Denver and the return trip to Salt Lake City and he was taking amphetamines. This combination of stress factors is not encountered by the average person in normal non-employment life, and therefore the Commission finds the facts support a conclusion of unusual exertion. Consequently, legal causation is established and the Administrative Law Judge therefore must be affirmed on this issue.

With respect to medical causation, the Commission also finds the Administrative Law Judge should be affirmed. Counsel for the Workers Compensation Fund asserts the Administrative Law Judge should have submitted the question of medical causation to a physician as the determination of medical cause requires medical expertise. The Commission notes however that no evidence, medical or otherwise, was submitted by the Workers Compensation Fund showing a different cause other than stress responsible for the arrhythmia which resulted in the heart attack. The only medical opinion submitted verifies that the amphetamines could have been a contributing cause to the arrhythmia and the Administrative Law Judge relied on consensus medical opinion that stress, fatigue and stimulants are all common precipitating causes of cardiac arrhythmias. The chart relied on by the Administrative Law Judge is reproduced below:

BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER DENYING MOTION
PAGE THREE

ARGUE - none of
these will
work

Table 34-4: Common precipitating causes of cardiac arrhythmias *

1. Emotional stress, particularly acute
2. Exertion, particularly under unusual conditions (e.g., cold, alcohol, fatigue, excitement, etc..)
3. Infection, thyrotoxicosis, and other noncardiac illness
4. Medication, especially digitalis over dosage
5. Stimulants such as tobacco, coffee, tea, or alcohol in sensitive subjects
6. Acute hypoglycemia (low blood sugar)
7. Therapeutic procedures such as surgery, anesthesia, and nerve block.
8. Trauma, particularly to the heart
9. Electric current as with accidental electric shock or electrotherapeutic procedures
10. Reflex vagal stimulation (e.g., acute gastritis, biliary colic, and other gastrointestinal disorders)
11. Acute cardiac disease (e.g., acute myocardial infraction, acute myocardial ischemia, cardiac contusion, congestive heart failure, etc.)

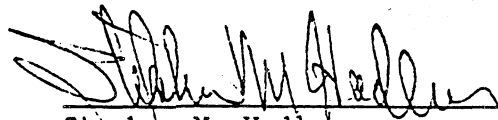
* adapted from Riseman, J. E.F., and Sagall, E.L.: Cardiac Arrhythmias; Electrocardiography, Diagnosis, Treatment. The Macmillian Co., New York, 1963.

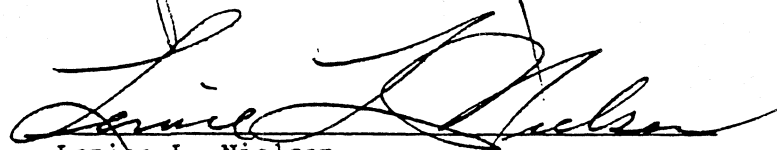
The Commission finds that in absence of evidence showing another cause, the Administrative Law Judge acted within his discretion in relying on a combination of medical evidence from the one medical opinion submitted and the consensus medical opinion as related in the medical text referred to by the Administrative Law Judge. As there is sufficient evidence to support the Administrative Law Judge's findings regarding both legal and medical causation, the Commission must deny the Workers Compensation Fund's Motion for Review and affirm the Administrative Law Judge.

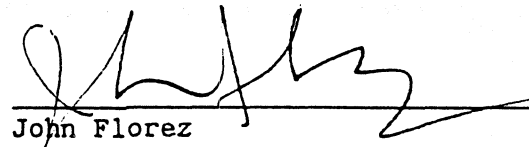
BERNICE STEWARD, Widow of
DALE W. STEWARD, Deceased
ORDER DENYING MOTION
PAGE FOUR

ORDER:

IT IS THEREFORE ORDERED that the Workers Compensation Fund's June 12, 1987 Motion for Review is denied and the Administrative Law Judge's May 28, 1987 Order is hereby affirmed and final with further appeal to the Court of Appeals only per U.C.A. 35-1-83.

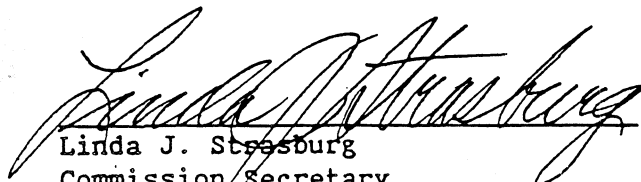

Stephen M. Hadley
Chairman


Lenice L. Nielsen
Commissioner


John Florez
Commissioner

Passed by the Industrial Commission
of Utah, Salt Lake City, Utah, this
20th day of August, 1987.

ATTEST:


Linda J. Stresburg
Commission Secretary

CERTIFICATE OF MAILING

059

I certify that on August 28th, 1987, a copy of the attached ORDER DENYING MOTION FOR REVIEW in the case of BERNICE STEWARD, Widow of DALE W. STEWARD, Deceased was mailed to the following persons at the following addresses, postage paid:

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Richard G. Sumsion, Administrative Law Judge

Janet L. Moffitt, Administrative Law Judge

INDUSTRIAL COMMISSION OF UTAH

By Pamela Hayes
Pamela Hayes

APPENDIX IV

WORKERS' COMPENSATION RULES AND REGULATIONS GUIDELINES FOR UTILIZATION OF MEDICAL PANEL

Workers' Compensation Rules & Regulations

- 1.2.33. GUIDELINES FOR UTILIZATION OF MEDICAL PANEL
- Pursuant to Section 35-1-77, U.C.A. the Commission adopts the following guidelines in determining the necessity of submitting a case to a medical panel:

(a) A panel will be utilized where:

1. One or more significant medical issues are involved.

Generally a significant medical issue must be shown by conflicting medical reports. The issues of permanent partial impairment will be considered significant if conflicting medical reports vary with a rating more than 5% of the whole person; or if the temporary total cut off date varies more than 90 days; or if the amount of medical expense in controversy is more than \$2,000.

2. In the opinion of the Commission the medical issues are so intertwined with the events that a determination of whether an accident has occurred cannot be made without first resolving medical consideration.

(b) Where in the opinion of the Commission, the evidence is insufficient for the Commission to make a final determination, the Commission may require an independent medical evaluation. Costs to be assessed against the employer and/or Second Injury Fund.

Workers' Compensation Rules & Regulations

- (c) A hearing on objections to the panel report may be scheduled if there is a proffer of conflicting medical testimony or an indication that all relevant medical evidence was not considered by the panel.
- (d) The Commission may authorize an injured worker to be examined by another physician for the purpose of obtaining a further medical examination or evaluation pertaining to the medical issues involved, and to obtain a report addressing these medical issues in all cases where:
 - 1. The treating physician has failed or refused to give an impairment rating.
 - 2. The employer or doctor considers the claim to be non-industrial.
 - 3. A substantial injustice may occur without such further evaluation.

APPENDIX V

1986 PHYSICIANS DESK REFERENCE
PRODUCT INFORMATION

PDR®
40
EDITION
1986

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a normal TSH during those two to eight

ns: Adverse reactions, other than those arthyroidism because of therapeutic over-ially or during the maintenance period are OSAGE).

allergic skin reactions have been reported thyronine sodium, SK&F) Tablets.

oms—Headache, irritability, nervousness, ardia, increased bowel motility and men-ies. Angina pectoris or congestive heart iduced or aggravated. Shock may also de-er dosage may result in symptoms resem-rm. Chronic excessive dosage will produce nptoms of hyperthyroidism.

er dosage—Dosage should be reduced or rily discontinued if signs and symptoms of ar. Treatment may be reinstituted at a normal individuals, normal hypothalamic- axis function is restored in six to eight id suppression.

te massive thyroid hormone overdose is g gastrointestinal absorption of the drugs g central and peripheral effects, mainly d sympathetic activity. Vomiting may be if further gastrointestinal absorption can everted and barring contraindications such ons, or loss of the gagging reflex. Treatment d supportive. Oxygen may be administered aintained. Cardiac glycosides may be indi- e heart failure develops. Measures to con- ylcemia, or fluid loss should be instituted if energic agents, particularly propranolol, advantageously in the treatment of in- etic activity. Propranolol may be admini- sly at a dosage of 1 to 3 mg. over a 10-minute 80 to 160 mg./day, especially when no con- sist for its use.

ministration: The dosage of thyroid hor- lined by the indication and must in every alized according to patient response and gs.

es are given orally. In acute, emergency table lev thyroxine sodium may be given hen oral administration is not feasible or he treatment of myxedema coma: during nutrition. Injectable liothyronine sodium is m Smith Kline & French Laboratories upon nvestigational status, for the treatment of . Intramuscular administration of these two not advisable because of reported poor ab-

liothyronine sodium, SK&F) Tablets once- ecommended; although liothyronine sodium f, its metabolic effects persist for a few days tinuance.

roidism: Recommended starting dosage is Daily dosage then may be increased by 12.5 ery one or two weeks. Usual maintenance ncg. daily. Smaller doses may be fully effec- atients, while dosage of 100 mcg. daily may others.

et and dissipation of action of liothyronine s compared with levothyroxine sodium (T₄), clinicians to prefer its use in patients who e susceptible to the untoward effects of thy- on. However, the wide swings in serum T₃ low its administration and the possibility of iced cardiovascular side effects tend to coun- e stated advantages.

thyronine sodium, SK&F) Tablets may be used o levothyroxine (T₄) during radioisotope edures, since induction of hypothyroidism in more abrupt and can be of shorter duration. e preferred when impairment of peripheral T₄ and T₃ is suspected.

Recommended starting dosage is 5 mcg. ay be increased by 5 to 10 mcg. daily every eeks. When 25 mcg. daily is reached, dosage increased by 12.5 or 25 mcg. every one or two maintenance dose is 50 to 100 mcg. daily. ome: Myxedema coma is usually precipi- ypothyroid patient of long standing by inter- s or drugs such as sedatives and anesthetics e considered a medical emergency. A 'Cyto- n Kit for the emergency treatment of myx- is available from Smith Kline & French Labo- 1 request, under investigational status. In- ick accompany this kit provide information ation.

Hypothyroidism: Recommended starting cg. daily, with a 5 mcg. increment every three until the desired response is achieved. Infants old may require only 20 mcg. daily for main- e year, 50 mcg. daily may be required.

Above three years, full adult dosage may be necessary (See PRECAUTIONS, Pediatric Use). Simple (non-toxic) Goiter: Recommended starting dosage is 5 mcg. daily. This dosage may be increased by 5 to 10 mcg. daily every one or two weeks. When 25 mcg. daily is reached, dosage may be increased every week or two by 12.5 or 25 mcg. Usual maintenance dosage is 75 mcg. daily.

In the elderly or in children, therapy should be started with 5 mcg. daily and increased only by 5 mcg. increments at the recommended intervals.

When switching a patient to Cytomel (liothyronine sodium, SK&F) Tablets from thyroid, L-thyroxine or thyroglobulin, discontinue the other medication, initiate 'Cytomel' at a low dosage, and increase gradually according to the patient's response. When selecting a starting dosage, bear in mind that this drug has a rapid onset of action, and that residual effects of the other thyroid preparation may persist for the first several weeks of therapy.

Thyroid Suppression Therapy: Administration of thyroid hormone in doses higher than those produced physiologi- cally by the gland results in suppression of the production of endogenous hormone. This is the basis for the thyroid sup- pression test and is used as an aid in the diagnosis of patients with signs of mild hyperthyroidism in whom baseline labora- tory tests appear normal or to demonstrate thyroid gland autonomy in patients with Graves' ophthalmopathy. ¹³¹I uptake is determined before and after the administration of the exogenous hormone. A 50 percent or greater suppression of uptake indicates a normal thyroid-pituitary axis and thus rules out thyroid gland autonomy.

Cytomel (liothyronine sodium, SK&F) Tablets are given in doses of 75-100 mcg./day for seven days, and radioactive io- dine uptake is determined before and after administration of the hormone. If thyroid function is under normal control, the radioiodine uptake will drop significantly after treatment. Cytomel (liothyronine sodium, SK&F) Tablets should be ad- ministered cautiously to patients in whom there is a strong suspicion of thyroid gland autonomy, in view of the fact that the exogenous hormone effects will be additive to the endog- enous source.

How Supplied: Cytomel (liothyronine sodium, SK&F) tablets: 5 mcg. in bottles of 100; 25 mcg. in bottles of 100 and 1000; and 50 mcg. in bottles of 100.

CY-L30

Shown in Product Identification Section, page 429

DARBID® TABLETS, 5 mg.

[dahr'bid]

(brand of isopropamide iodide)

Description: Darbid (brand of isopropamide iodide) is avail- able as a tablet for oral administration. Each round, pink, coated tablet is imprinted SKF and D62 and contains isopro- pamide iodide equivalent to 5 mg. of isopropamide. Inactive ingredients consist of acacia, calcium sulfate, FD&C Red No. 3, FD&C Yellow No. 6, gelatin, iron oxide, mineral oil, starch, stearic acid, sucrose, talc, titanium dioxide and trace amounts of other inactive ingredients.

Chemically, Darbid (isopropamide iodide, SK&F) is (3-carbam- oyl-3, 3-diphenylpropyl) diisopropylmethyl ammonium io- dide.

Actions: Isopropamide iodide is a synthetic anticholinergic that produces 10- to 12-hour gastric acid antisecretory effect and gastrointestinal antispasmodic response in man.

Indications: 'Darbid' is effective as adjunctive therapy in the treatment of peptic ulcer.

'DARBID' HAS NOT BEEN SHOWN TO BE EFFECTIVE AS SOLE THERAPY IN CONTRIBUTING TO THE HEAL- ING OF PEPTIC ULCER, DECREASING THE RATE OF RECURRENCE OR PREVENTING COMPLICATIONS. To be effective, dosage must be titrated to the individual pa- tient's needs.

Contraindications: Glaucoma; obstructive uropathy (e.g., bladder neck obstruction due to prostatic hypertrophy); ob- structive disease of the gastrointestinal tract (as in achai- sia, pyloroduodenal stenosis, etc.); obstructive or paralytic ileus, intestinal atony of the elderly or debilitated patient; unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis.

Warnings: In the presence of a high environmental temper- ature, heat prostration (fever and heat stroke due to de- creased sweating) can occur.

In patients with diarrhea due to incomplete intestinal ob- struction (especially those with ileostomy or colostomy), treatment with Darbid (isopropamide iodide, sk&f) would be inappropriate and possibly harmful.

'Darbid' may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activi- ties requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug.

Usage in Pregnancy: In pregnancy, lactation, and in women who may bear children, the potential benefits of the drug must be weighed against possible hazards.

Precautions: Use cautiously in elderly patients:

Since the iodine in isopropamide iodide may alter PBI test results and will suppress ¹³¹I uptake, it is suggested that therapy be discontinued one week prior to these tests. Also, iodine skin rash may occur rarely.

Use with caution in patients with:

Autonomic neuropathy.

Hepatic or renal disease

Ulcerative colitis (large doses may suppress intestinal moti- lity to the point of producing paralytic ileus, and the use of this drug may precipitate or aggravate the serious complica- tion of toxic megacolon).

Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmia, hypertension and nonobstruct- ing prostatic hypertrophy.

Hiatal hernia associated with reflux esophagitis (anticholin- ergic drugs may aggravate this condition).

It should be noted that the use of anticholinergic drugs in the treatment of gastric ulcer may produce a delay in gastric emptying time (antral stasis) and, thus, complicate therapy. Do not rely on the use of the drug in the presence of complica- tion of biliary tract disease.

Investigate any tachycardia before giving anticholinergic (atropine-like) drugs, since they may increase the heart rate. With overdose, a curare-like action may occur.

Adverse Reactions: Anticholinergics produce certain phar- macological effects which may be desirable or undesirable, depending upon the individual patient's response. The physi- cian must delineate these.

Adverse reactions which have occurred with Darbid (isopro- pamide iodide, sk&f) include: xerostomia (dry mouth); uri- nary hesitancy and retention; blurred vision; tachycardia; palpitations; mydriasis (dilatation of the pupils); cycloplegia; constipation; bloated feeling; nausea; dysphagia; fever; and nasal congestion.

Other adverse reactions possible with anticholinergics in- clude: increased ocular tension; loss of taste; headaches; ner- vousness; drowsiness; weakness; dizziness; insomnia; vomit- ing; impotence; suppression of lactation; severe allergic reac- tion or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons. Decreased sweating may occur. It should be noted that ad- renergic innervation of the eccrine sweat glands on the palms and soles makes complete control of sweating impossi- ble. An end point of complete anhidrosis cannot occur be- cause large doses of drug would be re- eed, and this would produce severe side effects from parasympathetic paralysis.

Dosage and Administration: Not for use in children under 12. Adults and children over 12—Usual starting dose is one 5 mg. tablet b.i.d. (every 12 hours). Patients with severe symptoms may require two 5 mg. tablets b.i.d., or more. Dos- age should be individualized and titrated to the patient's need for greatest therapeutic effect.

Overdosage: Involves the cardiovascular, respiratory, gas- trointestinal, central and peripheral nervous systems.

SYMPTOMS—May include dryness of mouth, dysphagia, thirst, blurred vision, dilated pupils, photophobia, fever, rapid pulse and respiration, disorientation. Depression and circulatory collapse may result from severe overdosage.

TREATMENT—Gastric lavage, repeated several times.

Respiratory depression should be promptly treated by the use of oxygen and stimulants. If marked excitement is pres- ent, one of the short-acting barbiturates, chloral hydrate, or gas anesthesia may be used. Otherwise do not administer sedation. Hyperpyrexia may be treated with physical cooling measures. Force fluids by mouth or, if necessary, by intrave- nous administration.

While pilocarpine or similar drugs are sometimes recom- mended for the relief of dry mouth, many authorities feel that these drugs are not indicated, since they relieve the minor peripheral effect but do not influence the more serious central effects and, thus, may merely mask signs of drug activity. If photophobia occurs, the patient should be kept in a darkened room.

How Supplied: Tablets, 5 mg., in bottles of 50.

Shown in Product Identification Section, page 429

DB-L16

DEXEDRINE®

[dex'eh-dreen]

(brand of dextroamphetamine sulfate)

SPANSULE® CAPSULES,

TABLETS and ELIXIR

Warning:

AMPHETAMINES HAVE A HIGH POTENTIAL FOR ABUSE. THEY SHOULD THUS BE TRIED ONLY IN WEIGHT REDUCTION PROGRAMS FOR PATIENTS IN WHOM ALTERNATIVE THERAPY HAS BEEN INEFFECTIVE. ADMINISTRATION OF AMPHETA- MINES FOR PROLONGED PERIODS OF TIME IN

Continued on next page

Smith Kline & French—Cont.

OBSESITY MAY LEAD TO DRUG DEPENDENCE AND MUST BE AVOIDED. PARTICULAR ATTENTION SHOULD BE PAID TO THE POSSIBILITY OF SUBJECTS OBTAINING AMPHETAMINES FOR NON-THERAPEUTIC USE OR DISTRIBUTION TO OTHERS, AND THE DRUGS SHOULD BE PRESCRIBED OR DISPENSED SPARINGLY.

Description: Dexedrine (dextroamphetamine sulfate, SK&F) is the dextro isomer of the compound *d,l*-amphetamine sulfate, a sympathomimetic amine of the amphetamine group. Chemically, dextroamphetamine is *d*-alpha-methylphenethylamine, and is present in all forms of 'Dexedrine' as the neutral sulfate.

Spansule® capsules

Each 'Spansule' sustained release capsule is so prepared that an initial dose is released promptly and the remaining medication is released gradually over a prolonged period.

Each capsule, with brown cap and natural body, contains dextroamphetamine sulfate as follows: 5 mg. imprinted SKF and E12, 10 mg. imprinted SKF and E13, 15 mg. imprinted SKF and E14. Inactive ingredients consist of acacia, benzyl alcohol, calcium sulfate, cetylpyridinium chloride, FD&C Blue No. 1, FD&C Red No. 40, FD&C Yellow No. 5 (tartrazine), FD&C Yellow No. 6, gelatin, glyceryl distearate, glyceryl monostearate, sodium lauryl sulfate, starch, sucrose, wax and trace amounts of other inactive ingredients.

Tablets

Each triangular, orange, scored tablet is debossed SKF and E19 and contains dextroamphetamine sulfate, 5 mg. Inactive ingredients consist of calcium sulfate, FD&C Yellow No. 5 (tartrazine), FD&C Yellow No. 6, gelatin, lactose, mineral oil, starch, stearic acid, sucrose, talc and trace amounts of other inactive ingredients.

Elixir

Each 5 ml. (one teaspoonful) of clear, orange-colored, orange-flavored liquid contains dextroamphetamine sulfate, 5 mg., and alcohol, 10%. Inactive ingredients consist of benzoic acid, citric acid, FD&C Yellow No. 5 (tartrazine), FD&C Yellow No. 6, flavor, sucrose, water and wine.

Clinical Pharmacology: Amphetamines are non-catecholamine, sympathomimetic amines with CNS stimulant activity. Peripheral actions include elevations of systolic and diastolic blood pressures and weak bronchodilator and respiratory stimulant action.

There is neither specific evidence which clearly establishes the mechanism whereby amphetamines produce mental and behavioral effects in children, nor conclusive evidence regarding how these effects relate to the condition of the central nervous system.

Drugs of this class used in obesity are commonly known as "anorectics" or "anorexigenics." It has not been established, however, that the action of such drugs in treating obesity is primarily one of appetite suppression. Other central nervous system actions, or metabolic effects, may be involved, for example.

Adult obese subjects instructed in dietary management and treated with "anorectic" drugs lose more weight on the average than those treated with placebo and diet, as determined in relatively short-term clinical trials.

The magnitude of increased weight loss of drug-treated patients over placebo-treated patients is only a fraction of a pound a week. The rate of weight loss is greatest in the first weeks of therapy for both drug and placebo subjects and tends to decrease in succeeding weeks. The origins of the increased weight loss due to the various possible drug effects are not established. The amount of weight loss associated with the use of an "anorectic" drug varies from trial to trial, and the increased weight loss appears to be related in part to variables other than the drug prescribed, such as the physician-investigator, the population treated, and the diet prescribed. Studies do not permit conclusions as to the relative importance of the drug and nondrug factors on weight loss. The natural history of obesity is measured in years, whereas the studies cited are restricted to a few weeks' duration; thus, the total impact of drug-induced weight loss over that of diet alone must be considered clinically limited.

Dexedrine (dextroamphetamine sulfate, SK&F) Spansule capsules are formulated to release the active drug substance *in vivo* in a more gradual fashion than the standard formulation, as demonstrated by blood levels. The formulation has not been shown superior in effectiveness over the same dosage of the standard, noncontrolled-release formulations given in divided doses.

Pharmacokinetics

Elixir—Ingestion of 10 mg. of dextroamphetamine sulfate in elixir form by healthy volunteers produced an average peak dextroamphetamine blood level of 33.2 ng./ml. The half-life was 11.75 hours. The average urinary recovery was 38% in 48 hours.

Tablet—The single ingestion of two 5 mg. tablets by healthy volunteers produced an average peak dextroamphetamine

blood level of 29.2 ng./ml. at 2 hours post-administration. The average half-life was 10.25 hours. The average urinary recovery was 45% in 48 hours.

'Spansule' capsule—Ingestion of a 'Spansule' capsule containing 15 mg. radiolabeled dextroamphetamine sulfate by healthy volunteers produced a peak blood level of radioactivity, on the average, at 8-10 hours post-administration with peak urinary recovery seen at 12-24 hours.

Indications and Usage: Dexedrine (dextroamphetamine sulfate, SK&F) is indicated:

1. In Narcolepsy.

2. In Attention Deficit Disorder with Hyperactivity, as an integral part of a total treatment program which typically includes other remedial measures (psychological, educational, social) for a stabilizing effect in children with a behavioral syndrome characterized by the following group of developmentally inappropriate symptoms: moderate to severe distractibility, short attention span, hyperactivity, emotional lability, and impulsivity. The diagnosis of this syndrome should not be made with finality when these symptoms are only of comparatively recent origin. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present, and a diagnosis of central nervous system dysfunction may or may not be warranted.

3. In Exogenous Obesity, as a short-term (a few weeks) adjunct in a regimen of weight reduction based on caloric restriction, for patients refractory to alternative therapy, e.g., repeated diets, group programs, and other drugs. The limited usefulness of amphetamines (see CLINICAL PHARMACOLOGY) should be weighed against possible risks inherent in use of the drug, such as those described below.

Contraindications—Advanced states of hypertension, moderate to severe hypertension, hyperthyroidism, known hypersensitivity or idiosyncrasy to the sympathomimetic amines, glaucoma.

Agitated states.

Patients with a history of drug abuse.

During or within 14 days following the administration of monoamine oxidase inhibitors (hypertensive crises may result).

Warning: When tolerance to the "anorectic" effect develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued.

Precautions:

General: Caution is to be exercised in prescribing amphetamines to patients with cardiovascular disease.

The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdose.

These products contain FD&C Yellow #5 (tartrazine), which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals. Although the overall incidence of FD&C Yellow #5 (tartrazine) sensitivity in the general population is low, it is frequently seen in patients who also have aspirin hypersensitivity.

Information for Patients: Amphetamines may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or vehicles; the patient should therefore be cautioned accordingly.

Drug Interactions

Acidifying agents—Gastrointestinal acidifying agents (guanethidine, reserpine, glutamic acid HCl, ascorbic acid, fruit juices, etc.) lower absorption of amphetamines. Urinary acidifying agents (ammonium chloride, sodium acid phosphate, etc.) increase the concentration of the ionized species of the amphetamine molecule, thereby increasing urinary excretion. Both groups of agents lower blood levels and efficacy of amphetamines.

Adrenergic blockers—Adrenergic blockers are inhibited by amphetamines.

Alkalinizing agents—Gastrointestinal alkalinizing agents (sodium bicarbonate, etc.) increase absorption of amphetamines. Urinary alkalinizing agents (acetazolamide, some thiazides) increase the concentration of the non-ionized species of the amphetamine molecule, thereby decreasing urinary excretion. Both groups of agents increase blood levels and therefore potentiate the actions of amphetamines.

Antidepressants, tricyclic—Amphetamines may enhance the activity of tricyclic or sympathomimetic agents; d-amphetamine with desipramine or protriptyline and possibly other tricyclics cause striking and sustained increases in the concentration of d-amphetamine in the brain; cardiovascular effects can be potentiated.

MAO inhibitors—MAOI antidepressants, as well as a metabolite of furazolidone, slow amphetamine metabolism. This slowing potentiates amphetamines, increasing their effect on the release of norepinephrine and other monoamines from adrenergic nerve endings; this can cause headaches and other signs of hypertensive crisis. A variety of neurological toxic effects and malignant hyperpyrexia can occur, sometimes with fatal results.

Antihistamines—Amphetamines may counteract the sedative effect of antihistamines.

Antihypertensives—Amphetamines may have hypotensive effects of antihypertensives.

Chlorpromazine—Chlorpromazine blocks norepinephrine reuptake, thus inhibiting the latent effects of amphetamines, and can be used in phetamine poisoning.

Ethosuximide—Amphetamines may delay in action of ethosuximide.

Haloperidol—Haloperidol blocks dopamine reuptake, thus inhibiting the central stimulant effects of amphetamines.

Lithium carbonate—The antiobesity and stimulant effects of amphetamines may be inhibited by lithium. Meperidine—Amphetamines potentiate the effects of meperidine.

Methamphetamine therapy—Urinary excretions of methamphetamine are increased, and efficacy is reduced when agents used in methamphetamine therapy.

Norepinephrine—Amphetamines enhance the effects of norepinephrine.

Phenobarbital—Amphetamines may delay the sorption of phenobarbital; co-administration may produce a synergistic anticonvulsant effect of phenobarbital.

Phenytoin—Amphetamines may delay the absorption of phenytoin; co-administration of phenytoin may produce a synergistic anticonvulsant action.

Propoxyphene—In cases of propoxyphene or phetamine CNS stimulation is potentiated at doses which cause sedation.

Veratrum alkaloids—Amphetamines inhibit the effect of veratrum alkaloids.

Drug/Laboratory Test Interactions

- Amphetamines can cause a significant increase in plasma corticosteroid levels. This increase is not clinically significant.

- Amphetamines may interfere with urinary excretions.

Carcinogenesis/Mutagenesis: Mutagenicity long-term studies in animals to determine the potential of Dexedrine (dextroamphetamine sulfate) have not been performed.

Pregnancy—Teratogenic Effects: Pregnant women have been shown to have embryonic effects when administered to A/Jax mice in doses approximately 41 times the maximum recommended dose.

Embryotoxic effects were not seen in white rabbits given the drug in doses 7 times the maximum recommended dose. There are no adequate and well-controlled studies in pregnant women. 'Dexedrine' should be used during pregnancy only if the potential benefit justifies the potential risks.

Nonteratogenic Effects: Infants born to mothers on amphetamines have an increased risk of prematurity and low birth weight. Also, these infants may have symptoms of withdrawal as demonstrated by irritability, agitation, and significant lassitude.

Nursing Mothers: It is not known whether amphetamines are excreted in breast milk; efforts to measure amphetamines in breast milk have been unsuccessful. Because amphetamines are excreted in human milk, caution should be exercised when 'Dexedrine' is administered to a nursing woman.

Pediatric Use: Long-term effects of amphetamines have not been well established.

Amphetamines are not recommended for use in children under 12 years of age, or in children 3 years of age with Attention Deficit Disorder (ADD) described under INDICATIONS AND USAGE.

Clinical experience suggests that in psychotic patients, administration of amphetamines may exacerbate behavior disturbance and thought disorder.

Amphetamines have been reported to exacerbate phonic tics and Tourette's syndrome. Therapeutic evaluation for tics and Tourette's syndrome in their families should precede use of stimulant drugs. Data are inadequate to determine whether chronic administration of amphetamines may be associated with inhibition; therefore, growth should be monitored.

Drug treatment is not indicated in all cases of Attention Deficit Disorder with Hyperactivity and should only be given in light of the complete history and evaluation of the child. The decision to prescribe amphetamines should be based on the physician's assessment of the severity of the child's symptoms and their appropriateness for his/her age. Prescription should not depend on the presence of one or more of the behavioral characteristics. When these symptoms are associated with acute actions, treatment with amphetamines is usually indicated.

Adverse Reactions:

Cardiovascular: Increased heart rate, increased blood pressure, palpitations, and other signs of hypertensive crisis.

Central Nervous System: Psychotic episodes, increased doses (rare), overstimulation, restlessness, insomnia, euphoria, dyskinesia, dysphoria, tics, and Tourette's syndrome.

erabation of motor and phonic tics and Tourette's

istinal: Dryness of the mouth, unpleasant taste, constipation, other gastrointestinal disturbances, and weight loss may occur as undesirable effects. Amphetamines are used for other than the anorectic

Urticaria.

Impotence, changes in libido.

Use and Dependence: Dextroamphetamine sulfate is a controlled substance.

Amphetamines have been extensively abused. Tolerance, psychological dependence, and severe social disability have occurred. There are reports of patients who have the dosage to many times that recommended. Intoxication following prolonged high dosage administration results in extreme fatigue and mental depression; this is also noted on the sleep EEG.

Signs of chronic intoxication with amphetamines include dermatoses, marked insomnia, irritability, personality changes. The most severe sign of chronic intoxication is psychosis, often indistinguishable from schizophrenia. This is rare with amphetamines.

Adverse Effects: Individual patient response to amphetamines is widely variable. While toxic symptoms occasionally occur at doses as low as 2 mg., they are rare at less than 15 mg.; 30 mg. can produce severe effects; doses of 400 to 500 mg. are not necessarily fatal.

LD₅₀ of dextroamphetamine sulfate is 96.8 mg/kg.

Overdosage: Manifestations of acute overdosage with amphetamines include restlessness, tremor, hyperreflexia, hypertension, confusion, assaultiveness, hallucinations, and coma.

Depression: Usually follows the central stimulant effect.

Cardiovascular effects: include arrhythmias, hypertension, and circulatory collapse. Gastrointestinal effects include nausea, vomiting, diarrhea, and abdominal pain. Fatal poisoning is usually preceded by convulsions.

Management: Management of acute amphetamine intoxication is symptomatic and includes gastric lavage with a barbiturate. Experience with hemodialysis in the management of amphetamine poisoning is inadequate to permit recommendation. Acidification of the urine increases amphetamine excretion. If acute, severe hypertension complicates amphetamine overdosage, administration of intravenous phentolamine (Regitine®) has been suggested; a gradual drop in blood pressure will usually follow sufficient sedation has been achieved. Amphetamine antagonizes the central stimulant effects of amphetamines and can be used to treat amphetamine intoxication.

of the 'Spansule' capsule medication is coated for ease of administration directed at reversing the effects of the drug and at supporting the patient should be as long as overdosage symptoms remain. Saline cathartics are useful for hastening the evacuation of the bowels. Have not already released medication.

Administration: Regardless of indication, amphetamines should be administered at the lowest effective dose and should be individually adjusted. Late administration—particularly with the 'Spansule' capsule—should be avoided because of the resulting insomnia.

Dosage: Usual dose 5 to 60 milligrams per day in divided doses, depending on the individual patient response. Overdosage seldom occurs in children under 12 years of age; when it does, dextroamphetamine sulfate (Dexedrine®) may be used. The suggested initial dose for patients 6–12 is 5 mg. daily; daily dose may be raised in increments of 5 mg. at weekly intervals until optimal response is obtained. In patients 12 years of age and older, start with 5 mg. daily; daily dosage may be raised in increments of 5 mg. at weekly intervals until optimal response is obtained. If severe adverse reactions appear (e.g., insomnia, hypertension), dosage should be reduced. 'Spansule' capsules are used for once-a-day dosage wherever appropriate; tablets or elixir, give first dose on awakening; additional doses (1 or 2) at intervals of 4 to 6 hours.

Deficit Disorder with Hyperactivity: Not recommended in children under 3 years of age.

Children 3 to 5 years of age: start with 2.5 mg. daily, elixir; daily dosage may be raised in increments of 2.5 mg. at weekly intervals until optimal response is obtained.

Children 6 years of age and older: start with 5 mg. once or twice daily; dosage may be raised in increments of 5 mg. at weekly intervals until optimal response is obtained. In cases where it is necessary to exceed a total of 40 mg. per day

With tablets or elixir, give first dose on awakening; additional doses (1 or 2) at intervals of 4 to 6 hours.

Where possible, drug administration should be interrupted occasionally to determine if there is a recurrence of behavioral symptoms sufficient to require continued therapy.

Exogenous Obesity: Usual dosage is one 10 or 15 mg. 'Spansule' capsule daily, taken in the morning, or up to 30 mg. daily by tablets or elixir, taken in divided doses of 5 to 10 mg. 30 to 60 minutes before meals. Not recommended for this use in children under 12 years of age.

How Supplied:

'Spansule' capsules available: 5 mg., in bottles of 50; 10 mg. and 15 mg., in bottles of 50 and 500.

Tablets, 5 mg., available in bottles of 100 and 1000.

Elixir, 5 mg./5 mL., available in 16 fl. oz. (473 mL.) bottles.

Shown in Product Identification Section, page 429.

DX:L34

DIBENZYLIN® Capsules

[di-benz 'eh-leen]

(brand of phenoxybenzamine hydrochloride)

Description: Each 'Dibenzylin' capsule, with red cap and red body, is imprinted SKF and E33 and contains phenoxybenzamine hydrochloride, 10 mg. Inactive ingredients consist of benzyl alcohol, cetylpyridinium chloride, D&C Red No. 33, FD&C Red No. 3, FD&C Yellow No. 6, gelatin, lactose, sodium lauryl sulfate and trace amounts of other inactive ingredients.

'Dibenzylin' is N-(2-chloroethyl)-N-(1-methyl-2-phenoxyethyl) benzylamine hydrochloride. Phenoxybenzamine hydrochloride is a colorless, crystalline powder with a molecular weight of 340.3 which melts between 136° and 141°C. It is soluble in water, alcohol and chloroform; insoluble in ether.

Actions: Dibenzylin (phenoxybenzamine hydrochloride, SKF) is a long-acting, adrenergic, α -receptor blocking agent which can produce and maintain "chemical sympathectomy" by oral administration. It increases blood flow to the skin, mucosa and abdominal viscera, and lowers both supine and erect blood pressures. It has no effect on the parasympathetic system.

Indication: Pheochromocytoma, to control episodes of hypertension and sweating. If tachycardia is excessive, it may be necessary to use a beta-blocking agent concomitantly.

Contraindications: Conditions where a fall in blood pressure may be undesirable.

Warning: 'Dibenzylin'-induced α -adrenergic blockade leaves beta-adrenergic receptors unopposed. Compounds that stimulate both types of receptors may therefore produce an exaggerated hypotensive response and tachycardia.

Precautions: Phenoxybenzamine hydrochloride has shown *in vitro* mutagenic activity in the Ames test and in the mouse lymphoma assay; it has not shown mutagenic activity in the micronucleus test in mice. In rats and mice repeated intraperitoneal administration of phenoxybenzamine hydrochloride resulted in peritoneal sarcomas. Chronic oral dosing in rats has produced malignant tumors of the gastrointestinal tract.

The clinical significance of such test results is not established. Nevertheless, these results should be given consideration in determining the benefit-risk ratio as it applies to the individual patient.

Administer with caution in patients with marked cerebral or coronary arteriosclerosis or renal damage. Adrenergic blocking effect may aggravate symptoms of respiratory infections.

Adverse Reactions: Nasal congestion, miosis, postural hypotension, tachycardia and inhibition of ejaculation may occur. These so-called "side effects" are actually evidence of adrenergic blockade and vary according to the degree of blockade. Furthermore, they tend to decrease as therapy is continued. Gastrointestinal irritation, drowsiness and fatigue have also been reported.

Dosage and Administration: The dosage should be adjusted to fit the needs of each patient. Small initial doses should be slowly increased until the desired effect is obtained or the side effects from blockade become troublesome. After each increase, the patient should be observed on that level before instituting another increase. The dosage should be carried to a point where symptomatic relief and/or objective improvement are obtained, but not so high that the side effects from blockade become troublesome.

Initially, 10 mg. of Dibenzylin (phenoxybenzamine hydrochloride, SKF) twice a day. Dosage should be increased every other day, usually to 20 to 40 mg. two or three times a day, until an optimal dosage is obtained, as judged by blood pressure control.

Overdosage: Symptoms: These are largely the result of block of the sympathetic nervous system and of the circulating epinephrine. They may include postural hypotension resulting in dizziness or fainting; tachycardia, particularly postural; vomiting; lethargy; shock. **Treatment:** When

consideration. In cases of mild overdosage, recumbent position with legs elevated usually restores cerebral circulation. In the more severe cases, the usual measures to combat shock should be instituted. Usual pressor agents are not effective. Epinephrine is contraindicated because it stimulates both α and β receptors; since α receptors are blocked, the net effect of epinephrine administration is vasodilation and a further drop in blood pressure (epinephrine reversal).

The patient may have to be kept flat for 24 hours or more in the case of overdosage, as the effect of the drug is prolonged. Leg bandages and an abdominal binder may shorten the period of disability.

I.V. infusion of levarterenol bitartrate* may be used to combat severe hypotensive reactions, because it stimulates α receptors primarily. Although Dibenzylin (phenoxybenzamine hydrochloride, SKF) is an adrenergic blocking agent, a sufficient dose of levarterenol bitartrate will overcome this effect.

How Supplied: Dibenzylin (phenoxybenzamine hydrochloride, SKF) capsules, 10 mg., in bottles of 100.

*Available as Levophed® Bitartrate (brand of levarterenol bitartrate) from Winthrop Laboratories.

Shown in Product Identification Section, page 429.

DL:L21

DYAZIDE® Capsules

[dye-uh-zide]

('Dyazide' is a product of SK&F Co., Carolina, P.R. 00630, Subsidiary of SmithKline Beckman Corporation, Philadelphia, Pa.)

Warning:

This fixed combination drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual patient. If the fixed combination represents the dosage so determined, its use may be more convenient in patient management. The treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Description: Each red and white 'Dyazide' capsule contains 50 mg. of Dyrenium® (brand of triamterene), a potassium-sparing agent, and 25 mg. of hydrochlorothiazide.

'Dyrenium' is 2, 4, 7-triamino-6-phenylpteridine. Hydrochlorothiazide is 6-chloro-3, 4-dihydro-2H-1, 2, 4-benzothiadiazine-7-sulfonamide 1,1-dioxide.

At 50°C., triamterene is practically insoluble in water (less than 0.1%). It is soluble in formic acid, sparingly soluble in methoxyethanol, and very slightly soluble in alcohol. Hydrochlorothiazide is slightly soluble in water. It is soluble in dilute ammonia, dilute aqueous sodium hydroxide, and dimethylformamide. It is sparingly soluble in methanol.

Action: 'Dyazide' is a diuretic/antihypertensive drug product that combines the natriuretic, hydrochlorothiazide, and the potassium-sparing natriuretic, triamterene, each of which complements the action of the other. The hydrochlorothiazide component blocks the reabsorption of sodium and chloride ions, and thereby increases the quantity of sodium traversing the distal tubule and the volume of water excreted. A portion of the additional sodium presented to the distal tubule is exchanged there for potassium and hydrogen ions. With continued use of hydrochlorothiazide and depletion of sodium, compensatory mechanisms tend to increase this exchange and may produce excessive loss of potassium, hydrogen and chloride ions. Hydrochlorothiazide also decreases the excretion of calcium and uric acid, may increase the excretion of iodide and may reduce glomerular filtration rate. The exact mechanism of the antihypertensive effect of hydrochlorothiazide is not known.

The triamterene component of 'Dyazide' exerts its diuretic effect on the distal renal tubule to inhibit the reabsorption of sodium in exchange for potassium and hydrogen ions. Its natriuretic activity is limited by the amount of sodium reaching its site of action. Although it blocks the increase in this exchange that is stimulated by mineralocorticoids (chiefly aldosterone) it is not a competitive antagonist of aldosterone and its activity can be demonstrated in adrenalectomized rats and patients with Addison's disease. As a result the dose of triamterene required is not proportionally related to the level of mineralocorticoid activity, but is dictated by the response of the individual patient, and the kaliuretic effect of concomitantly administered drugs. By inhibiting the distal tubular exchange mechanism, triamterene maintains or increases the sodium excretion and reduces the excess loss of potassium, hydrogen, and chloride ions induced by hydrochlorothiazide. As with hydrochlorothiazide, triamterene may reduce glomerular filtration and renal plasma flow. Via this mechanism it may reduce uric acid excretion although it has no tubular effect on uric acid reabsorption or secretion. Triamterene does not affect calcium

of 30, 60, and 100. Also available, Rimac-
oniazid USP) Dual Pack containing 60 Rimac-
and 30 INH 300-mg Tablets

V, Kirby, W. M. M., Sherris, J. C., and Turck,
usceptibility testing by a standardized single
m J Clin Path 45:493-496, 1966

C83-8 (Rev 7/83)

Product Identification Section, page 408

hydrochloride

ite hydrochloride USP)

ite hydrochloride)
se tablets

Ritalin is a white, odorless, fine crystalline
ns of which are acid to litmus. It is freely solu-

Pharmacology: Ritalin is a mild central nervous
nt
tion in man is not completely understood, but
ably activates the brain stem arousal system
roduce its stimulant effect.
r specific evidence which clearly establishes
1 whereby Ritalin produces its mental and
cts in children, nor conclusive evidence re-
ese effects relate to the condition of the cen-
stem.
SR tablets is more slowly but as extensively
he regular tablets. Relative bioavailability of
ompared to the Ritalin tablet, measured by
retion of Ritalin major metabolite (α -phenyl-
etic acid) was 105% (49-168%) in children
52%) in adults. The time to peak rate in chil-
urs (1.3-8.2 hours) for the SR tablets and 1.9
ours) for the tablets. An average of 67% of SR
excreted in children as compared to 86% in

Disorders: ~~Narcolepsy~~ (previously known
ain Dysfunction in Children). Other terms
describe the behavioral syndrome below in-
netic Child Syndrome, Minimal Brain Dam-
Cerebral Dysfunction, Minor Cerebral Dys-

ated as an integral part of a total treatment
typically includes other remedial measures
educational, social) for a stabilizing effect in
a behavioral syndrome characterized by the
p of developmentally inappropriate symp-
e-severe distractibility, short attention
vity, emotional lability, and impulsivity. The
s syndrome should not be made with finality
ptoms are only of comparatively recent orig-
ing (soft) neurological signs, learning disabili-
tial EEG may or may not be present, and a
tral nervous system dysfunction may or may
ad.

Diagnostic Considerations

, of this syndrome is unknown, and there is
stic test. Adequate diagnosis requires the use
cal but of special psychological, educational,
rcees.

commonly reported include chronic history
on span, distractibility, emotional lability,
d moderate-to-severe hyperactivity, minor
ns and abnormal EEG. Learning may or may
l. The diagnosis must be based upon a com-
d evaluation of the child and not solely on the
or more of these characteristics.

is not indicated for all children with this
ulants are not intended for use in the child
mptoms secondary to environmental factors.
psychiatric disorders, including psychosis.
ational placement is essential and psycho-
ion is generally necessary. When remedial
are insufficient, the decision to prescribe
ation will depend upon the physician's as-
sessment of the child's chronicity and severity of the child's symp-

~~marked anxiety, tension, and agita-~~
~~indications to Ritalin.~~

Contraindications: Ritalin is contraindicated also in
to be hypersensitive to the drug, in patients
in patients with motor tics or with a fam-
agnosis of Tourette's syndrome.
alin should not be used in children under six
ty and efficacy in this age group have not

weight gain, and/or height) has been reported with the long-
term use of stimulants in children. Therefore, patients re-
quiring long-term therapy should be carefully monitored.
Ritalin should not be used for severe depression of either
exogenous or endogenous origin. Clinical experience sug-
gests that in psychotic children, administration of Ritalin
may exacerbate symptoms of behavior disturbance and
thought disorder.

Ritalin should not be used for the prevention or treatment of
normal fatigue states.

There is some clinical evidence that Ritalin may lower the
convulsive threshold in patients with prior history of sei-
zures, with prior EEG abnormalities in absence of seizures,
and, very rarely, in absence of history of seizures and no
prior EEG evidence of seizures. Safe concomitant use of anti-
convulsants and Ritalin has not been established. In the
presence of seizures, the drug should be discontinued.
Use cautiously in patients with hypertension. Blood pressure
should be monitored at appropriate intervals in all patients
taking Ritalin, especially those with hypertension.
Symptoms of visual disturbances have been encountered in
rare cases. Difficulties with accommodation and blurring of
vision have been reported.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine.
Use cautiously with pressor agents and MAO inhibitors.
Human pharmacologic studies have shown that Ritalin may
inhibit the metabolism of coumarin anticoagulants, anticon-
vulsants (phenobarbital, diphenylhydantoin, primidone),
phenylbutazone, and tricyclic antidepressants (imipramine,
desipramine). Downward dosage adjustments of these drugs
may be required when given concomitantly with Ritalin.

Use in Pregnancy

Adequate animal reproduction studies to establish safe use
of Ritalin during pregnancy have not been conducted. There-
fore, until more information is available, Ritalin should not
be prescribed for women of childbearing age unless, in the
opinion of the physician, the potential benefits outweigh the
possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unsta-
ble patients, such as those with a history of drug depen-
dence or alcoholism, because such patients may in-
crease dosage on their own initiative.

Chronically abusive use can lead to marked tolerance
and psychic dependence with varying degrees of abnor-
mal behavior. Frank psychotic episodes can occur, espe-
cially with parental abuse. Careful supervision is re-
quired during drug withdrawal, since severe depression
as well as the effects of chronic overactivity can be un-
masked. Long term follow-up may be required because
of the patient's basic personality disturbances.

Precautions: Patients with an element of agitation may
react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised
during prolonged therapy.

Drug treatment is not indicated in all cases of this behavioral
syndrome and should be considered only in light of the com-
plete history and evaluation of the child. The decision to pre-
scribe Ritalin should depend on the physician's assessment of
the chronicity and severity of the child's symptoms and
their appropriateness for his/her age. Prescription should
not depend solely on the presence of one or more of the be-
havioral characteristics.

When these symptoms are associated with acute stress reac-
tions, treatment with Ritalin is usually not indicated.

Long-term effects of Ritalin in children have not been well
established.

Adverse Effects: Nervousness and insomnia are the
most common adverse reactions but are usually controlled
by reducing dosage and omitting the drug in the afternoon or
evening. Other reactions include hypersensitivity (including
skin rash, urticaria, fever, arthralgia, exfoliative dermatitis,
erythema multiforme with histopathological findings of
necrotizing vasculitis, and thrombocytopenic purpura), anore-
xia, nausea, dizziness, palpitations, headache, dyskinesia,
drowsiness, blood pressure and pulse changes, both up and
down, tachycardia, angina, cardiac arrhythmias, abdominal
pain, weight loss during prolonged therapy. There have
been rare reports of Tourette's syndrome. Toxic psychosis
has been reported. Although a definite causal relationship
has not been established, the following have been reported in
patients taking this drug: leukopenia and/or anemia, a few
instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss
during prolonged therapy, insomnia, and tachycardia may
occur more frequently, however, any of the other adverse
reactions listed above may also occur.

Dosage and Administration: Dosage should be individu-
alized according to the needs and responses of the patient.

preferably 30 to 45 minutes before meals. Average dosage is
20 to 30 mg daily. Some patients may require 40 to 60 mg
daily. In others, 10 to 15 mg daily will be adequate. Patients
who are unable to sleep if medication is taken late in the day
should take the last dose before 6 p.m.

SR Tablets: Ritalin SR Tablets have a duration of action of
approximately 8 hours. Therefore, Ritalin SR tablets may be
used in place of Ritalin tablets when the 8 hour dosage of
Ritalin SR corresponds to the titrated 8 hour dosage of Rita-
lin.

Children (6 years and over)

Ritalin should be initiated in small doses, with gradual
weekly increments. Daily dosage above 60 mg is not recom-
mended.

If improvement is not observed after appropriate dosage
adjustments over a one-month period, the drug should be
discontinued.

Tablets: Start with 5 mg twice daily (before breakfast and
lunch) with gradual increments of 5 to 10 mg weekly.

SR Tablets: Ritalin SR tablets have a duration of action of
approximately 8 hours. Therefore, Ritalin SR tablets may be
used in place of Ritalin tablets when the 8 hour dosage of
Ritalin SR corresponds to the titrated 8 hour dosage of Rita-
lin.

If paradoxical aggravation of symptoms or other adverse
effects occur, reduce dosage, or, if necessary, discontinue the
drug.

Ritalin should be periodically discontinued to assess the
child's condition. Improvement may be sustained when the
drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and
usually may be discontinued after puberty.

Overdosage: Signs and symptoms of acute overdosage,
resulting principally from overstimulation of the central
nervous system and from excessive sympathomimetic ef-
fects, may include the following: vomiting, agitation, trem-
ors, hyperreflexia, muscle twitching, convulsions (may be
followed by coma), euphoria, confusion, hallucinations, delir-
ium, sweating, flushing, headache, hyperpyrexia, tachycar-
dia, palpitations, cardiac arrhythmias, hypertension, mydri-
asis, and dryness of mucous membranes.

Treatment consists of appropriate supportive measures. The
patient must be protected against self injury and against
external stimuli that would aggravate overstimulation al-
ready present. If signs and symptoms are not too severe and
the patient is conscious, gastric contents may be evacuated
by induction of emesis or gastric lavage. In the presence of
severe intoxication, use a carefully titrated dosage of a short
acting barbiturate before performing gastric lavage.

Intensive care must be provided to maintain adequate cir-
culation and respiratory exchange; external cooling proce-
dures may be required for hyperpyrexia.

Efficacy of peritoneal dialysis or extracorporeal hemodialy-
sis for Ritalin overdosage has not been established.

How Supplied:

Tablets: 20 mg—round, pale yellow, scored (imprinted CIBA
34).

Bottles of 100 NDC 0083-0034-30

Bottles of 1000 NDC 0083-0034-40

Tablets: 10 mg—round, pale green, scored (imprinted CIBA
3).

Bottles of 100 NDC 0083-0003-30

Bottles of 500 NDC 0083-0003-35

Bottles of 1000 NDC 0083-0003-40

Accu-Pak® Unit Dose (blister pack)

Box of 100 (strips of 10) NDC 0083-0003-32

Tablets: 5 mg—round, yellow (imprinted CIBA 7)

Bottles of 100 NDC 0083-0007-30

Bottles of 500 NDC 0083-0007-35

Bottles of 1000 NDC 0083-0007-40

SR Tablets: 20 mg—round, white, coated (imprinted CIBA
16).

Bottles of 100 NDC 0083-0016-30

Note: SR Tablets are color additive free.

Do not store above 86°F (30°C). Protect from moisture.

Dispense in tight, light resistant container (USP).

C84-29 (Rev 6/84)

Shown in Product Identification Section, page 408

Continued on next page

The full prescribing information for each CIBA drug is
contained herein and is that in effect as of September 1,
1985.

APPENDIX VI

EVALUATION BY DR. JOSEPH PERRY
July 29, 1986

21

J. JOSEPH PERRY, M.D., F.A.C.C.

CARDIOLOGY

COTTONWOOD MEDICAL TOWER
5770 SOUTH 250 EAST, SUITE 340
MURRAY, UTAH 84107

July 29, 1986

Shaun Howell
Attorney at Law
State Insurance Fund
560 South 300 East
P.O. Box 45420
Salt Lake City, Utah 84145-0420

RE: Dale W. Steward
#86-05663
Inj: 11-11-85

Dear Counselor:

I have evaluated all of the information sent to me on this somewhat complicated case.

It seems clear that Mr. Steward was not conscious when his rig swerved to the left and ran off coming to rest on top of the median wall without apparently overturning or coming to an abrupt stop as it would with a collision. Additionally on arrival in the Emergency Department there was no gross evidence of physical injury or trauma according to the emergency physician. Additionally, the photographs sent to me demonstrate very little trauma to the rig he was driving. Thus, I think trauma can be excluded with a reasonable degree of confidence in this case.

Complicating the issue is the mention of narcolepsy in the medical record. The patient had given a history of sudden sleep attacks while driving and had been treated with amphetamines by his physician for several years prior to his demise. I find no studies in the record to document narcolepsy, thus this diagnosis is not secure in this individual.

Of the two scenarios which may have occurred, that is falling asleep then suffering a cardiac arrest sometime after contacting the median wall, or having the cardiac arrest while driving, only the latter seems to have firm medical support. Had he suffered narcolepsy while driving he would have awakened when he left the road (I speak from experience) and there seemed to be no event which would have been of sufficient severity to cause his death. It is remotely possible that the shock and fear of waking up in the middle of a serious accident would have been sufficient to engender the fatal cardiac arrhythmia, but this does not seem very probable.

In terms of medical probability it is most likely that he experienced a fatal cardiac arrhythmia while driving, lost consciousness a few seconds later thus losing control of the vehicle and having the accident as reported. It is possible that his dextroamphetamine was related to his death because it may worsen arrhythmias in susceptible individuals.

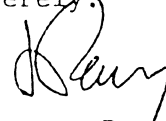
page 2 continued.....

An autopsy would have been supporting this diagnosis, but likely would not have confirmed it with an absolute degree of certainty. I suspect it would have shown amphetamines present and coronary artery disease present. At this point in time exuming the body would not shed any light on the presence of amphetamine. It would, however, document the presence or absence of coronary artery disease. In the absence of drugs it is extremely unusual for cardiac arrest to occur in a person with normal coronary arteries. If the absence of coronary artery disease could be documented, then the scenario of striking the median wall, waking up and then suffering a fatal arrhythmia would become somewhat more plausible. Whether or not that has any legal significance is of course not within my area of expertise.

In summary in terms of reasonable medical probability, the patient suffered a fatal arrhythmia while driving and the accident was simply the result of his death and subsequent loss of control of the vehicle. While other possibilities exist, they are far less likely. To exume and perform a post-mortum examination of the body would alter those probabilities to an extent, but it is highly unlikely it would provide definitive answer.

I hope this has been helpful to you.

Sincerely,



J. Joseph Perry, M.D.

JJP/jv